CASE REPORTS

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BRAIN ABSCESS IN A YOUNG FEMALE WITH UNDIAGNOSED EISENMENGER SYNDROME: A RARE CASE OF MISLEADING FINDINGS AND SEVERE COMPLICATIONS

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Background:
Brain abscesses in patients with congenital heart defects, such as those associated with Eisenmenger syndrome, are rare but highly dangerous, with significant morbidity and mortality. Recent studies show that brain abscesses in patients with cyanotic congenital heart disease have a high mortality rate, emphasizing the severity of this complication.

Case illustration:
We present a case of a 17-year-old female with untreated ventricular septal defect (VSD) who presented with severe headache, nausea, vomiting, abdominal pain, and altered consciousness. Clinical examination revealed cyanosis, digital clubbing, and severe hypoxemia, with peripheral oxygen saturation at 58%. Auscultation revealed a loud pulmonary component of the second heart sound without an audible murmur. Further diagnostics included an electrocardiogram showing right axis deviation, right ventricular hypertrophy, and lateral wall ischemia. Despite these findings, a chest X-ray revealed no cardiomegaly but indicated fibronodular infiltrates in the upper lobes, suggesting a differential diagnosis of tuberculosis. Echocardiography showed a sizable peri-membranous VSD with severe tricuspid regurgitation (maximum velocity 4.0 m/s), a significant right-to-left shunt, and multiple intracardiac emboli extending to the aortic arch. Given these findings, we performed a brain computed tomography scan, revealing a hypodense lesion with perifocal edema in the left parietal lobe, consistent with a brain abscess, alongside multiple hypodense lesions indicative of old infarcts in the basal ganglia. These misleading findings complicated the diagnosis, highlighting the importance of a systematic diagnostic approach. Early and accurate diagnosis is essential to avoid mismanagement and initiate appropriate treatment. This case emphasizes the need for heightened clinical suspicion and comprehensive evaluation in patients with cyanotic congenital heart disease presenting with neurological symptoms. Additionally, it underscores the severe impact of Eisenmenger syndrome on quality of life, particularly in young women of reproductive age.

Conclusion:
This case underscores the importance of early and prompt treatment of congenital heart defects to prevent severe complications like Eisenmenger syndrome and brain abscess. It emphasizes the need for vigilance in detecting septic embolic phenomena in patients with cyanotic congenital heart disease presenting with neurological symptoms and the critical role of systematic diagnostic pathways in early detection and prevention of misdiagnosis.

Keywords: Brain Abscess, Cyanosis, Embolism, Eisenmenger Syndrome, Ventricular Septal Defects

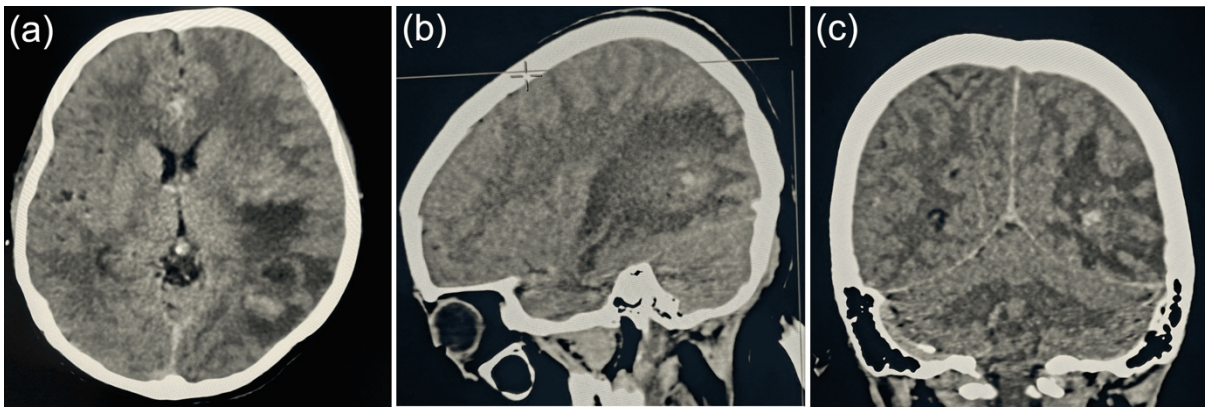

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Figure 1. Brain Computed Tomography Scan Showing Brain Abscess in (a) Axial View, (b) Sagittal View, and (c) Coronal View

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BRUGADA SYNDROME REVEALED BY DENGUE FEVER: A RARE CASE REPORT

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Background:
Brugada Syndrome (BrS) is a rare cardiac disorder caused by mutations in the SCN5A gene, affecting cardiac sodium channels and leading to abnormal heart rhythms and an increased risk of sudden cardiac death. This condition is characterized by ST-segment elevation in the right precordial leads on an ECG, often exacerbated by fever or metabolic imbalances. While it is known that fever can induce Brugada Syndrome, this case report presents a rare instance where Brugada Syndrome was specifically induced by Dengue Fever. Dengue Fever, a mosquito-borne viral infection, can worsen Brugada Syndrome due to high fever and electrolyte disturbances, posing significant diagnostic and therapeutic challenges. The aim of this case report is to emphasize the need for careful monitoring and management of arrhythmias in such patients.

Case illustration:
A 38-year-old male presented to Pasar Rebo General Hospital with a 2-day history of fever, weakness, shortness of breath, chest pain, nausea, and vomiting. The patient's socio-economic history revealed he is a heavy smoker and farmer living in a suburban area. With no significant past medical history, initial diagnosis was Dengue Fever without Warning Signs. Laboratory results indicated thrombocytopenia and leukopenia. ECG revealed ST elevation in V1-V3, characteristic of Type 1 Brugada Syndrome. The patient was managed in the CVCU (cardiovascular care unit) and, once stable, transferred to a regular inpatient ward for close monitoring and continued management of dengue. The patient's condition was moderately ill, and despite normal heart chamber visualization and valve function on TTE, the presence of fever unmasked the Brugada Syndrome. Transthoracic echocardiography showed normal heart chamber dimensions, global normokinesis with normal LV systolic function (EF 84%), normal diastolic LV function, normal heart valves, and normal RV systolic function (TAPSE 24 mm).

Conclusion:
This case highlights the importance of recognizing fever-induced Brugada Syndrome in patients with concurrent Dengue Fever. Monitoring ECG changes closely in such patients is crucial for managing potential arrhythmias and preventing sudden cardiac death.

Keywords: Dengue Fever, SCN5A gene, Arrhytmia, Brugada Syndrome, Fever-Induced

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A CASE OF POST PARTUM SEIZURE: MORE THAN JUST ECLAMPSIA

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Background:
Postpartum convulsion is a critical and intricate medical condition necessitating precise management. Initially, all patients presenting with this condition should be managed as eclampsia pending further investigation. Nonetheless, discerning the true etiology can be challenging due to its multifaceted nature, spanning from obstetric causes like eclampsia to neurological origins such as epilepsy. In this case study, we highlight a patient experiencing postpartum convulsions resulting from a staggering etiological factor.

Case illustration:
A 40-year-old Asian woman with a history of gestational hypertension and recent cesarean section presented with postpartum seizures necessitating hospitalization. She had a convulsive episode lasting 15-30 seconds the night before admission, characterized by rigidity, eye rolling, and transient loss of consciousness. Initial evaluation led to diagnosis of postpartum eclampsia, prompting immediate therapeutic intervention.During her hospital stay, she experienced recurrent seizures requiring transfer to the intensive care unit (ICU) for continuous monitoring. In the ICU, she had another seizure accompanied by polymorphic ventricular tachycardia resembling torsade de pointes on cardiac monitoring. Administration of anti-arrhythmic medication successfully terminated the seizure, and subsequent monitoring showed normalization of sinus rhythm with a prolonged QT interval. Following initiation of maintenance therapy, she remained seizure-free. Neurological and Imaging assessment revealed no abnormalities.

Conclusion:
Postpartum convulsion poses a diagnostic challenge due to the diverse range of underlying causes. Initially, all patients presenting with this condition are managed as potential cases of postpartum eclampsia until further evaluation reveals the etiology. Long QT Syndrome, along with other heart rhythm disorder, represents one of several differential diagnoses in such cases. Comprehensive electrocardiogram (ECG) assessment serves as the primary diagnostic tool for identifying this syndrome. However, detecting transient arrhythmias can prove challenging at times. Continuous 24-hour heart monitoring in the intensive care unit (ICU) can aid in detecting such arrhythmias, facilitating accurate diagnosis and appropriate management of this condition.

Keywords: Postpartum Convulsion, Seizure, Arrhythmia, Torsade de Pointes, Long QT Syndrome

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THE HIDDEN HAZARD: TOTAL ATRIOVENTRICULAR BLOCK IN YOUNG ADULT WITH CHRONIC ALCOHOLISM

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Background:
Total atrioventricular block (TAVB) is a relatively uncommon arrhythmia, however, becoming more prevalent among elderly individuals in developed nations as life expectancy rises. Conversely, TAVB is infrequently observed in healthy young adults without prior systemic symptoms.

Case illustration:
A 30-year-old male was referred to our emergency department with syncope during morning prayer at the mosque, with complaints of dizziness and chest discomfort a day prior. The patient denied any symptoms of fever during the past weeks. From in-depth history taking, it was revealed that the patient is an active smoker and has been consuming alcoholic beverages since he was 10 years old. The patient stated he consumed wine-type beverages with around 18% alcohol content regularly at least 1 bottle per week. There were no complaints of irregular heartbeat, syncope or chest pain in his adolescent years. Physical examination findings were unremarkable. The initial electrocardiogram showed TAVB with a P wave rate of 100 bpm and a Q wave rate of 75 bpm, without ST-T abnormality. Blood laboratory reported mild leukocytosis, a slight increase in CRP and lactic acid, otherwise within normal limits. Thorax x-ray showed left ventricular (LV) cardiomegaly. A temporary pacemaker was immediately implanted in our emergency department. The patient was admitted to the cardiac ward for further monitoring and received an inotropic infusion. Follow-up urinalysis disproves infection involvement. Subsequently, the patient underwent a permanent pacemaker (PPM) implantation with single chamber (VVIR) as a definite treatment following heart rate dependency on electrical support. Furthermore, our post-PPM echocardiography examination report revealed a normal LV ejection fraction with normal cardiac movement, with neither cardiac chamber enlargement nor valve dysfunction. The patient was discharged on the seventh day of hospitalization. Outpatient care is undergone for evaluation of symptoms recurrence and risk factors management.

Conclusion:
TAVB is a rare occurrence of arrhythmia in otherwise healthy young adults. This case underscores the potential for significant cardiac arrhythmias in younger individuals with a history of chronic alcohol consumption. A meticulous history-taking can be applied to assist in discovering differential causal of arrhythmia in this patient population.

Keywords: young adult, total atrioventricular block, alcoholism

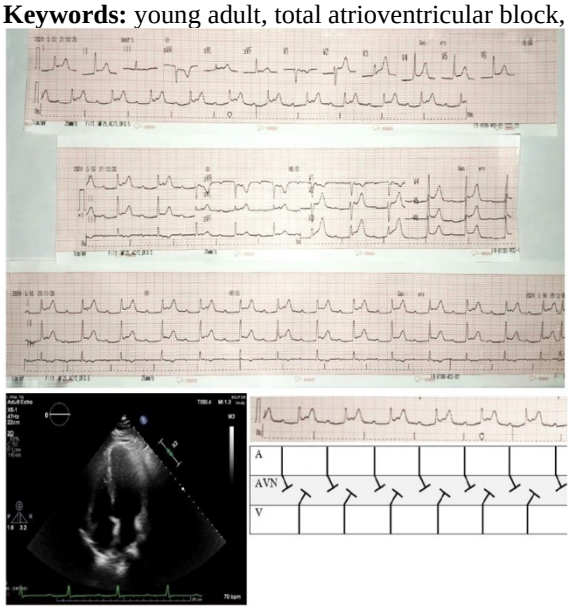

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Figure 1. Patient's electrocardiography and echocardiography findings

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19 YEARS OLD MAN WITH GENETIC DILATED CARDIOMYOPATHY: A CASE REPORT

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Background:
Dilated Cardiomyopathy (DCM) was defined as the presence of left ventricular or biventricular systolic dysfunction and dilatation that are not determined by abnormal loading conditions or coronary artery disease. The clinical management of DCM can be greatly affected by the identification of a specific underlying cause. The clinical decision-making primarily focusing on ejection fraction (EF) and NYHA classification, depending on the underlying causes. Dilated cardiomyopathy may be inherited as a monogenic trait, with multiple potentially causative mutations reported in high-throughput studies. Diagnostic method with multimodalities imaging, laboratory and genetic aspects was needed in managing DCM phenotype.

Case illustration:
A-19-years-old man came to cardiology clinic with chief complaints of recurrent coughing and shortening of breath during moderate activities. The patient's father and grandmother are suspected of having a cardiac problem and passed away at 5th decades of age. The physical examination found normal vital sign, normal electrocardiograph, cardiomegaly on chest x-ray, and transthoracic echocardiography (TTE) showed poor left ventricle (LV) systolic function with left ventricle ejection fraction (LVEF) 20%, global hypokinetic, moderate tricuspid regurgitation (TR), mild functional mitral regurgitation (MR), reduced right ventricle (RV) contractility, and intermediate probability of pulmonary hypertension (PH). Cardiac magnetic resonance imaging suggested dilated cardiomyopathy phenotype due to genetic cause.

Conclusion:
DCM was classified as genetic or non-genetic causes. The comprehensive examination included screening of relatives, symptoms, physical signs and multimodality imaging methods will increase the accurate diagnostic of DCM due to genetic cause. Further genetic testing should be performed in patient suspected genetic DCM.

Keywords: echocardiography, genetic testing, dilated cardiomyopathy, phenotype, magnetic resonance imaging

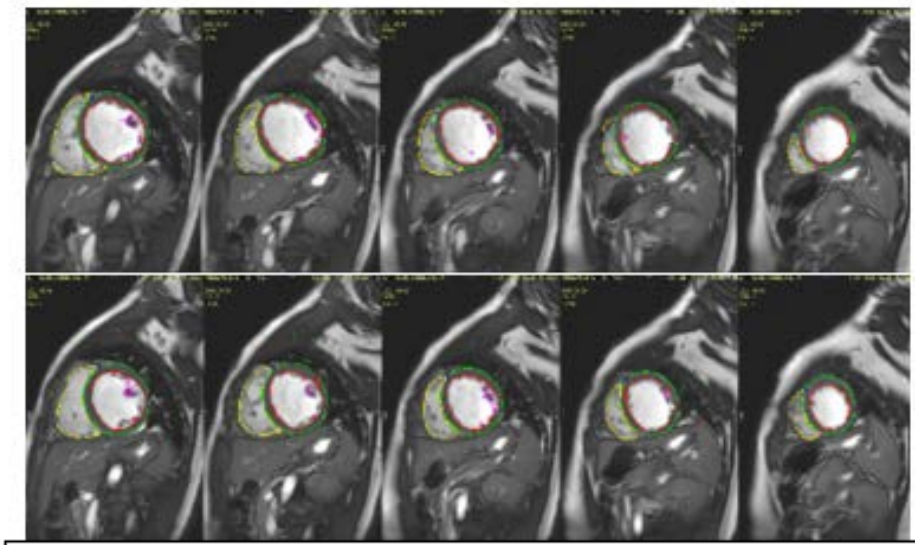

Figure 1. MRI during systolic and diastolic

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ACUTE MYOCARDIAL INFARCTION SUPERIMPOSED ON ANEMIA: A CLINICAL STRUGGLE OF MISMATCHED SUPPLY AND DEMAND

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Background:
Managing ACS in high bleeding risk is still challenging yet difficult. Anemia is a significant factor that exacerbates the prognosis of CAD, with the incidence of acute coronary syndrome (ACS) patients experiencing chronic anemia ranging from 11-40%. In cases of acute or chronic anemia, it can lead to poor prognosis.

Case illustration:
A 49-year-old female presented to the emergency room with complaints of generalized weakness persisting for one week. No fainting, palpitations, nausea, no vomiting. Her medical history was only previous blood transfusion, with no history of smoking, diabetes, or hypertension, family history is clear. No active bleeding was reported. On examination, her blood pressure was noted to be critically low at 60/32 mmHg. severe anemia with a hemoglobin level of 4.3 g/dL, Hematocrit level at 16%, then Platelet count at 1,342,000/uL. The result of peripheral blood smear suggests the presence of Macrocytic Anemia. While waiting for blood transfusion, she felt left-sided chest pain radiating to her left arm. Electrocardiogram (ECG) changed to inferoposterior ST-segment elevation, and Troponin I levels were significantly elevated at 10.32 ng/mL. PRC blood transfusions were done immediately. Antiplatelet was postponed until Hb increased, and the symptom was relieved after hemoglobin returned to normal. In a 2021 study, patients with AMI and anemia who underwent blood transfusions first showed good results. The American Heart Association (AHA) recommends prioritizing strategies to minimize bleeding risks in conjunction with standard ACS care.

Conclusion:
This case underscores the potential necessity of administering a blood transfusion before considering PCI in patients presenting with ACS and severe anemia, thus ensuring an appropriate balance between oxygen supply and myocardial demand. Given the rarity of such cases, further research is warranted to establish comprehensive guidelines for managing similar presentations.

Keywords: Anemia, STEMI inferoposterior, blood transfusion, PCI

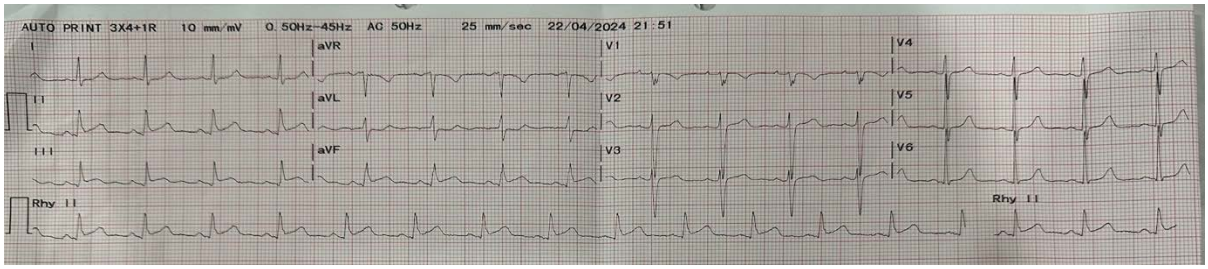

Figure 1. The inferior sided ECG

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SEPSIS – INDUCED BRUGADA PATTERN MISDIAGNOSED AS AN ACUTE MYOCARD INFARCTION

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[1]RSUD dr Soekardjo Tasikmlaya

Background:
Brugada Syndrome (BrS) is an inherited arrhytmogenic disease which increased risk of sudden cardiac death (SCD).

Case illustration:
A 59-year old male presented Transient Loss of Consciousness (TLOC) before came to ER around 5 minutes and woke up without neurologic deficits. He had blurred vision before TLOC, he unabled to walk due to pain and redness in his right leg. He had no chest pain, shortness of breath, palpitation and family history of TLOC.No significant risk factor. On physical examination he had fever (38^0C), tachypnea and tachycardia with no positive findings in either chest or cardiovascular exam. Laboratory findings were leukocytosis (37.060 WBC/mL) with 82% segmented neutrophile, normal troponin I (<0.01 ng/ml). He had normal chest radiograph. His initial ECG revealed elevated ST segment in V1-V3 with deep Q wave thus misdiagnosed as acute anterior myocardial infraction. Immediately administered loading antiplatelet, beta blocker, diuretic, morfin, sedative and laxative. In the ward, he repeated ECG there were no ECG evolution. Thus, it was a coved ST-segment elevation (J-point elevation with gradual down-sloping ST-segment) ≥2 mm in V_1, V_2 followed T wave inversion typical type – 1 Brugada pattern. He subsequently underwent TTE with normal heart chambers. Furthermore, the patient diagnosed with sepsis due to cellulitis, we administered antibiotic and antipyretic. After four days of hospitalization he was discharged with no fever or cardiac events and BrS pattern resolved.

Conclusion:
The importance suspicion risk factor of BrS, early diagnosis can help guide further management. BrS unmasked by fever shoud started antipyretic to prevent ventricular arrhythmias and SCD.

Keywords: acute myocard infarct, rare disease, brugada syndrome

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MYOCARDIAL BRIDGING CAUSING ISCHEMIA AND RECURRENT CHEST PAIN: A CASE REPORT

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Background:
Myocardial bridging occurs when a segment of the epicardial coronary artery runs intramurally through the myocardium. Although myocardial bridging is generally considered benign, it has been associated with myocardial ischemia. In this report, we present a case of myocardial bridging that manifested with recurrent chest pain and positive ischemic response during an exercise treadmill test.

Case illustration:
A 42-year-old man presented with recurrent retrosternal chest pain of 6 months duration. He had a history of smoking and dyslipidemia, otherwise, no physical abnormalities were detected by examination. Electrocardiogram and blood tests were normal apart from impaired glucose tolerance with elevated low-density lipoprotein and decreased high-density lipoprotein cholesterol. While doing the exercise treadmill test, the patient developed chest pain and significant ST-segment depression in leads II, III, aVF, V4, V5, and V6 that persisted for about 3 minutes through recovery. We decided to admit the patient to the catheterization lab to perform coronary angiography. Myocardial bridging was observed in the mid-segment of the left anterior descending coronary artery. In patients with myocardial bridging, symptoms often manifest during exercise and with tachycardia. Beta-blockers are the first-choice treatment for symptomatic patients. However, if refractory symptoms persist despite medical therapy, percutaneous intervention or myotomy must be considered as an alternative therapy. Medical treatment with an optimal beta-blocker was decided. At one year follow-up, our patient was healthy and had no cardiac complaints during medication.

Conclusion:
Myocardial bridging may predispose to coronary vasospasm that may lead to ischemic complications. Exercise treadmill test and further coronary angiography are appropriate diagnostic tools in cases of typical chest pain. Beta-blockers are the first-choice treatment for symptomatic myocardial bridging patients.

Keywords: Ischemic, Myocardial Bridging, Coronary Angiography, Exercise Treadmill Test

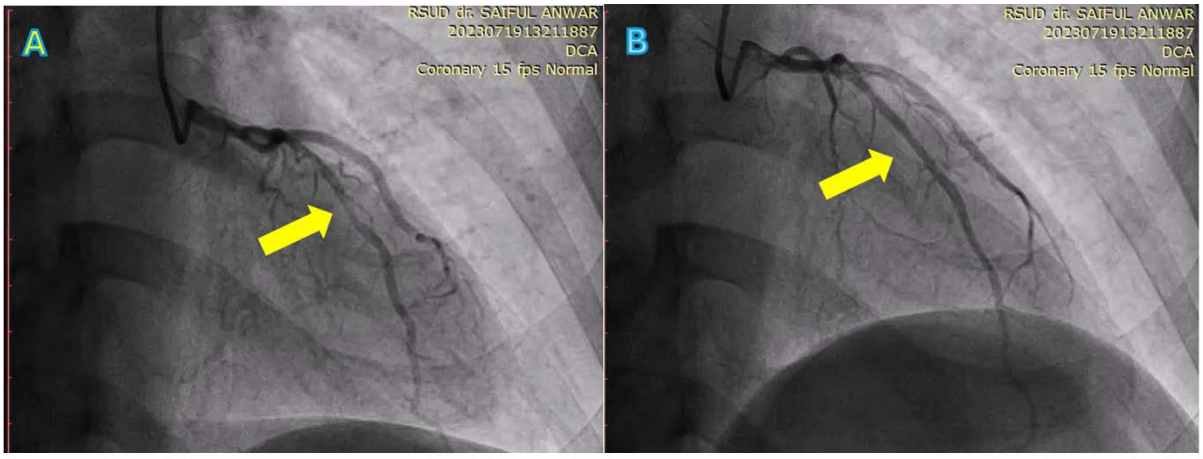

Figure 1. Coronary Angiography of the myoocardial bridging at the LAD coronary artery n the systolic (A) and diastolic (B) phases.

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THE UNSEEN ENEMY: PERICARDIAL EFFUSION IN HYPOTHYROIDISM

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Background:
Hypothyroidism is a widespread endocrine disorder that can manifest in various ways. It can be asymptomatic or present with non-specific symptoms, leading to delayed diagnosis. Pericardial effusions are a known complication of hypothyroidism, affecting 3-37% of cases. The most common cause is idiopathic pericarditis, often viral in origin. In contrast, metabolic disorders like hypothyroidism are relatively rare causes.

Case illustration:
A 55-year-old woman with hypertension was admitted to the emergency department for a massive pericardial effusion without tamponade signs. She reported persistent fatigue and bilateral lower extremity edema. Repeated echocardiography revealed cardiomegaly and pericardial effusion. Despite treatment with diuretics and antihypertensive medications, the effusion persisted for 3 years. Further evaluation revealed severe hypothyroidism, which was treated with levothyroxine. Follow-up echocardiography showed a reduction in pericardial effusion. The patient was discharged with a diagnosis of pericardial effusion due to severe hypothyroidism, chronic kidney disease, and controlled hypertension. Regular follow-up evaluations were scheduled in 4-6 weeks.

Conclusion:
Pericardial effusion linked to hypothyroidism is a relatively rare condition. Hypothyroid patients can develop a progressive effusion, which is more common in cases of clinical hypothyroidism. The symptoms exhibited are typically those related to the underlying disease that caused the effusion. Echocardiography is the most effective diagnostic tool for identifying pericardial effusion. Thyroid hormone replacement therapy is the recommended treatment, which often leads to the resolution of the effusion within one to 15 months after initiation. However, pericardiocentesis is necessary if the size of the pericardial effusion is moderate to large or cardiac tamponade is present.

Keywords: Pericardial disease, Hypothyroidism, Pericardial Effusion

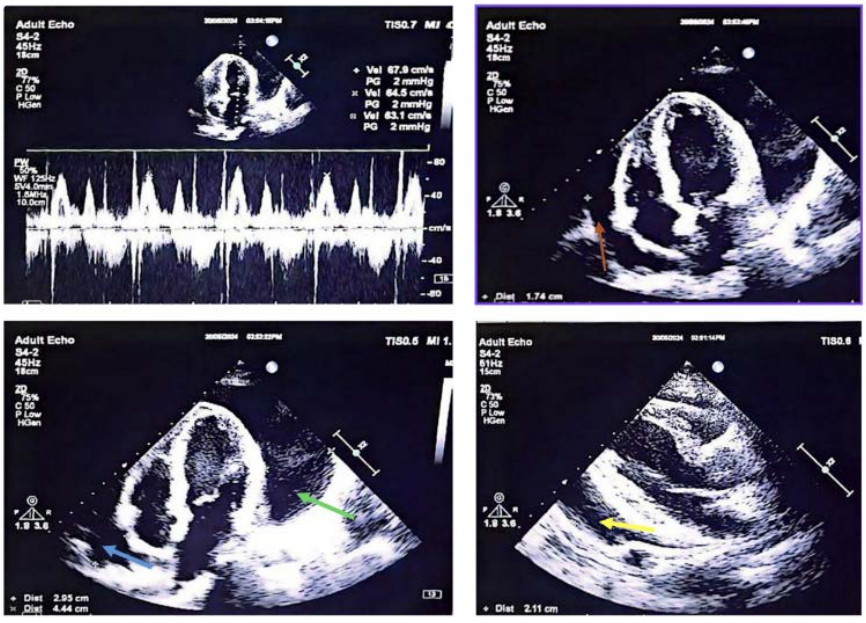

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Figure 1. Echocardiography shows circumferential pericardial effusion. (Arrow: Orange: lateral LV, Blue: lateral RA, Yellow: Posterior LV, Red: Lateral RV)

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OBESITY AND OSA: A "NIGHTMARE" FOR CARDIOVASCULAR SYSTEM

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Background:
OSA (Obstructive Sleep Apnea) has been proven be a risk for hypertension and vascular dysfunction and has been proposed to be causally related with cardiac arrhythmias and sudden cardiac death (SCD). OSA is a common sleep related breathing disorder affect obesity. OSA has mainly been associated with premature atrial complex, sinus bradycardia, sinus pauses, premature ventricular complexes, and paroxysmal atrial fibrillation. Bradyarrhythmias occur in up to 18% of patients with OSA, also atrial fibrillation occurs in up to 4.3% of patients with OSA compared to 2.1% obese patients without OSA.

Case illustration:
47-year-old women came to emergency, with chest discomfort and near-syncope, palpitation. No cardiovascular history before. On examination, BP 182/98 mmHg, pulse 54 bpm, with BMI 31.2 (Obesity). Chest x-ray result was cardiomegaly, and ECG result was sinus bradycardia 54 bpm, LVH, with normal PR interval. Laboratory results was NTproBNP 157 ng/L, pre-diabetic, dyslipidaemia, and microalbuminuria. Echocardiography was performed then, and the result was concentric LVH, dilated LA, global normokinetic, with LVEF 78,5%. CT coronary angiogram result was minimal stenosis without significant stenosis. Therefore, Holter ECG for 3 days was performed, and the result was found frequent isolated PACs (6,47%), and episode pauses two times (with duration 1,4 sec and 2,3 sec), no AF. Therefore, the patient was educated to lower her body weight, exercise regularly, dietary regulation, and medication to prevent any further more complications of cardiovascular.

Conclusion:
OSA is the most common sleep disorder linked to obesity with central adiposity can be a potent risk factor for OSA. Potential mechanisms underlying OSA with obesity and metabolic syndrome was intermittent hypoxia cause sympathetic activation, neurohumoral changes, and oxidative stress to cardiac cellular damage and alteration in myocardial excitability, recurrent arousals, resulting in sympathetic activation and coronary vasoconstriction. Some of studies OSA may be a causal factor for SCD, may result from malignant arrhythmias or ischemic heart disease. Early diagnosis can prevent several complications of OSA, and holistic approach for metabolic disorders is warranted.

Keywords: Sleep disordered breathing, OSA, Cardiac arrhythmia, Obesity

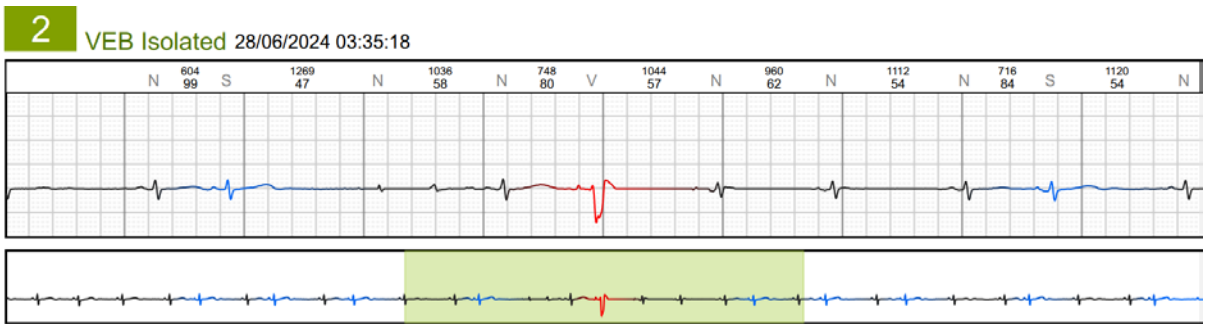

Figure 1. Non-conducted PAC with pauses

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MISDIAGNOSED ACUTE CORONARY SYNDROME: A CASE OF DIGITALIS-INDUCED VENTRICULAR ARRHYTHMIA IN DILATED CARDIOMYOPATHY

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Background:
Chest pain frequently prompts evaluation for acute coronary syndrome (ACS), but misdiagnosis can lead to inappropriate treatments and complications. This case underscores the importance of thorough diagnostics and highlights the potential side effects of digitalis, particularly ventricular arrhythmia (VA), in patients with dilated cardiomyopathy (DCM).

Case illustration:
A 33-year-old male with type 2 diabetes mellitus and dyslipidemia presented to the emergency room with chest pain and a foot ulcer. The patient exhibited tachycardia (107 bpm) but otherwise normal findings. Suspected ACS due to his chest pain and risk factors led to the administration of antiplatelet and nitrate therapy. He was previously treated with digoxin for two weeks for heart failure with reduced ejection fraction (HFrEF), presumed secondary to coronary artery disease. The patient's electrocardiography (ECG) on current admission revealed a prolonged QT interval (Fridericia corrected QT 497 ms). Echocardiography showed dilatation of all cardiac chambers, left ventricular ejection fraction of 25%, global hypokinesis, widened E-point septal separation, moderate mitral regurgitation due to annular dilation, and moderate to severe tricuspid regurgitation. Laboratory results indicated a Hemoglobin A1c of 9.9%, random blood glucose of 244 mg/dL, low-density lipoprotein of 71 mg/dL, and normal troponin and electrolyte levels. Digoxin was promptly discontinued based on these findings. The patient experienced a one-minute seizure in the ward, terminating spontaneously, with post-seizure evaluation showing no neurological deficits, thus excluding a neurological cause. Despite the undocumented ECG during the seizure, VA was deemed the most likely cause. Coronary angiography subsequently revealed normal coronary arteries, leading to a revised diagnosis of DCM as the underlying cause of HFrEF. Following Class IB indications, the consideration of an implantable cardioverter-defibrillator was recommended.

Conclusion:
Precise diagnosis and appropriate treatment are essential for patients presenting with chest pain and comorbid conditions. Misdiagnosis of ACS as the etiology of HFrEF led to unnecessary digoxin use, contributing to a prolonged QT interval and subsequent VA. This case highlights that not all HFrEF patients require digoxin, and those treated with it, especially in DCM, require meticulous monitoring.

Keywords: Heart Failure, Digitalis, Arrhythmia, Dilated Cardiomyopathy, Acute Coronary Syndrome

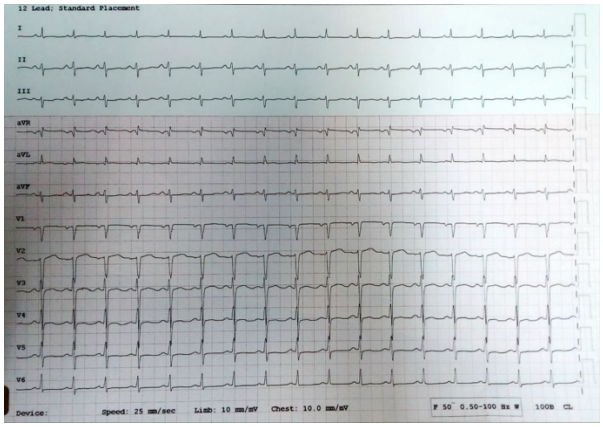

Figure 1. Electrocardiography Showing Sinus Tachycardia and Prolonged QT Interval

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PREMATURE VENTRICULAR CONTRACTIONS AS CARDIOVASCULAR MANIFESTATIONS IN A PREGNANT WOMAN WITH GESTATIONAL TRANSIENT THYROTOXICOSIS AND LOWN–GANONG–LEVINE ECG'S FEATURE: A CASE REPORT

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Background:
Lown–Ganong–Levine (LGL), also known as pre-excitation syndrome, is a rare cardiac conduction disorder. It is characterized by abnormal impulse propagation through an accessory pathway between the atria and ventricles, resulting in a short PR interval on electrocardiography. Meanwhile, premature ventricular contractions (PVCs) are early depolarizations of the ventricular myocardium that disrupt the normal cardiac cycle. In pregnancy, high thyroid hormone levels can lead to Gestational Transient Thyrotoxicosis (GTT) which may induce arrhythmia. We reported the concurrence of LGL , PVCs, and GTT in a pregnant woman.

Case illustration:
A 31-year-old pregnant woman complained of dyspnea and chest discomfort. Notably, the patient noted experiencing similar symptoms during her third pregnancy, which resolved after childbirth. Currently, she is in her fourth pregnancy at a gestational age of 11 weeks. The electrocardiogram exhibits a heart rate of 84 bpm with notable frequent PVCs originating from Anterior RVOT. Additionally, there are findings suggestive of LGL, characterized by a shortened PR interval without a delta wave. The patient's laboratory findings reveal free T4 thyroid hormone levels were at 31.81 pmol/L, while thyroid-stimulating hormone (TSH) levels were 0.071 mU/L, indicating a potential hyperthyroid state or thyroid gland overactivity. The patient was diagnosed with LGL with frequent PVCs, and GTT in a multigravida patient at 11 weeks of pregnancy with a single live intrauterine. The patient was given propranolol and propylthiouracil. In patients with LGL, the presence of GTT is known to potentially exacerbate arrhythmic disorders, impacting electrical conduction by increasing heart rate and irritability while influencing the heart's repolarization process.

Conclusion:
We reported the potential for exacerbation of underlying electrical and endocrine disorders amid the cardiovascular and hormonal fluctuations intrinsic to pregnancy, as well as how to manage arrythmia in pregnancy.

Keywords: Lown-Ganong-Levine, Arrythmia, Premature Ventricular Contractions, Gestational Transient Thyrotoxicosis

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MYOPERICARDITIS WITH MASSIVE PERICARDIAL EFFUSION IN TUBERCULOSE PATIENT : A RARE CASE REPORT

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Background:
The incidence of myopericarditis is rare which showed in 15% patients with pericarditis. Tuberculous pericarditis accounts for 4% of pericarditis and myocardial involvement is reported about 0.14-2%. This condition can lead to pericardial effusion (occurs in about 10% of cases of TB pericarditis) and increased mortality from cardiac tamponade. 1-2% patients with pulmonary TB are reported to develop TB pericarditis. This rare occurrence poses a challenge to make the diagnosis.

Case illustration:
A 25-year-old female presented to the emergency room with a week history of fever and a month of recurrent cough, accompanied by weight loss. The patient denies of anginal or positional chest pain, no evidence of shortness of breath and no comorbidities. On the third day of treatment, the patient experienced severe shortness of breath and transferred to ICU.Physical examination revealed the patient was agitated, blood pressure 80/40 mmHg, heart rate 132x/min, respiratory rate 40x/min, temperature 37.7 C, SaO2 94% with nasal canule. Auscultation revealed muffled heart sound and rales in the basal left lung, jugular venous pressure was increased, hepatomegaly. An electrocardiogram showed a low voltage.Laboratory results Hemoglobin 10.9 Leukocytes 11,000 Platelets 419,000 Ureum 42.8 Creatinin 0.9 Albumin 3.1 Blood gas analysis PH 7.43 PCO2 22.1, PO2 164, BE -7.9, HCO3 14.6, SaO2 99%, Electrolytes was normal.Thorax X-ray showed cardiomegaly with "water bottle sign" and bilateral apex infiltrate. The Echocardiography results was massive pericardial effusion with tamponade. An emergency pericardiocentesis was performed and obtained a serous xantocrome fluid up to 740 cc. The analysis of the pericardial fluid revealed an exudate, which is suggestive of a tuberculosis infection or systemic lupus erythematosus. Echocardiography evaluation after pericardiocentesis revealed minimal pericardial effusion with ejection fraction 28% and global hypokinetic characteristics.The patient received tuberculosis management, heart failure and myopericarditis treatment according to the suspected etiology based on the supporting examination. The patient showed clinical improvement and was discharged on the 11th day of treatment.

Conclusion:
Tuberculous myopericarditis is a rare condition and presents a significant challenge. Appropriate management can result in a reduction in morbidity and mortality.

Keywords: heart failure, cardiac tamponade, myopericarditis, tuberculose

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DECODING MYOCARDIAL INJURY: WHEN ST ELEVATION MISLEADS FROM STEMI TO MYOCARDITIS

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Background:
ST-elevation pattern on an ECG does not always indicate Myocardial Infarction, for the example is Myocarditis. Differential diagnosis between myocarditis and STEMI is crucial due to differing management strategies. Management of myocarditis focuses on supportive care, while STEMI requires reperfusion therapy.

Case illustration:
A 29-year-old male, presented to the emergency department with severe, persistent left-sided chest pain radiating to the left arm and back, accompanied by dyspnea and cold sweat. Initial vital signs revealed hypotension (BP 74/48 mmHg), bradycardia (heart rate 59 bpm), a respiratory rate of 22 breaths per minute, and a temperature of 36.9°C. The cardiovascular examination was notable for regular heart sounds without murmurs, and the respiratory system was clear. An electrocardiogram indicated sinus bradycardia with significant ST elevation across multiple leads, suggestive of an inferior myocardial infarction. Laboratory tests showed elevated white blood cell counts at 13.43 × 10^3/uL, elevated CRP at 318 mg/L, critically high troponin I levels over 40,000 ng/mL, elevated creatinine at 2.07 mg/dL, and low potassium at 3.1 mmol/L. Initial treatment was directed towards managing an acute myocardial infarction but no significant improvement. Subsequent diagnostic assessments were normal. The patient was given ACS therapy and supportive therapy. On the second day, there was resolution of ST elevation, but chest pain persisted, so it was decided to perform a CAG. The CAG results were within normal limits, thus ruling out the diagnosis of STEMI . The diagnosis was changed to myocarditis, and anti-inflammatory therapy was given, which lead to clinical improvement of the patient.

Conclusion:
Accurate diagnosis of myocarditis versus STEMI is crucial due to distinct management approaches. While both conditions can present with chest pain and EKG changes, careful consideration of clinical, imaging, and laboratory findings is necessary for differentiation.

Keywords: Diagnosis, Chest Pain, STEMI, Myocarditis

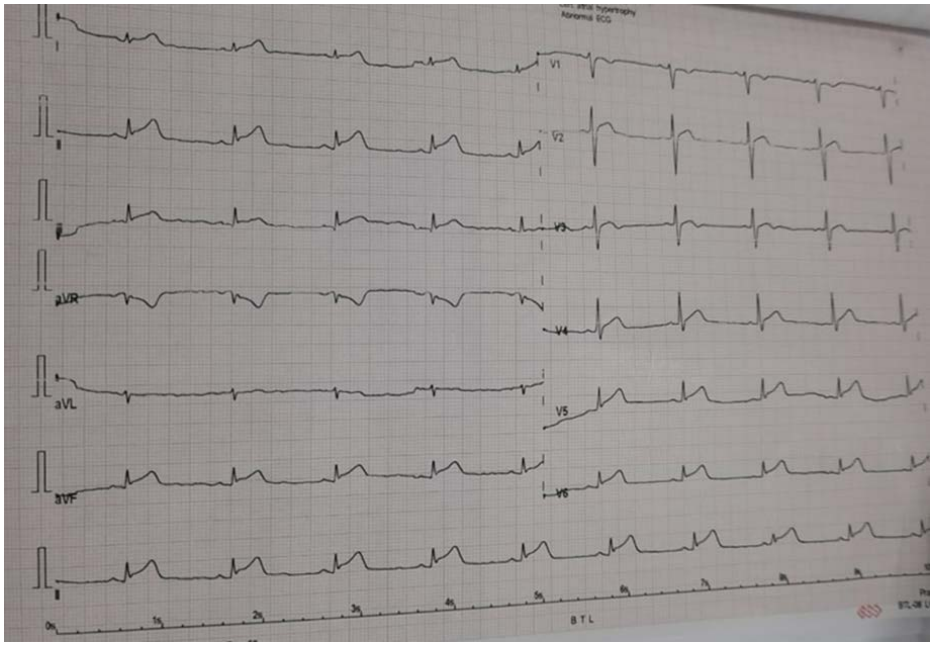

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Figure 1. ECG, Emergency Department

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MASSIVE RIGHT-SIDED INFECTIVE ENDOCARDITIS POST-CURETTAGE: A RARE OCCURRENCE IN A NON-IVDU PATIENT

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Background:
Infective endocarditis (IE) is a rare but serious infection of the heart's endocardial lining, with an incidence of about 5 per 100,000. Diagnosing IE is challenging and delayed treatment can be fatal. Right-sided infective endocarditis (RSIE) is especially rare, representing only 5-10% of cases, and is usually linked to intravenous drug use or cardiac devices. RSIE without these risk factors, such as post-curettage, is extremely uncommon and warrants further study.

Case illustration:
A 29-year-old female presented with a six-week history of intermittent fever, productive cough, shortness of breath, and bilateral leg swelling. Two months prior, she had a spontaneous abortion at an estimated gestational age of 9-10 weeks, followed by a curettage. Post-curettage, she experienced recurrent fever, chills, and nausea, necessitating multiple ICU admissions without improvement. She had no history of congenital heart disease, diabetes, hypertension, or renal disease. Initial examination revealed hypotension (80/66 mmHg), fever (39.4°C), conjunctival pallor, jugular vein distension, a grade III/6 systolic heart murmur at the LLSB, bilateral coarse crackles, hepatomegaly, ascites, and pitting edema in both legs. Laboratory tests showed significant abnormalities, including anemia (hemoglobin 6.1g/dL), leukocytosis ($17.24\times10^3/\mu L$), thrombocytopenia ($76\times10^3/\mu L$), hypoalbuminemia (2.3g/dL), hyponatremia (120.30mmol/L), and hypochloremia (84.72mmol/L). ECG revealed sinus tachycardia, incomplete RBBB, and occasional PVCs. Chest X-ray indicated cardiomegaly and right lower lobe infiltrates. Echocardiography showed right atrial and ventricular dilation, severe TR with vegetations on all three cusps and moderate pericardial effusion. Blood cultures confirmed Staphylococcus aureus, confirming a diagnosis of IE, necessitating cotrimoxazole, clindamycin, ramipril, and bisoprolol therapy. Despite temporary improvement, she underwent TVR based on indications of vegetation size >20 mm, right heart failure, and refractory bacteremia. A six-month postoperative follow-up showed excellent valve function and normal heart dimensions, with the patient maintained on warfarin, ivabradine, and bisoprolol.

Conclusion:
Early screening and cardiac evaluation in post-obstetric patients with symptoms of fever and heart failure play an important role in reducing the possibility of IE. Awareness of the incidence of RSIE in non-IVDU patients, especially those with a history of obstetric procedures is expected to increase along with the choice of therapeutic strategies including definitive antibiotics and even valve replacement.

Keywords: Curettage, Infective Endocarditis, Abortion, Case Report, Right-sided IE

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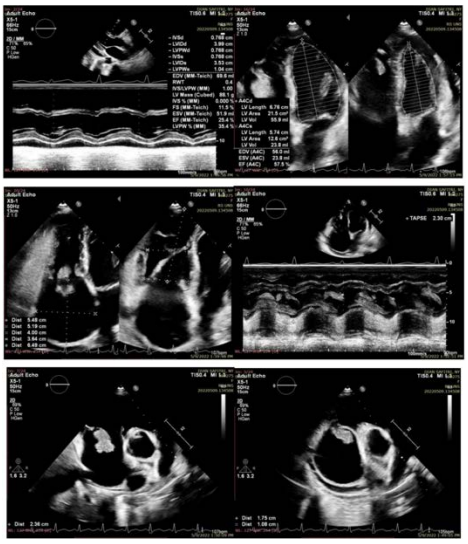

Figure 1. Transthoracic Echocardiography of the patient before intervention surgery.

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HYBRID STRATEGY WITH DRUG-COATED BALLOON AND DRUG-ELUTING STENT IN A YOUNG MALE WITH ACUTE LEFT MAIN TOTAL OCCLUSION AND BIFURCATION LESION

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Background:
Acute complete occlusion of the left main coronary artery (LMCA) accompanied by a bifurcation lesion is an extremely life-threatening condition. It can cause arrhythmias, cardiogenic shock, and sudden death. Revascularization in this condition is a very challenging procedures.

Case illustration:
A 27-year-old male was referred to the ER with a chief complaint of chest pain that had been felt 36 hours before admission. He also complained of shortness of breath that had been relieved since he was treated in hospital before. He was an active smoker for more than 10 years and used to smoke about 2-3 packs of cigarettes every day. His ECG showed ST-segment elevation in aVR and massive ST depression. Troponin level was increased significantly. We sent him to the cath lab. Contrast injection in the RCA showed retrograde filling of the LCx postero-lateral branch with a total occlusion at LMCA. After that, he was experienced pulseless ventricular tachycardia. He was given 360 joules of defibrillation, and had a return of spontaneous circulation. We also gave him Amiodarone IV 150 mg extra. We inserted guiding to the LM-LAD, administered Heparin 10.000 IU, and inserted wire to the proximal LAD. Predilatation was carried out several times in proximal LAD with a 1.0/8 mm balloon with was inflated up to 14 atm. After that, we were able to insert the wire into the distal. We implanted a 3.5/29 mm DES stent at LM-LAD which was inflated to 8 atm. After the successful implantation of DES at LM-LAD proximal, we found critical stenosis at osteal LCx. We opened the strut of DES with a 1.0/8 mm balloon which was inflated up to 12 atm. We performed double wiring to the OM1, continued by dilatation with 2.75/20 mm DCB which was inflated up to 8 atm at osteal-proximal LCx for 60 seconds. TIMI III flow was obtained. He was discharged 2 days later.

Conclusion:
Early diagnosis and quick decision in acute coronary syndrome caused by LMCA occlusion are critical for patient's survival. A hybrid strategy may be a safe and effective procedure, with persistence of good clinical outcomes.

Keywords: LMCA, Hybrid Strategy, DES, DCB, PCI

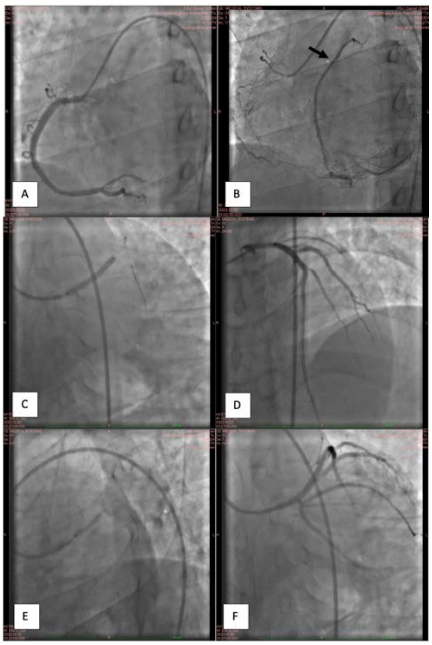

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Figure 1. A-B. Retrograde filling of the LCx postero-lateral branch (black arrows) after RCA visualization; C-D. Stenting with DES at LM-LAD, TIMI III flow; E-F. Dilatation with DCB at osteal-proximal LCx, TIMI III flow.

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MULTI – MODALITIES APPROACH OF DIAGNOSTIC AND THERAPEUTIC PERFORMANCE IN A CASE OF CHRONIC THROMBOEMBOLIC PULMONARY HYPERTENSION: A RARE CASE REPORT

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Background:
Chronic thromboembolic pulmonary hypertension (CTEPH) is one of several groups within pulmonary hypertension (PH). This condition is caused by long-term complications of pulmonary embolism (PE) that occur related to impaired thrombus resolution. Epidemiologically, the occurrence of CTEPH is very rare so early detection with various diagnostic modalities is necessary in determining therapy and reducing the morbidity and mortality of patients to heart failure and improving the ability of daily activities in patients. The use of diagnostic methods using both invasive and non-invasive methods is necessary to assess the patient's condition, besides PH cases are rare cases and not all hospitals have complete diagnostic methods.

Case illustration:
A 12-year-old male patient came to the emergency department looking weak with a complaint of shortness of breath. The patient had been complaining of shortness of breath for several days, especially when the patient was active. The patient has a history of heart disease but currently said that since some time the patient has not taken medicine. On physical examination, the patient was found to be conscious, with decreased oxygen saturation. Neither chest x-ray nor electrocardiography (ECG) examination revealed any abnormalities. Echocardiography examination showed abnormalities such as severe tricuspid regurgitation (TR), moderate pulmonary regurgitation (PR), and pericardial effusion. Pulmonary computed tomography (CT) scan was then performed with the results clearly illustrating that there was a thrombus that filled the entire lumen of the right pulmonary artery, and the left pulmonary artery had a thrombus that filled part of the lumen to the left upper lobe segmental branching. This patient was given symptomatic therapy to reduce symptoms, namely furosemide injection, and medical therapy as therapy for right heart failure, namely Sidenafil 20 mg twice a day.

Conclusion:
Multi-modality in the use of supporting examinations is important to determine the diagnosis and therapy in patients, as well as reduce more severe complications.

Keywords: Computed Tomography Scan Pulmonal, Pulmonary hypertension, Pulmonary Embolism, Pericardial Effusion, Chronic thromboembolic pulmonary hypertension

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HEART FAILURE DUE TO RHEUMATIOD HEART DISEASE IN 9 YEARS OLD MALE : A CASE REPORT

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Background:
Rheumatic heart disease (RHD) is a disease of poverty. RHD describes a group of short-term (acute) and long-term (chronic) heart disorders that occurs as a result of acute rheumatic fever. It is usually seen in children who are 5 to 15 years old. Rheumatic heart disease refers to long-term cardiac damage caused by either a single severe episode or multiple recurrent episodes of ARF. Rheumatoid heart disease patients are at increased risk of developing infective endocarditis, a condition with significant morbidity and mortality. This is a case of 9 years old male with heart failure caused by rheumatoid heart disease.

Case illustration:
Vital signs were blood pressure 133/87 mmHg, heart rate 100 times per minute, respiratory rate 30 times per minute, body temperature 37ºC, oxygen saturation 97% with 15 lpm NRM, and body weight 32 kg with no edema. There was not any murmur on every valves, lung: vesicular, rales +/+, no wheezing. Laboratory findings revealed hemoglobin 10.4 g/dL, leukocyte 8300/mm3, hematocrit 29.5 %, thrombocyte 139.000/mm3. MCV: 80.4 fl, MCH: 28.3 PQ, MCHC: 35.3 %. Chest X- ray examination presented cardiomegaly with lung edema in this patient. In echo, were found severe MR, Mild TR with dilatation on every chambers. EF level was 41%. The patient was given initial treatment. We administrate O2 3-4 lpm, injection Benzatine Penniciline 1,2 mil unit 1x IM , injection of furosemide 40 mg/ 12 hour, captopril 3 x 6,25 mg, spironolakton 1x25 mg, digoxin 1x 0,125 mg. There were no sign of lung oedem in his x- ray after treatment. The patient underwent 14 days of treatment before finally being declared ready for outpatient treatment. At the 6th month after leaving hospital treatment. Echocardiography was performed on the patient and the finding were trivial MR, Mild TR with no dilatation on every chambers. EF on LV level was 63 %..

Conclusion:
Althought patient's condition had improved. The patient's carditis still seemed reversible if we looked at the clinical and echo parameters. Regular monitoring should be performed to prevent recurrent carditis in these patient.

Keywords: echocardiography, regurgitation, Rheumatoid, mitral

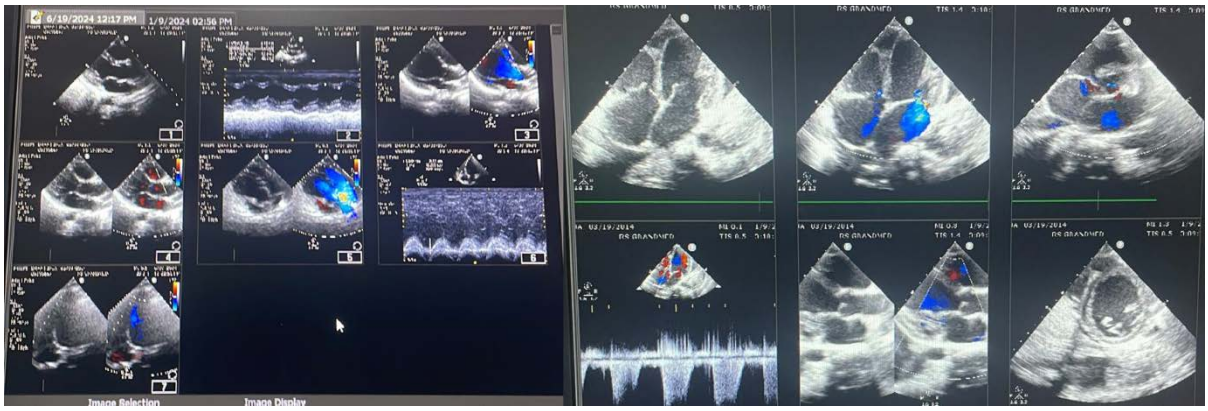

Figure 1. A. Echochardiography on his first day of admission. B. Echo on his 6th months of control after admission

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ACUTE ISCHAEMIC STROKE WITH LEFT VENTRICULAR APICAL THROMBUS IN VERY LOW EJECTION FRACTION HEART FAILURE PATIENT THE ULTIMATE DEADLY ALLIANCE

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Background:
Left ventricular (LV) thrombus is one of the most feared complications in patients with severe ischemic heart disease and heart failure, because it could carry significant risk for catastrophic complications including high risk for recurrent cardioembolic stroke and death. Risk factors for the development of LV thrombus include age, myocardial infarction location, infarct number, size and extention, and impairment in global or regional LV function (i.e. ejection fraction < 40%). There is a lack of consensus when it comes to combination therapy recommendations regarding to lethal combination of acute ischaemic stroke, heart failure and LV thrombus.

Case illustration:
A 61-year-old woman presented with a hemiplegia on the left side of her body since 1 day before admission. She also had slurred speech, paresis of right plica nasolabialis, oedema of left hand and foot, and also recurrent retrosternal chest pain episodes. She had no history of hypertension or diabetes. Brain CT scan revealed extensive infarction in the subcortical cortex of the right temporoparietal lobe, right corona radiata, right external capsule, right basal ganglia, right maxillary sinusitis. Electrocardiography showed Sinus Rhythm with left axis deviation, poor R wave progression and ischaemia in lateral leads. Our Echocardiography resultshowed dilated all chambers with eccentric LVH, Ejection Fraction 24% (Simpson's) with global hypokinetic, Mild MR, Moderate TR, withmoderate pericardial effusion, low stroke volume, LV apical Thrombus, and lung ultrasound revealed bilateral massive pleural effusion. No Mobile Thrombus found in bilateral carotid duplex ultrasound, other finding was only non-significant atheromatous plaque. Other findings was hypoalbuminemia (1.0 g/dl), acute kidney injury, and hyperglycemia (224 mg/dl). The patients was treated with intravenous anti-coagulant followed by oral anti-coagulant, decongestion with diuretics, and guideline-directed medical therapy (GDMT) for heart failure.

Conclusion:
Intracardiac LV thrombus is not an uncommon cause of cardioembolic stroke. We report a case of a woman with significant LV apical thrombus burden who had suggested cardioembolic ischaemic stroke event. Detection of LV thrombus by echocardiography in embolic stroke event and heart failure was fundamental, and anti-coagulation was still the mainstay treatment for LV thrombus followed by GDMT for the cornerstone pharmacological therapy for heart failure.

Keywords: Ischaemic Stroke, Heart Failure, LV Thrombus

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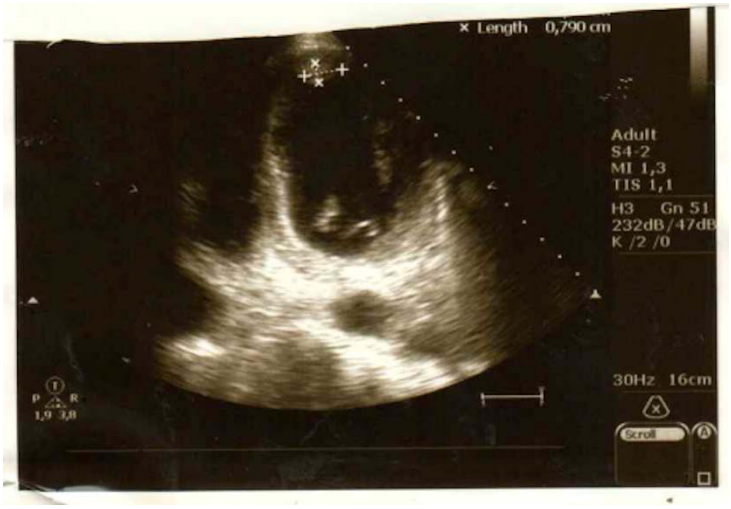

Figure 1. LV apical thrombus finding

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FACET OF RIGHT-SIDED HEART FAILURE: A CASE OF SEVERE CLINICAL COURSE DETERIORATION IN 47-YEAR-OLD WOMAN

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Background:
Many a time the right ventricle is regarded as the 'younger brother' of the left ventricle and is treated as a less important member of the contractile apparatus. Right heart failure as the primary presentation of acute decompensated HF and cause of hospitalisation accounted for 2.2% of HF admissions in the CHARITEM registry. As a distinctive disease with multiple aetiologies, PAH is recognized as a cause of right-sided heart failure. In-hospital mortality percentage is ranging from 26% up to 41%.

Case illustration:
A 47-year-old woman had progressive shortness of breath, presenting to the emergency department for several months. The patient also complained of bilateral leg edema, fever, and productive cough. Initial ECG showed RVH and RAD. Chest X-Ray revealed cardiomegaly. The patient was given norepinephrine to ensure adequate perfusion due to hypotension, as well as furosemide and dopamine to reduce congestion. However, on the 2nd of hospitalization, the patient was seen to have PVCs bigeminy due to arrythmogenic properties of vasopressors. After vasopressor substitution using milrinone and treatment with amiodarone, the patient still experienced frequent PVCs and subsequent episodes of run VT. Echocardiogram revealed enlarged right ventricular cavity (basal RVIDd 6.4 cm), decreased systolic function (TAPSE 1.7 cm) and pericardial effusion was also noted. There was significant severe tricuspid regurgitation (maxPG 38 mmHg; Vmax 3.1 m/s), pulmonal regurgitation (Sdec 1.9 m/s^2) with increased pulmonary artery diameter 3.1 cm and estimated mPAP was 58 mmHg. RA was enlarged (84 x 59 mm). Given of these abnormal findings suggestive of increased RAP accompanied by signs of HF, pulmonary artery dilation, high probability of PH and suspicious chest X-Ray, Thorax CT-Scan showed dilated inferior vena cava (5.4 cm), no visible thrombus in pulmonary vasculature. The Cardiac CT-Scan also found fluid density (8 Hounsfield Unit) in the pericardial space with a maximum thickness of 4.6 cm. Inevitable death by the 4th week of hospital stay due to refractory multifactorial shock and MODs.

Conclusion:
Management of isolated acute right HF remains more of an art than a science in the absence of robust randomised data. In addition to treating the specific cause, RV preload optimisation, the use of selective pulmonary vasodilators, RV inotropic support and temporary MCS form integral components of a comprehensive strategy to support the failing right heart.

Keywords: Cardiac CT, Pulmonary Hypertension, Right Heart Failure, Pericardial Effusion, Shock Cardiogenic

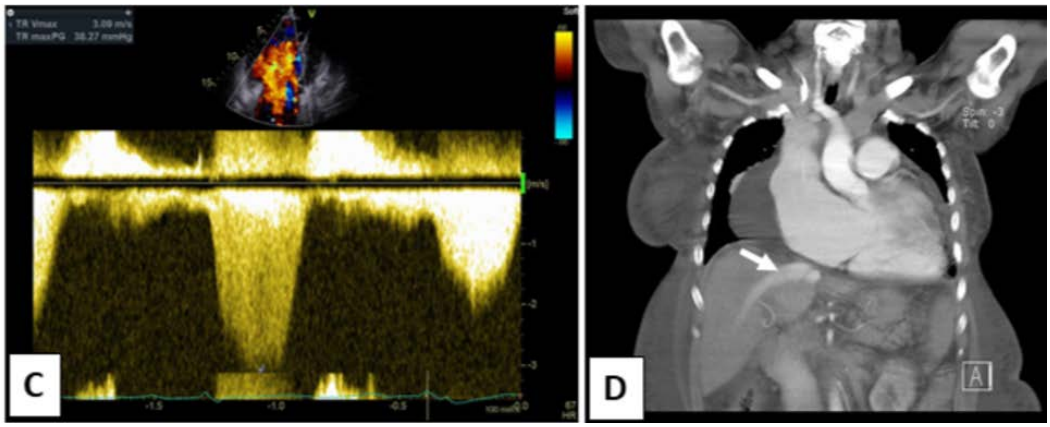

Figure 1.C. TTE 2D showed TR Severe and Main Pulmonary Artery Dilatation; Abdominal computed tomography findings revealed engorgement of the hepatic vein (black arrow, D

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FEMORAL ARTERY PSEUDOANEURISM POST CARDIAC CATHETERIZATION

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Background:
Pseudoaneurysm is the most common complication of arterial catheterization, and occurs most frequently in the femoral artery which is frequently used for diagnosis and interventional procedures.

Case illustration:
A 67-year-old female patient came with shortness of breath for the last 3 months and intermittent chest pain in the last 3 days. The patient underwent PCI in 2019. Based on the history and examinations, the patient was diagnosed with recent ST-elevation myocardial infarction (STEMI) anteroseptal Killip I TIMI 10/14 onset 3 days, coronary artery disease (CAD) history of PCI in 2019, functional congestive heart failure II. The patient was then scheduled for elective PCI.On March 13, 2024, PCI was performed with access from the femoral artery. After the procedure, the patient's condition was stable and without complaints. The first day after aff femoral sheath, the patient complained of swelling and pain in the right groin and a lump measuring 7x5 cm, the centre was bluish red, had a hard consistency, was fixed, there was tenderness, and a bruit was heard. Compressive splinting with sand pillows and elastic bandages, immobilization, and regular hematoma evaluation are performed on patients. During treatment, the pressure splint is continued until the swelling decreases. The patient was outpatient after 21 days of treatment with improving condition of the right groin which was no longer swollen.

Conclusion:
A pseudoaneurysm is a localized hematoma surrounded by soft tissue, appearing like a sac with a persistent connection with an artery, which appears like a narrow neck or channel. Femoral artery pseudoaneurysm is the most common complication after catheterization with femoral artery access. The incidence is approximately 0.2-0.5% after diagnostic procedures and 0.2-0.8% after interventional procedures. Arterial examination with doppler ultrasound is considered the gold standard in the diagnosis of pseudoaneurysms, with a sensitivity of 94% and a specificity of 97%. Treatment for pseudoaneurysms includes conservative or observation, ultrasound guided compression, ultrasound guided thrombin injection, and surgery.

Keywords: Femoral Artery Pseudoaneurism, Cardiac Catheterization

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REPERFUSION TREATMENT IN LATE ONSET ST-ELEVATION MYOCARDIAL INFARCTION (STEMI) ANTERIOR EXTENSIVE: IS TIME A MATTER OR NOT?

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Background:
Early reperfusion of the coronary artery in patients with STEMI has been the first choice of treatment. Myocyte death of the endocardium isproportionate to the duration of coronary artery occlusion which has been recognized since the 1970s.Dealing the patients who miss the time window for early reperfusion is still under debate.

Case illustration:
A 52-years-old male complained worsening chest discomfort and nausea, radiated to left arm 15 hours before admission. He is an active smoker and has uncontrolled hypertension. Electrocardiogram (ECG) showed ST-segment elevation in lead V1-V6, I and aVL. Laboratory results found elevated CK-MB (84 U/L), SGOT (104 U/L), SGPT (41 U/L), Hs-Troponin I (24256 ng/L) and NT-ProBNP (2878 pg/dL). He was diagnosed as STEMI anterior extensive late onset (>12 hours). He underwent coronary angiography. At left anterior descending (LAD), it revealed stenosis 70% proximal, total occlusion after diagonal branch and 80% mid diagonal. At left circumflex (LCx), it showed stenosis 80% mid OM 1 and OM 2. At right coronary artery (RCA), it was found stenosis 40% mid and stenosis 80% mid RPL. At ramus intermadiate showed myocardial bridging in mid. Percutaneous coronary intervention (PCI) was performed with two stents which were advanced at proximal and mid LAD, respectively. The summary was coronary artery disease 3 vessels disease (CAD3VD) post PCI LAD. After few days, he was discharged and treated with bisoprolol 1x1.25 mg, aspilet 1x80 mg, ticagrelor 2x90 mg, atorvastatin 1x20 mg, ramipril 1x5 mg and spironolactone 1x25 mg. At outpatient clinic, echocardiographic showed mild reduced left ventricular (LV) contractility (EF 43%), normal right ventricle contractility (RV), concentric left ventricular hypertrophic (LVH) and mild mitral regurgitation (MR). Nowadays, consensus as to whether PCI is beneficial to late onset STEMI patient (> 12 hours) is still absent. A nationwide observational studies in French showed that coronary revascularization of late onset STEMI patients is associated with better short and long-term clinical outcomes (within 12-48 hours).

Conclusion:
Reperfusion is still considered for late onset STEMI patients (within 12-48 hours) as it gives beneficial in short and long-term.

Keywords: late onset STEMI, reperfusion, myocardial infarction

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SHOCK CARDIOGENIC IN NSTE-MI FINDINGS, DOES THE SCAI SHOCK STAGING HELP WITH MANAGEMENT DECISION? : A CASE REPORT

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Background:
We sought to determine the outcomes of patients with cardiogenic shock (CS) complicating non-ST-segment elevation acute myocardial infarction (NSTEMI) with initial classification statement from the Society for Cardiovascular Angiography and Intervention (SCAI) regarding Grade D. These patients represent high risk (ST-segment depression), where suboptimal therapy, especially in the setting of shock, may lead to recurrent shock. However, the SCAI SHOCK Classification could provide a unique opportunity to provide individualized management by matching the degree of support to the severity of CS.

Case illustration:
Mr. ZM, aged 58 years, was referred from Meulaboh General Hospital with complaints of shortness of breath and left-sided chest pain. Physical examination revealed blood pressure of 88/60 mmHg, increased respiratory rate (24 breaths per minute), cold and pale extremities, 88% SpO2, and dyspnea suggestive of cardiogenic shock. In previous hospital, the patient also has sign of shock, such as blood pressure of 58/42 mmHg, Respiratory rate 32 per minutes, 85% SpO2, and dyspnea. The chest pain was described as a heavy object pressing on the left chest, radiating to the back and jaw, lasting > 20 minutes. The pain worsens with activity and does not improve with rest with active smokers and uncontrolled hypertention. Laboratory results showed elevated troponin levels. The EKG revealed ST deviasion in Leads I, II, III, aVF, V5, and V6. The patient was diagnosed with cardiogenic shock due to ACS with staging D SCAI Shock Classification, NSTEMI Very High Risk TIMI 4/7 GS 128.

Conclusion:
In this case study, we reported a case of a 58-year-old man with cardiogenic shock due to ACS with staging D SCAI Shock Classification, NSTEMI Very High Risk TIMI 4/7 GS 128 on CAD 3VD with CTO LAD and RCA pro CABG. However, the SCAI SHOCK Classification were rarely to use and remains speculative, it seems logical to evaluate this strategy objectively.

Keywords: SCAI SHOCK classification

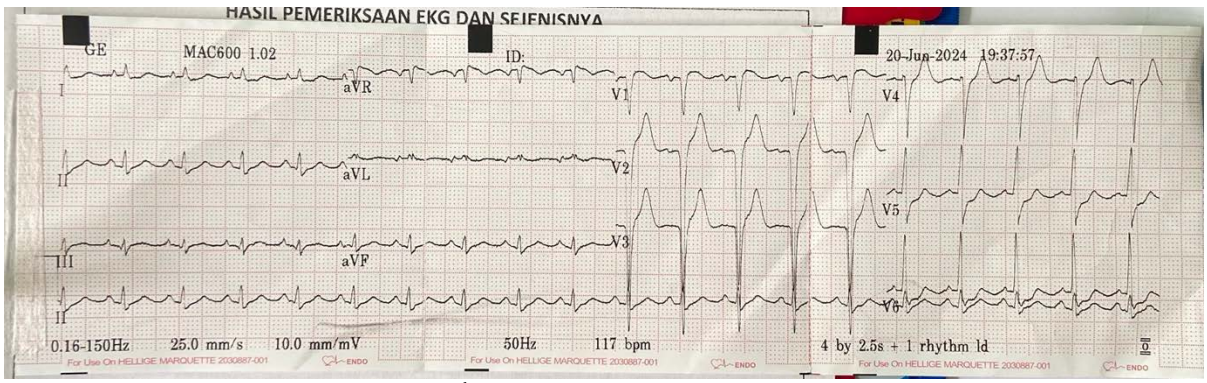

Figure 1: ECG findings in ER on June, 20th 2024, with ST-deviation refers to NSTE-ACS

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PULMONARY HYPERTENSION ASSOCIATED SYSTEMIC LUPUS ERYTHEMATOSUS

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[1]Rumah Sakit Pusat Angkatan Darat Gatot Soebroto

Background:
Pulmonary hypertension, defined as an elevation in blood pressure in the pulmonary arteries, is a heterogeneous group of disorders that result in an elevation in blood pressure in the pulmonary arteries (mean pulmonary artery [PA] pressure [mPAP] >20 mm Hg). Pulmonary Hypertension (PH) is a common manifestation in patients with Systemic Lupus Erythematosus (SLE) and varies from asymptomatic to life-threatening disease.

Case illustration:
A 43 years old female patient nonsmoker, nondiabetic, normotensive lady presented to us with shortness of breath and palpitation for one year. Physical Examination: Blood Pressure 138/80 mmHg, Heart Rate 83 beat/minute, Respiratory Rate 22 times/minute, Temperature 36.0 C, Electrocardiogram showed sinus ritme with low voltage. On chest X-ray examination, there were no abnormalities and there was cardiomegaly. Laboratory test displayed hypoalbuminemia, proteinuria, dsDNA (+) Sm (+++) CRP (-). Echocardiography results showed PA,RA, RV dilatation,LV concentric. Systolic function with an EF of 63%, TR moderate, PR moderate (PR PHT 469 ms) high probability pulmonary hypertension, disfunction diastolic grade I with normal LAP. mPAP 37.6 mmHg . Patient was diagnosed with pulmonary hypertension due to SLE, patient was checked to the hospital and said systemic lupus erythematosus was treated with myfortic 2x180mg, CACO3 3x500mg, cavit D31x1, revatio 3x20mg, Spironolacton 1x25mg, miniaspi 1x80mg.

Conclusion:
Pulmonary hypertension is a rare complication of lupus erythematosus, occurring within the first 5 years of the onset of SLE diagnosis in most cases.Doppler echocardiography is recommended for early detection of pulmonary hypertension in SLE. Anti-U1-RNP,anticardiolipin antibodies, and Raynaud's phenomenon are predictors of pulmonary hypertension in SLE patients. Patients with unknown or untreated pulmonary hypertension can progress to dilated right heart failure and can lead to death. Although no therapeutic regimen is highly effective in treating SLE with pulmonary hypertension, patients should be treated aggressively with immunosuppressants and anti-inflammatories, in combination with vasodilators because the disease can be progressive. The overall 5-year life expectancy of SLE patients with pulmonary hypertension is relatively good, about 83.9%. Pulmonary hypertension is a predictor of survival in SLE patients.

Keywords: Pulmonary hypertension

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MANAGEMENT DILEMMAS OF RHEUMATIC HEART DISEASE IN RURAL AREAS IN TEGAL REGENCY: THE IMPACT OF SOCIAL DETERMINANTS

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[1]RSUD Dr. Soeselo
[2]RSAD Wira Bhakti

Background:
Rheumatic Heart Disease (RHD) is a commonly acquired heart disease in children with especially high prevalence in Indonesia. Persisting high social determinants with a lack of culturally competent health system results in ineffective disease management. This case report aims to highlight the care challenges of paediatric RHD patients in Indonesia.

Case illustration:
A 15-year-old girl presented to the emergency room with an acute clinical presentation indicative of acute rheumatic fever (ARF) with carditis. She had visited community clinics twice before developing additional symptoms of dyspnea. A history of recurrent throat and joint pain was found. It was also often suffered by her roommates at a boarding school. Echocardiography revealed severe triple valve regurgitation. She received antibiotics to eradicate the inciting streptococcal infection and several carditis medications. The patient's condition substantially improved following the medications despite a few worsening episodes. However, notable distance to healthcare facilities and financial limitations led the patient to become unreachable and lost to follow-up, despite being advised to adhere to secondary prophylaxis treatment for the next ten years.

Conclusion:
A more holistic approach to care in RHD management, which includes earlier diagnosis, correct treatment regimen, and proper monitoring programs for secondary prophylaxis adherence should be established to ensure successful preventative measures despite persistent socioeconomic risk factors.

Keywords: Boarding School, Children, Rheumatic Heart Disease, Rheumatic Fever, Secondary Prophylaxis

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THE EFFICACY OF PERMANENT PACEMAKER IN MANAGING TAVB: A CASE REPORT AND STRONG RECOMMENDATION FOR IMPROVED PATIENT OUTCOMES

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Background:
Presyncope or near-syncope is one of clinical definitions of symptomatic bradycardia. The initial management of symptomatic bradycardia usually begins with the use of intravenous atropine or dopamine followed by pacemaker insertion if there was no improvement.

Case illustration:
A 78-years-old female came to Emergency Room at Pematang Siantar Army Hospital with chief complaints of previous presyncope and intolerance activity. There was no typical chest pain and shortness of breath. The physical examination was normal, blood pressure (BP) was 100/80 mmHg and heart rate was 42 bpm. The laboratory result showed complete blood count and electrolyte were in normal limit and renal function impairment was found (Ureum: 47 (N: 10-50), Creatinine: 1,4 (N: 0,6-1,2)). ECG showed Total AV Block. The patient was given dopamine and the heart rate did not improve. The patient then referred to Adam Malik Hospital for insertion of a temporary pacemaker followed by the placement of permanent pacemaker. Patient had been observed and we found the presyncope is clear, when the insertion of permanent pacemaker has done. The patient was observed for follow up presyncope after the insertion of PPM, and the result was the patient no longer experienced presyncope.

Conclusion:
Atropine or dopamine is rarely effective in increasing the heart rate in patients with total AV block. There is modest evidence and strong clinical consensus that patients with persistent second or total AV block must receive permanent pacemaker.

Keywords: Total AV Block, Dopamine, Permanent Pacemaker

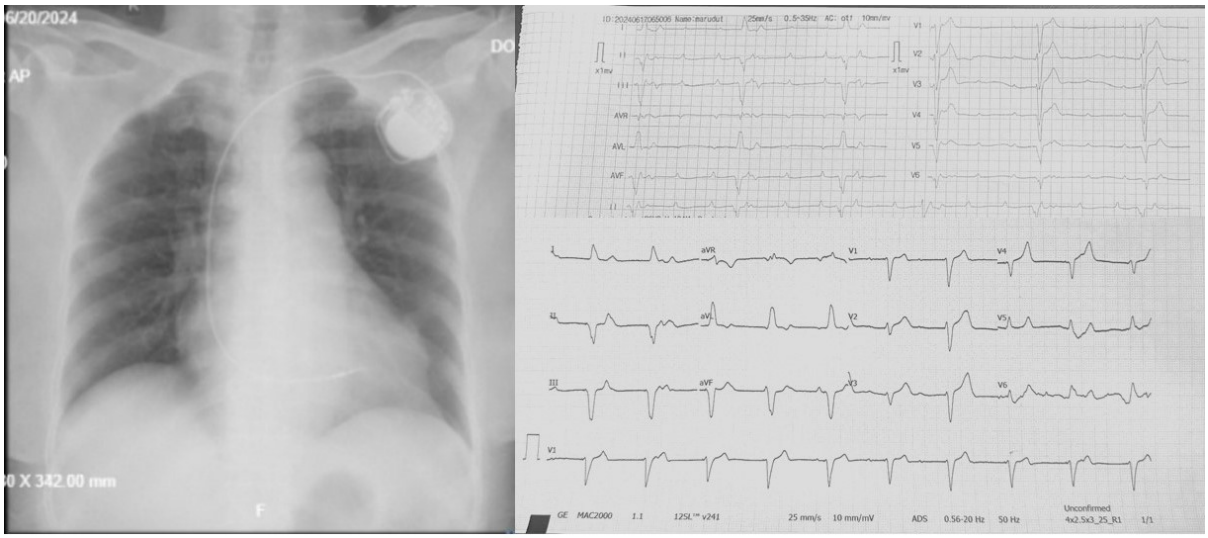

Figure 1. An X-ray (left) shows the PPM placement, and The ECG (upper right) when off The TPM and The ECG (lower right) when on The TPM.

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REFRACTORY VENTRICULAR ARRHYTHMIAS IN PACEMAKER-DEPENDENT PATIENT: CASE REPORT

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[1]Syiah Kuala University

Background:
Refractory ventricular arrhythmias are a clinical situation marked by three or more sustained episodes of ventricular arrhythmia occurring closely together, typically within a 24-hour period, with each episode separated by at least 5 minutes. A trial determined that pacing was deemed to be the cause in 2.6% to 5.2% of VT/VF episodes. This ventricular electrical storm manifests as recurrent episodes of hemodynamically unstable Ventricular Tachycardia (VT) or Fibrillation (VF), which often presents as a medical emergency. The primary goals are to alleviate symptoms and potentially improve prognosis.

Case illustration:
A 49-year-old woman was admitted to emergency room with chief complain loss of consciousness that occurred without warning like chest pain, shortness of breath, or palpitation. She had no regular medication. Her background is hypertension, and had implanted a VVIR pacemaker for total atrioventricular block since 2022. She did not response and monitor showed ventricular fibrillation. She was ROSC after given CPR. The ECG converted to the rhythm that programmed in the pacemaker. She continued to have run of sustained VT, which terminating after Amiodarone administration. Chest radiograph showed cardiomegaly and device placement was in the right place. The echocardiography demonstrated the decrease of LV function (EF 45-51%) with regional wall motion abnormality. Pacemaker interrogation revealed that patient had VT episode 2 days before admission. After the condition were stable, patient was discharged home and scheduled for elective cardiac angiography to evaluate the etiology of refractory ventricular arrhythmias due to coronary disease. The occurrence of ventricular electrical storms involves a complex interaction of three factors: the presence of an electrophysiological vulnerable substrate, triggers, and autonomic dysregulation. The possibility of cardiac pacing causing arrhythmias is uncommon. Conventional pacing modes like VVIR have been noted to potentially induce VT/VF.

Conclusion:
Our case designates that the potential of pacemaker-dependent-patient undergo refractory ventricular electrical arrhythmias. That condition can represent a medical emergency. Ventricular electrical storm requires a complex management approach that addresses multiple factors simultaneously, particularly in patients at risk of hemodynamic instability. It is important to investigate potential triggers such as ischemia or electrolyte abnormalities, although identifying causative factors may be challenging.

Keywords: Ventricular electrical storm, Permanent pacemaker, VVIR, Refractory ventricular arrhythmia

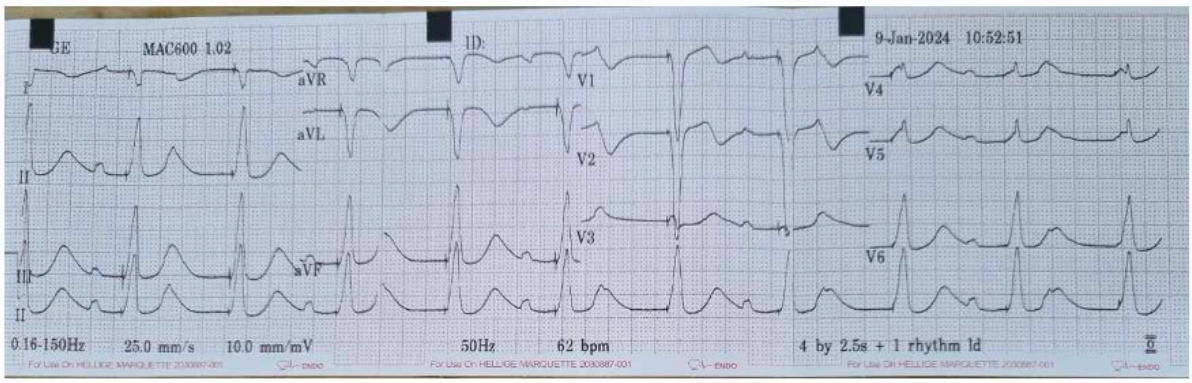

Figure 1. Electrocardiogram after returned on spontaneous circulation. The rhythm converted to sinus rhythm with complete AV dissociation and a paced ventricular rhythm at 60 beats per minute, as it was programmed in the pacemaker setting

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MULTIDISCIPLINARY MANAGEMENT OF ATRIAL SEPTAL DEFECT IN A HIGH-RISK PREGNANCY WITH NARROW PELVIS

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Background:
Managing atrial septal defects (ASD) during pregnancy presents significant challenges due to increased hemodynamic demands, which are further complicated by conditions like a narrow pelvis. This report examines a complex case involving these factors.

Case illustration:
A 25-year-old woman, G4P0A3L0, at 38-39 weeks gestation was referred for a cesarean section due to Atrial Septal Defect (ASD) and a narrow pelvis. ASD was diagnosed at age 12 but she declined ASD closure. She had three miscarriages at 4-5 weeks gestation. Current pregnancy check-ups showed a healthy fetus. Physical examination revealed a fixed split S2, a BMI of 17.5, height of 137 cm, pre-pregnancy weight of 33 kg, and pregnancy weight of 45 kg, with inadequate pelvic dimensions. Investigations showed ECG findings of sinus rhythm, RAD, RBBB incomplete, and RVH. Chest X-ray indicated cardiomegaly with increased pulmonary pressure. Echocardiography revealed an IAS gap of 1.7-2.2 cm with L-R shunt, EF of 53%, right heart dilation, RVOT dilation, Septal Flattening, and mild tricuspid regurgitation. Ultrasound confirmed an intrauterine live fetus with a normal biophysical profile. Patient underwent C-section under spinal anesthesia, delivered a male baby weighing 2980 grams, with APGAR scores of 8/9. Vital signs were stable. Post-operative care included a stay in the High Care Unit for 2 days, followed by the general ward for 4 days. Both mother and baby had no complications.

Conclusion:
Timely surgical intervention and multidisciplinary care are crucial for managing high-risk pregnancies with congenital heart defects like ASD, ensuring favorable outcomes for mother and baby.

Keywords: Narrow Pelvis, Spinal Anesthesia, High Risk Pregnancy, Multidisciplinary Care, Atrial Septal Defect (ASD)

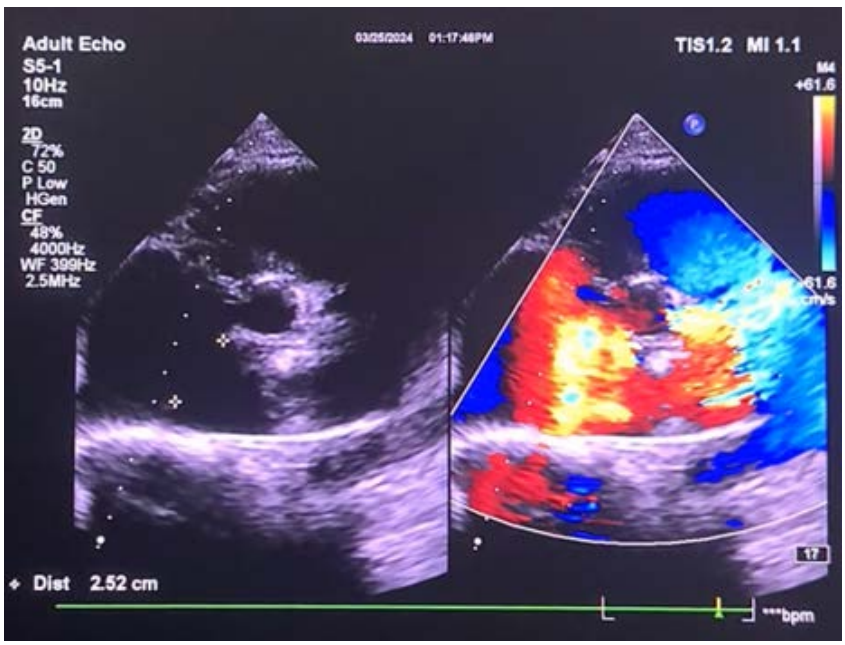

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Figure 1. Echocardiography. Parasternal Short Axis (PSSA) Aortic Valve Level .L-R Shunt due to Atrial Septal Defect

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10TH TIMES CARDIAC ARREST IN INFERIOR STEMI PATIENT IS A CHARM? A CASE REPORT

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Background:
Cardiac arrest mostly occurs in coronary artery disease, one that is often found is Inferior STEMI. Mostly ventricular tachycardia (VT) rhythm before changing to ventricular fibrillation (VF). Cardiopulmonary resuscitation (CPR) and electrical defibrillation is optimal therapy. Delay in using defibrillator reduces success rates by 7-10%.

Case illustration:
A 34 years old man came to ER with chest pain since ± 3 hours. Risk factor dyslipidemia, smoking, and alcoholic. The patient had seizure and cardiac arrest occurred due to VT. During CPR, the patient received seven defibrillator shocks after which the patient achieved return of spontaneous circulation (ROSC). CPR occurred for 37 minutes. ECG showed Inferior STEMI (Figure 1). Condition after CPR was E1M1V1 intubated on ventilator, BP 80/40mmHg, HR 63x/minute, extremities felt warm, no edema, Glucose ad random 374. Diagnosed with Cardiogenic Shock due to Inferior STEMI Killip IV TIMI 8 Post ROSC with stress hyperglycemia. After being transferred to ICCU, patient was given fluid resuscitation, vasopressors and inotropes. Because at that time the patient were still not eligible to underwent Primary PCI less than 120 minutes due to hyperglycemia, we decided to use alteplase as first line treatment. Three hours later the patient had another cardiac arrest due to VT. During CPR the patient received one defibrillator shock after which the patient ROSC. Antiarrhythmic intravenous drips were initiated. At the same time, the patient returned to cardiac arrest 8th times and received five defibrillator shocks until undergoing Rescue PCI. The patient successfully underwent Rescue PCI with 1 DES on pLCX 1DES on p-mRCA, double culprit STEMI. The patient was extubated on the seventh day with low dose of inotropes and was fully conscious. Echo showed left ventricular ejection fraction 40%, RMWA +, MR mild ec ischemic, est RAP 15mmHg.

Conclusion:
The use of immediate defibrillation during pulseless VT rhythm improves patient survival.

Keywords: defibrillation, ROSC, cardiac arrest, Inferior STEMI, VT

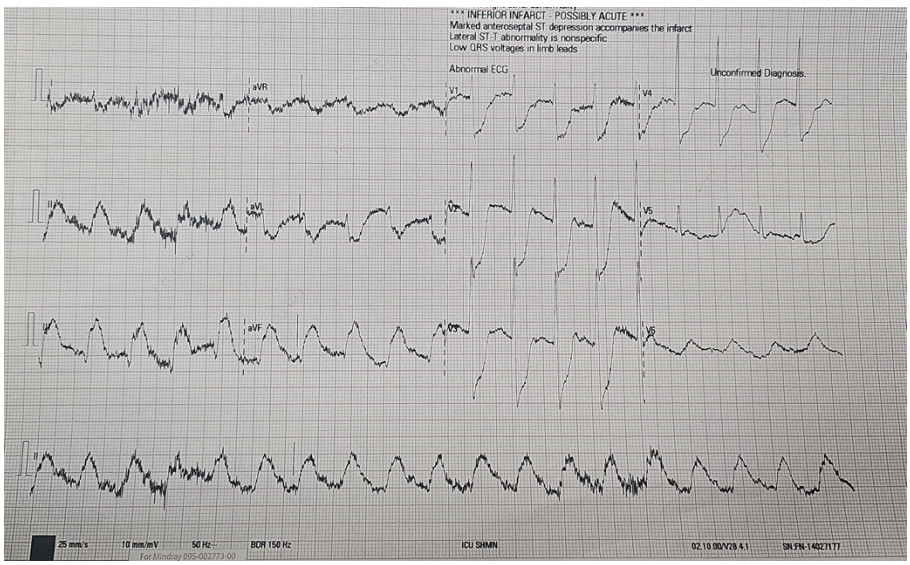

Figure 1. ECG showed Inferior STEMI

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ECG CLUES TO A SILENT CULPRIT: UNMASKING CENTRAL VASCULAR PROBLEM IN A HYPERTENSIVE PATIENT

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Background:
In clinical practice, electrocardiograms (ECGs) are predominantly used to diagnose cardiac conditions such as ischemia or arrhythmias. However, ECG changes can also signal non-cardiac issues, including central nervous system events. Recognizing these signs is crucial, especially in patients presenting with atypical symptoms that may mask underlying serious conditions like intracerebral hemorrhage.

Case illustration:
A 47-year-old female presented to the emergency department with a chief complaint of cephalgia for one day, accompanied by nausea, vomiting, and epigastric tenderness. Physical examination revealed a Glasgow Coma Scale score of 15, blood pressure of 210/110 mmHg, other vitals are stable, and no signs of lateralization or neurological deficits. She was admitted for hypertensive urgency and dyspepsia syndrome and consulted with internal medicine. Her medical history included uncontrolled hypertension. Initial ECG showed slight inverted T waves in leads V1-V4, prompting a cardiology consultation for suspected acute coronary syndrome. Serial ECGs revealed progressively larger inverted T waves, suggesting a central neurological issue. Despite the absence of neurological deficits, a neurology consultation was obtained, and a CT scan was performed. While awaiting the CT scan results, the patient became somnolent. The CT scan ultimately revealed an intracerebral hemorrhage in the left lobe.

Conclusion:
This case highlights the importance of considering central nervous system pathology in patients with significant ECG changes, even in the absence of typical neurological symptoms or detectable neurological deficits. The progressive ECG changes observed in this patient were instrumental in raising suspicion for a central vascular event, ultimately leading to the diagnosis of intracerebral hemorrhage. Additionally, this case underscores the need for heightened vigilance for neurovascular events in patients presenting with hypertensive emergencies, emphasizing the value of ECG as a diagnostic tool that extends beyond cardiac abnormalities.

Keywords: ICH, ECG, Hypertension, Giant inverted T-wave

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BUDDY WIRE TECHNIQUE FOR STENT PLACEMENT IN REMOTE RIGHT CORONARY ARTERY LESION THROUGH TURTOUSITY

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[1]Universitas Syiah Kuala

Background:
Percutaneus Coronary Intervention has become the preferred modality of revascularization for Coronary Artery Disease in majority of cases. However, despite advancements in technology, 2.7% to 3.3% of cases still experience difficulties during stent delivery. Vascular tortuosity, lesion severity, lesion length, chronic total occlusion (CTO), lesion located distal to previously implanted stent, stent length and structure, inadequate guiding catheter support in dilated aortic root, or an unadjusted angle of take-off of the proximal segment of the target coronary artery are among the factors linked to stent delivery failure. Buddy wire is one of the technique than can facilitate stent delivery in remote area lesion through tortuosity.

Case illustration:
A 65 years old man came to undergo elective staging PCI after previous PCI to LAD 3 month ago. We decided to use radial access despite history of severe turtousity on the subclavia, and Coronary angiogram revealed normal left main, stent patent on LAD with severe calcification on distal LAD, CTO on OM 2 in small vessel coronary artery, and stenosis 80-90% on osteal to proximal posterior descending artery with severe calcification. PCI was performed at Posterior Descending Artery (PDA). Canulation performed with guiding AL 3.5/6F, guidewire was inserted until distal PDA. Predilatation was performed several times. Stent DES Coroflex Isar Neo 2.2.5/24 mm was inserted with the help of buddy wire technique to reach target area. Stent was deployed properly, angiogram evaluation show TIMI III Flow.

Conclusion:
Buddy wire should be utilized first when more backup is required. The method entails inserting a second guide wire parallel to the first one that was put in. By straightening the vessel, it lessens friction between the inner surface of the vessel and the device—specifically, stents.

Keywords: Right Coronary Artery, Buddy Wire Technique, Coronary Artery Disease, Percutaneus Coronary Intervention, Turtuosity

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WHEN TO START ANTIPLATELET THERAPY AFTER PCI IN HEMORRHAGIC STROKE: A CLINICAL DILEMMA

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Background:
The incidence rate of intracranial hemorrhage (ICH) within the first 30 days after PCI procedureis small (2.1% per 1000 person-year), but fatal complication is high. Antithrombotic therapy after percutaneous coronary intervention (PCI) recently become challenging for doctors to withholding or restarting antithrombotic agents.

Case illustration:
A 45-year-old man with the history of intracranial haemoragic 1 weeks after elective Percutaneous Coronary Intervention (PCI). The Coronary angiography showed CAD one-vessel disease, he underwent coronary stenting in the proximal-mid LAD using DES EVROSURE 3.5 x 54 mm. Cineangiography evaluation showed TIMI Flow 3 and no residual stenosis. The Brain CT Scan demonstrated ICH punctate at lentiformis nuclei dextra, chronic SDH. He had history of hypertension and also consumed Aspilet and Ticagrelor as DAPT for CAD. Dual anti-platelet was postponed because of life threateaning bleeding. After discharged, the patient never controlled to cardiologist nor consumed cardiovascular drugs because he afraid of recurrent stroke. He was rehospitalization 3 month after that with seizure, slurred speech and weakness. There are no chest pain nor shortness of breath. The patient's electrocardiography revealed sinus rhythm with poor R progression. The cardiac enzyme level wasn't increased. We educated the patient to start Cardiovascular drugs, especially DAPT and joined care with Neurology Department to treat sequele stroke of the patient.

Conclusion:
Early resumption of antiplatelet therapy following ICH with safely should be address with close monitoring and without hesitate after considering the risk and benefit. However, guidelines do not currently define the optimal timing of antiplatelet (re)initiation following ICH but clinical benefit if resumed as early as within the first 2 weeks. Patient and family needs to be educated regarding it to prevent poor-compliance and it consequences.

Keywords: Dual Anti Platelet Therapy, Antithombotic Therapy, Percutaneous Coronary Intervention, Intracranial Haemoragic

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EXERCISE-INDUCED ARRHYTHMIAS IN FEMALE ATHLETES: INSIGHTS INTO ARVC AND CARDIAC REMODELING

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Background:
Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC) is a leading cause of sudden death among young athletes but can affect people of all ages and all activity levels. Intense sports activity leads to cardiac adaptations known as athlete's heart, which can mimic pathological conditions. We present two cases of female athletes experiencing ventricular arrhythmias related to intensive exercise.

Case illustration:
Case 1: A 57-year-old woman presented with skipped heartbeats without syncope or palpitations for a year. She had hypertension and engaged in endurance exercise. Holter monitoring revealed multifocal Premature Ventricular Contractions (PVCs) suggesting ARVC. Echocardiography showed all chambers dilated with normal systolic function. MRI revealed low RV volume with fibrofatty replacement of the RV myocardium.Case 2: An 18-year-old woman preparing for her police academy entrance exam reported chest discomfort post-exercise. Diagnostic tests, including electrocardiography and echocardiography, revealed left ventricular hypertrophy and frequent PVCs. An exercise stress test indicated arrhythmias during recovery. Holter monitoring confirmed the presence of ventricular arrhythmias suggesting from LCC origin (PVC burden 9%) with episode of trigeminy and bigeminy.

Conclusion:
These cases underscore the importance of differentiating between physiological athlete's heart and potential cardiomyopathies. ARVC is characterized by fibro-fatty infiltration of the RV myocardium and electrical instability. Intensive exercise can exacerbate these conditions, highlighting the need for careful assessment and management of arrhythmic symptoms in athletes.

Keywords: Ventricular Arrhythmia Cardiac Remodeling, exercise, Ventricular Arrhythmia, ARVC, exercise, Athlete's heart

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SYMPTOMATIC BRADYCARDIA IN YOUNG ADULT: A CONCOMITANT BRS, AVND & SND CASE?

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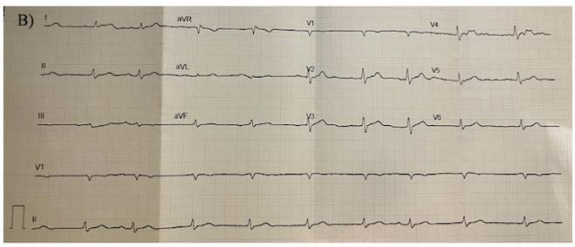
Background:
Brugada Syndrome (BrS) is an autosomal-dominant inherited arrhythmic disorder caused by *SCN5A* mutations which is characterized by coved-type ST elevation with inverted T wave in the right precordial leads without any structural cardiac abnormalities. Patients with BrS are at risk for sudden cardiac death (SCD) due to ventricular fibrillation (VF) or ventricular tachycardia (VT). However, *SCN5A* mutations are also implied in long-QT-syndrome (LQTS) type 3, progressive cardiac conduction disease (first degree to complete AV block), sick sinus syndrome, or combinations of these.

Case illustration:
A female, 25 years old, complained slow heart pulse and several episodes of presyncope since 2 months before admission. Family history of sinus node dysfunction (SND) and pacemaker implantation was confirmed. Physical examination and echocardiography showed normal cardiac findings. ECG tracing through *Apple iWatch* showed sinus bradycardia with episode of sinus pause. ECG showed sinus bradycardia with junctional escape beats. Patient was then referred for Holter ECG dan pacemaker implantation. Holter ECG showed a Brugada pattern type 1, sinus bradycardia with episode of sinus pauses and junctional escape beats. EP study was then planned to look for possibility of ventricular arrhythmia. EP study showed sinus node dysfunction (SND), AV node dysfunction (AVND) and several episodes of non-sustained VT. Patient was planned for dual chamber pacemaker or ICD implantation.

Conclusion:
SCN5A mutations are associated with BrS, LQTS type 3, AV node dysfunction and sick sinus syndrome. The diagnosis of BrS can be confirmed by ECG, Ajmaline test and genetic testing. Holter ECG and EP study are needed for evaluating AV node dan SA node function in BrS patient. Device implantation, such as pacemaker and ICD, is a therapeutic option for this patient.

Keywords: ICD, Sinus Node Dysfunction, Pacemaker, Brugada Syndrome



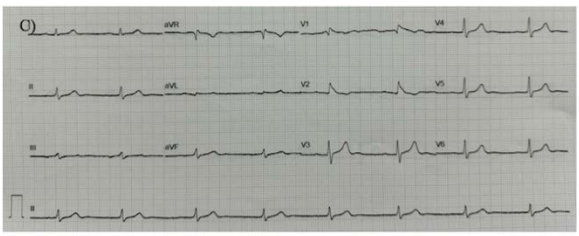


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Figure 1. Patient ECGs. A) *Apple iWatch* ECG tracing showed sinus bradycardia with episode of sinus pause. B) First ECG showed sinus bradycardia with junctional escape beats. C) Secondary ECG (V1-V2 leads located in 2nd ICS) showed sinus bradycardia and Brugada pattern type 1

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UNEXPECTED PRESENTATION OF WELLENS' SYNDROME IN PUBLIC HEALTH CENTERS: WE SHOULD BE AWARE OF IT

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Background:
Electrocardiography (ECG) is an essential tool for diagnosing and risk-stratifying acute coronary syndrome patients. Only 20% of acute ischemia ECG changes are recognized by emergency medical service (EMS) providers. Wellens' syndrome is an ECG characteristic, as certain ST-T segment abnormalities in the setting of impending myocardial infarction (MI) patients suggestively caused by critical stenosis in the proximal left anterior descending (LAD) artery. MI from a culprit lesion in the LAD artery is related to worse clinical outcomes.

Case illustration:
The first patient was a 27-year-old man smoker who presented with epigastric pain accompanied by shortness of breath in the past 1 hour. His blood pressure was 170/100 mmHg and physical examination revealed epigastric tenderness. ECG revealed biphasic T waves in leads V2-V5, suggestive of Wellens type A. The second patient was a 37-year-old man who presented after being stung by an insect 15 minutes before. He was given 0.5 mg dexamethasone intravenously and observed for 30 minutes. Upon observation, the patient suddenly experienced left-sided chest pain accompanied by diaphoresis. ECG revealed inverted T waves in leads V2-V4, suggestive of Wellens type B. Further history-taking revealed that he had experienced this kind of symptoms three months prior and had a history of hypertension, dyslipidemia, and a current smoker. Unfortunately, both patients refused to be referred for further examination and management.

Conclusion:
Physicians and EMS providers should be aware of Wellen's syndrome. Misinterpretation of this ECG characteristic could lead to fatal outcomes. Educating patients thoroughly about their condition is also important.

Keywords: Wellens syndrome, acute coronary syndrome, electrocardiogram, public health center, atypical presentation

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NAVIGATING THE COMPLEXITY : COMPLETE HEART BLOCK IN PREGNANCY ON DELIVERY

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Background:
A comprehensive management of pregnancy in a case of complete heart block, a condition of the heart characterized by dissociation between atrial and ventricular conduction. The assessment begins with thorough preconception counseling, which emphasizes assessment of maternal health, evaluation of cardiac function, and identification of potential risks.

Case illustration:
24-year-old female G1P0A0 38 weeks pregnant in labor phase 2, on examination found high blood pressure and ecg findings a complete heart block with junctional escape rhythm, the patient before and during pregnancy had no complaints. The patient was known to have rhythm disorders since 3 years ago and was advised to put a pacemaker but since there were no complaints, the family refused and did not have routine control. The patient was planned for vaginal delivery, but when she started pushing, her pulse dropped to 30x/minute, so a caesarean section was decided. A baby boy was born with a birth weight of 2450 grams, length 45 centimeters, APGAR score 8/9. The baby was in respiratory failure and required treatment in the NICU. The patient was transferred to the ICU for monitoring and with dopamine support 3mcg/kg/min, although the patient's ecg still showed complete heart block with a heart rate of 50-60x/min. On day 3 postpartum, dopamine support was stopped, the ecg still showed complete heart block with a heart rate of 45-50x/min but without any complaints, the patient was transferred to the ward and echocardiography was performed with no abnormalities found. The patient is planned to be placed with a permanent pacemaker.

Conclusion:
Total heart block is a rare condition and may be asymptomatic. A multidisciplinary team approach is essential, with collaborative efforts paving the way for improved patient outcomes.

Keywords: pacemaker, Pregnancy, Complete Heart Block

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UNMASKING DE WINTER SYNDROME: IS THE LEFT ANTERIOR DESCENDING ARTERY ALWAYS THE CULPRIT?

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Background:
De Winter syndrome is a rare form of acute myocardial infarction, distinguished by the absence of ST elevation and the presence of ST depression with hyperacute T waves which was firstly described in 2008 by de Winter et al. While this condition is commonly associated with complete obstruction of the proximal left anterior descending coronary artery (LAD), However, instances involving other coronary arteries, such as the right coronary artery (RCA) and the circumflex artery (LCX), have also been documented.

Case illustration:
A-53-year old male presented with non-radiating chest pain for 10 hours. The patient had a history of smoking. The initial 18-lead electrocardiogram (ECG) showed sinus bradycardia with incomplete RBBB and slightly elevation in J-point in inferior lead without any observable changes in the early serial ECGs. The patient was treated as non ST elevation myocardial infarction, with initial troponin I was 3009. However, serial ECGs conducted the following day revealed upsloping ST-segment depression with symmetrical tall T-waves in the anteroseptal leads (V1-V4), loss of R wave progression, and the presence of Q waves with slight ST elevation in the inferior leads. Surprisingly, early coronary angiography revealed occlusion in LCX without any lesions in LAD.

Conclusion:
This case highlights that de Winter syndrome is not exclusively linked to LAD occlusion. Serial 18-lead ECGs are crucial to prevent underdiagnosis, especially since the LCX territory is partially represented by leads V7-V9 on the ECG.

Keywords: Myocardial Infarction, Left Circumflex Artery, de Winter Syndrome

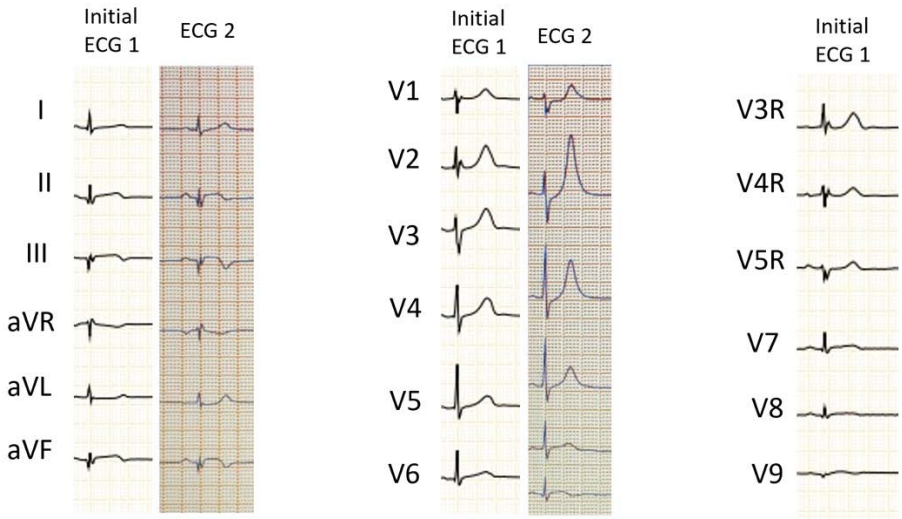

Figure 1. Initial ECG showing slight elevation in inferior leads. Serial ECG showing upsloping ST-segment depression with symmetrical tall T-waves in the anteroseptal leads

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HIGH TAKE-OFF RIGHT CORONARY ARTERY: INSIGHTS FROM CT IMAGING

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Background:
High take-off of the right coronary artery (RCA) is a rare anatomical variant that can pose challenges in clinical management and diagnosis. The main concern of this anatomical abnormality is decreased coronary perfusion which can result in myocardial ischemia. It is usually discovered incidentally on imaging modalities or during cardiac surgery for other indications. In such cases, care should be taken to avoid clamping or occlusion of the coronary arteries during interventional therapy or surgery. This case highlights the importance of awareness and careful evaluation of coronary artery anatomy variants in clinical practice.

Case illustration:
A 41-year-old female patient, came to the Outpatient Installation at the Cardiac Center H. Adam Malik Hospital with a history of short duration chest pain that was felt during activity and disappeared with rest. This complaint has been felt for the last month. She has hypertension and regularly takes anti-hypertension medication. Vital signs are within normal limits. The physical examination was unremarkable. ECG showed sinus bradycardia. There was no abnormality from the chest x-ray and laboratory results. It was decided to perform a CTCA on this patient, revealed a high origin of the RCA, located in the ascending aorta at distance of 12.2 mm from the sinotubular junction. The results also showed deep and long myocardial bridging in the mid LAD, and mild stenosis in three coronary arteries. The patient was treated with Ramipril 1x5mg, Amlodipine 1x5mg, Bisoprolol 1x15mg, Atorvastatin 1 x 20 mg. She must control risk factors and live a healthy lifestyle.

Conclusion:
This case underscores the significance of recognizing and understanding rare anatomical variations such as high take-off of the right coronary artery (RCA). Due to the rarity of this case, accurate diagnosis through advanced imaging modalities is crucial for appropriate clinical management.

Keywords: High Take Off RCA, Anatomical, CTCA, Imaging, RCA

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PATHOLOGICAL Q WAVE IN INFERIOR LEADS IN THE SETTING OF ACUTE ANTERIOR MI: IS THERE A POSSIBILITY OF PRIOR SILENT INFERIOR MI CAUSED BY TORTUOUS RCA OR MISSED WRAPAROUND LAD?

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Background:
Silent myocardial infarction (MI) indicated by Q wave on electrocardiogram (ECG) outside of the acute infarction area was associated with higher risk of major adverse cardiovascular events. Inferior MI is usually caused by RCA or distal LCx occlusion. No occlusion in those vessels prompts further investigation into other vessel anomalies.

Case illustration:
An 81-year-old female arrived at primary health care experiencing sudden loss of consciousness. Alloanamnesis revealed abdominal fullness since the night before and no history of chest pain. Physical examination showed GCS E2V2M3 with no lateralization, BP 80/60 mmHg with cold extremities and no improvement with fluid challenge. The ECG revealed ST elevation in leads V1-V4 and deep pathological Q waves in V1-V4, II, III, and aVF. She was diagnosed with anterior STEMI Killip IV and cardiogenic shock SCAI C, then transferred to tertiary healthcare for primary PCI. Pre-PCI echocardiography showed reduced global and segmental LV systolic function with EF 37%, LV diastolic dysfunction, reduced RV systolic function, and severe hypokinetic LV wall movement in basal, mid-cavity, and apical regions. Coronary angiography (CAG) revealed subtotal occlusion in the proximal LAD and tortuous but normal RCA. PCI was performed with 1 DES on proximal LAD. Further examination also found hypertension, hyperglycemia, hypokalemia, hypoalbuminemia, renal failure, and pneumonia. While the PCI was successful, she suffered from cardiac arrest 7 days after admission.

Conclusion:
The presence of Q/QS waves on inferior leads is pathognomonic for prior inferior MI. The inferior wall territory supplied either by RCA (70-90%) or distal LCx (8-15%). Inferior MI can also be associated with RCA tortuosity due to reduced perfusion pressure distal to the tortuous segment or concurrent spontaneous coronary artery dissection, which can be missed by CAG. Anatomical variants of LAD wrapping around the LV apex supplying more than 25% of inferior LV wall is another possible etiology associated with worse prognosis. Cardiac Magnetic Resonance Late Gadolinium Enhancement (cMRI-LGE) is beneficial for determining the location of myocardial scars and anomalous coronary vessels supplying the inferior wall.

Keywords: inferior MI, Q wave, wraparound LAD, silent MI, tortuous coronary artery

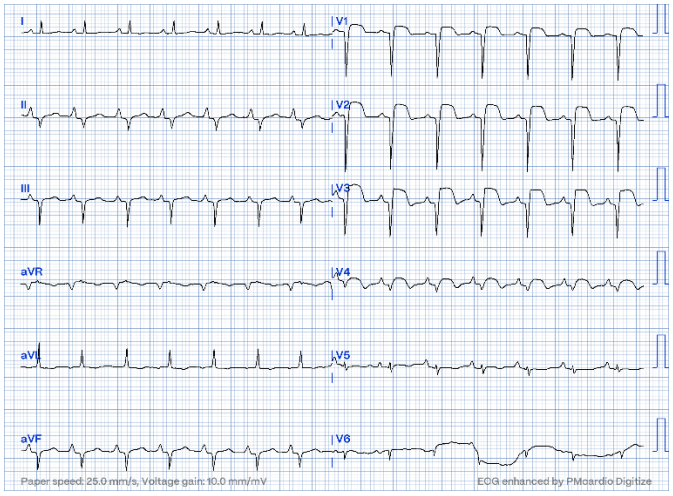

Figure 1. ECG upon arrival at the PHC

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ONE OF THE MOST URGENT VASCULAR CIRCUMSTANCES: ACUTE LIMB ISCHEMIA

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Background:
Acute limb ischemia (ALI) was a critical condition characterized by a sudden decrease in limb perfusion, which threatened the viability of the affected extremity. It was an emergency that required prompt diagnosis and intervention to prevent severe complications, including limb loss and mortality. The condition was often precipitated by underlying vascular diseases

Case illustration:
A 65-year-old male patient presented with acute limb ischemia, patient reported increasing pain in the left leg, which appeared bluish, numb, cold, and insensate. He had a medical history significant for diabetes mellitus and cardiac arrhythmias. The Doppler vascular bedside examination indicated a monophase waveform morphology from the left common femoral artery to the left profunda femoral artery, with a total occlusion at the level of the left superficial femoral artery and no blood flow below this point. The right femoral, popliteal, anterior tibial, and dorsalis pedis arteries showed biphasic Doppler waveforms. CT angiography (CTA) indicated a total occlusion of the left superficial femoral artery from the proximal third to the distal third, as well as the left anterior tibial artery from the proximal to distal segments. Percutaneous Transluminal Angioplasty (PTA) was performed on the left superficial femoral artery. This case underscored the importance of early recognition and prompt intervention in acute lower limb ischemia, particularly in patients with multiple comorbidities. The successful use of PTA in this patient highlighted the efficacy of endovascular techniques in managing acute ischemic events and improving patient outcomes

Conclusion:
The necessity of secondary prevention strategies to reduce the risk of recurrence and the importance of follow-up care to monitor patient progress and manage potential complications. Multidisciplinary approach in the management of acute limb ischemia, integrating prompt diagnostic assessments, effective endovascular interventions, and comprehensive post-procedural care to enhance patient prognosis and quality of life

Keywords: Percutaneous Transluminal Angioplasty, Acute limb ischemia

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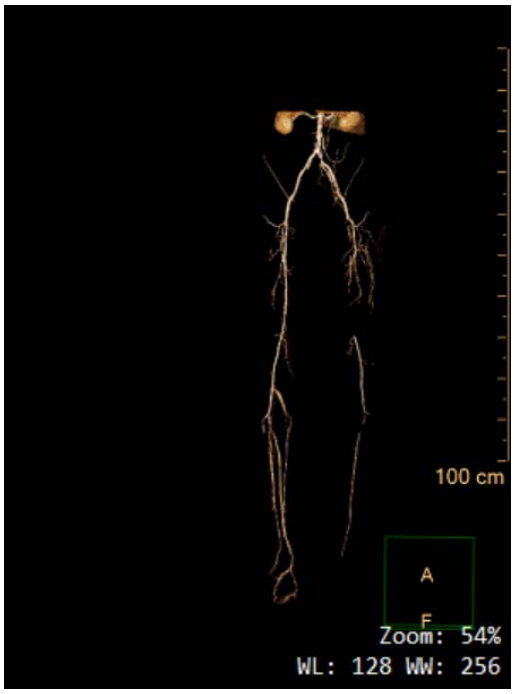

Figure 1. Computed Tomography Angiography (CTA)

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SEVERE LEFT VENTRICULAR DYSFUNCTION IN YOUNG MALE PATIENT: IN RURAL AREA

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Background:
Heart failure (HF) in young adults is rare, and is often misdiagnosed with a more common diagnosis such as lung infection. Identifying heart failure in young adults has many challenges, especially in recognizing the signs and symptoms of heart failure and the various underlying causes. Heart failure in young adults has different clinical characteristics including etiology, more severe left ventricular dysfunction and less severe symptom presentation. Early recognition of HF in young adults is important.

Case illustration:
We describe a 22-years-old man who experience shortness of breath since 1 week before. There was no specific risk factor. Physical examination, we found normal vital sign with sighly tachycardia, pansystolic murmur was found in the left RIC III-IV parastenal line, rales in both lung field and lower extremity edema. Initial ECG showed sinus tachycardia with LVH pattern. LV enlargement from chest x-ray. His Echocardiography showed dilated all chamber with global hipokinetik, decrease left ventricular ejection fraction of 19,5%, pericardial effusion and left ventricular (LV) thrombus discovered. He was eventually diagnosed with Acute Heart Failure due to Dilated Cardiomyopathy. Based on the European Society of Cardiology (ESC), Dilated Cardiomyopathy is defined as the presence of left ventricular dilatation and global or regional systolic dysfunction that cannot be explained by abnormal cardiac loading conditions (eg hypertension, valve disease and CHD (coronary heart disease). In these patients we did not find any specific risk factors and conditions of abnormal heart loading. This cardiomyopathy can be caused by genetic/familial or non-genetic (eg inflammatory, toxic and multisystem diseases. Approaches to diagnosis with various phenotypes in clinical presentation are more priority than determining etiology.

Conclusion:
While an uncommon diagnosis, young adults can present heart failure with different clinical characteristics including a variety of etiologies. Severe left ventricular dysfunction may be the initial clinical manifestation that physicians encounter. For this reason, a high suspicion for heart failure in young adults who present with respiratory symptoms is important. Young adults with newly diagnosed heart failure should be referred to a center capable of providing full diagnosed capabilities and advanced therapy.

Keywords: severe left ventricular dysfunction, Young Male, Dilated Cardiomyopathy

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DIFFERENT PRESENTATION OF CARDIAC INVOLVEMENT IN PATIENT WITH SYSTEMIC LUPUS ERYTHEMATOSUS: TWO CASES FROM SMALL DISTRICT

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Background:
Cardiac involvement in SLE (Systemic Lupus Erythematosus) are diverse and influence the patient's prognosis. Asymptomatic clinical presentations to fulminant heart failure leading to death can occur. We presented two cases (cardiomyopathy and pericarditis) to describe several cardiac involvement in patients with SLE.

Case illustration:
First case is a 17-years-old female presented with progresively worsening shortness of breath since 2 days before admission. Her symptoms developed 6 months prior to presentation. She was diagnosed with SLE since 2 years ago but never had any cardiac evaluation before. The patient was dyspneic and tachycardic at rest with stable blood pressure. On physical examination revealed marked jugular venous distention, basal bilateral pulmonary fine rales and a systolic murmur at apex. Electrocardiogram showed a sinus tachycardia with sign of left ventricle hypertrophy. Echocardiogram revealed dilatation of all heart chambers with decreased left ventricle and right ventricle function (EF of 14 %, TAPSE 15), mild-moderate pericardial effusion around the heart. We treated the patient as acute heart failure due to dilated cardiomyopathy with diuretic, sacubitril/valsartan, digoxin, mineralocorticoid receptor antagonist beside her SLE treatment.Second case is a 21-years-old female presented with shortness of breath since 1 week ago. She was diagnosed with SLE about 2 months before. Her physical examination showed normal vital sign with slightly tachypnea, normal breath sounds with muffled heart sounds. Electrocardiogram showed sinus tachycardia with low voltage and QRS alternance. Echocardiogram revealed massive pericardial effusion around the heart chamber without sign of tamponade with normal ventricular function. Laboratory findings showed increased CRP and ESR. We diagnosed the patient with acute pericarditis and treated her with colchicine as additional for steroid that already given for her SLE treatment.

Conclusion:
Cardiac involvement in SLE is not a rare finding and is usually detected in more severe clinical manifestations, especially in rural areas where diagnostic tools are limited. Both of our patient have different presentation. Routine screening to determine cardiac involvement in SLE patients is needed so that we can provide early treatment of heart disease to improve patient outcomes.

Keywords: Pericarditis, Cardiomyopathy, Systemic Lupus Erythematosus

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A RARE CASE OF ATYPICAL TYPE B AORTIC INTRAMURAL HEMATOMA : CT BEYOND LUMINOSITY

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Background:
Aortic intramural hematoma (AIH) is one of the acute aortic syndromes along with acute aortic dissection and penetrating aortic ulcer. It is a life-threatening emergency that involves aortic wall integrity. AIH is described by hemorrhage within the medial layer of the aortic wall without evidence of flow or clear luminal communication. It is proposed that AIH is caused by spontaneous rupture of vasa-vasorum in the medial layer due to the combination of wall stress and fragile vessels. Hypertension is one of main factor contributing to wall stress and fragile vessels. The incidence of AIH ranges from 5% to 25% of acute aortic syndrome and mostly in elderly patients. Most AIH patients (50%-85%) is Stanford type B. Symptoms of AIH are often very similar with aortic dissection, including: severe chest pain, severe abdominal pain, nausea, severe back pain, and shortness of breath. With the current imaging techniques, it is possible to observe small intimal disruptions in AIH patients.

Case illustration:
An 81-year-old male, was referred to our clinic with one-week atypical abdominal discomfort. Patient has comorbidity of uncontrolled hypertension and bilateral kidney cysts. Plain abdominal CT Scan was done to evaluate the kidney cysts and accidentally revealed suspicion of aortic aneurysm. Previous medication was nifedipine GITS 30mg once daily, ramipril 5mg once daily and bisoprolol 5mg once daily. Physical examinations were unremarkable. The ECG was sinus rhythm, right axis deviation and complete RBBB. Echocardiogram was not remarkable. CT Aortogram revealed type B aortic intramural hematoma on the infrarenal abdominal aorta with size of 65,4mm x 62mm with hematoma thickness of 40mm. This is a characteristic of high-risk type B intramural hematoma. Oral medication was started to control the hypertension and heart rate. Transcutaneous endovascular aortic repair (TEVAR) was planned

Conclusion:
AIH is a rare acute aortic syndrome with no intimal flap commonly presenting with severe pain. This is a case of AIH with atypical presentation. It is important to strictly monitor the blood pressure. TEVAR could be the choice for high risk AIH

Keywords: Aortic Intramural Hematoma, Aortic Intramural Hematoma, Acute Aortic Syndrome

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ACUTE ISCHEMIC STROKE UNDER 24 HOURS AFTER PRIMARY PERCUTANEOUS CORONARY INTERVENTION IN PATIENT WITH ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION: A CASE REPORT

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Background:
Ischemic stroke after percutaneous cardiac intervention (PCI) is a rare but catastrophic complication. Previous studies have investigated predictors of stroke after PCI. Most strokes are reported to occur within 48 hours after PCI.

Case illustration:
We present a 57-year-old diabetic male who suffered from sudden neurologic deficits under 24 hours after PCI in our catheterization laboratory. Ischemic stroke was proven by brain computed tomography (CT) scan and how to manage this serious complication in clinical practice. The initial electrocardiogram showed acute inferior myocardial infarction. The troponin-I was elevated to 7.89 ng/mL. Primary PCI was performed for the occluded right coronary artery to achieve recanalization of the right coronary artery. The patient then admitted to the intensive care unit for further observation, we noticed that the patient presented with a lethargic appearance with sudden right limb weakness within 20 hours after PCI. Brain CT was performed and showed ischemia at the left upper temporal cortex and posterior frontal lobe, the diagnosis of acute ischemic stroke was confirmed. Despite intensive medical care with inotropic agents, the patient died after gradual blood pressure drop and progression of multi-organ failure. Although rare, stroke is a serious complication of PCI. Studies have shown an association between postprocedural stroke and high morbidity and mortality rates, with in-hospital mortality ranging from 22%-37%. In most studies, the majority of postprocedural strokes were ischemic. Many of the aforementioned studies identified diabetes mellitus, older age, hypertension, and history of cardiovascular and cerebrovascular disease as clinical predictors of stroke after PCI. Dislodgement of atherosclerotic debris or other material from the aorta, the aortic valve, or the left ventricle during the procedure may lead to an embolization, which in turn can precipitate an ischemic stroke.

Conclusion:
Acute ischemic stroke is one of the most catastrophic complications of PCI. Careful consideration of the precipitating factors for postprocedural stroke after PCI is essential. Coronary interventional procedures must be performed with meticulous attention to technical detail. Immediate brain CT is critical to assess any neurological deficits of patients during or after procedures.

Keywords: Myocardial infarction, Percutaneous coronary intervention, Ischemic stroke

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SEVERE ASYMPTOMATIC HYPERTENSION INDUCED BY LONG-TERM USE OF DEPO MEDROXYPROGESTERONE ACETATE (DMPA)

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Background:
Depo Medroxyprogesterone Acetate (DMPA) is progesteron-only contraceptive injection which widely used and generally safe for women worldwide. DMPA is known for its minimal side effects, high effectiveness, and cost-efficiency. Although it has been reported that it may slightly increase blood pressure in patients with a history of hypertension and cardiovascular disease, but there was limited information suggesting that DMPA injections can lead to severe hypertension. The following case describes severe asymptomatic hypertension induced by DMPA.

Case illustration:
A 49-years-old woman with one-year history of hypertension admitted to the clinic. The patient reported no symptoms. Her medical records showed that the blood pressure was consistently high on every visit. She stated that she was regularly taking anti-hypertensive medications (25 mg captopril and 10 mg amlodipine). There was no known family history of hypertension or cardiovascular disease. The patient had been using DMPA injections for the past 12 years.On physical examination, she appeared in good general condition, conciousness state was alert and oriented, blood pressure was 224/126 mmHg, heart rate was 94 beat per minutes with regular rhythm, temperature was 36.8°C, respiratory rate was 19 breaths per minute, and oxygen saturation was 97%. No murmurs were detected on cardiac auscultation. The patient's nutritional status was classified as obese. Laboratory tests showed elevated total cholesterol levels. She was diagnosed with Severe Asymptomatic Hypertension and Dyslipidemia. Two antihypertensive medications and a statin were prescribed. Monitoring in the emergency department was conducted for one hour, after that the patient was discharged. She was provided with education regarding therapy, monitoring, diet, exercise, and weight loss.

Conclusion:
It is crucial to recognize that long-term use of DMPA potentially cause hypertension. Regular blood pressure monitoring and screening for other abnormalities are essential for patients who have been long-term users of DMPA. Alternative contraceptive methods should be considered for high risk or poorly controlled hypertension patients.

Keywords: Depo Medroxyprogesterone Acetate (DMPA), Severe Asymptomatic Hypertension

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UNDIAGNOSED SJÖGREN SYNDROME PRESENTING AS ACUTE CORONARY SYNDROME IN YOUNG FEMALE: A CASE REPORT

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Background:
Patients with autoimmune diseases have a high cardiovascular risk. Sjögren syndrome (SS) is an autoimmune disorder primarily affecting exocrine glands, leading to keratoconjunctivitis and xerostomia. It is associated with various extra-glandular manifestations, including cardiovascular complications. Chronic inflammation in SS can disrupt endothelial function, increase reactive oxygen species (ROS) production, and predispose individuals to cardiovascular diseases such as coronary artery disease.

Case illustration:
:A 30-year-old female presented to the ER with a history of syncope 7 hours earlier, accompanied by chest pain radiating to her back, diaphoresis, and nausea. She had no history of hypertension, diabetes, dyslipidemia, or smoking. Initial examinations revealed an ECG showing sinus rhythm with inverted T waves in lead III and aVF, normal LVEF, and elevated high sensitivity troponin T (495 ng/L). She was diagnosed with NSTEMI and underwent early PCI.During the procedure, the left circumflex (LCx) artery and left anterior descending (LAD) artery spasmed, causing blood pressure to drop and rapidly changed to pulseless ventricular tachycardia (VT), necessitating defibrillation and intubation. ROSC was achieved during procedure, without any significant stenosis left in coronary arteries.Post-cardiac arrest, her ECG rhythm changed to total AV block. Further evaluation revealed the diagnosis of Sjögren syndrome (SS), supported by mild dry eye symptoms (Schirmer test: 5 mm in the left eye, sialometry: 0.08 ml/minute) and strongly positive serological markers (anti-SS-A, anti-Ro52, anti-SS-B, ANA 1:1000). Besides standard ACS therapy, she was treated with high dose IV methylprednisolone 500 mg tapered off into 125 mg. Her condition gradually improved and successfully discharged without any further angina.

Conclusion:
Managing SS-related ACS involves addressing the acute cardiac event with standard treatments like PCI and medications, alongside immunomodulatory therapies to control the underlying autoimmune activity. This dual approach is essential to stabilize cardiac function and mitigate the systemic effects of SS, improving patient outcomes.

Keywords: Endothelial dysfunction, Chronic Systemic inflammation, autoimmune diseases, Sjögren Syndrome, Acute Coronary Syndrome

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MULTIDISCIPLINARY MANAGEMENT OF RECURRENT ACUTE RHEUMATIC FEVER IN A YOUNG ADULT WITH RHEUMATIC HEART DISEASE: A CASE STUDY

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Background:
Acute rheumatic fever (ARF) is an inflammatory disease that can develop after a Group A Streptococcus (GAS) infection, such as streptococcal pharyngitis. It primarily affects children and young adults and can lead to rheumatic heart disease (RHD), a chronic condition resulting from damage to the heart valves. The management of ARF and RHD requires a multidisciplinary approach, including eradication of the causative agent, symptomatic treatment, and prevention of relapse. This case study aims to highlight the diagnosis and management of recurrent ARF in a young adult.

Case illustration:
A 19-year-old male with a known history of rheumatic heart disease presented to the hospital with symptoms of shortness of breath, fever, sore throat, and joint pain. Clinical examination and diagnostic criteria, including the Jones criteria, confirmed a diagnosis of recurrent acute rheumatic fever. The patient exhibited carditis, erythema marginatum, and other minor criteria. He was also diagnosed with suspected mitral and aortic regurgitation and a 1st-degree AV block. The treatment regimen included antibiotics to eradicate the streptococcal infection, prednisone for anti-inflammatory effects, and other symptomatic medications. Despite recommendations for surgical intervention, such as valve replacement, the patient and his family declined the procedure. Over an eight-day period, the patient showed steady improvement with significant resolution of symptoms. He was educated on the importance of regular follow-up and strict adherence to medication to prevent recurrence of rheumatic fever.

Conclusion:
This case underscores the critical importance of a comprehensive and multidisciplinary approach in managing recurrent acute rheumatic fever and its complications. Effective treatment involves not only addressing the acute symptoms with antibiotics and anti-inflammatory medications but also educating the patient on the necessity of long-term follow-up and adherence to prophylactic measures. While surgical interventions may be required in severe cases of rheumatic heart disease, patient and family preferences must be considered. Regular monitoring and timely intervention are essential to prevent the progression of rheumatic heart disease and improve patient outcomes.

Keywords: recurrent rheumatic fever, young adult, rheumatic fever, rheumatic heart disease

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DEGENERATIVE LA MYXOMA: THE HIDDEN DANGER BEHIND STROKES IN YOUNG-AGE PATIENT

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Background:
Ischemic stroke is uncommon in the young-age population and presents unique diagnostic challenges compared to older patients. Myxoma a primary cardiac tumor most commonly found in adults and rarely in young-age. Cardiac myxoma are noncancerous tumors but can lead to serious morbidity and mortality when left untreated. Complications of cardiac myxoma include mitral valve obstruction, which may result in pulmonary artery hypertension and right ventricular dysfunction, and embolization that cause ischemic stroke.

Case illustration:
We present a case of a 17-year-old female with no risk factors for atherosclerosis, who was admitted to the emergency department of the H. Adam Malik Hospital due to decreased consciousness after slipping in the bathroom. Initial vital signs revealed somnolence (E3M5V2), blood pressure readings of 102/75 mmHg, heart rate of 110 beats per minute, and a respiratory rate of 20 breaths per minute. Physical examination showed MDM murmur grade 2/4 at apex, Laboratory results revealed increased of D-dimer (2.04) and other laboratory results were within normal limits. Electrocardiogram showed sinus tachycardia and a rightward axis. Chest X-ray showed normal CTR, Brain CT-scan showed a massive hypodense lesion with indistinct borders in the left occipital lobe. Due to the deteriorating condition of the patient, the patient was admitted to the ICU. Echocardiography was done and revealed a degenerated giant mass in the left atrium with decreased LV function (EF 30%), akinetic throughout mid-apical segment and apical LV ballooning. ECG changes on the fourth day of treatment showed anterolateral ischemic features without troponin elevation. Unfortunately, the patient died before getting the myxoma resected.

Conclusion:
Cardiac myxomas are rare in young patients but may present with neurological symptoms. Routine echocardiography is helpful in the diagnosis of asymptomatic myxoma and recommended when a young patient presents with unusual neurological deficits

Keywords: Echocardiography, LA myxoma, Young-age stroke

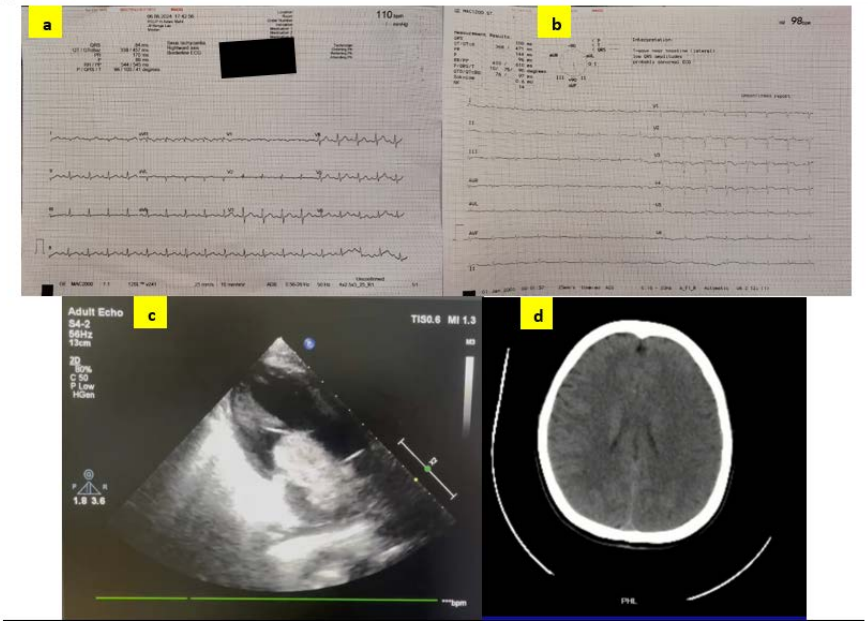

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Figure 1. (a) Initial ECG with Sinus Tachycardia and Right Axis Deviation, (b) ECG on day four with anterolateral ischemic impression, (c) Echocardiography showing LA myxoma, (d) CT scan of the head showing extensive hypodence lesion.

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SUCCESSFUL PHARMACOINVASIVE STRATEGY IN INFERIOR ST-ELEVATION MYOCARDIAL INFARCTION WITH DELAYED ATRIOVENTRICULAR BLOCK WITH RECURRENT SYNCOPE MANIFESTATION IN ELDERLY : A CASE REPORT

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Background:
Transient atrioventricular (AV block) is relatively common complication of inferior ST-segment elevation myocardial infarction (STEMI). AV conduction disturbances due to interruption of blood flow through the AV node branch of right coronary artery. This case to review rapid diagnosis and other practical treatment consideration of AV block in inferior myocard, especially in area with remote PCI centre

Case illustration:
A 72 years old gentleman with no documented cardiovascular history, poorly controlled type-2 diabetes mellitus, presented to non-PCI centre with recurrent syncope. Patient reported multiple short-duration of syncope (<10 minute) three hours prior, followed by typical chest pain. Initial electrocardiogram (ECG) was done, showed inferior STEMI with right ventricular (RV) involvement and first degree AV block. On presentation, vital signs were stable; blood pressure 121/79 mmHg, pulse 69 beats per minut with SpO2 98% in ambience air. Cardiac biomarker was normal. Referral for Primary PCI was suggested but the patient refused. Patient was given thrombolytic therapy as pharmacoinvasive strategy of reperfusion. Patient had successful fibrinolytic therapy with resolution of STEMI in ECG. Subsequent to reperfusion, patient developed total AV block with period of unstable haemodynamic. Patient was managed conservatively with fluid challenge also given atropine and dopamine. After 24 hours close monitoring in intensive unit, rhythm resolved with spontaneous recovery. Until discharge, patient was hemodynamically stable and had no further chest pain. AV block associated with inferior MI was a supra-Hisian in origin. Treatment of inferior STEMI and high degree AV block may involve mechanical or medical reperfusion to optimize blood flow and cardiac function.

Conclusion:
Earlier and prompt reperfusion may associated with earlier recovery of AV block and better morbidity and mortality. Development of total AV block in several cases still not common, but this case highlighted a response to reperfusion and conservative treatment.

Keywords: Reperfusion Strategy, cardiac syncope, inferior STEMI, atrioventricular block

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UNDERSTANDING THE FUTURE ANTIARRHYTHMIC POTENTIAL OF SGLT-2 INHIBITORS IN HIGH-DEGREE AV BLOCK CAUSED BY HEART FAILURE: A CASE REPORT IN A NON-ELECTROPHYSIOLOGY CENTER

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Background:
According to WHO's Top 10 Global Health Threats, non-communicable diseases such as heart failure contribute to more than 70% of all deaths worldwide. Several studies have shown that SGLT-2 inhibitors can considerably reduce heart failure. As research progresses, SGLT-2 inhibitors may lessen the risk of arrhythmias in heart failure patients.

Case illustration:
A 57-year-old female with uncontrolled hypertension came to the ED after collapsing at home. The vital signs and physical examination are normal. The preliminary ECG showed a high degree AV Block with multiple multifocal PVC. Early laboratory revealed mild hypokalemia. The patient experienced a seizure the next day, and an ECG showed total AV block with a non-sustained VT episode with PVC R on T. Following critical cardiac care and stabilization, the patient underwent echocardiography, which showed mild MR and AR with a reduced ejection fraction (47.7%). Once the patient's condition has stabilized, the patient is given 1x10mg Empagliflozin PO for 4 days in the ward. Following the treatment, the patient no longer complained of breathlessness, no seizures, and felt better. The latest ECG results revealed sinus rhythm with 50 BPM. The patient's condition improved, and she was discharged.

Conclusion:
As research progresses, it has been discovered that SGLT-2 inhibitors may significantly reduce the risk of arrhythmia and heart failure by inhibiting sodium-hydrogen exchange in myocardial cells and the sympathetic nerve system.

Keywords: high-degree AV block, sodium-glucose co-transporter-2 inhibitors, syncope, heart failure

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ACUTE CORONARY SYNDROME MIMICKING CASE : A RARE CASE OF TYPHOID PERICARDITIS

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Background:
Pericarditis is an inflammation disease involving the pericardium that is caused by both infections and non-infectious causes. In some cases, clinical presentations are numerous and distinction may be difficult since it may manifest as other clinical entities. We report a rare case of typhoid pericarditis that mimics acute coronary syndrome case.

Case illustration:
A 62 year old male with underlying diabetes mellitus and cardiovascular disease referred by local hospital to our center with inferior STEMI in order to receive percutaneous coronary intervention (PCI). Patient complained of chest pain like burning sensation and epigastric pain for 4 day before admission, accompanied by fever, cough and shortness of breath. Physical examination was unremarkable. ECG revealed a widespread ST elevations in limb and precordial leads with reciprocal ST depression and PR elevation in lead aVR and V1, suggesting a pericarditis type ST elevations. PCI was then deferred and a thorough assessment was then performed. Transthoracic echocardiography showed a mild pericardial effusion with thickened pericardium. Chest X-Ray showed a left paracardial infiltrates suggesting a pneumonia. Laboratory revealed mild anemia (Hb 9,2 g/dL) with leukositosis (17.420/uL), normal troponin and elevated IgM salmonella (+6). Diagnosis of typhoid pericarditis was then established. Intravenous antibiotics and colchicine were then initiated. On the 3rd day of treatment, the ECG returned to normal. The patient was then discharged with no recurrent symptoms.

Conclusion:
Pericarditis may mimic ACS cases, awareness and a thorough investigation should be perform to confirm the diagnosis and prevent unnecessary medication or intervention.

Keywords: pericarditis, thypoid, myocardial infarction, chest pain, salmonella

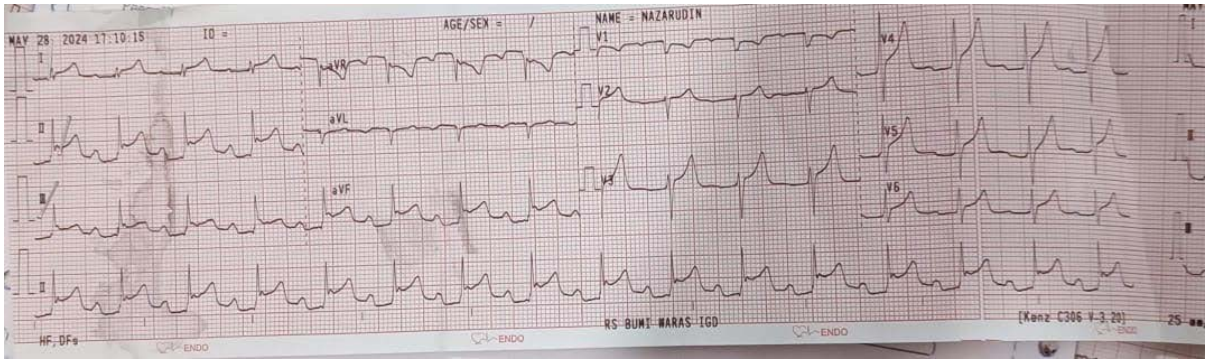

Figure 1. ECG of the patient shows an inferior STEMI

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COVID-19 VACCINE-INDUCED MYOCARDITIS: HOW ARE PATIENTS FARING AFTER 2 YEARS?

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Background:
COVID-19 vaccine-induced myocarditis is a mild disease with fast clinical recovery, characterized by uncontrolled cytokine-mediated inflammation, possibly involving the interleukin-6 signaling pathway. Most cases with a favorable prognosis and quick clinical recovery, but our case didn't resolve within a year and may have persistent myocardium injury.

Case illustration:
A 26-year-old woman reported severe fatigue, chest pain, cold sweat, and palpitations after receiving the second COVID-19 vaccine three weeks prior. There is no family history with the same symptoms. Physical examination revealed blood pressure 90/60 mmHg, pulse 112 bpm, temperature 36.6oC, breathing 28 bpm, and bilateral pitting edema in both lower extremities. The ECG showed tachycardia sinuses, heart rate 115 bpm, left axis deviation, T inverted I, aVL, and V6. Echocardiography showed left ventricle enlargement, ejection fraction 30%, global hypokinesia, and grade III diastolic dysfunction. During the 24-hour Holter ECG examination, sinus rhythm was obtained, and tachycardia was predisposed with a HR 74 – 123 bpm, maximal interval RR 1280 ms, 5 isolated VES, and 1565 isolated SVES. Cardiovascular MRI revealed hiposignal T1-mapping, global diffuse fibrosis, with adenosine no perfusion interrupted, and late galodinium enhancement (LGE) achieved intramural lesions in the posteromedial and on the RV inferoseptal inserts. Laboratory tests showed increased levels of hsTroponin T (300 ng/L), proBNP (11436 ng/L), and CRP (0.14 mg/dl) without increased leukocytes. The patient was diagnosed with acute myocarditis due to the COVID-19 vaccine and was treated with spironolactone 1 x 50 mg, metoprolol succinate 1 x 47.5 mg, sacubitril/valsartan 2 x 49 mg/51 mg, and dapaglifosine 1 x 10 mg. Patients are routinely checked every two to six months for significant changes in hemodynamic parameters. The patient experienced dyspnea (NYHA III) after discontinuing ARNI and SGLT2i in the 20th month of treatment, but regained asymptomatic state after resuming treatment with SGLTi and ARNI.

Conclusion:
These findings indicate that myocarditis can occur as a result of COVID-19 vaccination. However, the long-term outcomes for patients are not consistently positive. The maximum of goal-directed medical therapy is needed to avoid the progression of heart failure.

Keywords: Myocarditis, Heart Failure, Covid-19 Vaccine

Table 1. Patient 2 years evaluation

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Parameter	Evaluation	Duration of Post Therapy					
		2 months	4 months	6 months	14 months	20 months	26 months
Symptoms	Palpitation	Ocasionally	Ocasionally	Never	Never	Never	Never
	Chest Pain	Often	Often	Often	Sometimes	Ocasionally	Rarely
	NYHA	IV	III	III	II	III	I
Drug Therapy	Beta Blockers	Metoprolol 1 x 47.5 mg			Bisoprolol 1 x 5 mg	Bisoprolol 1 x 2.5 mg	Bisoprolol 1 x 1.25 mg
	ARNI	Sacubitril/Valsartan 2 x 49/51 mg			Sacubitril/Valsartan 2 x 49/51 mg	-	Sacubitril/Valsartan 1 x 49/51 mg
	MRA	Spironolactone 1 x 50 mg			Spironolactone 1 x 50 mg	Spironolactone 1 x 25 mg	Spironolactone 1 x 25 mg
	SGLT2i	Dapaglifozin 1 x 10 mg			Dapaglifozin 1 x 10 mg	-	Dapaglifozin 1 x 10 mg
Echocardiography	Ejection Fraction	30%	35%	35%	54%	53%	56%
	GLS				-14%	-13.5%	-16.3%
	LVEDV	218 ml	186 ml	144 ml	83 ml	104 ml	86 ml
	LVESV	153 ml	120 ml	93 ml	38 ml	49 ml	37 ml
	E/A				0.82	1.09	0.68
	E/e'				6.69	5.8	5.2
	Wall Motion	Global hypokinesia			Inferoseptal basal, anteroseptal basal, inferior basal-mid hypokinesia		
	LVEDD	72mm	58mm	58mm	47mm	39mm	36mm
	TAPSE	15mm	16mm	16mm	18mm	22mm	20mm
	RV S'				10 cm/s	12 cm/s	10 cm/s
	LAVi	26.8 ml/m^2	25.15 ml/m^2	25.15 ml/m^2	13.84 ml/m^2	15.31 ml/m^2	17.7 ml/m^2

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CASE REPORT FROM A TYPE B HOSPITAL: THERAPY CHALLENGES IN A LATE-TERM PREGNANCY PATIENT WITH ASD SECUNDUM AND ADHF

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Background:
Late detection of congenital heart diseases like Atrial Septal Defect (ASD) in combination with other serious condition often results in high fatality rates. ASD compromises the heart's ability to pump sufficient oxygenated blood, especially in pregnant women leading to acute decompensated heart failure (ADHF). This report explores challenges and therapeutic strategies for late-term pregnant patient with ASD and ADHF.

Case illustration:
A 21-year-old pregnant woman at 35/36 weeks gestation (G1P000) presented to ER with respiratory distress and vulval oedema over three days. She denied any history of heart conditions and previously passed physical endurance tests without dyspnea. Examination revealed blood pressure of 151/101 mmHg, heart rate of 133/min, respiratory rate of 29/min, and oxygen saturation of 60% on 6lpm simple mask. Auscultation exhibited rales in the lung bases, wide fixed splitting, and murmur in S2. Cyanotic extremities and clubbing fingers were observed.Oral medication given was 25mg erythromycin four times daily; 20mg nifedipine thrice daily. After being moved to ICU, her oxygen saturation improved to 74% on 12lpm non-rebreathing mask. ECG exhibited right ventricular hypertrophy (RVH). Transthoracic echocardiography (Figure 1.) revealed ASD Secundum with 34mm diameter, severe tricuspid regurgitation, right atrium and right ventricle dilatation, pericardial effusion, and high probability pulmonary hypertension (PH).The patient was diagnosed with ASD Secundum and Eisenmenger Syndrome, severe tricuspid regurgitation, ADHF, moderate pericardial effusion, RVH, and late-term pregnancy. She was treated with iloprost nebulization, Revatio. Fluid balance monitoring resulted in -37cc/13hr, furosemide was changed on pump 5mg/hr. The patient later chose cesarean section (SC) for delivery after stabilizing but succumbed three days post-operation due to post-partum hemorrhage and cardiac insufficiency.

Conclusion:
This case highlights the challenges of mediating therapies on patient with newly detected ASD complicated with late-term pregnancy. Desaturation and PH crisis can be moderated with iloprost (ventavis) and adequate oxygenation while fluid balance monitoring is vital for ADHF. Delivery method viability relies on SC with inevitable probability of PH crisis and other complications post-op. Timely identification of ASD and pregnancy prophylactics is crucial for better patient outcomes.

Keywords: ADHF, Inpartu, Pregnancy, ASD

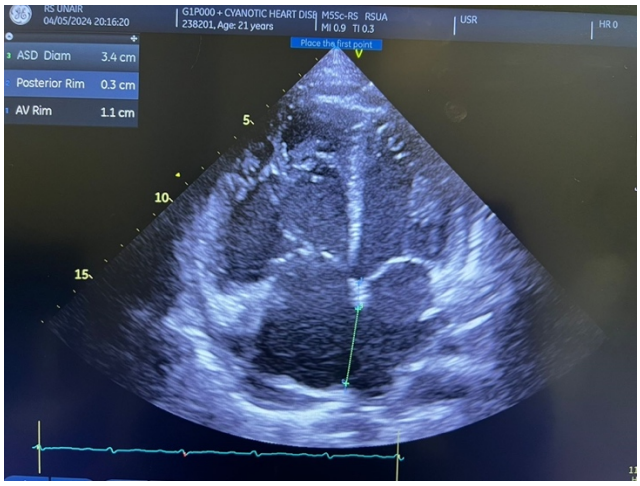

Figure 1. Echocardiograph

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VERY HIGH RISK NSTEMI WITH TOTAL OCCLUSION OF THE LEFT MAIN CORONARY ARTERY: REVISITING THE USE OF TEMPORARY MECHANICAL SUPPORT FOR HIGH-RISK PCI AND CABG

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[1]RSJPD Harapan Kita

Background:
Temporary mechanical circulatory support (MCS) currently being a debatable support treatment in intensive care especially in ACS setting. Current evidence showed no significant difference and benefit from MCS but still there are some pros and cons. This case report wants to bring a perspective and local evidence regarding MCS use in ACS patient.

Case illustration:
A 46-year-old male smoker admitted to emergency department (ED) with a chief complaint of retrosternal chest pain in the last 5 hours prior admission. Upon arrival, his vital sign was within normal limit. Rales are obtained in 1/3 of bilateral lung basal area. The electrocardiogram showed ST elevation in aVR lead and ST depression in V3-V6. Laboratory tests revealed significant increase of cardiac enzyme. Bedside echocardiography showed euvolemia status with reduced LV hemodynamic (VTI 13.5 cm, SV 34.3 mL, CO 3.3 L/min and SVR 2109 dyn.s/cm^{-5}), and RWMA in mid anterolateral and inferolateral segments. Lung ultrasound and chest radiography showed congestion without pleural effusion. Diagnosis of very high risk NSTEMI was established. Immediate invasive strategy was chosen with finding of total occlusion in the left main artery. During RCA canulation, the blood pressure was drop, the procedure decided to stop and the patient admitted to ICVCU with inotrope. After that, prophylactic temporary mechanical circulatory support was chosen (intra-aortic balloon pump) to support hemodynamic before high-risk PCI (POBA) procedure and bridging for surgical revascularization. Balloon angioplasty was done in LM-LAD and LM-LCx, with angiography finding showed critical LM stenosis and CAD3VD. Inotrope was stopped on the following day. Chest pain and dyspnea were relief. The vital sign was stable with IABP support. The bedside echo showed euvolemia status with improved left heart hemodynamic (LVOT VTI 20.2 cm SV 51 mL CO 3.8 L/min and SVR 1409 dyn.s/cm^{-5}). On the 9th day of admission, the patient underwent CABG and then the patient was discharged with good result.

Conclusion:
Although its benefit remains a topic of debate, this case provides local evidence that temporary mechanical support continues to enhance intensive care treatment in this patient setting, leading to optimal outcomes.

Keywords: IABP, MCS, left main artery total occlusion, Myocardial infarction

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VENTRICULAR ARRHYTHMIA IN AN ATHLETE'S HEART: A CASE REPORT OF AN 18-YEAR-OLD FEMALE

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[1]RS dr Kariadi Semarang

Background:
Athlete's heart, characterized by cardiac remodeling due to intense physical training, can mimic pathological conditions. We present a case of an 18-year-old female experiencing ventricular arrhythmia post-exercise.

Case illustration:
An 18-year-old female reported chest discomfort post-exercise, having adopted a strenuous regimen in preparation for her police academy entrance exam. Physical exams and diagnostics, including electrocardiography and echocardiography, revealed left ventricular hypertrophy and frequent premature ventricular contractions. An exercise stress test indicated arrhythmias during recovery, leading to further evaluation via 24-hour Holter monitoring. The case emphasizes careful differentiation between physiological athlete's heart and potential cardiomyopathies.

Conclusion:
The case underscores the importance of cautious interpretation of arrhythmic symptoms in athletes. While beta-blockers were prescribed and exercise modifications recommended, further research is warranted to understand the persistence of certain cardiac alterations.

Keywords: Athlete's Heart, Exercise Test, Ventricular Arrhythmia, Cardiac Remodeling, Exercise-Induced Cardiomyopathy

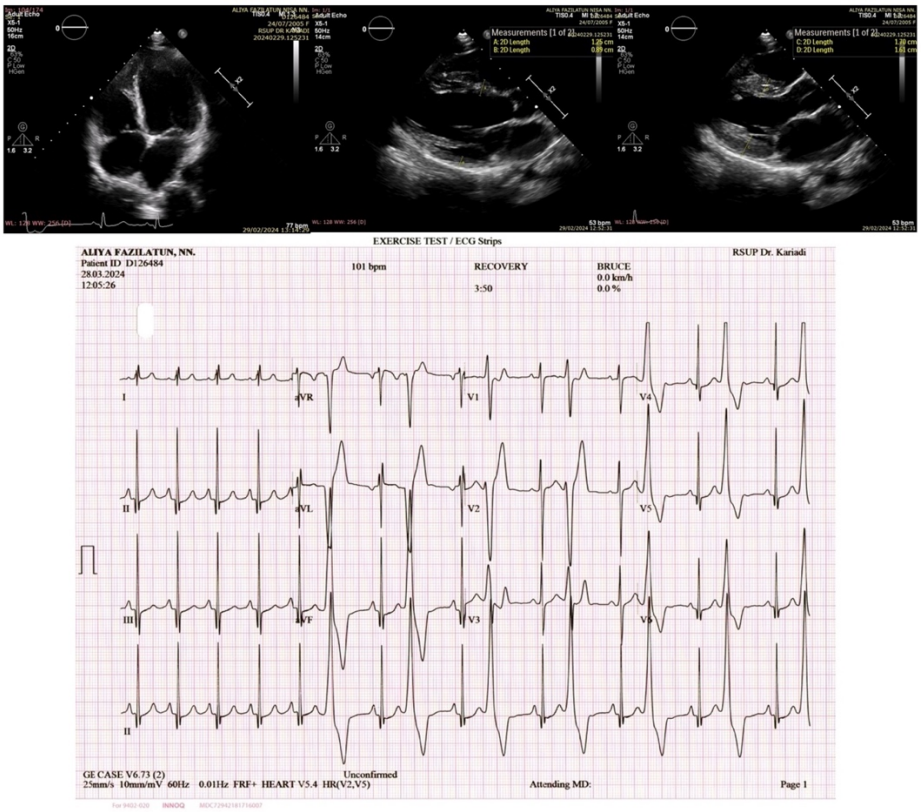

Figure 1. Echocardiogram and ECG

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TREATMENT DILEMMAS IN CARDIOGENIC SHOCK COMPLICATING STEMI WITH MULTIVESSEL DISEASE: A CLINICAL CASE REPORT

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Background:
Almost 75% of patients with cardiogenic shock secondary to AMI present with multivessel disease. Multivessel coronary artery disease in the setting of ST elevation myocardial infarction (STEMI) poses significant challenges in clinical management. Optimal revascularization strategy remains a subject of debate, with options including culprit-only percutaneous coronary intervention (CO-PCI), immediate multivessel PCI (MV-PCI), or staged MV-PCI.

Case illustration:
A 66-year-old male, with unknown medical history, presented with sudden onset chest pain, confusion, and shortness of breath lasting 3 hours. Upon arrival, he was hemodynamically unstable with elevated troponin I, blood glucose, liver enzymes, and creatinine levels. His electrocardiogram indicated inferior STEMI and total atrioventricular block (TAVB). Diagnosis included inferior ST elevation myocardial infarction, TAVB, cardiogenic shock, diabetes, and secondary hepatic and kidney injury. Coronary angiography revealed acute total occlusion in the proximal right coronary artery (RCA) and a significant stenosis in the left anterior descending artery (LAD). Primary percutaneous coronary intervention (PPCI) was done. Given the patient's condition and risk profile, CO-PCI was chosen as the initial strategy, focusing on the RCA as the culprit lesion, with planned staged PCI to the LAD. The choice of strategy depends on balancing the benefits of revascularization against potential procedural risks and long term impact, particularly in patients presenting with complications such as cardiogenic shock or multiple organ involvement. Post-procedural evaluation showed TIMI flow III. The patient received a temporary pacemaker for rhythm support and showed gradual improvement, with significant reduction in chest pain and removal of the temporary pacemaker on the 7th day of hospitalization. After 10 days, he was discharged home in stable condition, continuing with optimal medical therapy and scheduled for elective PCI evaluation.

Conclusion:
In this case, CO-PCI followed by staged evaluation and PCI for remaining ischemic areas remains preferable for STEMI patients with multivessel disease and complicated with cardiogenic shock. Literature suggests no significant benefit in STEMI with cardiogenic shock (STEMI-CS) for MV-PCI compared to CO-PCI, emphasizing careful consideration of procedural risks versus benefits in revascularization decisions.

Keywords: Complete revascularization, ST-Elevation Myocardial Infarction, Acute coronary Syndrome, Cardiogenic Shock, Culprit vessel-only revascularization

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THE DILEMMA OF SINGLE CORONARY OSTIUM IN ACUTE MYOCARDIAL INFARCTION: PCI OR CABG?

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Background:
Single ostium coronary artery (SCA) is a rare coronary artery anomaly concerning its origin and path, with an observed prevalence of just 0.024% to 0.066% in patients undergoing routine coronary angiography. Although it can manifest in various clinical scenarios, it typically remains asymptomatic in most individuals. However, individuals with SCA, particularly postpulmonary pattern, face marked increase risk of sudden cardiac death due to acute myocardial infarction. The use of coronary angiography and coronary computed tomography angiography is crucial for the classification of these coronary anomalies. Here, we present a rare case of Lipton type RIIP in which the patient underwent coronary artery bypass graft for severe triple vessel disease presenting as acute myocardial infarction.

Case illustration:
A 60-year-old man presented to emergency ward with Non ST-elevation Acute Coronary Syndrome. ECG revealed Wellen's Type A pattern in leads V1-V2. Troponin was elevated with 9803 pg/ml. Previous history of coronary computed tomography angiography (CCTA) identified a single coronary ostium with an anomalous origin of the left anterior descending artery (LAD) arising from the proximal right coronary artery (RCA), absence of the left circumflex (LCx) artery, and atresia of the left main coronary artery (LM). We classified this anomaly with Lipton-Yamanaka RIIP (LAD after arising from RCA courses posterior to the aorta). Diagnostic coronary angiography confirmed that the LM and LCx were not visualized and the LAD arise from the proximal RCA. Trial to penetrating distal LAD from proximal RCA was unsuccessful. Based on these findings, heart team discussion planned a surgical intervention. The patient underwent successful coronary artery bypass grafting and was discharged after uneventful recovery.

Conclusion:
Single coronary arteries are extremely rare but carry significant prognostic implications, increasing the risk of future myocardial ischemia, infarction, and sudden cardiac death. As the course of the coronary artery was between the aorta and pulmonary artery, surgery was recommended in this case.

Keywords: Single coronary artery, Percutaneous coronary intervention, Coronary artery bypass grafting, Acute myocardial infarction

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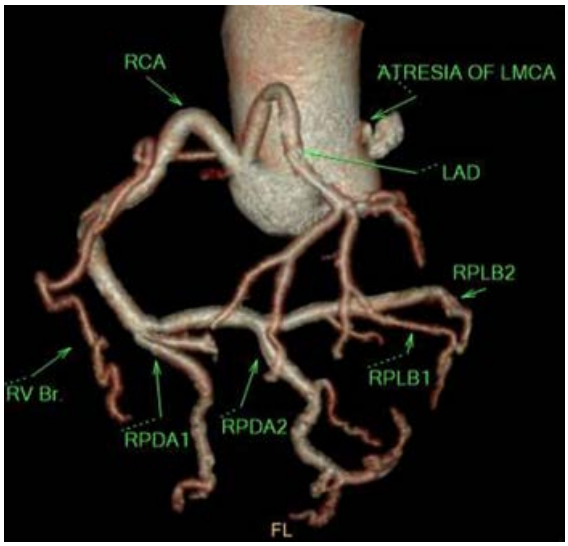

Figure 1. Coronary computed tomography angiography (CCTA) identified the anomaly single coronary ostium

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ROLE OF ECHOCARDIOGRAPHY IN DIAGNOSING HEPATOPULMONARY SYNDROME

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Background:
Hepatopulmonary syndrome (HPS) is defined as the presence of a defect in arterial oxygenation caused by *intrapulmonary vascular dilation* (IPVD) in *chronic liver disease* (CLD). This condition is characterized by the presence of CLD, gas exchange disorders that cause hypoxemia and the presence of IPVD. *Transthoracic Echocardiography* (TTE) has an important role as the gold standard for diagnosing the HPS. We are reporting cases of suspected HPS in patients with CLD, along with the role of TTE in the enforcement of diagnosis.

Case illustration:
A 48-year-old male consulted from internal medicine for evaluation of *dyspnea on effort* (DOE). This complaint has worsened in the last 1 month and has not improved with positional changes. Patient with past medical history hepatic cirrhosis and esophageal varicose veins (EVV). Vital signs showing decrease in blood oxygen saturation (88% on room air) and physical examination were unremarkable. ECG showing atrial fibrillation. Esophagogastroduodenoscopy (EGD) showing EVV and *fibroscan* showing Metavir F4, which confirm hepatic cirrhosis. Conventional TTE showing no *atrioventricular* (AV) shunt nor microbubbles. After agitated saline contrast injection, more than 20 microbubbles on left ventricle (LV) were detected after 5 beats. The presence of microbubbles after 5 beats indicates hepatopulmonary shunt detectable via TTE. This finding is important in differentiating it from an intracardiac shunt, which requires a shorter time for the bubbles to appear.

Conclusion:
HPS is a defect in arterial oxygenation caused by diffusion and perfusion mismatch in presence of IPVD in patients with CLD. This condition appears because of portal hypertension. Clinical signs leading to HPS accompanied by a history of CLD should be planned for further diagnostic examinations. TTE with contrast is supporting qualitative examination, which is non-invasive and sensitive, with important role in enforcing diagnosis of HPS

Keywords: Hepatopulmonary syndrome, Echocardiography Hepatopulmonar, Chronic liver disease

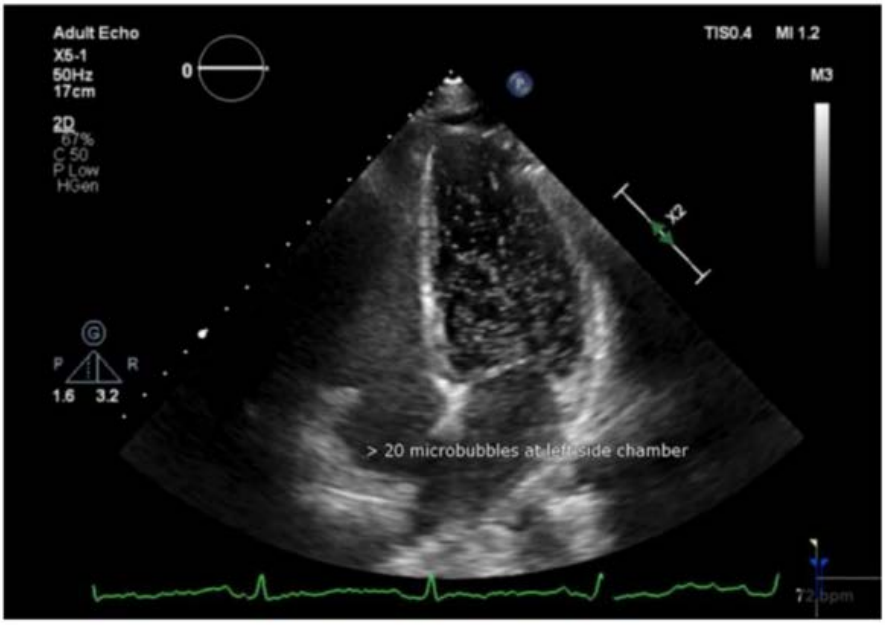

Figure 1. More than 20 microbubbles was seen at LV after saline contrast injection

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ABSENCE OF THE LEFT MAIN CORONARY ARTERY, RARE CONGENITAL CARDIAC MALFORMATION: A CASE REPORT

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Background:
Absence of the coronary arteries has a low incidence and prevalence in society, with a 0.3-5.6% prevalence. The incidence of absent left main coronary artery (LMCA) is between 0.02% to 0.07% in individuals undergoing coronary angiography.

Case illustration:
A 36-year-old man presented with chest pain since 1 day before admission. The electrocardiogram showed there is pathological Q wave at V1-V4. The first coronary angiography of left side showed normal vessel of left circumflex (LCx) but LMCA and left anterior descending (LAD) was not visualized. Angiography catheter was retracted to left sinus of Valsalva and then contrast was injected (non selective coronary angiography). The result was there is absence of LMCA and total occlusion a t mid LAD. LAD and LCX separated from the left sinus of Valsalva. The coronary angiography of right side showed critical stenosis at proximal right coronary artery (RCA). Percutaneous transluminal coronary angioplasty (PTCA) was performed using drug eluting stent (DES) 3.5 mm x 29 mm that placed at mid to distal LAD with good flow in the end of procedure.

Conclusion:
A 36-year-old man presented with chest pain since 1 day before admission. The electrocardiogram showed there is pathological Q wave at V1-V4. The first coronary angiography of left side showed normal vessel of left circumflex (LCx) but LMCA and left anterior descending (LAD) was not visualized. Angiography catheter was retracted to left sinus of Valsalva and then contrast was injected (non selective coronary angiography). The result was there is absence of LMCA and total occlusion a t mid LAD. LAD and LCX separated from the left sinus of Valsalva. The coronary angiography of right side showed critical stenosis at proximal right coronary artery (RCA). Percutaneous transluminal coronary angioplasty (PTCA) was performed using drug eluting stent (DES) 3.5 mm x 29 mm that placed at mid to distal LAD with good flow in the end of procedure.

Keywords: congenital cardiac malformation, PTCA, Absence of LMCA

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AVRT MIMICKING SINUS TACHYCARDIA IN WOLFF-PARKINSON-WHITE SYNDROME: A CASE REPORT

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Background:
Wolff-Parkinson-White (WPW) syndrome is a congenital pre-excitation syndrome that involves an accessory pathway (AP) allowing the bypass of electrical currents aside from the AV node. Most patients are asymptomatic and may remain undetected until it manifests as a paroxysmal supraventricular tachycardia (PSVT).

Case illustration:
A 30-year old woman presented to the ER complaining of palpitations and mild chest tightness persisting for two days. She has history of controlled asthma but denied previous episodes of palpitation. She denied smoking, alcohol and caffeine consumption, nor illicit drug use. She had no family history of sudden cardiovascular death.

Upon examination, her vital signs were stable, and physical examination revealed normal heart and lung sounds. Initial ECG showed regular narrow QRS-complex tachycardia of 137 bpm with upright P- wave in leads I and II, and negative P-wave in lead aVR, suggesting sinus tachycardia. Her labs showed mild hypokalaemia and normal thyroid function. The patient was given oral beta blocker, and the tachycardia episode resolved spontaneously. However, a follow-up ECG revealed change in morphology, now presenting classic signs of WPW syndrome, including a shortened PR-interval and distinct delta waves. St George's algorithm suggests a right anteroseptal accessory pathway. This case presents a patient with regular narrow complex tachycardia, which mimicked sinus tachycardia, but was actually a manifestation of AVRT. The episode of AVRT suggests an orthodromic circuit but curiously, did not show the telltale signs of a retrograde P-wave nor corresponding ST depressions. It was narrowly dismissed as benign sinus tachycardia, but conversion to sinus rhythm also showed different electrical axis. In hindsight, if the ESC diagnostic algorithm for narrow complex tachycardia had been applied, we would've recognised a short RP interval tachycardia with RP <90 ms, prompting consideration of AVRT, atypical AVNRT, or focal AT. Patients with AVRT can be managed similarly to other regular narrow complex SVTs. Vagal manoeuvres and adenosine not only treat but can also aid in clinical diagnosis, particularly when the initial ECG is unclear.

Conclusion:
This case highlights the use of diagnostic algorithm for the evaluation of narrow complex tachycardias, and the importance of post-arrhythmia treatment follow-up.

Keywords: Wolff-Parkinson-White, PSVT, Tachyarrhythmia, AVRT

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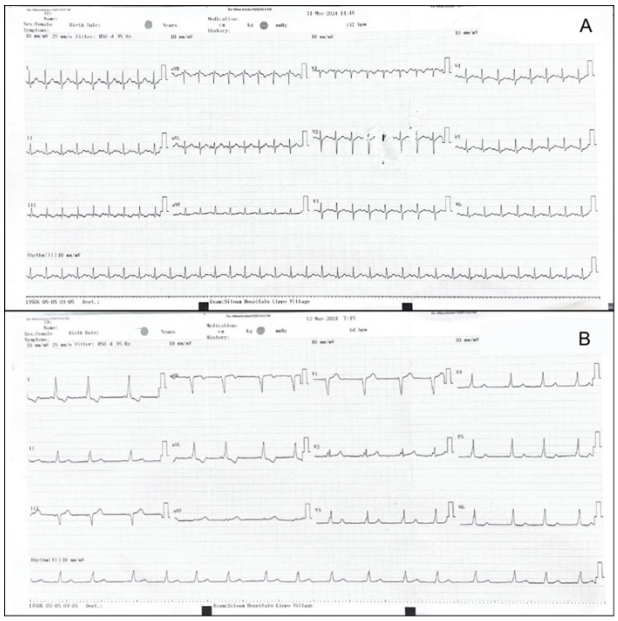

Figure 1. Initial and Follow-up ECG of WPW syndrome mimicking Sinus Tachycardia

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DEVELOPMENT OF STEMI POST CARDIAC ARREST WITH SHOCKABLE RHYTHM DUE TO ELECTRICAL INJURY: A RARE CASE REPORT

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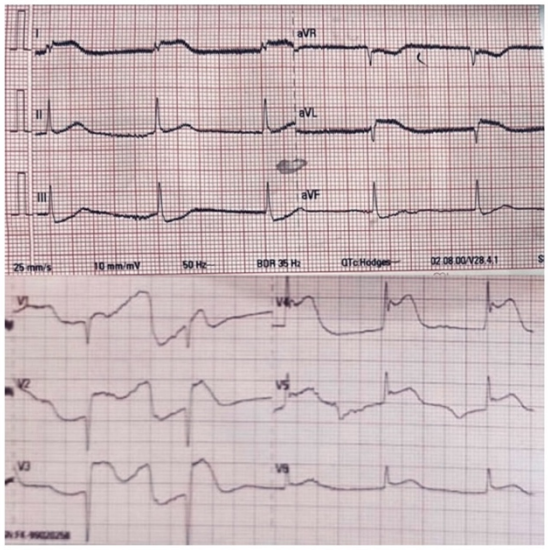
Background:
The most susceptible organ to electrical injuries is the heart. Arrhythmias, conduction disturbances, and myocardial infarction are among the complications. STEMI after electrical injury is a rare clinical condition. In this case, we present our experience treating STEMI Post Cardiac Arrest Due to Electrical Injury.

Case illustration:
A 39-year-old male was brought to the emergency room following electrical exposure. On admission, the patient was unconscious with an unpalpable pulse and absent respiration. No entrance or exit site wounds were identified. The initial rhythm was ventricular fibrillation. After three cycles of CPR, alternating with three DC Shocks 200 J, one intravenous dose of epinephrine (1 mg), one intravenous dose of amiodarone (300 mg), and intubation, return of spontaneous circulation (ROSC) was attained. The ECG showed ST-segment elevation in leads I, aVL, V1-V6,suggesting extensive anterior myocardial infarction. Dual antiplatelet therapy (DAPT) was administered. Troponin I was elevated up to 15 µg/L. The echocardiography showed an EF of 63%, TAPSE 2.0 cm, E/A ratio >1, and eRAP of 15 mmHg. A chest x-ray revealed pulmonary edema. Unfortunately, a coronary angiography could not be performed due to its unavailability in our region. The patient received subcutaneous administration of 0.6cc / 12 hours of enoxaparin, IV diuretics, as well as oral beta-blocker, ace-inhibitor, antiplatelets through the nasogastric tube. Four hours later ECG showed a significant reduction in ST Elevation. On the second day of ICU admission, the patient was fully conscious and extubated. The patient had no significant symptoms and eventually safely discharged after four days of hozpitalitation.

Conclusion:
The diagnosis and management of STEMI after electrical injury can be challenging. Troponin I, echocardiography and ECG findings should be the manjor considerations in detecting myocardial injury. A coronary angiography must thus be performed if there is any doubt as to the possibility of underlying coronary artery disease. Immediate resuscitation in cardiac arrest due to electrical injury can result in long-term survival and successful complete recovery.

Keywords: Electrical Injury, Myocardial Injury, STEMI

The initial ECG post ROSC



Four Hours Later ECG

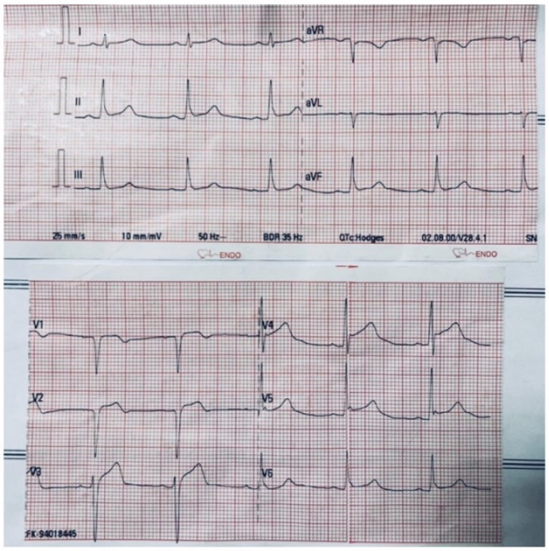


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Figure 1. Electrocardiography Findings

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HEPARIN AS AN ALTERNATIVE BRIDGING FOR NON-CARDIAC SURGERY IN HIGH-RISK STENT THROMBOSIS PATIENT: AN ISSUE IN THE ERA OF NATIONAL HEALTH INSURANCE

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Background:
Patients with high-risk stent thrombosis who are scheduled for non-cardiac surgery with high-risk bleeding, it is advised to stop the P2Y12 inhibitor and substitute to bridging medication with glycoprotein IIb/IIIa inhibitors (GPI) or cangrelor. Regrettably, Indonesia's national formulary for national health insurance does not include either GPI or cangrelor. In this article, we will present a case of heparin bridging in a patient with a high-risk stent thrombosis who will undergo non-cardiac surgery.

Case illustration:
A 58-year-old man with a history of coronary artery disease with triple vessel disease, post-PCI total of five stents, heart failure with mildly reduced ejection fraction, hypertensive heart disease, mild aortic regurgitation, first-degree AV block, diabetes mellitus type 2, and fourth-stage chronic kidney disease came to our hospital for umbilical hernia surgery. Low-risk bleeding operations, which include hernia repairs, typically recommend continuing dual antiplatelet therapy, but our surgeon disagreed. Another option is to stop taking P2Y12 inhibitors and use bridging therapy, such as GPI or Cangrelor. Unfortunately, National Health Insurance does not cover both therapies. Therefore, we decided to stop the patient's Clopidogrel consumption on the fifth day before surgery and we provided heparin bridging therapy as an alternative. We started with a bolus dose of 60 units/body weight (BW), continued with 12 units/BW/hour for 18 hours, and stopped 6 hours before surgery. The patient did not report any chest pain or tightness prior to the procedure. During the procedure, there were no reports of severe intraoperative bleeding. Similarly, no complaints of tightness, chest pain, or bleeding from the post-operative site were reported two days following surgery. The patient was checked into the cardiac polyclinic one month later. For thirty days following surgery, the patient did not report any chest pain or dyspnea.

Conclusion:
Heparin bridging, as an alternative to GPI or Cangrelor, may be considered for patients with a low risk of non-cardiac surgery bleeding and a high risk of stent thrombosis. We found no clinical incidence of thromboembolic events or stent thrombosis within 30 days after surgery.

Keywords: Heparin, Briding, Surgery, Stent, Thrombosis

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WHY PREVIOUS PRIMARY PCI MISSED THE TARGET ?

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Background:
It is important to understand the anatomic variants of coronary artery anatomy by performing coronary angiography from different views. Sometimes, side branch may mimic main branch in term of size and direction, but from specific angle it can be differentiated clearly.

Case illustration:
A 60 years old female with history of Anterior STEMI 3 weeks before and Primary PCI was done at different hospital. Her co-morbidities were hypertension, dyslipidemia and menopause. Recurrent chest pain remains occurred after discharge. She was referred to our hospital due to unstable angina pectoris. ECG showed anterior wall ischemia, negative troponin T. Echocardiogram showed LVEF 52% with hypokinetic at anterior wall.Left coronary angiography from LAO cranial view showed that the previous stent was actually implanted from the mid LAD to the diagonal branch. Tiny flow to the LAD confirmed that the previous PCI missed the target. A 6 Fr EBU Guide catheter was used to engage LM ostium. A 0.014" Fielder XT-R guidewire encounter difficulty in crossing LAD lesion despite changing tip of guide wire. Decision was made to perform open sesame technique, by inflating 2.5/10 mm NC balloon at 20 Atm proximal to stiff angle, to modify anatomical geography. The Fielder XT-R guide wire was finally able to cross the lesion. Enlarging stent strut was performed using 1.5/15mm compliance balloon, followed by a 2.5/10 mm NC balloon. a 2.5/38 mm DES was implanted, small overlapping with previous stent. The stent was deployed at 18 Atm at mid LAD.

Conclusion:
LAD may run a regular course in the anterior interventricular sulcus (AIVS) but gives off a large diagonal branch that runs parallel to the LAD which does not re-enter the distal AIVS. This form of anatomy is colloquially known as a parallel LAD where a large diagonal branch supplies septal arteries and if, however, it does not give septal branches, it should be labeled as parallel diagonal. An appropriate angle may help to realistically picture the target vessel with no, or very little bias caused by the image acquisition, otherwise we may wrongly treat the target lesion.

Keywords: PCI, Coronary Artery Variation, Angiographic Projections

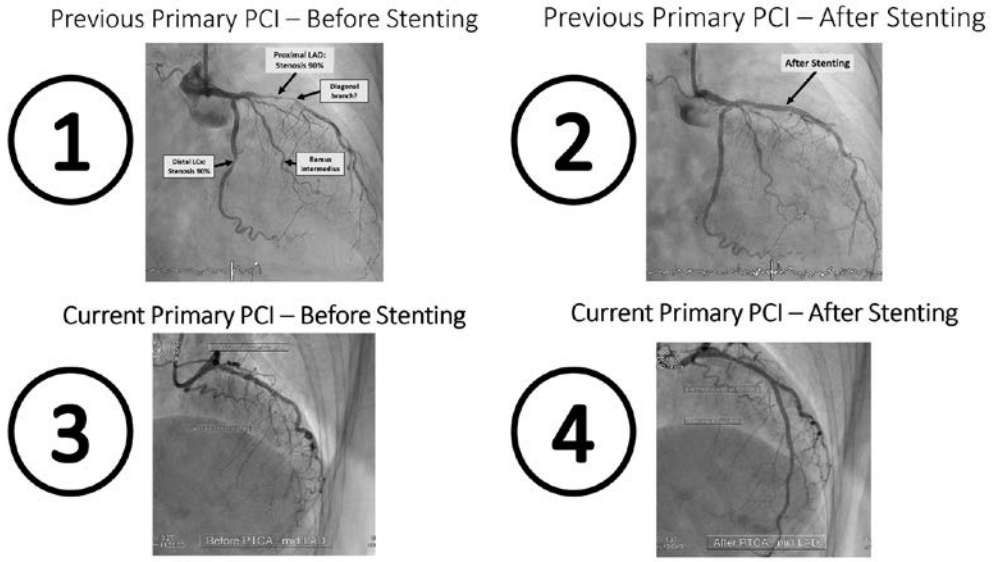

Figure 1. 1) Previous primary PCI showed 90% stenosis in distal LCX, 90% stenosis in proximal LAD. 2) Previous primary PCI showed DES from the mid LAD to the diagonal branch. 3) Current primary PCI showed

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99% stenosis in mid LAD with patent stented segment from mid LAD to the diagonal branch. 4) Current primary PCI after stenting showed improved in distal LAD circulation.

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ANOTHER PERSPECTIVE ON ATYPICAL CHEST PAIN: A COMPREHENSIVE CASE WITH MULTIPLE AORTIC ANEURYSMS

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Background:
Atypical chest pain is the most common symptom in adults, often indicating severe cardiovascular conditions. Aortic aneurysms can cause chest pain or tearing sensations. The incidence and mortality rates are increasing due to factors like smoking, hypertension, and connective tissue disorders. Ruptured aortic aneurysms may lead to death. Hence, it is imperative to conduct thorough examinations, accurate diagnoses, and effective treatment in order to prevent mortality.

Case illustration:
A 69-year-old male patient presented to the emergency room with right-sided chest pain. He defined the pain as sudden and stabbing. He stated the pain was 8/10 on NRS. He exhibited risk factors such as smoking, hypertension, recurrent stroke, obesity, and a prior diagnosis of prostate cancer. Upon admission, the patient had stable vital signs. His physical examination showed neither pulsatile nor bruit sounds in the abdominal region. The initial laboratory results were unremarkable; the troponin T level was normal (< 0.04 ng/mL). Chest radiology revealed cardiomegaly and elongation of the aorta. These findings led to the diagnosis of aortic dissection. The patient underwent thoraco-abdominal CT angiography, which confirmed multiple aneurysms in the aortic arch and abdominal aorta. The aortic arch showed dilatation at 6.4 cm in diameter with no signs of dissection. It also showed dilatation in the abdominal aorta with a diameter of 3.8 cm, extending from below the left renal artery to the right and left common iliac arteries. There was no evidence of blood-clot development or dissection. He was administered beta-blockers, statins and consulted with the vascular surgeon. He was released from the hospital with instructions to quit smoking and manage his blood pressure. The patient planned to be referred to a national center hospital for surgical consideration. In addition, he scheduled an imaging follow-up every 3 years for abdominal aortic aneurysm.

Conclusion:
In patients with atypical chest pain, clinicians should be able to refine their diagnosis because it usually implies a serious heart condition. The diagnosis and treatment of multiple aneurysms must entail a multi-disciplinary approach.

Keywords: Atypical Chest Pain, Aortic Arch Aneurysm, Abdominal Aortic Aneurysm, CT Angiography

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CERVICAL AND THORACIC HERPES ZOSTER INDUCED UNSTABLE BRADYCARDIA: AN UNUSUAL CARDIOVASCULAR MANIFESTATION OF A VIRAL INFECTION

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Background:
Herpes zoster is well-known for its painful neuro-dermatological manifestations, but its impact on the cardiovascular system is less understood. Recent studies suggest that varicella zoster virus (VZV) reactivation can lead to bradycardia through direct viral effects on cardiac innervation and inflammation. Herein we present a symptomatic bradycardia induced by VZV reactivation.

Case illustration:
A 53-year-old man presented to our emergency room with a stabbing headache, dizziness and burning pain along the distribution of rash on his posterior neck, back, and left anterior chest wall. Syncope, palpitations, shortness of breath and chest pain as well as family history of premature sudden cardiac death were denied. He mentioned having a childhood infection of chickenpox. Physical examination upon admission revealed blood pressure of 133/68 mmHg, heart rate 30 beats/minute, and non-confluent grouped vesicles on erythematous base along the distribution of left C5, C6, C7, and T1 dermatome, clinically diagnosed as herpes zoster reactivation. Initial electrocardiogram (ECG) showed sinus pause with atrial escape beat (fig 1). A 1 mg dose of sulfas atropine was administered, resulting in an increased pulse to 109 beats/minute. However, hours later, his heart rate and blood pressure dropped to 36 beats per minute and 80/40 mmHg, respectively, with no significant response after repeated doses of SA. Dopamine was then initiated at 5 mcg/kg/minute and titrated accordingly. All laboratory parameters and electrolytes were unremarkable. After a few days of observation, bradycardia persists although increased to 55 beats/minute (fig 2). Echocardiography showed normal cardiac structure and function. He was treated with acyclovir, gabapentin, mecobalamin, and cetirizine, then discharged 3 days later in stable condition with a sinus rhythm and improved heart rate of 61 bpm .Herpes zoster can cause bradycardia through mechanisms such as direct viral invasion of cardiac tissue, systemic inflammation, neural spread or direct VSV reactivation within cardiac sympathetic or parasympathetic ganglia.

Conclusion:
Recognizing potential uncommon viral infection manifestations, including autonomic nervous system and cardiovascular system involvement, is essential for diagnosing and managing bradycardia in the setting of recent VZV reactivation. This ensures effective treatment and prevents complications, leading to optimal patient outcomes.

Keywords: Bradycardia, Herpes Zoster

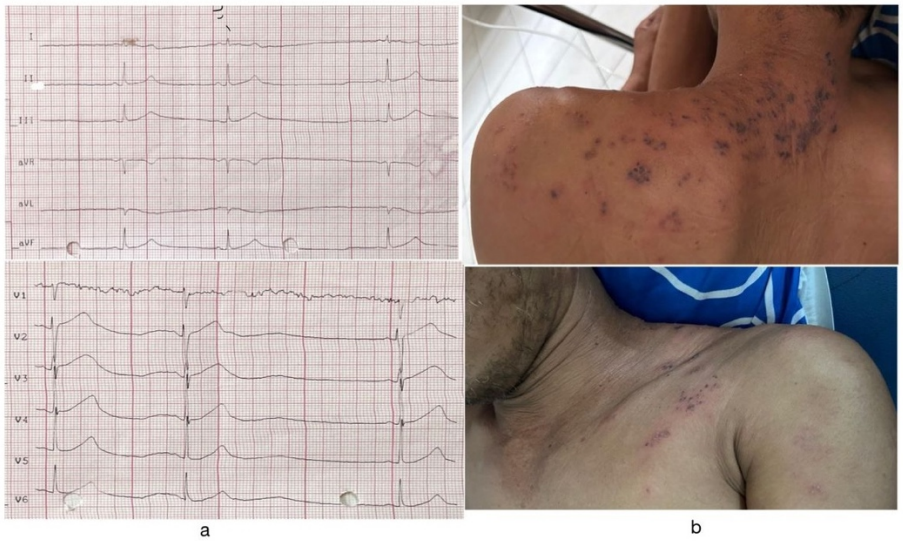

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Figure 1. Initial ECG at ED showed a sinus pause with atrial escape beat (a). Physical findings of patient's herpetic lesion (b)

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VALVE REPAIRED, HEART UNHEALED: THE COMPLEX BATTLE WITH INFECTIVE ENDOCARDITIS AND TUBERCULOSIS

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Background:
Right heart failure (RHF) is a multifaceted condition with various etiologies, often beyond cardiac causes. Non-cardiac factors, such as tuberculosis (TB), are frequently overlooked but significantly impact RHF. TB can lead to severe pulmonary complications, like bullous lung disease, which strains the right heart and can directly cause RHF.

Case illustration:
A 31-year-old woman presented to our ER with recurrent shortness of breath and abdominal bloating. The patient has a history of RHF with preserved ejection fraction (62.5%) presenting with ascites and splenomegaly, secondary to severe TR caused by infective endocarditis, characterized by large vegetations on the tricuspid valve and isolation of Staphylococcus aureus from cultures. In June 2022, she underwent excision of the vegetations and TVR with a St. Jude mechanical valve. Postoperatively, she was managed with warfarin, ivabradine, and bisoprolol. In April 2023, she was hospitalized for tuberculosis, having experienced dyspnea for two months with a 2 kg weight loss and 3 cm neck lymphadenopathy. Tuberculosis was confirmed through cultures, and she completed a six-month course of 2HRZE/4HR therapy, achieving negative acid-fast bacillus (AFB) results at the end of treatment. Her routine medications included warfarin, and spironolactone. Physical examination revealed increased jugular venous pressure and cardiac enlargement. A coronary CT scan showed normal coronary arteries, and a normally functioning mechanical tricuspid valve. However, multiple bullae in the right lung, measuring 7.22x3.49 cm in the superior lobe and 8.14x1.57 cm in the inferior lobe, contributed to her persistent RHF. Further evaluation via chest CT revealed cystic bronchiectasis and fibrosis in both lungs. The patient was diagnosed with RHF secondary to bullous lung disease and bronchiectasis. Her medications were adjusted to include methylprednisolone, aminophylline, and salbutamol. She was referred to a cardiothoracic surgeon for potential further intervention.

Conclusion:
Although TVR surgery has been done, an etiological search must continue using multimodal imaging to identify other potential causes of RHF beyond valve dysfunction. It highlights the importance of considering non-cardiac factors in the comprehensive management of RHF. Thorough diagnostic evaluation and interdisciplinary collaboration are essential for effectively addressing the multifactorial causes of heart failure.

Keywords: tuberculosis, infective endocarditis, right heart failure, valvular heart disease, bullous lung disease

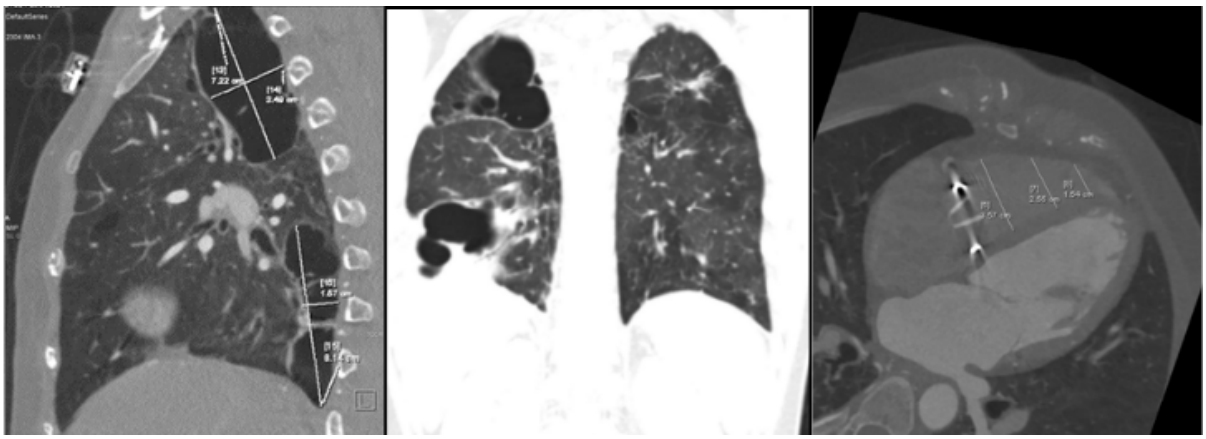

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Figure 1. CT Scan of the patient, showing multiple bullae and a mechanical tricuspid valve

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5 YEARS OUTCOMES OF HEART FAILURE COMPLICATING ACUTE CORONARY SYNDROME IN YOUNG SOLDIER UNDER 30S: A CASE REPORT

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[1]RSPAU dr S Hardjolukito

Background:
Acute coronary syndrome at a young age is uncommon. Smoking was the most common conventional cardiovascular risk factor among patients with premature coronary artery disease contributes to aggressive atherosclerosis and premature death. Heart failure (HF) development after hospital discharge in patients with myocardial infarction (MI) is very prevalent. Major predictors of cardiovascular death of acute coronary syndrome were a history of hospitalization for HF and other comorbidities that need to be more carefully targeted and treated.

Case illustration:
A 32-year-old young man came to the emergency room with a swallowed stomach since last week. He also complained of getting tired easily and having shortness of breath during moderate activities. The patient had a history of stent placement in the Left Anterior Descending (LAD) artery and Right Coronary Artery (RCA) 5 years before admission and experienced Heart Failure with Reduced Ejection Fraction (HFrEF) with 45% ejection fraction (EF) 1 year after stent placement. The patient is active in the military, has been a heavy smoker since a teenager but quit smoking after stent placement, and has no history of medical illness or familial history with cardiovascular problems. On examination, vital signs were found to be within normal limits. On physical examination, an S3 gallop was found on the tricuspid and mitral valves. The electrocardiography showed sinus rhythm, heart rate at 77 beats per minute, RBBB, and frequent VES in V1-V3. Echocardiography showed HFrEF at 33%, severe mitral regurgitation, and moderate tricuspid regurgitation. Laboratory findings were normal and abdominal ultrasound showed ascites. Even though he takes regular medication and has regular check-ups with a cardiologist, the patient feels his quality of life is decreasing.

Conclusion:
Younger patients had similar incidences of mortality when they had identical risk factor profiles with older adult patients. Conventional cardiovascular risk factors were also predictors of poor outcomes, highlighting that prevention and treatment of these factors need to be more carefully targeted and treated. The population of patients after MI represents a high-risk group for HF development. Development of HF after MI is associated with adverse events, impaired quality of life, and lower survival.

Keywords: younger adult, heart failure, outcomes, acute coronary syndrome, myocardial infarction

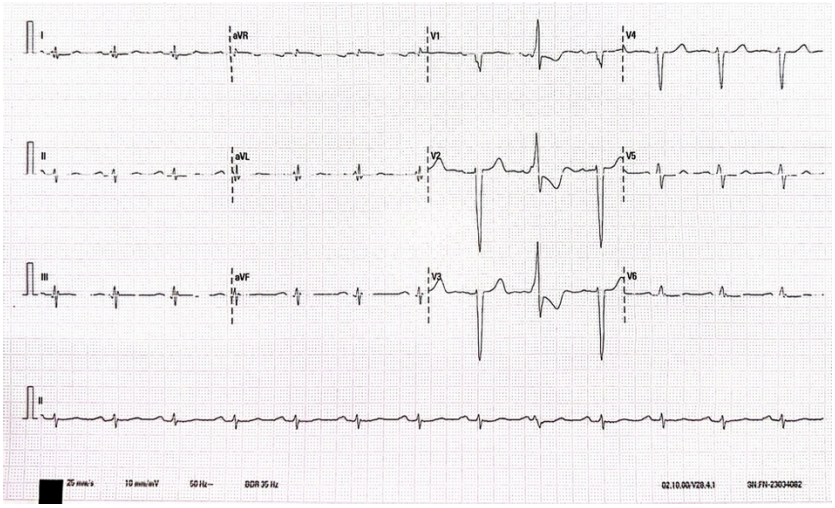

Figure 1. Electrocardiography in the Emergency Room

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MANAGING AF RAPID VENTRICULAR RESPONSE IN HFREF PATIENT DURING DENGUE HEMORRHAGIC FEVER

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Background:
Atrial fibrillation (AF) and heart failure (HF) often coexist and can significantly impact each other in terms of prognosis and management. AF and HF frequently coexist due to shared risk factors such as hypertension, coronary artery disease, valvular heart disease, and aging. The presence of one condition increases the likelihood of developing the other and its management can be challenging, especially in the setting of concurrent dengue haemorrhagic fever.

Case illustration:
A 50-year-old male patient, referred from another hospital with AF RVR, Acute Lung Oedema and was intubated because of desaturation of oxygen. The blood pressure was 90/60 mmHg, with pulse rate 150-160 bpm, irregular. There was crackles sound almost throughout the lung field and minimal pitting oedema at the extremities, indicating an acute lung oedema condition. ECG showed AFRVR. The X-ray results showed cardiomegaly with congestion. In echocardiography found ejection fraction was only 19%, with LA dilatation and severe mitral regurgitation. From the laboratory results found thrombocytopenia and positive IgM and IgG anti-dengue. The patient was given rate control therapy for the AFRVR condition, and diuretics to reduce congestion. Due to the thrombocytopenia condition, the administration of anticoagulants was delayed until the thrombocyte within normal limit. During treatment, patients are closely monitored for fluid balance, and also treated with the internist. On the 2nd day of treatment at the ICCU, the patient was extubated, signs of congestion decreased, the patient was transferred to regular ward. In the outpatient clinic, the thrombocyte was already within normal range so anticoagulants began to be administered, to prevent stroke conditions. The patient is also given optimal therapy for the condition of Heart Failure.

Conclusion:
Managing atrial fibrillation in a patient with heart failure with reduced ejection fraction during dengue haemorrhagic fever presents a significant clinical challenge. This complex scenario requires close monitoring, careful assessment of risks and benefits, and individualized treatment decisions are vital.

Keywords: Dengue Hemorrhagic Fever, Atrial Fibrillation, Heart Failure Reduced Ejection Fraction

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CHAOTIC ARRHYTHMIA IN PERIPARTUM CARDIOMYOPATHY: A CASE REPORT

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Background:
Peripartum cardiomyopathy (PPCM) is a dilated cardiomyopathy defined as systolic cardiac heart failure in the last month of pregnancy or within five months of delivery. In this peripartum period, there is an increased incidence of arrhythmias, which may range from 50% to 90%, even in women without structural heart disease. This case report examines the arrhythmic variations in a patient diagnosed with PPCM two weeks postpartum, emphasizing the need for timely diagnosis and comprehensive management.

Case illustration:
A 34-year-old woman arrived at the ED with severe dyspnea lasting two days. She couldn't lie flat due to orthopnea and found some relief by leaning forward. She had undergone a caesarean section two weeks earlier, had no complications during pregnancy or delivery, and had no known cardiovascular risk factors. On examination, the patient had a blood pressure of 177/122 mmHg, a pulse rate of 116 beats per minute, a respiratory rate of 42 breaths per minute, and an oxygen saturation of 87% on room air. Lung examination revealed bilateral fine basal crackles. An ECG indicated sinus tachycardia. A chest radiograph showed cardiomegaly and pulmonary edema. The patient has a normal magnesium count and an increased prolactin level. Echocardiography revealed a significantly reduced LVEF of 32% with a dilated LV, indicative of heart failure due to PPCM.Initial treatment with diuretics provided temporary stabilization. However, within 12 hours, the patient experienced a seizure with polymorphic VT, which was observed on the monitor. Prompt defibrillation restored sinus rhythm and consciousness. Within 24 hours, she developed SVT, which reverted to sinus rhythm following amiodarone administration. Bromocriptine was then initiated, and her condition gradually improved during hospitalization

Conclusion:
This case highlights the complexity of PPCM and the need for vigilant monitoring for arrhythmic complications. The range of arrhythmic presentations—from sinus tachycardia to life-threatening VT and SVT—demonstrates the need for a comprehensive approach to managing PPCM. Early recognition and diagnosis are crucial, particularly for postpartum women with unexplained dyspnea. The treatment regimen for PPCM typically includes standard heart failure therapies, and bromocriptine, as a prolactin inhibitor, is fundamental to improving outcomes.

Keywords: heart failure, maternal morbidity, postpartum cardiac complications, peripartum cardiomyopathy, arrhythmia

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PACING INDUCE CARDIMYOPATHY

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Background:
A pacemaker is a definitive treatment tool for symptomatic bradyarrhythmia to reduce morbidity and mortality. However, chronic right ventricular (RV) pacing has the potential to cause pacing induced cardiomyopathy (PICM), which results in remodeling of the left ventricle that triggers heart failure, atrial fibrillation and increased mortality.

Case illustration:
This is a case report on a patient who was treated at the Central General Hospital (RSUP) Prof. Dr. R. D. Kandou Manado with pacing induced cardiomyopathy. A 79-year-old woman with complete atrioventricular block was admitted to Prof. Dr. R. D. Kandou hospital Manado to undergo pacemaker implantation. Echocardiography prior to pacemaker implantation showed dilated LA, concentric LVH, LVEF 73% with no regional wall motion abnormalities, mild – moderate TR. Patient underwent DDDR pacemaker implantation with ventricular lead was placed in mid-septal RV. Duration of QRS complex after pacemaker implantation was 150 ms. During 7 months follow-up, patient developed shortness of breath. Echocardiography showed reduction of LVEF to 59%, mild – moderate hypokinesia at basal – mid anterior, anteroseptal, septal. Coronary angiography showed non-significant coronary artery stenosis with 30% stenosis at distal LAD. Pacemaker was then interrogated and revealed that pacing burden was 100%. Based on the history, wide QRS complex on ECG, echocardiography changes, high pacing burden, and coronary angiography results, patient was diagnosed with heart failure with preserved ejection fraction due to PICM. Patient was planned to upgrade the pacemaker to a CRT or conduction system pacing.

Conclusion:
Pacing induced cardiomyopathy (PICM) is generally defined as a decrease in left ventricular ejection fraction (LVEF) in the setting of chronic high burden RV pacing. It has been reported that approximately 20% of patients will experience PICM within 3-4 years after implantation of a pacemaker in the RV. Treatment of PICM is primarily focused on upgrading to cardiac resynchronization therapy / CRT when the left ventricular ejection fraction decreases.

Keywords: Right Ventricular Pacing, Pacing Induced Cardiomyopathy (PICM), Pacemaker

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A 53-YEAR-OLD MALE WITH STEMI INFERIOR AND APLASTIC ANAEMIA : THERAPEUTIC CHALLENGES IN RURAL AREA

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Background:
STEMI is a cardiac emergency caused by coronary artery occlusion, leading to heart tissue necrosis. Aplastic anaemia (AA), characterized by bone marrow failure to produce blood cells, complicates STEMI management. This report discusses a patient with both conditions, highlighting therapeutic challenges because of the increased bleeding risk and limited drugs of choice in rural area.

Case illustration:
A 53-year-old male, was referred to our hospital with a typical angina complaint. His symptoms began approximately 15 minutes before admission. Three months before admission, Anemia Aplastic was diagnosed based on the patient's bone marrow biopsy report and methylprednisolone 3x24 mg was prescribed for daily medication. He was suspected to have a history of type 2 diabetes (random blood glucose 211 mg/dL at our Emergency Room). His family had no early onset history of cardiovascular diseases. Physical examinations showed normal blood pressure (120/70 mmHg), and chest auscultation revealed no lung rales. Ecchymosis appeared in the upper and lower extremity. ECG showed ST elevation on lead II, III, aVF and ST depression on lead I, AVL, V5 and V6. Laboratory results revealed pancytopenia. Therefore, he was diagnosed with STEMI inferior KILIP I and Aplastic Anaemia with platelet count, 4x103/uL, Hb 10.4 g/dl and white blood cell count 3.05 x 103/uL. He received ISDN 3x5mg, bisoprolol 1x1.25 mg, tranexamic acid 3x500 mg IV, gliclazide 1x80, dexamethasone 3x10 mg and platelet transfusion. The antithrombotic was omitted from patient's therapy until his platelet count >5x104/uL. After he received 4 bags of TC in 2nd day of hospitalization, his platelet count decreased to 1x103/uL. However, 3rd day of hospitalization the patient passed away because of cardiogenic shock.

Conclusion:
This case underscores the complexity of managing a patient with STEMI Inferior and aplastic anaemia due to the increased bleeding risk. A comprehensive, multidisciplinary approach is essential to optimize outcomes and manage the interplay between these conditions effectively.

Keywords: Rural Area, Aplastic Anaemia, STEMI Inferior

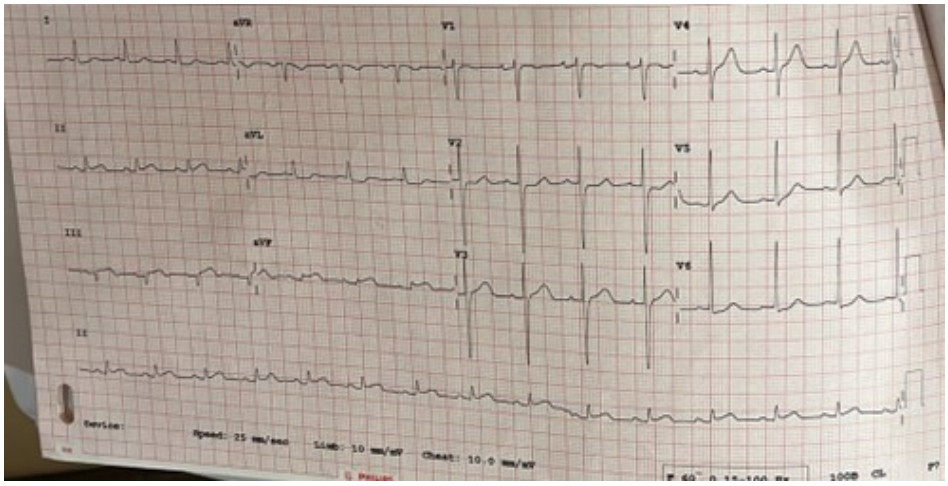

Figure 1. ECG showed ST elevation on II, III, aVF leads and ST depression on lead I, AVL, V5 and V6.

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ACUTE UPPER LIMB ISCHEMIA WITH THROMBECTOMY EMERGENCY: A CASE REPORT

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Background:
Acute limb ischaemia (ALI) is defined as the sudden and significant reduction of blood flow to the limb which promote various complications, including the risk of major amputation and death. Acute Upper Limb Ischemia (AULI) is not as common as acute lower limb ischemia with cardiac embolism as the most common cause. It is the vascular emergency and urgent revascularization is mandatory.

Case illustration:
A 77-year-old man was admitted with pain at the left hand since 5 days before admission that occured suddenly when he was sleeping. The pain was felt at rest and the tips of all fingers of the left hand were blackish. The sensation of the left hand were decreased and it felt cold. The patient explained that the complaint started in the middle finger only then radiated to all fingers. The patient had a history of hypertension and absence of prior traumatic incidents. History of chest pain and palpitation were denied. Physical examination of the left hand presented pulselessness of left radialis artery, pallor, poikilothermy, paresthesia and painful with left fingers oxygen saturation of digiti I 68%, digiti II 68%, digiti III 66%, digiti IV 80% and digiti V 82% with Ankle Brachial Index (ABI) were 0,57. The motoric of the fingers were decline. Electrocardiography showed sinus rhythm with heart rate 95 beats per minute and normoaxis. Echocardiography found left ventricle ejection fraction (LVEF) 71%, TAPSE 2.3 cm, trivial tricuspid regurgitation, mild pulmonal regurgitation, no visible intracardiac thrombus/vegetation, global normokinetics, and left ventricle diastolic dysfunction grade 1. Emergency thrombectomy were performed using Fogarty catheter and found thrombus at the left axillaris artery, thrombus at left ulnaris artery with slow flow, and thrombus with total occlusion at left radialis artery. Furthermore, the patient received anticoagulant, antihypertensive, and analgetic. The patient showed clinical improvement in the left arm including relief symptoms, the blackish of the fingers decreased and adequate arterial pulse.

Conclusion:
Acute Upper Limb Ischemia is a vascular emergency that requires early recognition and appropriate treatment. Urgent revascularization enhances patient outcome and decreases morbidity and mortality.

Keywords: Peripheral artery disease, Acute upper limb ischemia, Thrombectomy

Figure 1. Clinical improvement of the left hand after thrombectomy

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CONQUERING INCESSANT VENTRICULAR TACHYCARDIA: A CASE REPORT ON LIFE-SAVING MANAGEMENT STRATEGIES

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Background:
Ventricular arrhythmias (VA) are common in the initial phases of ischemia and remain a major cause of sudden death during acute myocardial infarction. The yearly occurrence of refractory VA can reach up to 0.6 cases per 100,000 people, with a mortality rate as high as 97%. The treatment is determined by the related symptoms, underlying pathology, hemodynamic consequences, and long-term prognosis. Due to their complexity, physicians face considerable challenges in initial care, risk classification, and treatment of ventricular arrhythmias

Case illustration:
A 67-year-old man with hypertension and ischemic heart disease, known cardiovascular risk factors, presented with rapid and sustained monomorphic ventricular tachycardia (VT) and poor clinical and hemodynamic tolerance. Six months prior, he had suffered a heart attack and was admitted to the intensive care unit, though there was no history of revascularization. Despite six external cardioversions, electrocardiographic monitoring showed persistent arrhythmia organized as VT (Figure 1). Initial treatment with full-dose lidocaine was ineffective. Two cycles of defibrillation followed by intravenous amiodarone successfully terminated the VT. Later the electrocardiogram showed complete RBBB and ST-segment depression in V4-6. The serum electrolytes were normal with elevation of serial cardiac troponin. We managed to perform coronary angiography that revealed significant stenosis in two coronary vessels.

Conclusion:
Ventricular tachycardia is a dangerous condition that requires rapid treatment and often leads to poor outcomes despite available therapies. Effective interventions include the prompt recognition of the condition, addressing reversible causes, the use of antiarrhythmic drugs, sedation, and catheterization.

Keywords: incessant ventricular tachycardia, anti-arrhythmic drug, refractory ventricular tachycardia, Ventricular arrhythmias

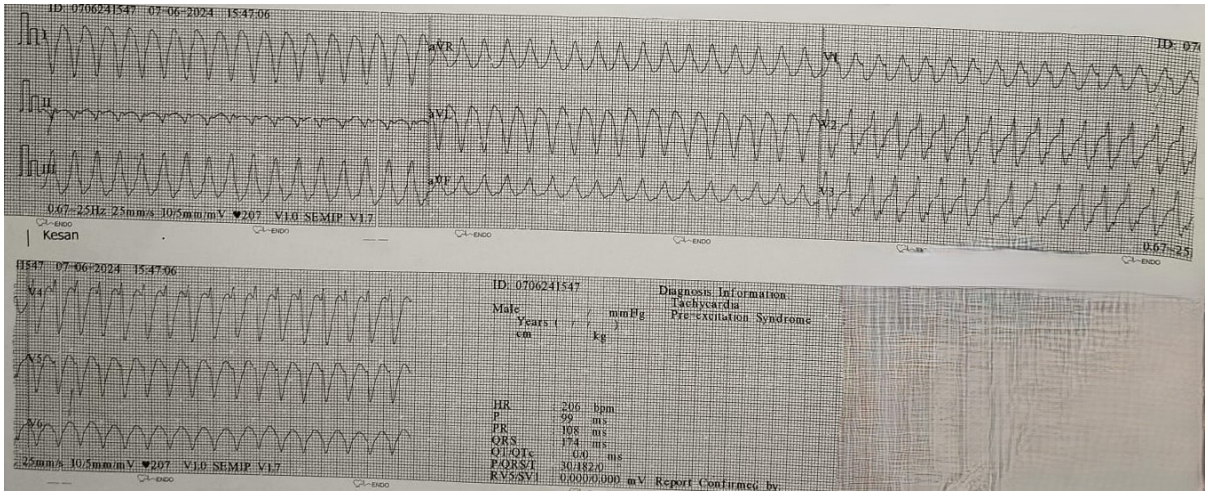

Figure 1. ECG showed Monomorphic Ventricular Tachycardia (VT)

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FIXED COUPLING INTERVAL OF PREMATURE VENTRICULAR CONTRACTION: IS IT FOCAL OR RE-ENTRY?

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Background:
Premature Ventricular Contractions (PVCs) are common both in patients with coronary artery disease. One of the basic ECG characteristics of PVCs is the coupling interval (CI), which is defined as the distance between the onset of the preceding sinus QRS and that of the premature beat. The variability of CI is influenced by several factors (e.g, variation of the preceding cycle length, fluctuations in rhythmic distribution patterns, intermittent parasystole, and precipitancy of another ectopic source.

Case illustration:
A 58 years-old man presented to the Emergency Department (ED) with chest pain that occurred since 4 hours before admission. The chest pain was described as a tightness radiates to the back. A 12 Lead ECG showed Sinus Rhythm with PVC present in a pattern of bigeminy with coupling interval 460 ms. The originating from RVOT. Laboratory tests showed within a normal limit. The echocardiogram showed LVEF 44 % with Mild Pulmonal insufficiency. Amiodarone administration was stopped due to junctional bradycardia. PCI showed three vessel disease and one drug-eluting stent was deployed in LAD. Temporary Pacemaker is used in these instances, but the PVCs were still frequent. There was a reduction in PVCs after combination of continuous Amiodarone and Lidocaine were given. The patient was discharged and planned for catheter ablation. These Phenomenon could be caused by myocardial scar tissue which can create conditions such as unidirectional block and slow conduction, which facilitate the formation of re-entry circuits. These circuits can lead to the generation of PVCs with a fixed coupling interval, indicating a consistent exit site for the re-entry circuit and suggesting a specific area of scar tissue as the origin of these abnormal beats.

Conclusion:
PVCs with a fixed coupling interval suggest that a re-entry circuit origin . In such circuits, the electrical impulse follows a specific pathway within or around scar tissue in the myocardium. Due to the anatomical and physiological properties of this pathway (such as conduction velocity and refractory periods), the time interval between each sinus beat and subsequent PVC remains constant.

Keywords: re-entry, fixed coupling, lidocaine, Premature Ventricular complex, temporary pacemaker

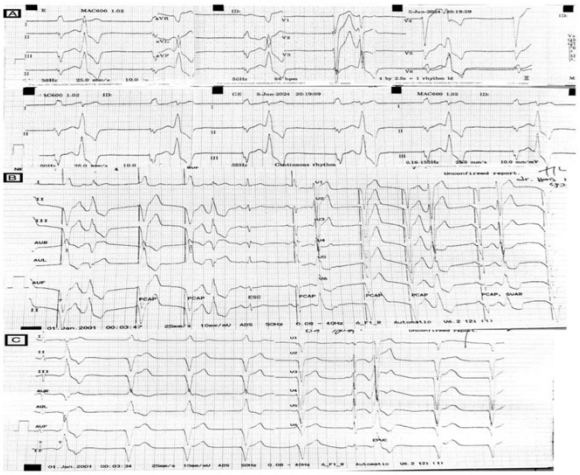

Figure 1 A) Initial ECG show PVC bigeminy with fixed CI. B) ECG on TPM, ventricular pacing with junctional rhythm and SND, fixed CI. C) ECG on TPM after continuous Amiodarone and Lidocaine

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RADIOTHERAPY-INDUCED PERIMYOCARDITIS

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Background:
Radiotherapy-induced perimyocarditis is a severe and rarely overlooked complication in patients undergoing treatment for breast cancer. Early detection through advanced imaging techniques and appropriate management is crucial to prevent irreversible damage.

Case illustration:
A 42-year-old female patient experienced dyspnea on exertion (DOE), paroxysmal nocturnal dyspnea (PND) and orthopnea during her radiotherapy sessions. She underwent six cycles of chemotherapy with anthracycline and cytarabine from Desember 2022 to March 2023 and continued with radiotherapy from Desember 2023 to January 2024.Heart Failure Association and International Cardio-Oncology Society (HFA-ICOS) baseline risk score was high before starting radiotherapy. Echocardiographic findings before chemotherapy showed normal chambers and left ventricular ejection fraction (LVEF) 52%. However, echocardiographic and cardiac magnetic resonance imaging (CMR) post-radiotherapy revealed significant deterioration. Echocardiography and CMR showed dilated LV and dramatically decreased LVEF with regional wall motion abnormality (LVEF 25%). Global subendocardial enhancement more prominent at base to mid anterior, anteroseptal and sub-epicardial enhancement at basal to mid inferolateral were seen on CMR. Moderate-severe circumferential pericardial effusion with increased of pericardial intensity were also seen at anterior, lateral, and inferior wall base to apical. T1, T2, and T2 STIR were also increased. The CMR impression is perimyocarditis probably due to radiotherapy-induced cardiomyopathy. The patient was given furosemide 40 mg, spironolactone 25 mg, ramipril 5 mg, and bisoprolol 2.5 mg once daily.

Conclusion:
Radiotherapy-induced perimyocarditis should be prevented. Regular evaluation of cardiac function is required. Utilizing multimodal imaging and tailored pharmacotherapy is essential in mitigating the adverse cardiac outcomes associated with cancer treatments.

Keywords: Radiotherapy-induced perimyocarditis

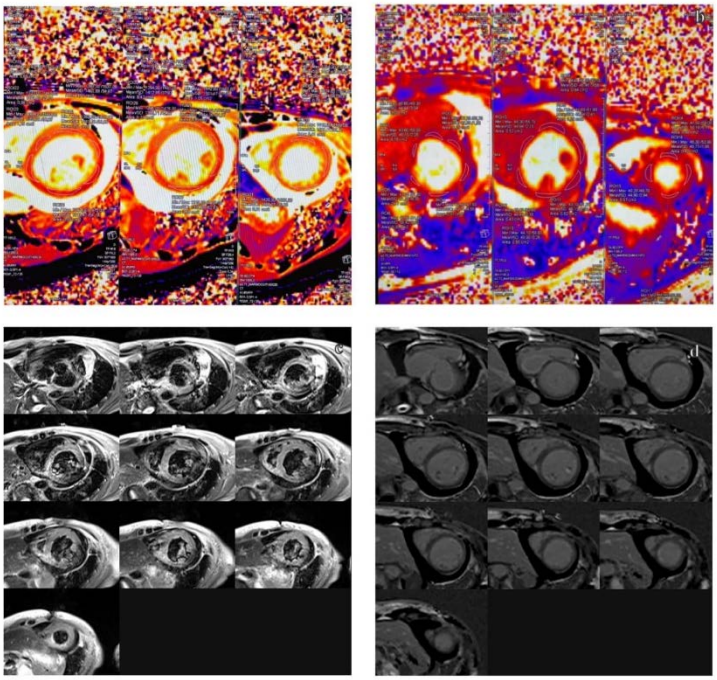

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Figure 1. Cardiac MRI findings: (a) T1 mapping, (b) T2 mapping, (c) T2 STIR, (d) LGE.

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MORE THAN JUST MUCOUS CLEARING: TERMINATION OF WIDE COMPLEX TACHYCARDIA BY COUGHING

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[1]RSUD Inche Abdoel Moeis

Background:
A wide complex tachycardia (WCT) is defined as a cardiac rhythm with a rate >100 beats per minute and a QRS width >120 ms. The most common cause of WCT are ventricular tachycardia (VT) and supraventricular tachycardia (SVT) with aberrancy. Apart from using well known criteria such as Brugada or Vereckei criteria, vagal maneuvers are also may be useful in distinguishing between VT and SVT with aberrancy. However, most patients with WCT should be managed as though they have VT until proven otherwise.

Case illustration:
A 64 years old male was referred to our ED with typical chest pain. The onset was 1 hour. The patient had previous history of STEMI anterior. The patient was haemodynamically stable. On physical examination, the patient was sweating profusely and breathing rapidly. On ECG (Fig 1), we found WCT (HR: 212 bpm). Upon closer inspection, using Brugada and Vereckei criteria, the ECG was classified as SVT and VT respectively. Diagnosis of stable VT was made and synchronized cardioversion was planned using midazolam as sedating agent. Upon injection of midazolam, patient then started to cough for about 2 minutes. After the cough stopped, the patient's rhythm was converted to normal sinus rhythm (HR: 100 bpm). Coughing is comparable to an exaggerated valsalva maneuver because both conditions elicit an increase in intrathoracic and a decrease in venous return. Termination of WCT by coughing suggest that this was SVT with aberrancy. However, research conducted by Waxman et al shows that vagal maneuvers can also terminate VT repeatedly.

Conclusion:
We present a case of a 64 year old male with initial ECG of WCT. After administration of sedative agent, this patient was coughing excessively, and the ECG was then turned into sinus rhythm. Most patients with WCT should be managed as though they have VT until proven otherwise. However, in hemodynamically stable patients, we suggests trying vagal maneuvers, as these maneuvers may be useful in distinguishing between SVT and VT. Vagal have also been shown to terminate VT repeatedly.

Keywords: VT, vagal, SVT, WCT, wide complex tachycardia

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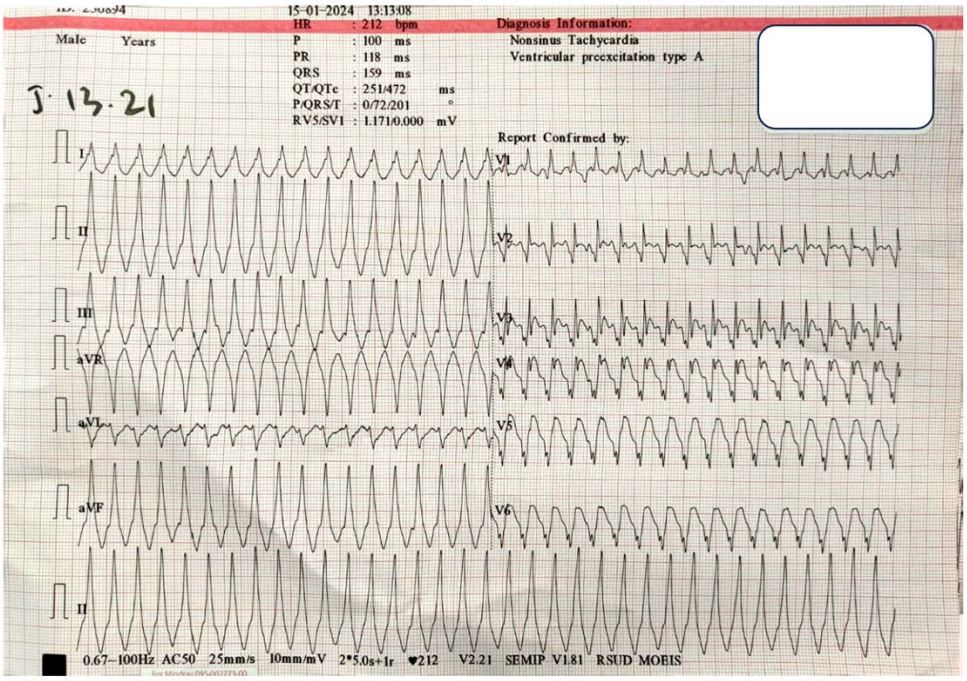

Figure 1. Initial 12-lead ECG of the 64 year old male upon presentation in Emergency Department

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AORTOPULMONARY WINDOW WITH PULMONARY ATRESIA AND VENTRICULAR SEPTAL DEFECT: A VERY RARE CONGENITAL COMBINATION IN CHILDREN

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Background:
Aortopulmonary window (AP window) is an uncommon conotruncal defect characterized by communication between the ascending aorta and main pulmonary artery. It comprises only 0.1% of all congenital cardiac defects. Pulmonary atresia (PA) with ventricular septal defect (VSD) is encountered more frequently. Combination of PA-VSD in AP window is extremely rare. To the best of our knowledge, there have been only 8 cases reported in medical literature.

Case illustration:
A 17-day-old baby girl was referred to the pediatric outpatient clinic with chief complaint of poor feeding since birth. Patient often looked diaphoresis and dyspneic while feeding. Patient had cyanosis in her fingernails, especially when she cried. Cardiac auscultation revealed normal first sound, and single, non-accentuated second heart sound. There was continuous murmur heard best at left-upper sternal border. We found cardiomegaly, upward apex, and normal pulmonary vasculature in chest radiograph. Echocardiography showed PA, VSD (right-to-left shunt), and large aortopulmonary window (diameter 6 mm, left-to-right shunt). Patient was diagnosed with AP window type IV, pulmonary atresia, and ventricular septal defect. She was given captopril and furosemide. Staged surgical approach was preferred due to her small weight. AP window banding was done when she was 2 months old to reduce lung overflow and prevent pulmonary vascular disease. Post-operative echocardiography evaluation showed banded AP window (diameter 3.3 mm). In the long-term, definitive surgery by Rastelli procedure and AP window repair is planned when the patient reaches age 1 year old.

Conclusion:
We reported a very rare case of AP window in association with PA-VSD. Despite the pulmonary atresia, this patient presented early in infancy with signs of heart failure without cyanosis due to large shunt volume from systemic to pulmonary circulation. Diagnosis can be established by comprehensive examination including echocardiography. Management of AP window consists of medical management with vasodilators and diuretics, followed by palliative AP window banding. Surgery should not be delayed to avoid development of pulmonary vascular disease in later age.

Keywords: Aortopulmonary window, pediatric cardiology, pulmonary atresia, congenital heart disease, conotruncal defect

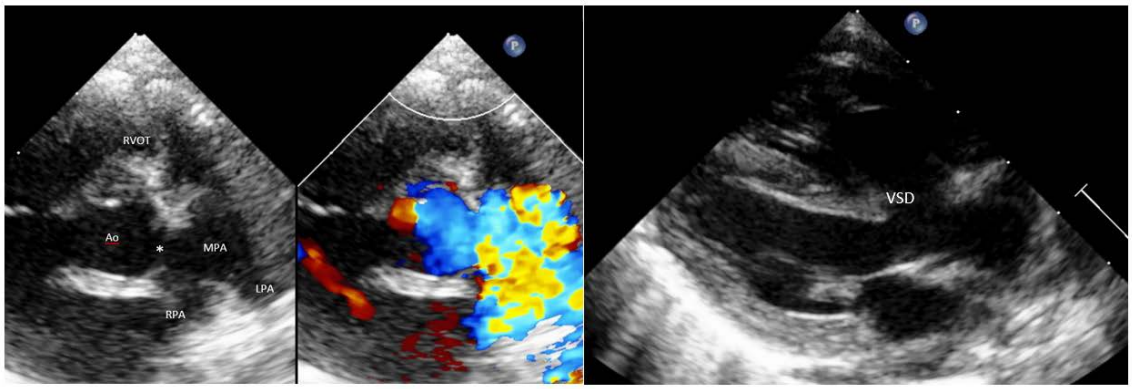

Figure 1. Echocardiography showing AP window (asterisk) type II with left-to-right shunt. RPA and LPA originate from the MPA. There was no flow from the right ventricle to MPA, indicating pulmonary atresia. There was coexisting subaortic VSD

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CHALLENGES IN POST-CORONARY ARTERY BYPASS GRAFTS RECOVERY AND REHABILITATION OUTCOMES: TWO DIVERGENT CASES

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Background:
Coronary artery disease (CAD) is the leading global cause of mortality. Coronary disease with complex lesions is prone to having bypass grafting as a choice intervention but it could be a challenging prognosis regarding duration time being hospitalization. The length of stay (LOS) after Coronary Artery Bypass Grafting (CABG) procedure is influenced by factors like patient attributes, pre-and intraoperative treatment, complications, surgical events, and hospital protocols.

Case illustration:
We presented 2 patients diagnosed with CAD who underwent CABG. The first was a 55-year-old man with a risk factor of being an ex-smoker and hypertension. The duration of hospital stay for 20 days of care. The patient suffered respiratory failure due to pneumonia et causa *Pseudomonas aeruginosa* and *Methicillin-resistant Staphylococcus aureus* infections on the fourth day after CABG. The patient received one gram of Amikacin and 500 mg of Meropenam three times per day via intravenous. In addition, the patient also developed hyperkalemia and paroxysmal atrial fibrillation making the longer hospitalization. The patient's condition was improving, and the infection was being managed. The second case was a 56-year-old woman who had risk factors of dyslipidemia and obesity, the duration after surgery lasting 7 days of LOS. A decrease in hemodynamic and RV contractility, as well as acute lung edema, occurred after the administration of protamine post-off cardiopulmonary bypass. Following iloprost 2.5 mcg nebulization and furosemide 20 mg intravenous, clinical and hemodynamic conditions were improved. During treatment, both patients went through early Phase I Rehabilitation Programs. The six-minute walking test results of the two CABG postoperative patients were not very different; patients with long-term hospitality obtained a 420-meter distance, VO2 max 16.5 ml/kg/min, and maximum workload of 4.7 METs, while patients with shorter LOS received a 355-meter distance, VO2 max 14.6 ml/ kg/min and a max workload of 4.2 METs.

Conclusion:
Several variables involved the duration of hospitalization in patients with CAD following CABG. Patients who participate in a Phase I Rehabilitation Program while receiving therapy will see positive effects on SMWT outcomes after the CABG procedure.

Keywords: Coronary Artery Bypass Grafts, Coronary Artery Disease, Length of Stay, Six-Minute Walking Test

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BREAKING THE AGE BARRIER: ACUTE CORONARY SYNDROME IN YOUNG ADULTS

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Background:
Acute Coronary Syndrome (ACS) typically affects patients over the age of 45. However, it can also occur in younger individuals. Even though it's rare, it can still be a severe issue for the patient and the managing physician. Traditional risk factors of ACS are hypertension, diabetes, smoking, and dyslipidemia.

Case illustration:
We describe 2 cases of ACS in young adult patients. The first patient was a 28-year-old male with no history of hypertension, diabetes, and dyslipidemia who was admitted to the emergency department due to chest pain. The only risk factor for this patient is smoking. On physical examination, he was fully alert and had normal blood pressure. The ECG revealed atrial fibrillation rapid ventricular response with ST-segment elevation in anterior and lateral leads. Laboratory testing showed cardiac Hs-Troponin I elevation at 17229,5 ng/L. The patient was then administered a loading dose of dual antiplatelet and high-dose statin (Atorvastatin 80mg). We have decided to use potent P2Y12 inhibitors as an antiplatelet agent for this patient. The patient then underwent Primary Percutaneous Coronary Intervention (PCI). Coronary angiogram showed acute total occlusion in osteal to mid Left Anterior Descending artery (LAD) and 95% stenosis at proximal Right Coronary Artery (RCA). The second patient was a 26-year-old male admitted with chest pain. The risk factors for this patient are smoking, hypertension, dyslipidemia, and Smoking. Upon initial assessment, his blood pressure was 170/100 mmHg. Laboratory testing showed elevated cardiac HS- troponin I. The patient then underwent fibrinolytic therapy. On the second day of treatment, PCI was performed. It demonstrated 95% stenosis in distal Left Circumflex artery (LCx) and 80% in proximal RCA. In this patient with multiple risk factors, we found a worsening of stenosis four months after the initial PCI.

Conclusion:
ACS and Coronary Artery Disease (CAD) in young adults is rare compared to elderly individuals. In our case, both patients have several traditional risk factors. For young individuals, its important to consider non-traditional risk factors that could contribute to CAD. This disease has a disruptive effect on the patient’s quality of life, survival, cost of health and social care.

Keywords: Coronary Artery Disease, Young Adult, Risk factors, Acute Coronary Syndrome

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NAVIGATING SYNCOPE: A CASE OF MANAGEMENT AND REFERRAL

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Background:
Syncope is a transient loss of consciousness characterized by a temporary inability to maintain posture, followed by spontaneous recovery. While often benign, it can signal potentially life-threatening conditions. Elderly individuals and those with cardiac disease are more likely to experience syncope from cardiac causes, which carry higher mortality rates. Therefore, thorough investigations are essential in these cases.

Case illustration:
A 48-year-old woman presented at the emergency department following an episode of syncope occurring six hours prior to admission. She suddenly experienced a brief loss of consciousness lasting less than one minute, precipitated by a fall resulting in abrasions on the occipital region. The episode occurred while she was queuing at a stall, without any reported changes in position or aura beforehand. The patient reported no headache, nausea, or vomiting, and had no chest pain, palpitations, or shortness of breath. She acknowledged a previous similar episode but had not previously sought medical evaluation. The patient mentioned a cardiovascular diagnosis from 15 years ago without specifying the exact condition and had a history of dyspepsia syndrome.Initial assessment revealed normal vital signs and physical examination findings. The 12-lead ECG revealed the presence of ventricular tachycardia (see Picture 1).The patient was admitted to the high-care unit (HCU) for monitoring and started on Amiodarone 200mg three times daily (TDS). Over the 24-hour observation period in the HCU, the patient remained stable without any syncope episodes. Consequently, the patient was discharged from the hospital. Three days later, the patient returned to our outpatient clinic for follow-up. After confirming normal echocardiography findings, we decided to refer the patient to an electrophysiologist for further evaluation.

Conclusion:
Progressive premature ventricular contractions (PVCs) can be triggered by exercise or stress, potentially leading to syncope or death due to its progression into ventricular tachycardia (VT) or ventricular fibrillation (VF).

Keywords: ventricular tachycardia, Syncope

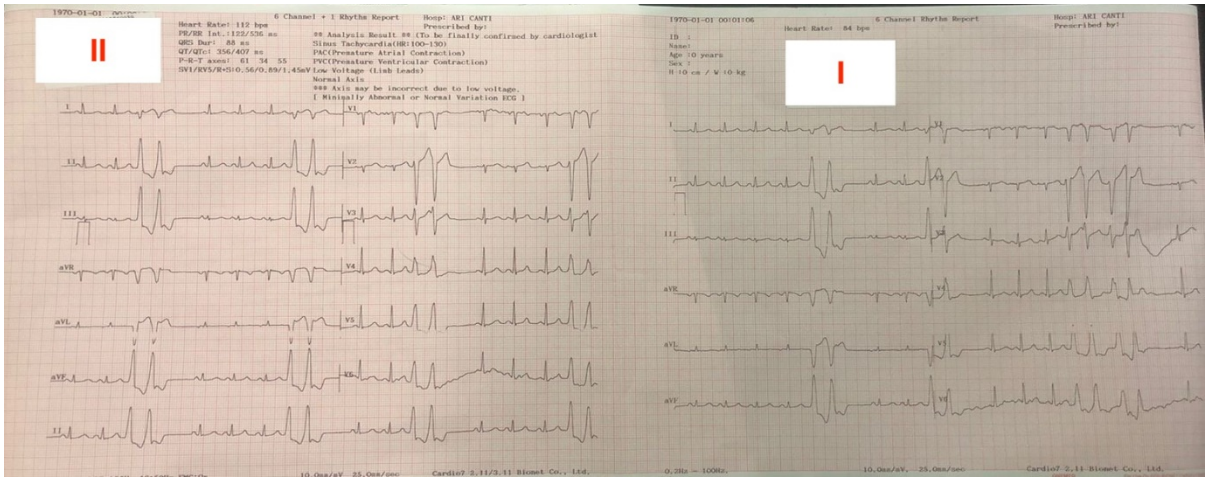

Figure 1. Initial ECG showing ventricular tachycardia

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IMMEDIATE STAGED PERCUNATEOUS CORONARY INTERVENTION IN MULTIVESSEL STEMI WITH WORSENING CARDIOGENIC SHOCK

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Background:
Primary percutaneous coronary intervention (PCI) is the most optimal management in ST elevation myocardial infarction (STEMI) patients. Optimal revascularisation can reduce mortality and major adverse cardiovascular events. However, in cases of multivessel disease, the choice of culprit lesion only or multivessel PCI is still controversial. The optimal timing of non-target-vessel PCI is also debatable. In this case report, we discuss immediate staged PCI in a case of STEMI with cardiogenic shock and three-vessel disease.

Case illustration:
Male, 46 years old admitted to the emergency room with a chief complaint of chest pain since 90 minutes ago. The complaint was associated with dyspnea, nausea, vomiting, and sweating. The physical examination revealed a blood pressure level 80/60, signs of shock, and ronchi in both lung fields. ECG revealed inferolateral ST elevation. Coronary angiography was performed with 90% stenosis in right coronary artery (RCA) and chronic total occlusion (CTO) in left anterior descending (LAD) and left circumflex (LCX) artery. PCI was performed on the RCA. Echocardiography examination after primary PCI showed decreased heart function (LVEF 21%), akinetic inferolateral and inferior, large of apex area, and hypokinetic of the other segments. 12 hours after PCI, the patient developed acute lung edema and the patient was intubated. The patient underwent staged PCI at LAD and Intra-Aortic Balloon Pump (IABP) insertion 1 day after primary PCI. 2 weeks after treatment the patient was discharged with improved condition. 30 days follow up, vital sign within normal limit, no sign pulmonary nor systemic congestion.

Conclusion:
Immediate staged PCI can be performed in STEMI patients with cardiogenic shock who have worsened after primary PCI and showed good results. Based on the CULPRIT-SHOCK trial and guidelines from ACC/AHA/SCAI in 2021, immediate PCI is not recommended for non-culprit lesions because it can increase the risk of death and renal failure. However, a large trial in Korea showed that immediate multivessel PCI in STEMI cases with severe cardiogenic shock requiring venoarterial-extracorporeal membrane oxugenation can reduce 30-day mortality and 12-month follow-up mortality.

Keywords: cardiogenic shock, staged PCI, STEMI

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TRANSIENT ST-ELEVATION DURING PERCUTANEOUS ATRIAL SEPTAL DEFECT DEVICE CLOSURE IN A 1 YEAR OLD CHILD: A RARE CASE REPORT

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Background:
Atrial septal defect (ASD) is one of the most common congenital heart diseases (CHD), accounting for 6-10% of all CHDs. The treatment of ASD continues to evolve following the times and technology. Fifty percent of these defects require percutaneous intervention or surgery Trans-catheter device closure of ASD is considered to be safe but sometimes complication could arise and one of them is transient ST elevation.

Case illustration:
A 1-year-old 11 months male with intermittent shortness of breath and difficulty gaining weight. He had a history of recurrent of upper respiratory tract infection. Hemodynamic parameters were stabled and no cyanosis found. The chest X-ray showed cardiomegaly, transthoracic echocardiography and right heart catheterization examination showed large secundum ASD left to right shunt with defect size approximately 7 – 9 mm and all the rims were suitable for device closure. During percutaneous atrial septal defect closure device implantation procedure, monitor electrocardiogram showed ST elevation in the inferior derivations for 1 minutes and 5 seconds and return to normal after the device was released. Echocardiography evaluation showed the device well seated, and it did not interfere with the movement of the cardiac valves and no residual shunt present.

Conclusion:
Although trans-catheter device closure of ASD is relatively safe and convenient for the patient,transient elevation of the ST segment has been described as a rare complication during percutaneous ASD device closure and it was reported caused by several different possible mechanism which still needs further investigation. Manipulation of the catheter, the occluder device and also inflation deflation technique of the septal device occluder procedure should be done more careful and efficient by all operators.

Keywords: Transient ST-elevation, Atrial septal defect, Transcatheter device closure

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THE INTERLINK OF AORTIC DISSECTION AND ACUTE STEMI : CHALLENGING DIAGNOSIS AND TREATMENT IN LIMITED CARDIOVASCULAR FACILITIES AREA

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Background:
Aortic Dissection and STEMI are two dangerous clinical entities that could be rapidly fatal if not detected and treated promptly. These two provide similar clinical symptoms but with different management of therapy, so a holistic examination is needed to confirm the diagnose and determine patient's treatment.

Case illustration:
A 44 year old male was referred to the emergency room of Ben Mboi General Hospital diagnosed with anteroseptal STEMI due to sharp, tearing chest pain, and changes in ECG showed QST elevation. Presenting in Ben Mboi, on cardiac auscultation, we found diastolic murmur at the 2nd intercostal space of right parasternal, radiating to the apex. Laboratory findings showed HS-Troponin I enzyme level has increased. Echocardiography revealed dilation of the entire aorta, dissection flap from the ascending to descending aorta and severe aortic regurgitation, so we suspected STEMI as a complication of Aortic Dissection. The contrast-enhanced thoracic CT scan as the gold standard examination revealed Aortic Dissection Standford Type A,De Bakey 1, with STEMI due to possible flap dissection. In this condition, surgery is the treatment of choice. Due to limited facilities and patient's preference, surgery or catheterization couldn't be performed, so we optimized the conservative therapy by giving oral medication, targeting at blood pressure and rate control, also optimizing the heart failure therapy. The patient has shown improvement and discharged from the hospital with a stable condition. In cases like this, optimizing conservative therapy is a crucial step to give patients time to receive definitive therapy, also reduce the progression of heart failure.

Conclusion:
Aortic dissection and STEMI are interlinked diseases with fatal consequences if not detected, a quick and accurate diagnosis can bring the patient to the appropriate treatment. In rural areas, without cardiovascular surgical facilities, conservative therapy is a crucial step for the patients before receiving definitive therapy.

Keywords: De Bakey 1, STEMI, Aortic dissection, Stanford Type A

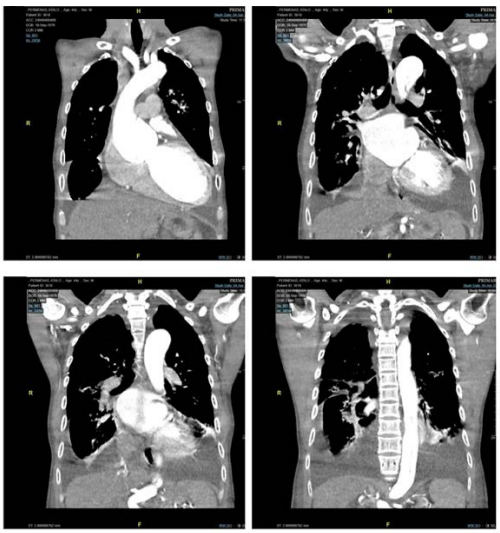

Figure 1. Contrast-enhanced thoracic CT scan

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EXERCISE ELECTROCARDIOGRAPHIC STRESS TEST IN DETECTING CORONARY MICROVASCULAR DYSFUNCTION: A CASE REPORT

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Background:
When evaluating patients with new-onset angina, electrocardiographic stress testing is a common, non-invasive, and affordable functional test. In many clinical settings, the prevalence of coronary microvascular dysfunction (CMD) is higher than previously believed, and its presence is linked to worse clinical outcomes, particularly when myocardial ischemia or nonsignificant coronary artery disease (CAD) are present. This case report describes a patient who showed a positive ischemia response on an exercise stress test but had a normal coronary arteries result when a coronary angiography was conducted.

Case illustration:
A 19-year-old male came to the cardiovascular clinic with complaints of palpitations, fatigue, and chest discomfort during activities. Smoking, asthma, and other respiratory histories were denied. Vital signs in the clinic were within normal limits. A type B Wolff-Parkinson-White syndrome from the anteroseptal was discovered during the patient's ECG evaluation and 24-hour Holter monitoring. EPS and 3D ablation were performed successfully. After the procedures, the patient underwent a stress test to evaluate his exercise capacity. The results revealed ST depression in the inferior and lower lateral leads. The patient then underwent a coronary angiography, which showed normal coronary arteries.

Conclusion:
CMD is recognized as the cause of positive stress tests that indicate ischaemia without obstructive coronary arterial disease. Near-normal angiograms may be seen in patients with diffuse coronary atherosclerosis. On angiography, diffuse coronary atherosclerosis without localized stenosis results in a graded, continuous pressure fall, which contributes to myocardial ischaemia. A thorough history-taking and physical examination, followed by appropriate supportive examinations, can help clinicians diagnose CMD. Exercise stress testing should remain the primary method for clinicians to detect CMD.

Keywords: coronary microvascular dysfunction, coronary artery disease, electrocardiographic stress test

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UNEXPECTED PERICARDIAL EFFUSION : A RARE CARDIAC COMPLICATION OF HYPOPARATHYROIDISM

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Background:
Hypoparathyroidism-induced hypocalcemia remains an unusual cause of cardiovascular complications. In rare instances, this may result in the formation of a pericardial effusion, which carries a high mortality rate. This case report presents a case of pericardial effusion caused by secondary hypocalcemia resulting from hypoparathyroidism.

Case illustration:
A 31-year-old woman was referred to our hospital with dyspnea. She had undergone a total thyroidectomy two years earlier. She was diagnosed with cardiogenic shock, with a blood pressure of 102/56 mmHg while on norepinephrine (50 mcg) and dobutamine (5 mcg/kg/min). A physical examination revealed rales in the lower third of both lungs. The electrocardiogram (ECG) showed slow R wave progression in leads V1-3, accompanied by a prolonged QT interval. Laboratory findings indicated elevated thyroid-stimulating hormone (TSH) levels and hypocalcemia. The chest X-ray revealed cardiomegaly and pleural effusion. Echocardiography showed dilated cardiomyopathy with left ventricular dysfunction (ejection fraction (EF) 38%), right ventricular dysfunction, and a massive pericardial effusion, without right ventricular or right atrial collapse. The patient was started on diuretics (furosemide), calcium gluconate, and levothyroxine, and was discharged after seven days of treatment. Sixteen days later, the patient returned with the same complaint. A recurrence of hypocalcemia was identified, accompanied by an EF reduction and pericardial effusion.

Conclusion:
The role of calcium in excitation-contraction coupling in heart muscle has been identified. In this case, hypocalcemia associated with hypoparathyroidism led to pericardial effusion. Although the mechanism is not yet clear, calcium may affect the heart, contributing to hypertension, left ventricular hypertrophy (LVH), heart failure, and calcific disease, all of which increase cardiac morbidity and mortality. Early treatment of parathyroid disease has the potential to decrease or reverse the adverse effects on the cardiovascular system.

Keywords: pericardial effusion, hypoparathyroidism, heart failure, hypocalcemia

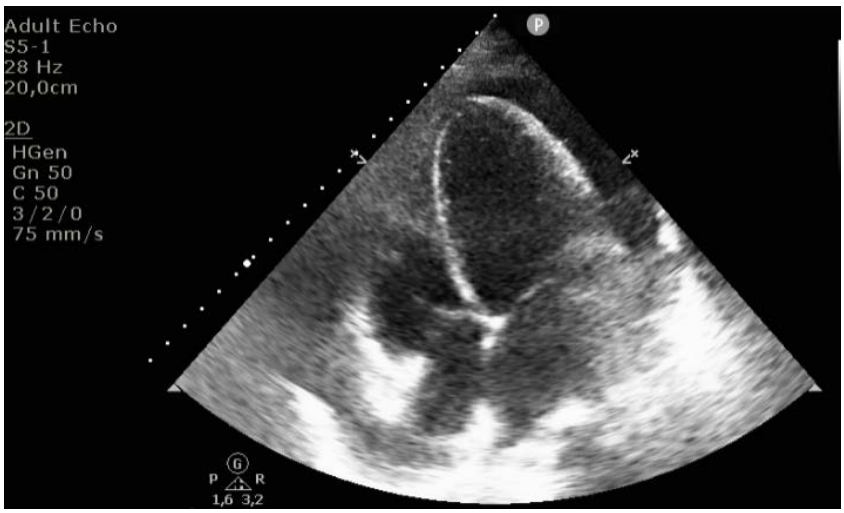

Figure 1. There is massive pericardial effusion in posterior area, without right ventricular or right atrial collapse

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INVERTED T WAVE, IS IT ACS OR SOMETHING ELSE? HYPERTROPHIC CARDIOMYOPATHY (HCM): A CASE REPORT

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Background:
Inverted T waves ECG were not always suggest myocardial ischemia, but also identified in other cases, such as Hypertrophic cardiomyopathy (HCM). HCM is the most common non-ischemic cardiomyopathy, characterized by increased left ventricular wall thickness ≥15 mm.

Case illustration:
A 49-year-old male came to Emergency Department complaining of shortness of breath, fatigue with marked limitation of physical activity. History of syncope or palpitations was denied. There was no history of sudden cardiac death in family member. The blood pressure was 120/80 mmHg, pulse rate 72 bpm reguler, respiratory rate 22 x/min. ECG findings were sinus rythm with inverted T wave in almost leads, especially giant inverted T-wave in leads V4-V6. The echocardiography showed apical left ventricular wall thickness 16mm without systolic anterior motion (SAM), diastolic dysfunction grade I , normal systolic function (LVEF 67%) that suggest apical HCM. The patient discharged after treated several days and prescribed bisoprolol 1.25mg once daily. The patient also recommended echocardiography evaluation follow-up every 6 month or any progressivity of symptoms. Family screening echocardiography was suggested for the other family member.

Conclusion:
Giant inverted T-wave is the classic finding in suggest apical HCM. Echocardiography continues to be the foundational imaging modality for patients with HCM.

Keywords: Hypertrophic Cardiomyopathy, Inverted T Wave

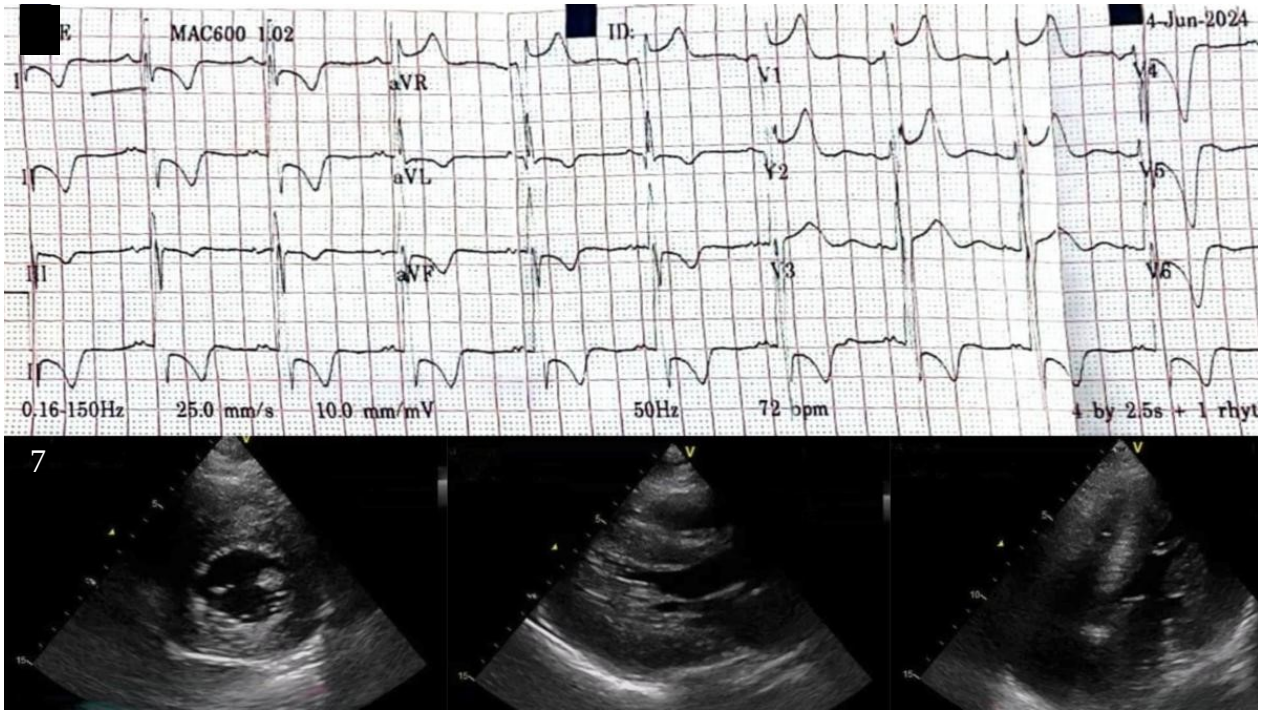

Figure 1. **A.** ECG shows sinus rythm, voltage criteria for left ventricular hypertrophy with *"Giant inverted T-wave"* **B.** Echocardiography of Hypertrophic Cardiomyopathy (HCM).

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CARDIOMYOPATHY WITH TOTAL ATRIOVENTRICULAR BLOCK IN CHILDREN

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Background:
Cardiomyopathy with Total Atriovetricular Block (TAVB) in children is a rare case and sometimes asymptomatic that can lead to serious complications and even sudden death. Cardiomyopathy causes damage to tissue around the heart, as well as heart muscle cells. In severe cases the heart becomes to weakness that it can't pump blood properly and can lead to heart failure, irregular heartbeats (arrhythmias). Cardiomyopathy often occurs in children with Dilated Cardiomyopathy (DCM), incidencies in the United State of America (USA) is 1 of 100,000 children per year aged <20 years, in Indonesia is still unclear but it is believed that the incidency will be greater than in the USA.

Case illustration:
A 13 year old girl was taken to the Emergency Department (ED) with chief complaint of difficult carrying out daily activities. There is history of the mother's sudden death at the age of 45. On Vital Sign, Blood pressure within normal limit, pulse 40 bpm. Electrocardiography (ECG) showed TAVB accompanied by Right Axis Deviation (RAD). Physical examination was within normal limits. Chest X-Ray showed cardiomegaly. Echocardiography showed LV dilatation, Global hypokinetic, LVEF 45%, with conclusion is cardiomyopathy, Laboratory result within normal limit. She given antibiotic therapy, steroid, loop diuretic, MRA, low dose ACE-Inhibitors. Working diagnose is Cardiomyopathy with TAVB DD Suspect Myocarditis, then consulted to the arrhythmia division for planning to install a pacemaker. On second day of treatment, the patient underwent Permanent Pacemaker (PPM) DDDR setting 80-150 ppm, the ECG results after PPM showed a pacing rhythm at 95 bpm. On the third day of treatment, the patient complaints was improved and she was moved to a non-intensive room. On the fifth day, the patient was able to go home with well condition.

Conclusion:
Cardiomyopathy and TAVB are two types of dangerous diseases and it can cause sudden death, especially if these two diseases appear simultaneously or related to each other, it will certainly increase mortality. Pacemaker installation and heart failure management immediately to prevent sudden cardiac death.

Keywords: Cardiomyopathy, Total Atrioventricular Block (TAVB)

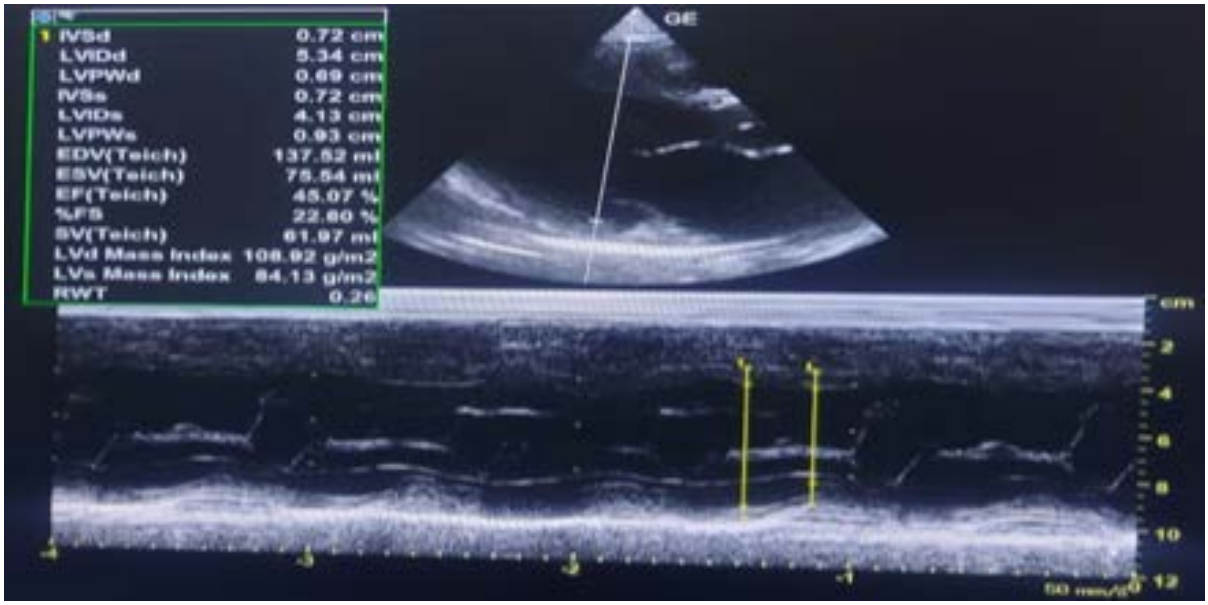

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Figure 1. Echocardiography: Dilated LA, RA, LV, Eccentric LVH, Global hypokinetic, Reduce LV systolic LVEF 45 %.

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SUPRAVENTRICULAR TACHYCARDIA TRIGGERED BY GASTROCARDIA SYNDROME IN YOUNG WOMEN WITH HISTORY OF GASTROESOPHAGEAL REFLUX DISEASE: A CASE REPORT

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Background:
Gastrocardiac syndrome is a gastrointestinal-symptoms that associated with cardiac arrhythmias secondary to foregut stimulation. Supraventricular tachycardia (SVT) is a narrow complex dysrhythmia which origin above or at AV node. Gastroesophageal reflux induced autonomic imbalance associated with vagus nerve stimulation or local inflammation nearby of the left atrium due to the anatomical location of the left atrium near to the esophagus.

Case illustration:
A young woman, 21 years old, presented with complaints of heartburn since approximately 2 days before and vomiting over 7 times a day. She had a previous history of GERD. She had been brought to the ER and received gastric medications. The complaints had decreased a while and she was given home medication. The complaints worsen with heartburn began to radiate to the chest accompanied by a feeling of tightness since the morning. Vomiting became more frequent and she began to feel chest palpitations. her state when she arrived was appeared conscious and looked pale with cold sweat. Vital signs were obtained with BP 96/58 mmHg, SpO2 93-93% and pulse 154 bpm. The pulse was fast, weak, and regular. ECG examination showed supraventricular tachycardia. The patient was fitted with a monitor, not long after she seemed agitated thus she was diagnosed with unstable SVT. Her family refused cardioversion. The patient received oxygen, intravenous fluid loading 250 cc followed by maintenance, and 1 ampoule of fargoxin injection. She also received an extra injection of farmabes 1 ampoule. About 10-15 minutes after giving extra farmabes, she seemed more stable with consciousness increasing to compos mentis, but her BP reached 208/124 mmHg with a pulse 129 bpm, and SpO2 96% on NC 3 lpm. The patient was admitted to the HCU for 3 days. Patient's and her family history of heart disease, diabetes, or thyroid disorder was denied. There were no abnormalities in routine blood tests, electrolytes, blood sugar, and thyroid function.

Conclusion:
Tachyarrhythmias in young adults need further consideration as to the cause. In patients with no cardiac history and risk factors, gastrointestinal symptoms often experienced in young women may trigger arrhythmia.

Keywords: Supraventricular tachycardia, Young adult, Gastroesophageal Reflux, Gastrocardia Syndrome

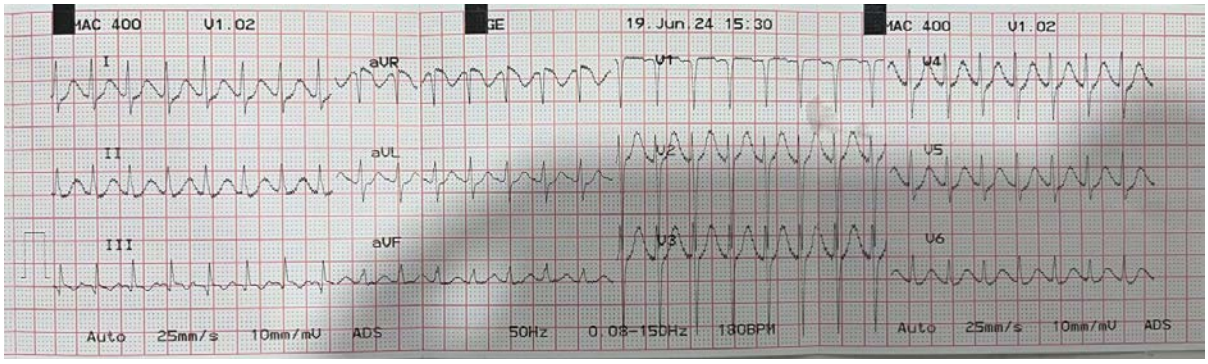

Figure 1. Patient ECG showed Supraventricular tachycardia with HR 188 bpm

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THROMBOLYTIC THERAPY IN PATIENT WITH STEMI ANTERIOR POST CARDIAC ARREST, CAN DO?: A CASE REPORT

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Background:
Thrombolytic after cardiopulmonary resuscitation (CPR) in patients with acute ST-segment elevation myocardial infarction (STEMI) is controversial. The recommendations of the American College of Cardiology and the American Heart Association refer to the thrombolytic therapy trialists study showed that thrombolytic therapy during and in the early phase after CPR has been contraindicated because of the risk of bleeding. We present case report describes a thrombolytic after resuscitation.

Case illustration:
A 60- years- old female came to the emergency room with decreased consciousness. She did not respond to the calls, there was loss of carotid pulsation. cardiopulmonary resuscitation was performed immediately. After successful resuscitation, ECG showed ST elevation in leads V2 to V6, I AvL was significantly elevated. The patient was diagnosed with extensive anterior STEMI. The patient received full dosed streptokinase. Two hours of post thrombolytic with ST-segment regression which indicated that the thrombolytic effect was successful. Norepinephrine injection was maintained and withdrawn for 2 days, and oxygenation was good with ventilator support. No bleeding was observed during or after thrombolysis, and the patient's neurological function recovered well.

Conclusion:
Thrombolytic in the early phase after cardiac arrest in patient return of spontaneous circulation (ROSC) and indicating acute myocardial infarction can be carried out, especially in certain conditions where it is not possible to make a referral and there are limited facilities. In this case report, thrombolytic performed after CPR provided good outcomes, including no risk of bleeding and the patient's neurological function recovered well. Accuracy and immediate decision making influence the success of thrombolytics in patients who are not transportable for Primary Percutaneous Coronary Intervention (PPCI).

Keywords: Cardiac Arrest, Thrombolytic, STEMI, Primary Percutaneous coronary Intervention

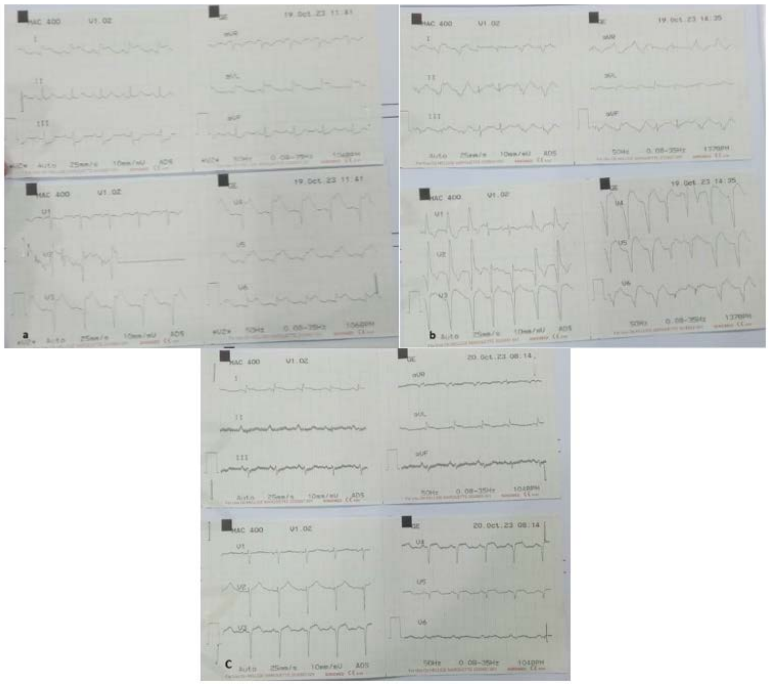

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Figure 1. A. ECG before Thrombolytic (ST elevation in leads V2 to V6, I, AVL). B. ECG after Thrombolytic (Sinus Rhythm with tachyarrhythmia induced RBBB). C. ECG 20 hours after Thrombolytic (Sunys Rhythm 98 bpm, with pathological Q wave in leads V3-V6, I, AvL, and decreased ST elevation in leads V2-V6, I, AvL).

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ANTIPLATELET THERAPY MANAGEMENT IN NSTEMI PATIENTS WITH THROMBOCYTOPENIA: WHAT IS THE RECOMMENDED THERAPY APPROACH?

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[1]RSUD Indrasari Rengat

Background:
Cardiovascular disease (CVD) is the most common cause of mortality globally. Cardiovascular disease encompasses various diseases, including acute coronary syndrome (ACS). Non-ST-segment elevation myocardial infarction (NSTEMI) is more prevalent than ST-segment elevation myocardial infarction (STEMI) among individuals with acute coronary syndromes of the myocardial infarction type. The purpose of this case report is to investigate the clinical presentation of individuals diagnosed with NSTEMI and the therapeutic therapy of NSTEMI patients who also have thrombocytopenia.

Case illustration:
A 70-years-old male who has been experiencing symptoms of heartburn since yesterday afternoon. The discomfort of heartburn radiated to the chest. Previously, patients frequently reported experiencing dyspnea after physical exertion. Based on the patient's anamnesis, physical examination, and supporting tests, the patient has been diagnosed with NSTEMI (Non-ST Segment Elevation Myocardial Infarction) with a GRACE Score of 153. Additionally, the patient has CHF (Congestive Heart Failure) classified as NYHA IV, Acute Left Ventricular Failure, and Thrombocytopenia. The patient's diagnosis of non-ST segment elevation myocardial infarction (NSTEMI) was confirmed not only by the study of the ECG, but also by the elevated levels of Troponin I-3, specifically at 1.00ng/mL. Antiplatelet therapy is a viable treatment option for patients with thrombocytopenia in ACS cases. A thrombocytopenia count of less than 100x10^9/L, specifically 50x10^9/L, indicates that a monotherapy of clopidogrel and PPI is advised. On the fourth day of treatment and follow-up, the patient suffered from respiratory arrest and cardiac arrest. Despite undergoing five cycles of CPR, the patient did not show any signs of response. Five minutes later, the patient was pronounced dead.

Conclusion:
The rarity of the NSTEMI with thrombocytopenia case presentation should be the primary focus to increase knowledge and provide a practical guidance on the appropriate antiplatelet therapy for NSTEMI patients with thrombocytopenia, who are susceptible to bleeding complications based on the latest studies and evidences. According to the ESC, it is advisable to administer antiplatelet monotherapy and a proton pump inhibitor in these patients to minimize bleeding risk.

Keywords: Acute Coronary Syndrome, Thrombocytopenia, NSTEMI, Antiplatelet

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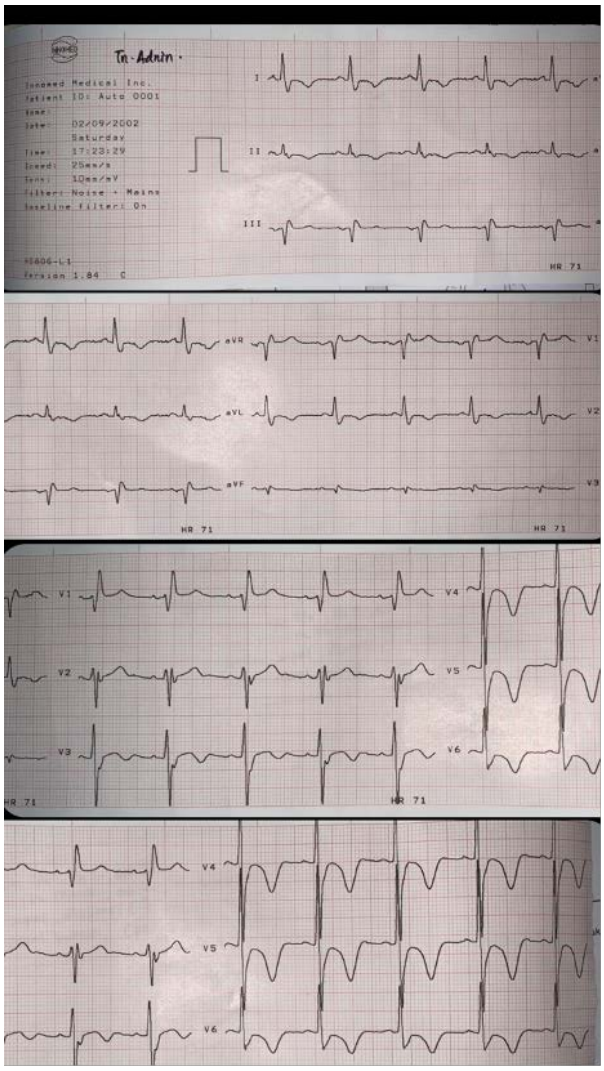

Figure 1. ECG in NSTEMI patient

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FINDINGS OF DE WINTER PATTERN COVERING AN INFERIOR STEMI: A CASE STUDY

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[1]Rumah Sakit Jantung Diagram

Background:
The De Winter sign is a unique ECG pattern distinguished by tall, symmetrical T waves in the precordial leads, upsloping ST depression at the J point, and the lack of ST elevation. This pattern is commonly seen in individuals with acute anterior wall myocardial infarction (STEMI), necessitating urgent reperfusion treatment. In contrast, inferior STEMI is a subtype of acute coronary syndrome (ACS) characterized by obstruction of the right coronary artery (RCA) or left circumflex artery (LCx), which affects the heart's inferior wall.

Case illustration:
A 53-year-old guy presented with recurring retrosternal chest discomfort and cold chills, which were consistent with ACS. The patient was referred with signs of an acute myocardial infarction, including radiation to the left jaw and arm. On admission at Emergency Room, the patient's ECG revealed sinus rhythm, slightly ST segment depression, and hyperacute T waves in V2-V4, indicating the Winter sign. Subsequent tests indicated a severe stenosis in the distal LCx. Immediate reperfusion treatment with a drug-eluting stent in the LCx produced good results.

Conclusion:
The de Winter sign is a specific ECG pattern indicating acute LAD occlusion in ACS, characterized by upsloping ST-segment depression in leads V1-V6 without ST elevation, accompanied by tall T waves in precordial leads. It is equivalent to STEMI and requires immediate reperfusion therapy. In contrast, inferior STEMI involves RCA or LCx occlusion with ST elevation in II, III, and aVF. Differentiating these patterns is crucial for prompt ACS management.

Keywords: Inferior STEMI, Acute Coronary Syndrome, De Winter, ECG

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DESCENDING AORTIC PSEUDOANEURYSM AFTER PATENT DUCTUS ARTERIOSUS LIGATION IN A CHILD: A RARE CASE

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Background:
Aortic pseudoaneurysm is a life-threatening complication after Patent Ductus Arteriosus (PDA) closure with only a few cases reported in literature. This article aims to report a rare case of descending aortic pseudoaneurysm after PDA ligation in pediatric setting.

Case illustration:
. A twenty-two-month-old child with a known case of PDA a 4 mm undergoing PDA ligation five weeks prior, presented to our hospital with fever for 2 consecutive weeks, shortness of breath, and cough. The prior post-operative outcome was uneventful, with no complication reported. Based on vital signs, temperature was 39^{o} Celsius, respiratory rate was 40 beats per minutes, heart rate was 150 beats per minutes, and saturation oxygen was 87% in room air. Physical examination obtained chest wall retraction, crackles, and machinery murmur. Laboratory result revealed anemia, leukocytosis, and high C-Reactive Protein. Chest X-ray showed homogenous opaque shadow in the paratracheal to paracardial with suspected pulmonary abscess or even vascular abnormality. Contrast-enhanced chest multi sliced computed tomography confirmed saccular aortic pseudoaneurysm measuring about 7.18 x 6.72 x 5.63 cm. Echocardiography showed descending aortic pseudoaneurysm and residual shunt of PDA. After several days, machinery murmur was heard even louder at the third intercostal space to the right of sternal border. Recanalization of PDA was found from echocardiography evaluation. Sputum culture resulted in Acinetobacter baumanni bacteria. The patient was diagnosed with descending aortic pseudoaneurysm, recanalization of PDA, and sepsis with suspected pulmonary abscess. The patient was admitted to the Pediatric Intensive Care Unit. The management included adequate oxygenation using continuous positive airway pressure, antibiotic meropenem, paracetamol, captopril, spironolactone, inhalation therapy, and transfusion of packed red cells. Due to our hospital's limited resources, we discussed with National Cardiovascular Center Harapan Kita for further management. After clinical improvement, the patient was transferred for urgent surgery.

Conclusion:
Aortic pseudoaneurysm following prior PDA ligation is a rare complication with high mortality and morbidity. Any similar case needs to be documented for a better understanding in future cases.

Keywords: Descending Aortic Pseudoaneurysm, Patent Ductus Arteriosus

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THE IMPORTANCE OF PROVOCATIVE MANUEVERS IN HYPERTROPHIC CARDIOMYOPATHY (HCM) : A SERIAL CASE REPORT

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[1]Fakultas Kedokteran Universitas Diponegoro

Background:
Hypertrophic cardiomyopathy (HCM) was characterized by the presence of left ventricular (LV) hypertrophy in the absence of another cardiac or systemic etiology. It was a genetic condition with an autosomal dominant inheritance, affecting 1 in 500 individuals of the general population. Apart from myocardial hypertrophy, mitral valve and papillary muscles abnormalities had been well described in this condition. These structural changes could contribute to the degree of left ventricular outflow tract obstruction (LVOTO) and mitral regurgitation (MR).

Case illustration:
We had two cases of HCM. A 44-year-old woman with HCM and a 33-year-old woman with HCM and LVOTO. The first patient came with chief complaint of shortness of breath and the other came with chief complaint of palpitation which was found to be caused by rapid atrial fibrillation. Echocardiographic examination performed and identified septal wall thickness more than 15 mm in both cases. The first case showed LA, RA and RV dilatation, concentric LVH with septal hypertrophy 21 mm in thickness, severe mitral regurgitation (MR) with vena contracta 10 mm, SAM (+), dynamic LVOTO with resting LVOT peak gradient 19 mmHg and provoked LVOT peak gradient 73 mmHg. Dynamic LVOTO in this patient was exacerbated by Valsava maneuver. The second case showed LA dilatation, concentric LVH with septal hypertrophy 17 mm in thickness, mild mitral regurgitation (MR) due to prolaps of anterior mitral leaflet (AML), SAM (-), and no obvious LVOTO (resting LVOT peak gradient 10 mmHg and no obstruction of LVOT during systolic phase on Apical 5-chamber view). Unfortunately, we were unable to perform a provoked LVOTO examination due to the patient's condition.

Conclusion:
Various echocardiographic methods such as M-mode, two-dimensional echocardiography, color Doppler, and continuous-wave (CW) Doppler were employed to diagnose and assess the severity of LVOTO in individuals with HCM. For symptomatic patients whose resting LVOT gradient was less than 30 to 50 mm Hg, additional tests were conducted to provoke a higher LVOT gradient. Medical treatment was then initiated to alleviate symptoms related to LVOTO.

Keywords: Hypertrophic cardiomyopathy (HCM), Left ventricular hypertrophy (LVH), Left ventricular outflow tract obstruction (LVOTO), Mitral regurgitation (MR), Echocardiography

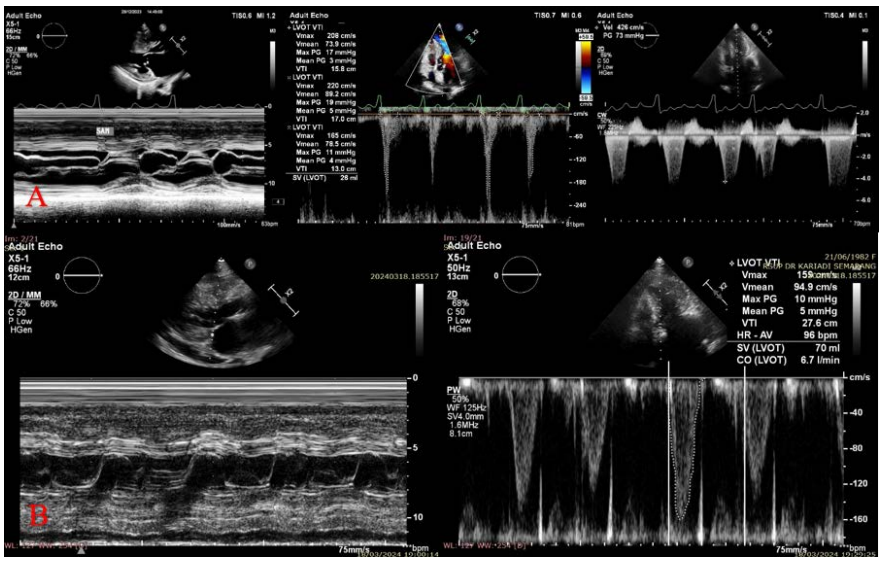

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Figure 1. (A) Echocardiographic finding of case 1 with SAM (+) and dynamic LVOTO with LVOT peak gradient during resting (19 mmHg) and Valsava manuever (73 mmHg) and (B) case 2 with SAM (-) and resting LVOT peak gradient (10 mmHg)

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A UNIQUE CASE OF AORTIC REGURGITATION MIMICKING ACUTE CORONARY SYNDROME

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Background:
Aortic regurgitation (AR) is a valvular heart disease characterized by backward blood flow from the aorta into the left ventricle during diastole. Without intervention, AR can cause significant morbidity and mortality, presenting with chest pain and dyspnea. Causes include rheumatic heart disease (RHD) leading to dilatation of the ascending aorta. We describe a 63-year-old man initially suspected of acute coronary syndrome (ACS), later diagnosed with AR and treated accordingly.

Case illustration:
A 63-year-old male patient diagnosed with STEMI was referred to the Cardiac Center H. Adam Malik Hospital for percutaneous coronary intervention (PCI) strategy. At our hospital, the patient presented with dyspnea, diaphoresis and vomiting. Vital signs showed a blood pressure of 166/95 mmHg, heart rate of 95 beats per minute, and respiratory rate of 24 breaths per minute. Physical examination revealed a diastolic murmur, 3/4 in intensity, best heard in the upper right sternal border. Laboratory results showed thrombocytopenia (133,000 /µL), hypokalemia (3.27 mmol/L), and troponin-I within normal limits (0.03 ng/mL). ECG showed left axis deviation, left atrial enlargement, left ventricular hypertrophy, and ST-segment elevation in anterior leads. Chest X-ray showed cardiomegaly and aortic dilatation. Coronary angiography revealed non-significant lesions. A transthoracic echocardiography revealed reduced ejection fraction (EF) of 36%, global hypokinesia, eccentric left ventricular hypertrophy, moderate AR due to RHD (AR VC: 0.6cm; AR PISA 0.6cm PHT 474ms; AR EROA 0.2cm^2 AR RVol 34 ml), and severe mitral regurgitation (MR VC 0.5 cm, MR EROA 0.4 cm^2, MR Rad 0.6 cm, MR RVol 66 ml, MR Vmax 5.57 m/s, MR VTI 188.7 cm). Thoracoabdominal CTA revealed dilatation of the aorta. The patient was diagnosed with heart failure with reduced EF, moderate AR due to RHD and severe MR. The patient was started on heart failure medications and prepared for the Bentall procedure.

Conclusion:
We report a case of AR mimicking ACS with chest discomfort and dyspnea. ST-T changes on ECG, suggesting myocardial ischemia, often lack specificity and can resemble ACS. This report highlights the importance of thorough examination when evaluating chest pain to ensure the best patient outcomes.

Keywords: Aortic Regurgitation, Acute Coronary Syndrome, Heart Failure, Echocardiography

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WHEN CHEST PAIN STRIKES: IDENTIFYING AORTIC DISSECTION VS. ACUTE CORONARY SYNDROME – A CASE REPORT

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[1]andalas university
[2]Universitas Andalas
[3]Andalas University

Background:
Acute aortic dissection is a serious emergency often linked to hypertension (72%) and atherosclerosis (30%). It is more common in Western countries (5,200 cases per 100,000) and in men (3:1 ratio). In Asia, the incidence is lower, with 2.8 cases per 100,000 in China. Ascending dissections occur in those aged 50-60, while descending dissections are more common in those aged 60-70. Men are twice as likely to experience aortic dissection as women. Symptoms range from mild to severe chest pain, and about 15% of cases are initially misdiagnosed, often as acute coronary syndrome (ACS). This report discusses a 73-year-old man with persistent chest pain, initially diagnosed with unstable angina, later confirmed to have an aortic dissection. It highlights the need to distinguish between aortic dissection and ACS in patients with acute chest pain.

Case illustration:
A 73-year-old man presented with seven days of sharp, persistent chest pain radiating to his back, unrelieved by rest, and accompanied by cold sweats. Initially treated for unstable angina at a private hospital, he was referred for further evaluation. His vital signs were stable: BP 148/74 mmHg, HR 75 bpm, RR 22 bpm, SpO2 98% on 2L nasal cannula. Exams showed normal heart sounds, bilateral crackles, anaemia, and hypocalcaemia. An ECG indicated left ventricular hypertrophy with inverted T waves, and a chest X-ray showed mediastinal widening and bilateral infiltrates. Elevated D-dimer levels also suggested aortic dissection. Echocardiography showed reduced left ventricular ejection fraction (40%) and hypokinesia in several segments, with minimal pericardial effusion but no clear aortic dissection. CT scan confirmed descending aortic dissection (Stanford type B, DeBakey III) with thrombus and atherosclerosis in the aorta and iliac arteries. The patient was managed with medications, including beta-blockers, calcium channel blocker, diuretics, MRA, and analgetics. The patient is scheduled to undergo thoracic endovascular aortic repair as the definitive treatment.

Conclusion:
Differentiating between ACS and aortic dissection is crucial due to their overlapping symptoms but different treatments. This case underscores the importance of distinguishing aortic dissection from ACS in patients presenting with acute chest pain. Aortic dissection typically presents with sudden severe chest pain radiating to the back, while ACS usually manifests as uncomfortable chest pain. Thorough history taking, physical examination, and imaging are vital for accurate differentiation. Early and accurate diagnosis using CT angiography is crucial for effective treatment and reducing complications.

Keywords: acute chest pain, aortic dissection, acute coronary syndrome

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RECURRENT INFECTIVE ENDOCARDITIS AS A COMPLICATION OF PATENT DUCTUS ARTERIOSUS IN YOUNG WOMEN WITH POOR DENTAL HEALTH

Billy J. Tando[1], R. Sukarya[1]

[1]RSUD Ciawi

Background:
Infective Endocarditis (IE) is infection of endocardium, often including cardiac valves. IE is uncommon with recurrence rate of 3% per year. Therefore we present this case.

Case illustration:
A 17-year-old female presented with intermittent fever for two-weeks and minor respiratory distress. She was diagnosed with Patent Ductus Arteriosus (PDA) and IE four months prior and successfully treated with antibiotic, then scheduled to Harapan-Kita's Hospital for surgical intervention. While she waited, she began to experiencing the same symptoms as before. Before, she failed to undergo a tooth extraction procedure as result of her low blood pressure. She was mildly lethargic, with blood pressure of 90/60 mmHg, heart-rate of 105 bpm, respiratory-rate of 23 per-minutes and temperature of 38^{o}C. She had 3/6 continuous murmur at left sternal border. Laboratories showed Hb 7.7 g/dL, WBC 14.100 /uL and CRP 57.7 mg/dL. Transthoracic-echocardiography (TTE) showed vegetation at pulmonary valve (Figure-1) and L-R Shunt PDA. It would be challenging to diagnose IE because the presentation could mimic other infectious disease. Modified-Duke's criteria for IE was used with 1 major (TTE finding for vegetation) and two minor finding (PDA and fever). She had recurrent IE which episodes of relapse/reinfection caused by the same organism or different microorganism detected within the first six-months after completing the initial treatment. PDA is the main risk factor for this case, with prior episodes of IE and poor dental health. Standard treatment for two-week duration with ceftriaxone and gentamisin was chosen because she has no allergic to penicillin, normal renal function and suspicion of involvement of oral Streptococci group. Later we referred the patient for surgical intervention because enlargement of the vegetation to 17x10 mm diameter. Patient education on the risk of recurrence and preventive measures, with emphasis of dental health and based on individual risk profile is recommended during follow up.

Conclusion:
Risk factor management for IE is essential and it should remain under close surveillance for potential long-term complications. Partnership between cardiologists, infectious disease specialists, cardiac-surgeons, general practitioners, and dentists is encouraged to improve patient care and reinforce prophylaxis measures.

Keywords: infective Endocarditis, Patent Ductus Arteriosus, Dental Health

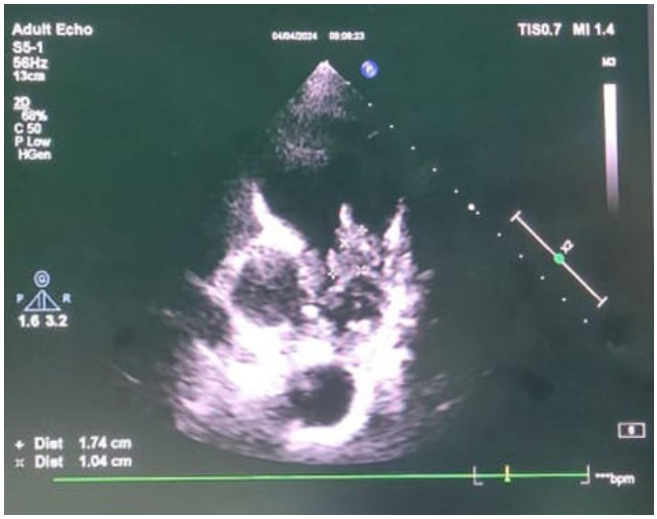

Figure 1. Vegetation Finding with Transthoracic Echocardiography

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A CASE OF ONE-MONTH REHOSPITALIZATION FOR ACUTE HEART FAILURE IN MEDICATION NONCOMPLIANT PATIENT WITH SEVERE AORTIC STENOSIS

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[1]RSU Bunda Margonda

Background:
Medical therapy is imperative for all heart failure patients with severe aortic stenosis to optimize one's quality of life regardless of the choice of therapy, which includes aortic valve replacement or palliative care. Therefore, medication adherence and routine monitoring by cardiologist is crucial. In this report, we present a case of a 73-year-old female with heart failure caused by severe aortic stenosis, who was on palliative approach yet had poor adherence, being rehospitalized within one month.

Case illustration:
A 73-year-old female was brought to our emergency department with chief complaints of worsening dyspnea over four days and swollen lower limbs. She was just being admitted one month before her presentation with acute heart failure, severe aortic stenosis, and unstable angina, and was favored for palliative care upon discharge. However, she did not take her medication regularly and missed follow-up visits. She presented with blood pressure of 106/65 mmHg, respiratory rate of 22 breaths/min., and oxygen saturation of 94% room air. Electrocardiogram showed sinus tachycardia with heart rate of 107 beats/min., old myocardial infarction in the anteroseptal leads, and left ventricular hypertrophy. Laboratory investigation showed stage 2 chronic kidney disease with eGFR of 69.2 mL/min/1,73 m^2 and mild hyponatremia of 134 mmol/L. Chest x-ray demonstrated cardiomegaly, pulmonary edema, pneumonia, and right pleural effusion. Her past echocardiogram revealed severe aortic stenosis (low flow, low gradient), left ventricular eccentric hypertrophy with diastolic dysfunction, left ventricular ejection fraction of 28%, and good right ventricular contractility. She was directed to the cardiology service and diagnosed with acutely decompensated chronic heart failure, coronary artery disease, and pneumonia. She was treated with furosemide, clopidogrel, atorvastatin, spironolactone, and started on bisoprolol once deemed euvolemic. On the second day of care, she was hypotensive with blood pressure of 86/57 mmHg without hypoperfusion and was managed with dobutamine. Consultation to pulmonologist was made, and she was given ceftriaxone and thoracocentesis was performed. She improved after one week of therapy and then discharged with guideline-directed medical therapy.

Conclusion:
Medical noncompliance is associated with a high risk of one-month readmission for acute heart failure in patients with severe aortic stenosis.

Keywords: severe aortic stenosis, noncompliance, rehospitalization, acute heart failure

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PULMONARY EMBOLISM MIMICKING ACUTE INFEROANTERIOR STEMI: A RARE CASE REPORT

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[1]RSUP DR M DJAMIL PADANG

Background:
ST segment elevation is a common electrocardiogram (ECG) sign of acute transmural myocardial elevation. Symptoms of pulmonary embolism (PE) and acute myocardial infarction can be similar. ST segment elevation is a rare ECG finding in acute Pulmonary Embolism

Case illustration:
A-58-Years Old Female Present with chest pain and shortness of breath since 15 hours before admission. From physical examination revealed blood pressure 105/78 mmHg, regular heart rate 93 beats/min, respiratory rate 26 times/min, oxygen saturation 98% and temperature 37o C. In Cardiac auscultation was heart pansystolic murmur at Left lower sternal border RIC 3th-4th. In ECG showed sinus rythm with Right axis Deviation, Q- pathologis with ST segments elevation 1-3 mm in III, aVF, V3R-V4R and V1-V6. Coronary Angiography was performed and showed non significant lession at Left Anterior Descending Artery. In the next day Transthoracic echocardiography revealed dilatation of Right atrium and right ventricle, decreased TAPSE, plethora IVC, elevated of pulmonary arterial pressure and positive McConnel's sign. D-dimer examination value was 8635 ng/mL and dopler vascular revealed deep vein thrombosis at level poplitea sinistra. Computed tomography pulmonary angiography concluded thrombus at bilateral pulmonary artery. Patient was treated with subcutaneus anticoagulant and initiation of oral anticoagulant before discharge. Patient was discharged with stable condition

Conclusion:
Pulmonary Embolism can be difficult and challenges to be diagnose. The symtomps and ECG in pulmonary embolism can be mimicking of acute transmural myocardial infarction. Multiple modalities was needed to determined diagnostic of pulmonary embolism.

Keywords: Pulmonary Embolism, Acute transmural myocardial Infarction

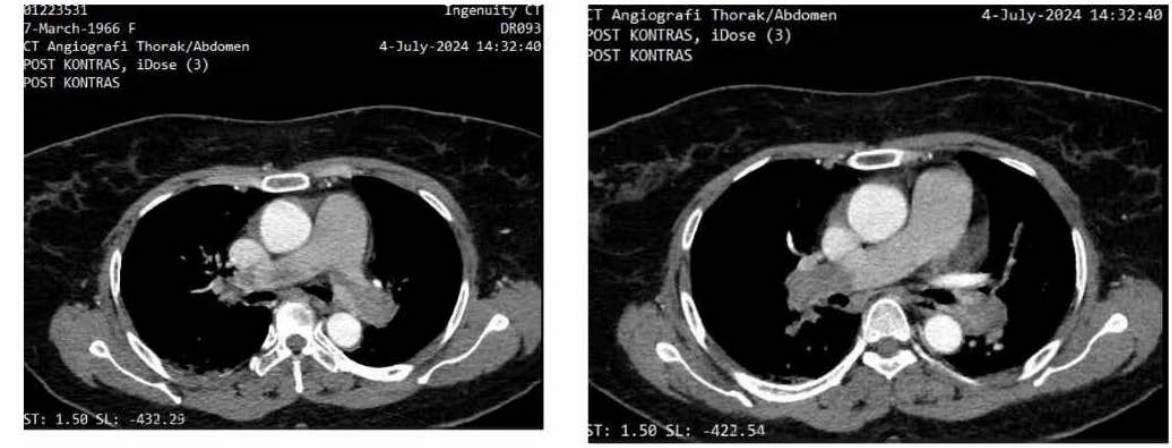

Figure 1. Thrombus at bilateral pulmonary artery

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A CASE SERIES OF WELLENS SYNDROME: AN OMINOUS SIGN OF LEFT ANTERIOR DESCENDING ARTERY CRITICAL STENOSIS

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Background:
The pre-infarction stage of coronary artery disease is known as Wellens syndrome. Acute anterior wall myocardial infarction (MI) will occur shortly from high-degree stenosis of the proximal left anterior descending (LAD) coronary artery. This condition is indicated by T-wave abnormalities (biphasic T wave or deep T inverted) on a patient's ECG during painless period. Based on de Zwann et al 1982 and Haines et al 1983, patients with Wellens syndrome who don't receive invasive strategies within 1 week will end up with anterior MI.

Case illustration:
First Case: We reported a 70-year-old male with intermittent right chest pain for 1 hour. The patient had a history of hypertension for 10 years. Vital signs indicated stable. ECG showed deep inverted T waves in leads V2-V6 and II. In echocardiography concentric LV remodelling, with grade I LV diastolic dysfunction, LV EF 48%, mid-anteroseptal and anterior basal hypokinetic. The patient was carried out elective PCI with the results of one culprit lesion in the LAD, 80-90% proximal-distal stenosis. Final results showed TIMI flow III with 1 Drug Eluting Stent (DES) in the LAD. Patient concluded with Wellen type B.Second Case: 44 year-old female with recurrent chest pain that has worsened in last 10 days. The patient had a history of chest pain and improved with nitrates 2 years ago. History of hypertension for 12 years and type 2 diabetes for the past 2 years. The patient's vital signs showed blood pressure 176/92. ECG showed biphasic T waves in V1-V3. Echocardiography examination EF 56%, TAPSE 17 mm. Coronary angiography showed one vessel disease with subtotal occlusion at proximal LAD with no thrombus, and 70% stenosis at mid RCA. 1 DES was implanted with a good result. With this patient having Wellen type A.

Conclusion:
Early recognition is crucial for clinicians to avoid the development of anterior MI. Immediate invasive strategy is needed.

Keywords: critical stenosis, LAD, typical T wave, Wellens syndrome

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CATHETER-DIRECTED THROMBOLYSIS IN CHRONIC THROMBOEMBOLIC PULMONARY HYPERTENSION: TRIALS OR TRIUMPHS?

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Background:
Chronic Thromboembolic Pulmonary Hypertension (CTEPH) is a rare and serious form of pulmonary hypertension characterized by chronic obstruction of the pulmonary arteries due to unresolved blood clots leading to progressive damage to the heart and lungs. CTEPH treatment varies from anticoagulants, Balloon Pulmonary Angioplasty (BPA), and surgical Pulmonary Endarterectomy (PEA). Thrombolysis is generally not the treatment of choice in CTEPH due to the nature of the thrombus and can only considered as an adjuvant therapy along with mechanical or surgical intervention. This study presents a case of progressive CTEPH that underwent catheter-directed thrombolysis (CDT) as a last-resort given the limitations of other treatment modalities.

Case illustration:
A 53 year old woman was diagnosed with CTEPH and received oral anticoagulant therapy for 6 months. Despite anticoagulant treatment, the patient experienced a downward trend in peripheral oxygen saturation. An imaging evaluation showed a progressive thrombus in the proximal main pulmonary artery accompanied by findings of UIP pattern interstitial lung disease (ILD), a form of ILD that relentlessly progressive, and usually leading to respiratory failure and death without a lung transplant. A comprehensive evaluation was then carried out to look for other etiology of the CTEPH progression, however both the presence of malignancy and autoimmune related disease showed negative results. Heart team discussions were carried out and the choice of surgical intervention by PEA was hampered by many factors, including the readiness of personnel and facilities and financial issues. With various considerations, it was decided to perform CDT on the patient by administering Alteplase to the pulmonary artery, but unfortunately there was also no significant clinical improvement after the procedure. The patient finally succumbed to the disease after 31 days of treatment by the multidisciplinary team.

Conclusion:
The treatment algorithm for CTEPH employs a multimodal strategy that address the different anatomical lesions. In proximal obstruction, surgical PEA remains the treatment of choice in operable patients with accessible PA lesions. An expert multidisciplinary team including an experienced surgical team is mandatory. In the absence of these requirements, other strategies such as thrombolysis can continue to be investigated for their role in inoperable CTEPH.

Keywords: CTEPH, Catheter-Directed Thrombolysis, Interstitital Lung Disease, Pulmonary Endarterectomy

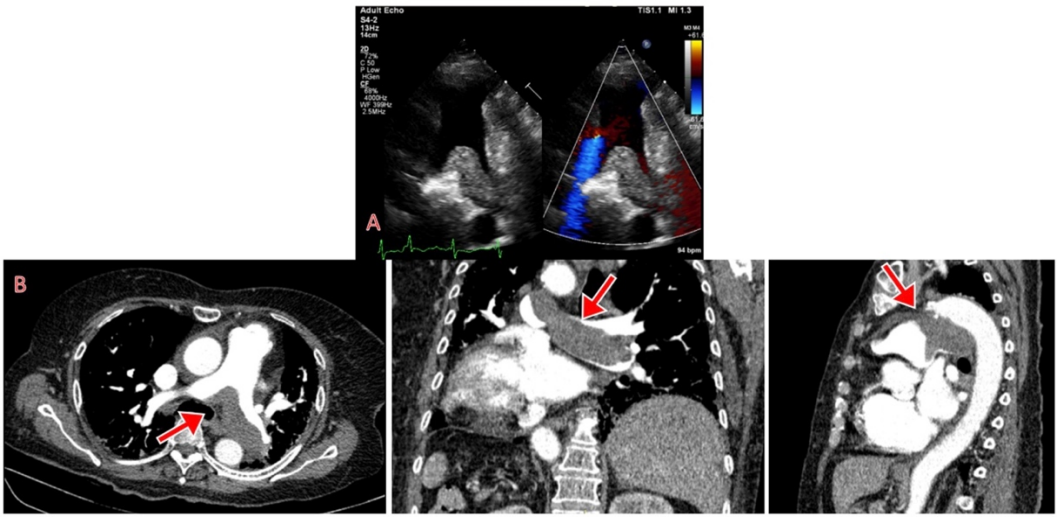

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Figure 1. (A) Thrombus seen in main pulmonary artery and both branches on transthoracal echocardiography; (B) Thrombus seen in proximal pulmonary artery in computed tomography (CT) evaluation

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PATENT DUCTUS ARTERIOSUS-ASSOCIATED INFECTIVE ENDOCARDITIS COINCIDENT WITH PULMONARY TUBERCULOSIS: AN EXTREMELY RARE CASE

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Background:
Patent ductus arteriosus (PDA) left-to-right shunt (L-R) occurs when the ductus arteriosus, a small hole between the aorta and pulmonary artery, does not close after birth. Untreated congenital heart defect (CHD) is a risk factor for infective endocarditis (IE). The risk of IE in the CHD population from birth to 18 years old is 4.1/10,000 people per year, especially in cyanotic conditions. PDA-IE is a rare event since advanced interventions are combined with early antibiotic therapy. Tuberculosis is a complication that occurs 2.5 times more often in someone with CHD compared to the normal population and is more often found in cyanotic conditions. This clinical condition has significant adverse effects if not treated appropriately. The recommended treatment is early surgery plus long-term therapy (6 weeks) with a combination of beta-lactam bactericides and aminoglycosides. Early diagnosis with transthoracic echocardiography and antimicrobial therapy for IE is recommended to minimize multiorgan failure and severe pulmonary embolism.

Case illustration:
A 25-year-old woman with CHD; PDA L-R came with a high fever for almost a month accompanied by coughing. Echocardiography results showed vegetation around 1.3x0.5cm. The patient underwent blood cultures and found positive growth with the *Acinetobacter baumannii* and the antibiotics ampicillin-sulbactam and gentamicin were administered. The chest x-ray showed cardiomegaly and a right perihilar nodule suspicious for tuberculoma with the results of a negative rapid molecular test and acid-fast bacilli test. She was given antitubercular drugs (ATD) based on clinical and radiological diagnosis. A similar case occurred in a 9-month-old baby girl who received combination surgery therapy with antibiotics for PDA-EI and ATD. Another similar case of PDA-EI without tuberculosis in a 33-year-old woman was successfully cured with antibiotics alone.

Conclusion:
Closure of large, hemodynamically significant PDAs requires minimal intervention and can be performed carefully and efficiently with either surgical or transcatheter procedures. Malformation repair and combination with proper antibiotics are recommended to eliminate or reduce the possibility of IE. Tuberculosis in this case could be associated with complications of but could also be a coincidence considering the patient had acyanotic congenital heart disease.

Keywords: patent ductus arteriousus, infective endocarditis, tuberculosis

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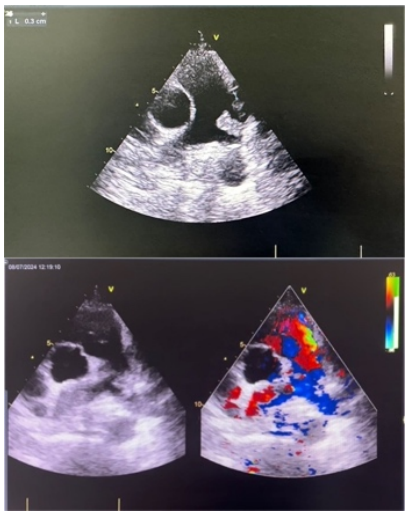

Figure 1. Endocarditis vegetation size 1.3x0.5cm and jet PDA in patient echocardiography

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DEEP VEIN THROMBOSIS IN LUNG CANCER PATIENTS: UNRAVELING THE MALIGNANCY-THROMBOSIS CONNECTION - A CASE REPORT

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[1]RSUD Kraton

Background:
Deep vein thrombosis (DVT) is a significant clinical concern, often associated with malignancies such as lung cancer. Understanding the interplay between thrombosis and cancer is crucial for effective management and improved patient outcomes.

Case illustration:
A 39-year old male presented with unilateral left leg swelling for three days. He also reported low-grade fever, cough, and shortness of breath persisting for a year. He denied chest pain, recent major surgery, prolonged immobilization, and had no history of diabetes or hypertension. He had a long history of heavy smoking, consuming 1-2 boxes of cigarettes per day for over 10 years. Physical examination revealed a fever (38 C) and notable swelling and tenderness of the entire left limb, with prominent superficial veins and a positive Homans sign. Chest x-ray revealed right perihilar reticular opacity suggestive of a lung tumor with a pneumonia reaction, Laboratory tests indicated leukocytosis and elevated D-dimer levels (3066 ng/mL). Color Doppler ultrasound of the leg confirmed acute DVT in the common femoral and popliteal veins. Contrast-enhanced chest CT showed a unilateral left pulmonary hilar tumor. The patient was diagnosed with DVT, scoring 6 points on Wells's score. Anticoagulant therapy was started with low-molecular-weight heparin (LMWH) for five days, showing a good response, and was eventually discharged on oral anticoagulation. The hypercoagulable state associated with neoplastic diseases like lung cancer significantly predisposes patients to thrombotic events such as DVT. Cancer can lead to a prothrombotic environment through mechanisms, including the release of procoagulant factors by tumor cells, activation of coagulation pathways, and inflammatory responses.

Conclusion:
This case underscores the critical association between DVT and lung cancer, highlighting the need for vigilant assessment of thrombotic events in cancer patients. Early detection and appropriate anticoagulation therapy are pivotal in managing DVT in the context of malignancy, improving patient prognosis.

Keywords: Deep vein thrombosis, Anticoagulation, thrombosis, lung cancer

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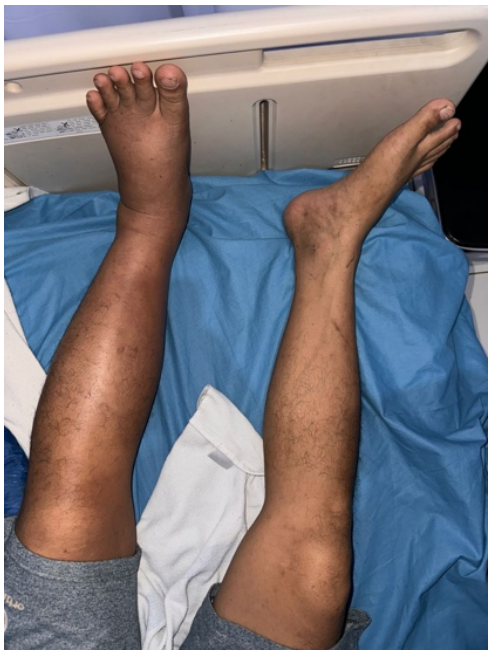

Figure 1. Unilateral leg swelling with prominent superficial veins

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SUDDEN CARDIAC DEATH IN MITRAL VALVE PROLAPSE: WHAT ARE THE CLUES?

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Background:
Mitral valve prolapse (MVP) is a common valvular abnormality characterized by displacement of one or both mitral valve leaflets into the left atrium during systole. However, the outcome may vary greatly, with complications ranging from mitral regurgitation and atrial fibrillation to ventricular arrhythmias leading to sudden cardiac death (SCD).

Case illustration:
A 20-year-old male came to the clinic with complaints of fatigue, atypical chest pain, and occasional palpitations. He took no medication and had no previous hospitalizations. The physical exam revealed normal vital signs and a low body mass index (BMI) of 12.5 kg/m2. On auscultations, a midsystolic click occurs just prior to grade 3 systolic murmur, best heard over the apex. Handgrips maneuvers during dynamic auscultation accentuated murmurs.The ECG showed incomplete RBBB and inferolateral T wave inversion. The echocardiogram revealed a normal EF, along with 6 mm thickened mitral valves, 3 mm billowing of both leaflets into the left atrium during systole, displaced mitral annular, and moderate mitral regurgitation. He was scheduled to undergo Holter monitoring. However, the patient passed away from a cardiac arrest at home three months later.

Conclusion:
MVP is an underestimated cause of SCD. Echocardiography remains the cornerstone for diagnosing high-risk MVP and monitoring its progression. The presence of arrhythmogenic substrates, such as inferior depolarization abnormality, mitral annular disjunction, and severe myxomatous features might indicate a malignant course of the disease and an increased risk of SCD, independent of mitral regurgitation severity. Thus, it is crucial to promptly identify and vigilantly monitor high-risk MVP patients who may benefit from aggressive management strategies.

Keywords: Mitral regurgitation, Myxomatous disease, Sudden cardiac death, Mitral valve prolapse

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DOUBLE OUTLET RIGHT VENTRICLE WITH SEVERE COMPLICATION: A CASE REPORT

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[1]RS elizabeth Situbondo

Background:
Double Outlet Right Ventricle (DORV) is rare congenital heart disease where both aorta and pulmonary artery originate from the right ventricle. It is often associated with other cardiac anomalies. Radiological examination is essential to localize and characterize the type of congenital malformation. In this report, we present a girl with double outlet right ventricle who presented with dyspnea on exertion

Case illustration:
A 3 years old girl presented to emergency department with swelling all over the body since 5 days with no history like this before. In addition to her chief complaint, her mother also complained shortness of breath since yesterday with no history of fever, cough, and wheezing. The patient had no night sweats but had complained loss of appetite, vomiting, and weight loss. Patient has not been able to walk properly, cannot speak fluently, and had history of incomplete immunization. The clinical examinations show blood pressure was 90/50 mmHg, heart rate of 90 beat per minute, a respiratory rate of 30 breath per minute with oxygen saturation was 84% in the room air. There was swelling in all of her limb and eye lid. Slightly ascites was found in abdominal examination. the laboratorium examination showed albumin 3.31gr/dL, urinary examination showed protein urine +1(30), leukosit +10-12 therefore initial diagnose was suspect nephrotic syndrome.Chest radiograph showed cardiomegaly with aorta dilatation accompanied by pulmonary hypertension and congestive pulmonary. hence echocardiogram was advised with cardiology consultation, which revealed right atrium and right ventricle dilatation. Defect Intact Ventricular Septum with diameter 0,66cm. The aorta and pulmonary artery empty to right ventricle. Overriding aorta 50%. Moderate-severe tricuspid regurgitation was found with pulmonary hypertension. Multiple thrombosis in right atrium. patient received sildenafil 3x4mg and was advised to refer to bigger hospital.

Conclusion:
DORV is complex form of congenital cardiac heart disease. Early diagnosis, appropriate management and surgical intervention, vigilant post operative care and on going follow up will be essential for the outcome and reduce mortality rate.

Keywords: VSD, DORV

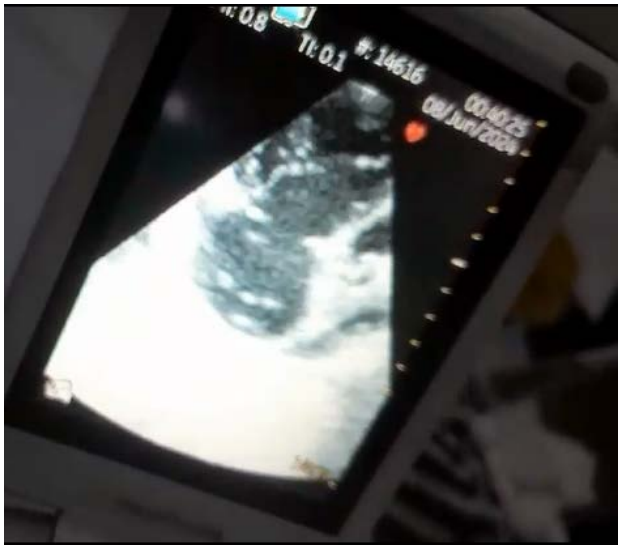

Figure 1. Chest Xray showed cardiomegaly with minimal left pleural effusion

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ST ELEVATION IS NOT ALWAYS STEMI: BRUGADA ECG PATTERN IN SEVERE HYPERKALEMIA CASE REPORT

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Background:
Hyperkalemia is associated with various ECG changes. An ST-segment elevation in V1–2 due to hyperkalemia can be mistaken for acute myocardial infarction or the ST elevation associated with the Brugada electrocardiogram. These ECG changes can be challenging when the laboratory test results are unavailable. Severe hyperkalemia with a Brugada ECG pattern is related to malignant arrhythmias and mortality.

Case Illustration:
A 51-year-old man came to the emergency room, reportedly complaining of a fever for two weeks, accompanied by multiple itchy ulcers on his arms and legs. The patient also complained of pain in the epigastrium and shortness of breath for the past week. The patient has a history of diabetes but has never taken medication or controlled blood sugar. While in the emergency room, an ECG examination showed RBBB and coved ST elevation at V2-V3 with T inversion, which showed a Brugada ECG Pattern. Laboratory results in the emergency room showed potassium 7.7 mmol/L (3.5-5.5 mmol/L), BUN 121.7 mg/dL (6-20 mg/dL), and creatinine 22.04 mg/dL (<1.17mg/dL). After hemodialysis, the potassium value dropped to 5.0 mmol/L, and the ECG results showed that the sinus rhythm and brugada ECG pattern disappeared.

Conclusion:
ST elevation is not always caused by myocardial infarction. Electrolyte balance disorders must be considered. Patients with ECG ST elevation type Brugada pattern must be considered, whether it is Brugada Syndrome or Brugada Phenocopy, by investigating the history further. There are significant differences in management between the two. Brugada phenocopy can be resolved by eliminating the underlying cause, while symptomatic Brugada Syndrome requires ICD placement.

Keywords: Hyperkalemia, Brugada

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SINUS BRADYCARDIA IN YOUNG ADULT: UNCOMMON MANIFESTATIONS OF DENGUE FEVER

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Background:
Dengue myocarditis is considered an uncommon complication of dengue, although its reported incidence is likely an underestimation. In general, most cases of dengue myocarditis are self-limited, with only a minority at risk of progressing to heart failure. Some symptoms suggestive of cardiac involvement (such as chest pain, dyspnea, tachycardia,etc).

Case illustration:
A 35-year-old man was admitted to hospital reported having high fever, headaches, muscle pain, joint pain, nausea for five days without any chest pain. BP 120/80mmHg, HR 40-50x/min, RR 20x/min, SpO2 98% Room air. In ECG showed sinus bradycardia, 50x/m, normoaxis, ST elevation anterior with ST depression in multiple lead. Laboratorium findings were Hemoglobin 17.6 g/dl, hematocrit 50.2%, leukocyte count 6280 cells/µl, platelet 18.000/µl. Patient was assessed as dengue fever by internist. One day later patient feels chest pain and shortness of breath with HR 40-45 bpm. Patient was consulted to cardiologist and echocardiography was done with results as EF was 49%, dilated all heart chambers, global hypokinetic at rest, LVH eccentric hypertrophy with diastolic dysfunction due to constrictive impairment, MR mild ec functional, TR mild, low probability PH, good RV contractility, eRAP:8. The troponin I level was 2.72 ng/L. Because our hospital was not facilitated with Cath lab, we performed Coronary Computed Tomography Angiography but the result unremarkable. Patient was assessed as bradycardia ec Myocarditis Dengue. The patient was treated with Atropin Sulfat 3mg not responded then dopamine was start 5-10 mcg/kg/min with other symptomatic treatment. On the 3rd day of hospitalization after intiation of salbutamol, dopamin was stopped, On the 4th days patient discharged in stable condition.

Conclusion:
Dengue virus can have several atypical presentations, one of which is acute myocarditis. Fortunately, this complication is usually self-limited. Medical personnel must maintain a high index of suspicion for patients with epidemiological risk factors and presenting with symptoms suggestive of cardiac involvement (such as chest pain, dyspnea, tachycardia,etc.)

Keywords: dengue, myocarditis, bradycardia

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PERICARDIAL DECOMPRESSION SYNDROME: CATASTROPHIC COMPLICATION FOLLOWING PERICARDIOCENTESIS IN TUBERCULOUS CARDIAC TAMPONADE

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[1]RS UMMI

Background:
Pericardial decompression syndrome (PDS) is a rare but potentially fatal complication, characterized by a paradoxical hemodynamic deterioration after pericardial drainage. Effusion can occur due to pericardial conditions, such as tuberculous pericarditis, particularly in countries with a high tuberculosis burden like Indonesia, and 10% can develop into cardiac tamponade. We reported a case of PDS after pericardiocentesis in tuberculous cardiac tamponade.

Case illustration:
A 16-year-old female was admitted with pulmonary tuberculosis, presenting with symptoms such as cough, shortness of breath, weight loss, fever, anemia, and an enlarged heart. Transthoracic echocardiography revealed a significant pericardial effusion with impending cardiac tamponade. The patient underwent an emergency pericardiocentesis, evacuating 750ml of blood-stained pericardial fluid. The patient is continuously monitored in the ICU. Six hours later, the patient's condition deteriorated, and bedside echocardiography showed left ventricular function was severely impaired, leading to acute heart failure and shock. Despite our efforts, she passed away several hours later.
Tuberculous pericarditis is rare but can lead to serious complications, including cardiac tamponade. PDS occurs in 4.8% of cases after draining pericardial fluid and may cause acute heart failure. Echocardiography is crucial for recognizing PDS and should be readily available when PDS is suspected. The treatment for PDS is mainly supportive including close monitoring for the first 24 hours in the ICU, breathing support, inotropic, and aggressive heart failure treatment. Current guidelines advise against immediately removing large amounts of pericardial fluid to prevent PDS. Fluid should be removed until cardiac tamponade resolves, and prolonged drainage can be considered for gradual fluid removal.

Conclusion:
Clinicians must be vigilant about PDS. Gradually draining effusion and continuous monitoring using echocardiography is important. We recommend all clinicians, especially general practitioners in Indonesia, become familiar with basic echocardiography for monitoring heart function and collaborate with cardiologists when necessary.

Keywords: echocardiography, cardiac tamponade, pericardial effusion, pericardial decompression syndrome, tuberculous pericarditis

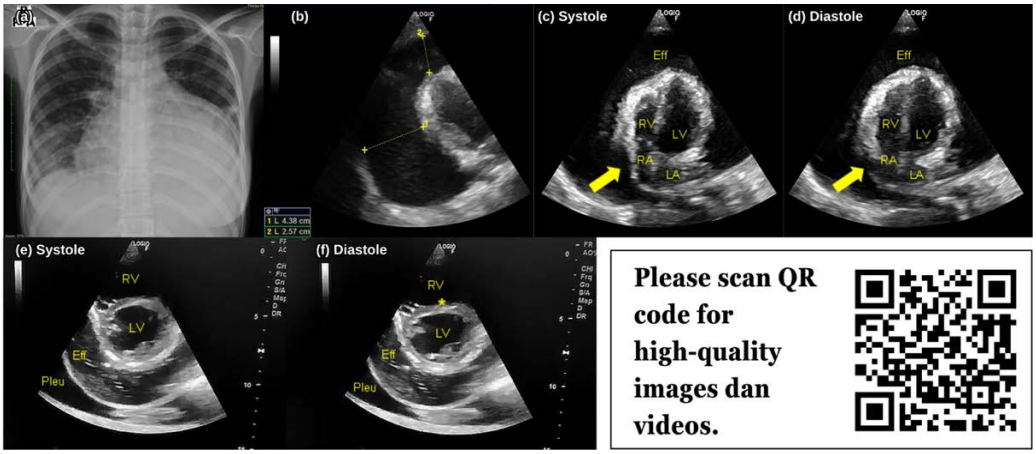

Figure 2. (a) Typical pulmonary TB with an enlarged heart (water bottle appearance). (b) Subxiphoid view shows large pericardial effusion. (c-d) Apical 4-chamber view demonstrating systolic right atrial collapse (yellow arrow) indicating cardiac tamponade. (e) & (f) Limited bedside parasternal short axis view performed by our general practitioner in ICU six-hours after drainage reveal residual pericardial effusion, dilated right ventricle with

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flattening of the interventricular septum (D-sign) in diastole indicating volume overload (marked by asterisk/*). Note that left ventricle also dilated and severely impaired in systolic function. (RV: Right Ventricle; RA: Right Atrium; LA: Left Atrium; LV: Left Ventricle; Eff: Pericardial Effusion; Pleu: Pleural Effusion).

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TRIPLE THREAT: AVNRT IN THE CONTEXT OF HYPERTHYROIDISM AND TYPE 1 DIABETES MELLITUS

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Background:
Hyperthyroidism is a common thyroid disease that can be accompanied by other autoimmune diseases, such as Diabetes Mellitus type 1, and is followed by atrioventricular nodal reentrant tachycardia (AVNRT). The simultaneous presence of various diseases poses distinct problems in terms of diagnosis and therapy, making them difficult to treat. Multiple modalities and comprehensive treatment will be needed.

Case illustration:
The patient complained of palpitations that started 24 hours prior to admission and intensified 6 hours later. The patient has been routinely experiencing palpitations for the previous decade and has experienced dyspnea within the past day, exacerbated by exertion. There is no history of paroxysmal nocturnal dyspnea, chest pain, fever, or coughing. The patient exhibits consistent tremors and has experienced significant weight loss in the past month. In addition, the patient has been suffering from nausea for the past two days accompanied by ongoing weakness. In physical examination, the patient was mildly agitated with blood pressure of 110/70 mmHg, heart rate of 187 bpm, respiratory rate of 24 bpm, a body temperature of 37 °C, and SpO2 97%. There was palpable diffuse thyroid gland enlargement. The electrocardiogram revealed atrioventricular nodal reentrant tachycardia (AVNRT) with a heart rate of 176 bpm. The laboratory tests results were; leukocyte count 18.4 x 10^3/µl, random blood sugar level 430 mg/dl, Sodium Level 130 mmol, HbA1c level 15.4%, FT4 level 3.79 ng/dl, and TSH level 0.01 mIU/ml. The patient was administered 5 mg of metoprolol maintenance with 20 mg of propranolol, and 10 mg of thiamazole every 8 hours and started the Yale Protocol. Metoprolol therapy converts the heart rhythm to normal sinus rhythm. The patient was discharged after a six-day hospitalized.

Conclusion:
This case showed the complex relationship between AVNRT, hyperthyroidism, and type 1 diabetes mellitus, which is challenging in both diagnosing and treating the conditions. The statement emphasizes the importance of using a comprehensive and multidisciplinary strategy to effectively deal with difficult problems. The success of treatment highlights the need for customized therapeutic approaches that address these complex coexisting medical conditions, with a focus on early intervention to improve patient outcomes.

Keywords: AVNRT, Hyperthyroidism, Diabetes Mellitus Type 1

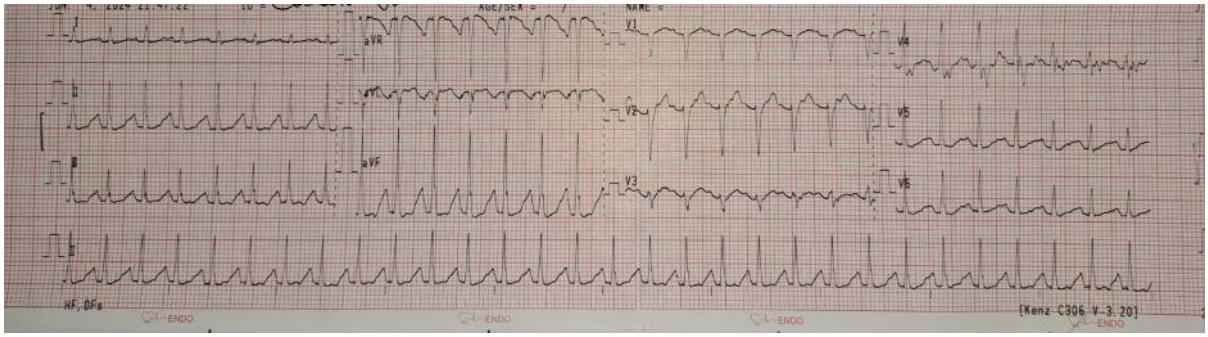

Figure 1. AVNRT ECG Before Metoprolol Administration

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THROMBOLYSIS AFTER CARDIOPULMONARY RESUSCITATION IN MYOCARDIAL INFARCTION COMPLICATED WITH VENTRICULAR FIBRILLATION

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[1]Panti Rahayu Hospital

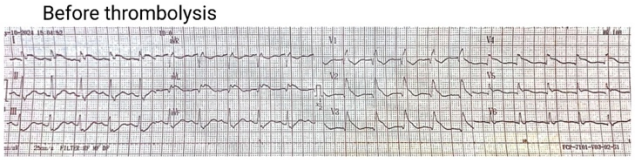
Background:
Thrombolysis after cardiopulmonary resuscitation (CPR) in patients with acute ST-segment elevation myocardial infarction (STEMI) is controversial. This case report describes thrombolysis after resuscitation in STEMI complicated with ventricular fibrillation.

Case illustration:
A 45-year-old man presented with an hour-episode of chest pain. Instantly after arriving at the emergency room, he suffered a sudden cardiac arrest with ventricular fibrillation on the monitor. CPR was immediately performed with repeated defibrillation, administration of epinephrine and amiodarone, and intubation. In less than 10 minutes, the patient returned to spontaneous circulation. Electrocardiography revealed a right bundle branch block (RBBB) with ST elevation in lead V2-V6, 1, and aVL. The diagnosis of anterolateral STEMI was made. The patient received streptokinase thrombolytic therapy despite the anticipated bleeding risk caused by traumatic CPR which is known as a relative contraindication of thrombolysis, considering several factors supporting thrombolysis for such patients, those are the younger age, the very early onset, and the fact that primary percutaneous coronary intervention (PPCI) was not available in a timely manner because of our rural area setting. Moreover, the patient had a higher risk of death shown by the RBBB morphology, and the possible benefit of thrombolysis might exceed the risk. Several minutes after finishing thrombolysis, he regained consciousness. With effective vasopressor, antiplatelet, heparin, and statin treatment, continued with diuretic, angiotensin-converting enzyme inhibitor, beta-blocker, and nitrate treatment, his general condition quickly improved. The patient was then referred to another city with a catheterization laboratory and treated with a drug-eluting stent (DES) in the left anterior descending artery and a DES in the left circumflex artery. The patient recovered pretty well with regional wall motion abnormality and left ventricular dysfunction on echocardiography.

Conclusion:
Thrombolysis after successful resuscitation is still an effective treatment for patients with STEMI. Prompt thrombolysis would lead to a better prognosis if spontaneous circulation can be restored within ten minutes.

Keywords: thrombolysis, cardiopulmonary resuscitation, acute myocardial infarction

Before thrombolysis



After thrombolysis

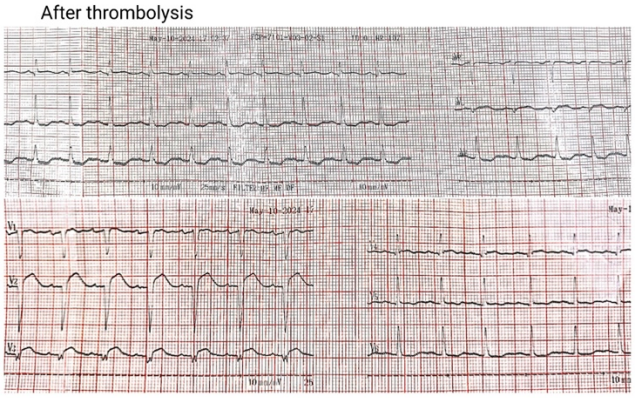


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Figure 1. Electrocardiography of the patient before and after thrombolysis

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DEHYDRATION-INDUCED ATRIOVENTRICULAR NODAL REENTRY TACHYCARDIA (AVNRT) IN A 1.5-YEAR-OLD BOY WITH SINUS VENOSUS ATRIAL SEPTAL DEFECT (SVASD) : A CASE REPORT

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[1]RS JHC Tasikmalaya

Background:
Supraventricular tachycardia is the most common type of arrhythmia seen in children. If prolonged, it may cause heart failure and progress to cardiogenic shock warranting prompt treatment.

Case illustration:
A 1.5 year-old presented to Emergency Departement with 3-day history of palpitation, intermittent vomiting 2 times/day and diarrhoea 2-3 times/day. He had decreased urine output, irritability, lack of water intake and nutrition without history of fever and prior congenital heart disease. On physical examination he had body weight was 10.9 kg, height 100 cm with tachycardia (173-290 bpm) and signs of moderate dehydration such as dry mucosa oral, decreased anterior fontanelle and skin turgor. At first auscultation, murmur was hardly identified. Laboratory findings were leukocytosis (16.20 $10^3/\mu L$), hypokalemia (3.0 mmol/L) and hyponateremia (132 mmol/L). An ECG demonstrated a narrow-complex tachyarrhytmia (RP interval< 70 ms) pseudo S in lead II, and pseudo R in V1 likely typical AVNRT (slow-fast) (Image 1). Valsava manuvers were unsuccessful, thus adenosine rapid intavenous (IV) with double syringe method dose of 0.05-0.1 mg/kg (0.55mg) administered but unsuccesfull in restored to sinus rhytm, after followed by two more doses of the same value, the arrhytmia terminated and the patient returned to sinus rhytm (149 bpm). After it terminated, on auscultation a wide fixed splitting S2 and a systolic ejection murmur was heard in left upper sternal border grade 2/6. He continued treatment with rehydration fluid, antibiotics, antipyretic, beta-blocker, probiotics also hypokalemia and hyponatremia were corrected. The patient underwent Transthoracic Echocardiogram (TTE) revealed an Atrial Septal Defect (ASD) type of Sinus Venosus 2 mm, L to R shunt. CXR revealed normal chest radiograph. The patient was transferred to PICU, after 4-days of hospitalization he discharged and maintained normal sinus rhytm without any cardiac events.

Conclusion:
SVASD with prolonged dehydration could triggered AVNRT due to electrolyte imbalance. Intravenous (IV) adenosine is the drug of first choice in hemodynamically stable patients if vagal manouvers fail in a young children with congenital heart disease.

Keywords: hypokalemia, SVT, Congenital Heart Disease, ASD

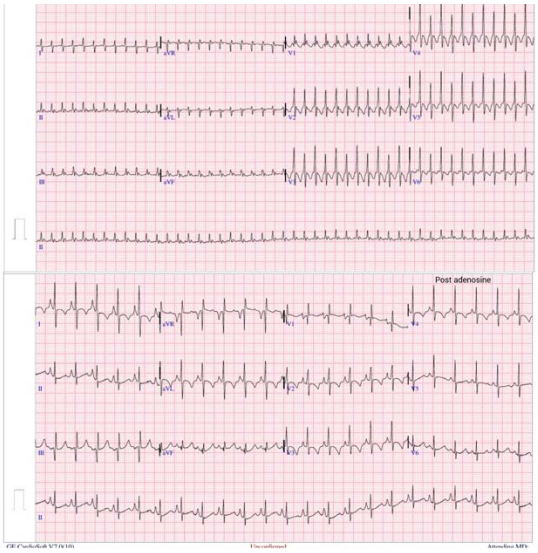

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Figure 1. Supraventricular tachycardia, RP interval< 50 ms pseudo S in lead II, and pseudo R in V1 likely typical AVNRT (slow-fast)

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WOLFF-PARKINSON-WHITE SYNDROME PRESENTING AS PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA IN FEBRILE PEDIATRIC PATIENTS

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[1]RSPAU dr. Suhardi Hardjolukito

Background:
Wolff–Parkinson–White (WPW) syndrome is a congenital abnormality which characterized by additional accessory pathway between atrium and ventricle, bypassing the AV node. In some cases, this abnormal electrical conduction remain unnoticed until it presents as potentially life-threatening arrythmias, such as Paroxysmal Supraventricular Tachycardia (PSVT), as the most common type seen in children. Fever could provoke the re-entry mechanism in WPW syndrome and in this case presenting as PSVT.

Case illustration:
A 14-year-old girl with a history of asthma was brought with shortness of breath, which had started 2 hours prior to the ER. She complained of fever, sore throat, and palpitation since the previous day. She recalled having history of palpitations associated with increasing activity since 1 year ago, which resolved spontaneously.
Upon admission patient was febrile with temperature 38.8ºC, tachycardia, blood pressure was 120/86, respiration rate 24-26 times per minute, saturation 98% with room air. The cardiac monitor showed narrow complex tachycardia with heart rate 210 bpm and an 12-lead ECG was performed afterward with SVT. Physical examination found tonsil hyperemia, pharyngitis, clear lung, and mild epigastric pain. The laboratory analysis showed mild hypokalemia and leukocytosis, suggestive acute tonsillopharyngitis as source of infection.
The patient received oxygen, IV line, and metamizole injection due to history of paracetamol allergy. Fever resolved but the monitor showed Premature Ventricular Contractions Bigeminy. Shortly after, the patient developed itchy rash followed by angioedema, lip swelling, and hoarseness, indicating anaphylactic shock. She was treated with IM adrenaline and epinephrine nebulization due to laryngeal edema. After stabilization, she was transferred to the ward for further observation and treatment.
Later the next day, after the fever had already subsided, a 12-lead ECG was performed. It showed normal sinus rhythm with delta wave, indicating WPW pattern, which was suspected as the cause of PSVT that presented in ER.

Conclusion:
We report a case of a girl with WPW, that initially diagnosed with PSVT. Fever may trigger the re-entry mechanism leading to PSVT in WPW syndrome. In this case, PSVT resolved when the fever subsided. Further evaluation and management should be done to prevent another episode of life-threatening arrhythmia.

Keywords: Paroxysmal Supraventricular Tachycardia, Wolff–Parkinson–White

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ANTIPLATELET AND ANTICOAGULANT STRATEGIES IN CHRONIC KIDNEY DISEASE PATIENTS WITH ACUTE CORONARY SYNDROME AT NON-PCI CENTRE HOSPITALS

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Background:
Patients with CKD are at a higher risk of ACS, significantly higher mortality, and increased bleeding problems. Multiple registries also reveal that medical therapy alone does not enhance the outcome of ACS patients with severe CKD. Antiplatelet and antithrombotic drugs are the cornerstone of ACS management but should be used with caution in patients with CKD

Case illustration:
A 69-year-old man with CKD reported left chest pain for three weeks, which was exacerbated within six hours before admission. Vital signs and cardiac examination were normal. A 12-lead ECG revealed a sinus rhythm of 85 BPM, normoaxis, and ST depression leads I, V4, V5, and V6, with pathological Q waves in leads III and aVF. Echocardiography revealed mild mitral and aortic regurgitation, with reduced ejection fraction (44.68%). Renal ultrasonography also detected contractions in the left kidney. The patient was given ISDN 5mg, Aspilet 80mg, Clopidogrel 300mg, Carvedilol 6.25mg, and Atorvastatin 40mg orally, as well as Furosemide 20mg and Heparin 4000IU intravenously followed by 750 IU/hour during 48 hours. Then, the patient was observed in the ICU for three days. After being stabilized, the patient got hemodialysis and was discharged after three days on the ward.

Conclusion:
According to the recent guidelines, there is no different medication and dose selection of antiplatelet and anticoagulant between CKD stage V and normal renal function. In this case, the selected management strategy involved categorizing patients based on the degree of CKD. However, there is insufficient evidence regarding the administration of clopidogrel and high-intensity statin in CKD stage V.

Keywords: Antiplatelet, Chronic Kidney Disease, Acute Coronary Syndrome, Anticoagulant

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A CASE REPORT OF FAILED THROMBOLYSIS AMONG INFERIOR STEMI PATIENT: RESCUE PCI, CONSERVATIVE TREATMENT OR REPEATED THROMBOLYSIS?

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[1]Rumah Sakit Pusat Angkatan Udara dr. Suhardi Hardjolukito

Background:
Thrombolysis remains the reperfusion strategy of choice for many countries treating patients presenting with acute myocardial infarction (AMI). But there are around 30% of patients with AMI fall into failed thrombolysis.

Case illustration:
A 52-year-old male came to emergency room with dyspnoea and diaphoretic an hour before admission. He has uncontrolled hypertension and he is an active smoker. Blood pressure (BP) was 141/88 mmHg and electrocardiogram (ECG) found ST segment elevation in inferior leads then he was given dual antiplatelet therapy (DAPT). CHADS-VASc and CHADSVASc-HS scores were 2 and 4, respectively. Thrombolysis using alteplase was performed. During thrombolysis, haemodynamic altered (BP 91/58 mmHg), ongoing chest discomfort and no resolution in ST segment elevation (< 50%). It was stated as a failed thrombolysis/reperfusion. He was transferred to percutaneous coronary intervention (PCI) centre for advanced management. Rescue PCI had been performed successfully to the patient and the result was successful thrombolysis at right coronary artery (RCA) and CAD2VD LM Disease Medina 1-1-0. Then he was treated with heparinization and DAPT. Thrombolytic therapy is still the first choice of AMI management at non-PCI centres in many regions, even though it doesn't give complete reperfusion. CHADS-VASc and CHADSVASc-HS scores can be useful in predicting failed thrombolysis, especially for those who work in non-PCI centres. In a randomized controlled trial study, rescue PCI after failed thrombolysis gave significantly higher event-free survival than conservative treatment or repeated thrombolysis. In 2017 ESC guideline, cases of successful thrombolysis are still recommended to transfer to PCI centre for following initiation (2 – 24 hours after thrombolysis). In cases of failed thrombolytic or reinfarction with recurrence STEMI, immediate angiography and rescue PCI is indicated. Repeated thrombolysis should be discouraged even if it is likely that it will be successful but an early angiography is recommended.

Conclusion:
In a region with nearest PCI centres that can be reached, a case of failed thrombolysis should be treated with rescue PCI rather than conservative treatment or repeated thrombolysis. Angiography evaluation is recommended for successful thrombolysis.

Keywords: myocardial infarction, conservative treatment, failed thrombolysis, rescue PCI, repeated thrombolysis

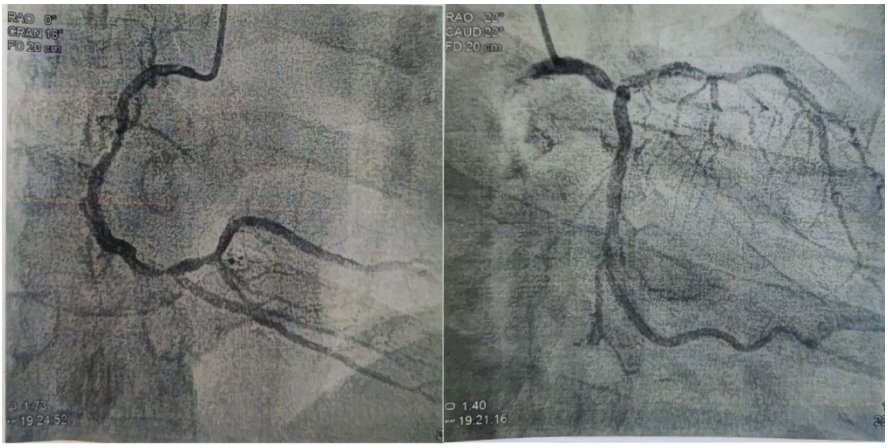

Figure 1. Coronary angiogram (CAG_ with fractional flow reserve (FFR) was performed and it showed midstenosis 50% (eccentric plaque) at LAD. Functional test FFR using adenosine was 1.0 (>0.8) so that the patient had CAD non-significant

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WIDE COMPLEX TACHYCARDIA: APPROACH TO MANAGEMENT IN EMERGENCY DEPARTMENT

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Background:
Wide complex tachycardia (WCT) refers to conditions of tachycardia >100 beats per minute (bpm) with QRS duration at least 120 milliseconds. Ventricular tachycardia (VT) accounts for 80% of cases and approximately 15% of WCTs had supraventricular tachycardia (SVT) with aberrancy. It is still challenging to quick and precisely diagnose WCT subtypes due to the numerous algorithms and intricate criteria that may be hard to recall in an emergency situation. Accurate diagnosis is crucial in distinguishing between VT and SVT with abberancy since they have distinct mechanisms and patophysiologies in order to obtain proper management.

Case illustration:
A 43-year-old man was presented to emergency department with recurrent and persistent palpitations after exercise. Physical examination were blood pressure of 97/65 (76) mmHg, heart rate of 190 beat per minute regular, respiratory rate was 18 times per minute with oxygen saturation 98% room air. Cardiac and pulmonary examination were within normal limits. Laboratory results showed renal insufficiency. Electrocardiography (ECG) revealed regular WCT, 190 x/minute, right axis deviation, left bundle branch block morphology with discordant precordial, no atrioventricular (AV) dissociation. Brugada, Vereckei, and RPWT algorithm were applied suggesting supraventricular tachycardia (SVT) with abberancy. Modified valsava manuever was failed to convert the rhythm. However after administered adenosine, the rhythm become sinus. Electrophysiology study and radio-frequency ablation was proposed. Unfortunately the patient refused. He was prescribed verapamil when severe palpitation occur.In regular WCT, tachycardia can be VT, SVT with bundle branch block, or antegrade conduction through bypass tract. Since ECG remains the primary modality for establishing the diagnosis of WCT, many algorithms have been proposed to help establish the diagnosis. If SVT with aberrancy was confirmed, it may be treated in the same regimen as narrow complex SVT, with vagal manuever or adenosine or AV node blocking agents.

Conclusion:
Accurate, non-invasive differentiation of WCT into SVT or VT is fundamental to delivers promp and appropriate therapy. Patients with unstable haemodynamic need synchronized cardioversion. However many patients presenting with stable WCT should be diagnosed carefully before giving therapeutic interventions. Proper recognition and management will help to treat SVT with abberancy effectively.

Keywords: wide complex tachycardia, supraventricular tachycardia, ventricular tachycardia, abberancy

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HEALING THE UNCOMMON, RECURRENT BLEEDING IN POST-TRAUMATIC LOWER EXTREMITY ARTERIOVENOUS MALFORMATION: A CASE REPORT

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Background:
Arteriovenous malformations (AVMs) occur congenitally in less than 2 cases per 100,000 person-years and typically remain asymptomatic until triggered by trauma. Post-traumatic extremity AVMs are extremely rare and frequently misdiagnosed. Even after proper diagnosis and treatment, they have a high recurrence rate.

Case illustration:
A 60-year-old male experienced painless blood leakage from his right knee. Two months prior, he had a closed right leg fracture and was treated with blood clot aspiration and a cast. Seven weeks later, blood leaked from the aspiration site, and debridement was performed. The patient was first clinically diagnosed as chronic venous insufficiency (CVI), hence the endovenous laser ablation (EVLA) and phlebectomy procedure was done at the previous hospital. Four days later, blood leaked from the EVLA puncture site, causing referral to our hospital and was suspected for lower extremity AVM. On admission, the patient had blood seeping from the right knee and contracture without leg edema. The patient experienced limited movement, tenderness, warmth, with adequate pulse on the right lower extremity. The patient had a history of hemangioma excision on his right leg at the age of 24. Veno-arteriography revealed an AVM in the popliteal, anterior and posterior tibial, and peroneal arteries, which was treated with embolization, then the bleeding resolved. Six months later, the patient had completely healed. AVM is a rare condition that can mimic the symptoms of other vascular diseases, resulting in a high prevalence of misdiagnosis (42.5%). The current treatment for AVM is embolization with or without surgical excision. However, the recurrence of AVM symptoms remains high (50%). Therefore, accurately diagnosing AVM and providing adequate treatment is very important.

Conclusion:
Accurate diagnosis and treatment of post-traumatic AVM are challenging, with high recurrence rates. Initially diagnosed with CVI, the patient experienced recurrent bleeding after EVLA and phlebectomy. After proper identification of the AVM, embolization of the feeding arteries was performed, leading to remission.

Keywords: Endovenous Laser Ablation, Phlebectomy, Post-traumatic arteriovenous malformation, Arterial Embolization

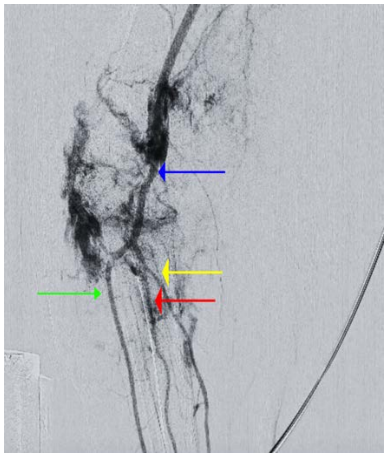

Figure 1. Right leg veno-arteriography. Feeding arteries forming AVM nidus in colored arrow; popliteal (blue), posterior tibial (yellow), peroneal (red), and anterior tibial (green).

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WHEN LEGS GIVE WAY AND THE HEART RACES: A CASE OF ATRIAL FLUTTER WITH LOWER LIMB PARALYSIS IN A YOUNG MALE PATIENT

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Background:
Atrial flutter may manifest in diverse clinical contexts, occasionally even presenting in uncommon situations such as lower limb paralysis caused by electrolyte imbalance. Research indicates that hypokalemia can significantly increase the risk of atrial arrhythmias. We present a thyrotoxic hypokalemic periodic paralysis case, characterized by periodic episodes of muscle weakness and concomitant hyperthyroidism.

Case illustration:
A 24-year-old soldier was admitted to the emergency department with an increasing palpitation, weakness in all four limbs, tingling sensations, and blurred vision that started 30 minutes prior. The patient had experienced similar episodes four times in the past year and was taking thiamazole, propranolol, and KCl. Upon physical examination, the patient was fully conscious with a blood pressure of 149/86 mmHg, irregular pulse rate, respiratory rate of 16 bpm, SpO2 of 98%, and tetraparesis 4444/1111. An EKG showed a supraventricular rhythm, a heart rate of 110 bpm, normoaxis, flutter waves with negative deflection in leads II, III, avF, and variable conduction. Echocardiography revealed an ejection fraction of 70% and grade 1 diastolic dysfunction. Laboratory tests showed a potassium level of 2.1 mmol/L, blood glucose level of 103 mg/dL, and free T4 >100 pmol/L. The patient was diagnosed with thyrotoxicosis periodic paralysis with typical atrial flutter. The patient underwent potassium correction, intravenous digoxin, furosemide, spironolactone, propranolol, and thiamazole. All symptoms resolved, and an EKG showed sinus rhythm with a heart rate of 88 bpm and normoaxis within a day.

Conclusion:
The rapid resolution of severe symptoms following targeted treatment highlights the importance of early recognition and comprehensive management of this complex condition.The association between thyrotoxicosis and atrial flutter underscores the importance of considering endocrine disorders in the differential diagnosis of cardiac arrhythmias, particularly in young male patients presenting with atypical symptoms such as periodic paralysis. A prompt and accurate diagnosis can restore the patient's functional status, leading to a sense of satisfaction for doctors, families, and patients. This is particularly true for emergency department physicians, as it fosters a sense of satisfaction and positively impacts the work atmosphere.

Keywords: Hypokalemia, Atrial Flutter, Thyrotoxicosis Periodic Paralysis

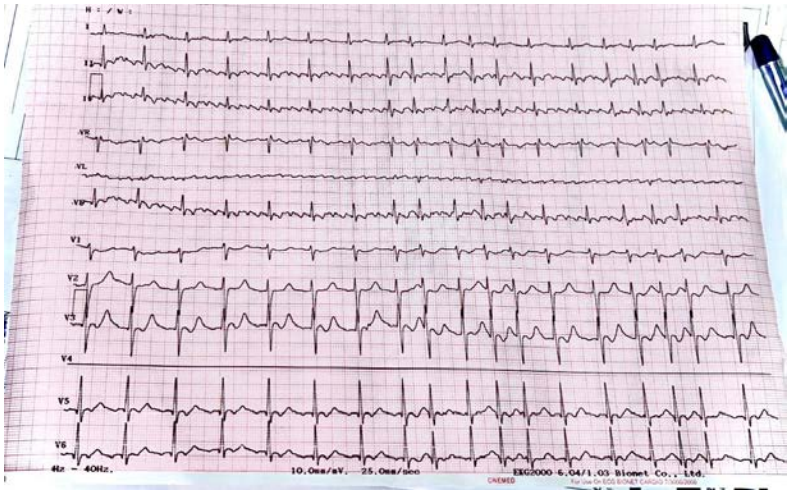

Figure 1. ECG on admission

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FREQUENT PREMATURE VENTRICULAR CONTRACTIONS DUE TO HYPOKALEMIA IN SUSPECTED HEMORRHAGIC STROKE PATIENTS AT REGIONAL GENERAL HOSPITALS WITH LIMITED SUPPORTING INVESTIGATION AND THERAPY OPTIONS : A CASE REPORT

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Background:
Premature Ventricular Contractions (PVCs) can be caused by low potassium levels in the blood, especially in patients with hemorrhagic stroke. Managing such cases in regional hospitals with limited resources is challenging. This case report explores frequent PVCs due to hypokalemia in a suspected hemorrhagic stroke patient, emphasizing diagnostic and treatment limitations in resource-constrained settings. Understanding this connection is crucial, especially in hospitals with restricted access to advanced medical care.

Case illustration:
A 37-year-old man with a history of uncontrolled hypertension presented with unconsciousness. Previously the patient complained of headaches and vomiting. Physical examination revealed high blood pressure, bradycardia, tachypnea, weak pulse quality, and desaturation. On physical examination, lateralization was found to the left. Electrocardiography results showed more than 5 PVCs per minute with frequent PVC effects. Laboratory results showed hypokalemia and leukocytosis. Chest x-ray were found segmental atelectasis in the right upper lobe of the lung. He was stabilized in the emergency unit with intubation, installation of a nasogastric tube and a dower catheter, as well as administering intravenous therapy of furosemide, nicardipine, lidocaine, and Potassium chloride. After stabilization, he was transferred to the intensive care unit for further management. The case report emphasizes the crucial link between electrolyte imbalances and cardiac arrhythmias in hemorrhagic stroke patients, especially in resource-limited settings. Managing such cases in regional general hospitals is challenging due to limited access to advanced diagnostic tools and treatments. Despite these limitations, timely recognition and correction of hypokalemia were essential in reducing arrhythmia frequency and stabilizing the patient. This case highlights the significance of basic electrolyte monitoring and management in improving patient outcomes and underscores the need for improved resource allocation and support for regional hospitals to handle complex cases involving metabolic and cardiac complications.

Conclusion:
This case report emphasizes the impact of hypokalemia on PVCs in a hemorrhagic stroke patient in a regional hospital with limited resources. Managing the patient's condition through electrolyte correction highlights the importance of monitoring and addressing imbalances in stroke patients and the need for improved resource allocation in regional hospitals.

Keywords: Hypokalemia, Stroke, Premature Ventricular Contraction

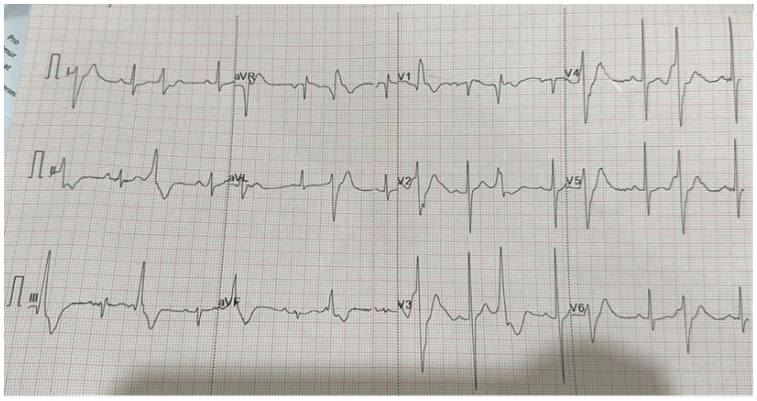

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Figure 1. Frequent PVC ECG

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THROMBOCYTOPENIA IN ATRIAL FIBRILLATION, IS THIS DIGOXIN-INDUCED THROMBOCYTOPENIA?

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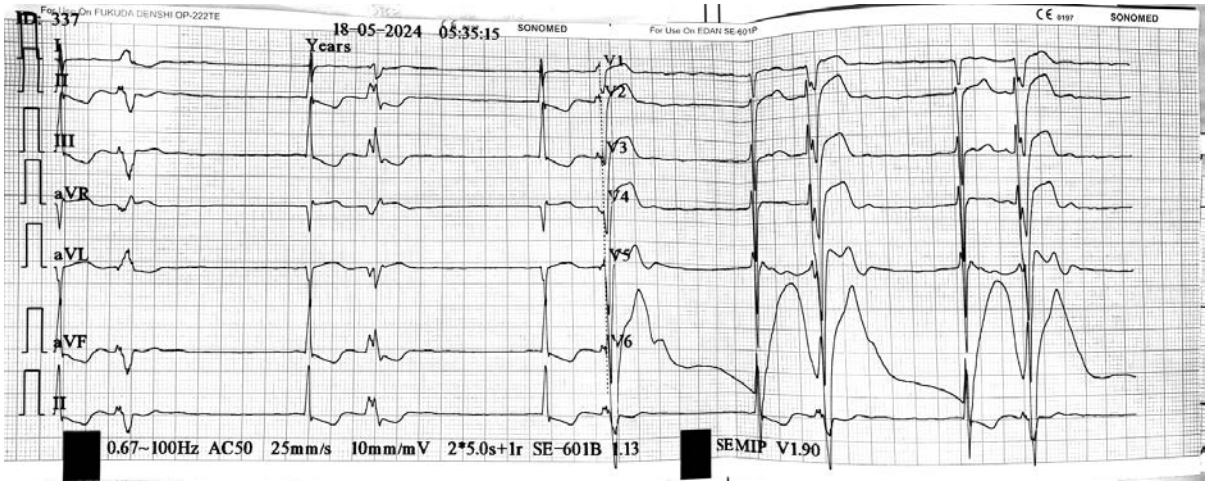
[1]RSUD Cicalengka

Background:
Long-term use of digoxin has been reported to cause thrombocytopenia, although it is very rare.

Case illustration:
A 68-year-old woman came to the emergency room with complaints of discomfort in epigastric with nausea for the last 1 month. The patient had a history of AF and heart failure with treatment with digoxin 0.25mg od, furosemide 40mg od, amlodipine 5mg od, spironolactone 25mg od, and acetylsalicylate 100mg od. Bilateral rhales were found, systolic heart murmur at the apex also bilateral pretibial oedema. Laboratory examination revealed platelets of 86,000 cells per mm^3. ECG revealed atrial fibrillation with SVR, right axis deviation, PVC bigeminy with CI ±520ms, digoxin effect sign, LVH, and persistent S in lead V5-V6.
Thrombocytopenia can be caused by various causes including autoimmune diseases, drug-induced thrombocytopenia, infections, chronic liver disease, blood disorders and cancer. However, suspicion of thrombocytopenia due to digoxin increases because of gastric complaints accompanied by digoxin effect. Digoxin has a very narrow therapeutic and toxic dose, also several factors can increase theoccurrence of digoxin toxicity such as hepatorenal disorders.

Conclusion:
Thrombocytopenia can be caused by digoxin by various mechanisms. Knowing the signs of digoxin toxicity will be very helpful in making a further diagnosis.

Keywords: digoxin toxicity, thrombocytopenia, digoxin effect, digoxin



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A CASE OF TYPE A AORTIC DISSECTION COMPLICATED WITH SUPERIOR MESENTERIC DISSECTION: CARDIAC CT BEYOND IMAGING

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Background:
Acute aortic dissection (AD) is a serious condition affecting the aorta causing significant morbidity and mortality. Symptoms often resemble those of acute coronary syndromes (ACS). Predisposing factors such as hypertension, atherosclerosis, and connective tissue diseases increase the risk of AD. A high degree of suspicion and imaging modalities such as CT scans play a pivotal role in diagnosis. We report a case of a 53-year-old man who was initially suspected with ACS. However, further investigations revealed an ascending and descending AD.

Case illustration:
A 53-year-old male patient, initially diagnosed with ACS was referred to the Cardiac Center H. Adam Malik Hospital and already given antiplatelet. The patient presented with acute epigastric pain described as a tearing sensation radiating to the patient's lower back, associated with cold sweat, nausea and vomiting. Initial vital signs showed blood pressure in normal limit, heart rate of 89 beats per minute. ECG shows ischemic in lateral leads. Lab results showed a slightly increased troponin. After we do transthoracic echocardiography showed a reduced LV systolic function, eccentric LVH, LA-RA dilatation, severe AR due to aortic aneurysm and dissection, MR moderate due to tenting, and intimal flap. An aortic CT was performed revealing an AD from the ascending aorta through to the descending aorta and superior mesenteric artery; classified as a Stanford type A (DeBakey type I) The patient was started on heart failure medications including a beta blocker to reduce heart rate with a target heart rate ≤65 x/i and prepared for the surgical procedure.

Conclusion:
We report a case of Stanford type A (DeBakey type I) AD with superior mesenteric artery dissection presenting with symptoms mimicking ACS such as epigastric pain and dyspnea and identified by CT. CT therefore plays a major role in determining important causes of acute chest pain.

Keywords: CT Aorta, Aortic Dissection, Superior Mesenteric Dissection

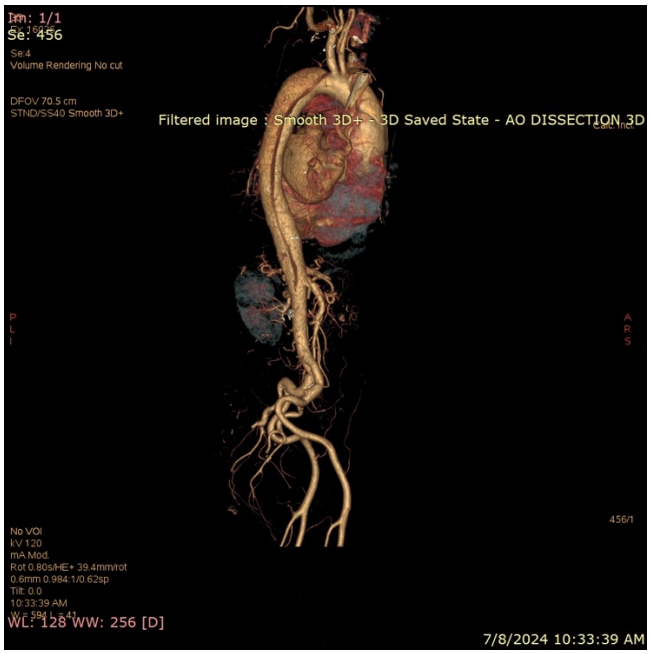

Figure 1. 3D volume rendering showed dissect at ascending aorta until superior mesenteric artery

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AN UNCOMMON CONTINUATION: BIATRIAL MYXOMA RECURRENCE IN AN 18-YEAR-OLD MALE POST-LEFT ATRIAL MYXOMA RESECTION

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Background:
Cardiac myxomas, a rare primary cardiac tumors, predominantly benign, were often removed due to its severe complications. Recurrence of myxomas after surgical intervention is very common, while biatrial reccurence is extremely rare. We report a case of biatrial reccurence of a myxoma in a patient who had undergone resection of a left atrial myxoma

Case illustration:
A 18-year old man with a history of left atrial myxoma presented with dyspnea on exertion, bloated abdomen and swelling in extremities for the last 1 month before hospitalization. He had undergone successful resection of the left atrial myxoma 3 years ago. He was asymptomatic until one year before current presentation. Physical examination revealed apical holosystolic murmur, tumor plop, ascites, and legs edema. Transthoracic echocardiograms and cardiac computed tomography images showed large mobile mass occupying almost the entire right atrium and left atrial mass that protruded to left ventricle causing severe mitral valve regurgitation (Fig.1). We planned to perform repeat resection surgery to removed both masses and to replace the damaged valves.

Conclusion:
The biatrial recurrence of previously resected cardiac myxomas are very rare. Myxomas predominantly present as benign tumors, but some may undergo malignant transformation, spreading to others. Complete surgical removal followed by chemoradiotherapy are needed to prevent recurrences.

Keywords: cardiac tumors, myxoma, heart failure, biatrial, cardiac surgery

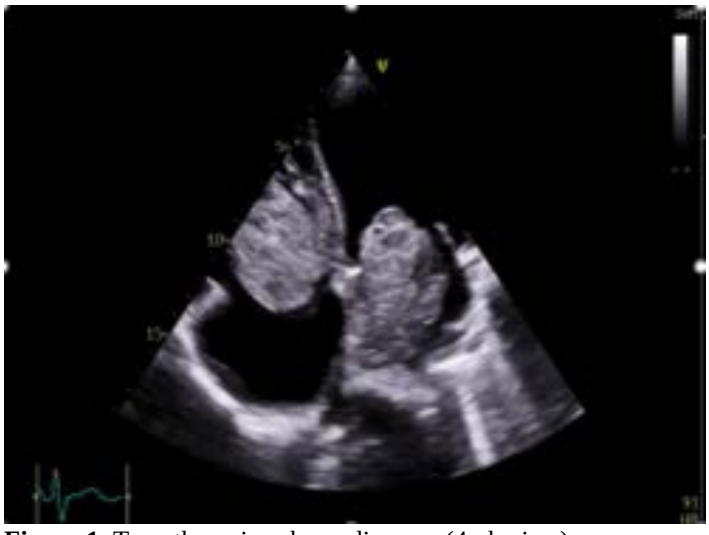

Figure 1. Transthoracic echocardiogram (4-ch view)

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CONQUERING OR DEFERRING? – A PERSPECTIVE ON VULNERABLE CORONARY PLAQUES MANAGEMENT

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Background:
Coronary plaques presenting as stable disease or asymptomatic were treated conservatively. A portion of these coronary plaques were considered “vulnerable” for rupture despite being non-flow-limiting in nature, and intervening them as a countermeasure act is debatable as previous studies suggested that there was no reduction of MACE risk from invasive procedures. This case series of silent vulnerable plaques aims to highlight clinical and CTCA features, and the final decisions in managing them.

Case illustration:
In Table 1, we presented four stable patients with plaque findings assumed as non-flow-limiting since they did not experience any ischemic symptoms. Plaque vulnerability was determined through features found in their CTCA, mostly being low-attenuation lesions with positive remodelling and or spotty calcifications. Patients with critical location of atherosclerotic lesions (pLAD and LM) and suspected significant stenosis (pLAD ≥70% or LM ≥50%) underwent invasive angiography to further establish the significance of these plaques (Case 1-4). Three of them (Case 1,2, and 3) were decided to undergo PCI guided with intravascular imaging, whilst one patient (Case 4) was deferred due to outweighed risk of complex PCI in the non-obstructive ostial LAD lesion. In Case 4 where angioplasty was deferred, conservative treatment with antiplatelet and aggressive lipid control were given to achieve LDL <55 mg/dL. Routine visits were always aimed for monitoring disease progression and looking out for alarming unstable cues such as decreasing physical capacity since anginal chest pain might not always be present.

Conclusion:
Coronary plaque vulnerability can be screened through features found in CTCA. With trials yet to conclude guidelines to manage these plaques, when vulnerable plaques are found with heavy prognostic value according to Duke Coronary Artery Index, invasive angiography may aid in deciding final management of either angioplasty or conservative with optimised medical therapy and close monitoring of disease.

Keywords: conservative, PCI, vulnerable coronary plaque, stable angina

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LATE ONSET OF TRIPLE ATRIAL TACHYARRHYTHMIAS AFTER ATRIAL SEPTAL DEFECT CLOSURE: A CASE REPORT

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Background:
Atrial Septal Defect (ASD) is the most common congenital heart disease in adults. Atrial arrhythmias are frequent in ASD patients, either before or after ASD closure, especially in patient who undergo surgical atrial septal defect closure. However, de novo atrial arrhythmia is rare in patients over 40 years of age following device closure of an atrial septal defect, while atrial arrhythmias are less common in those under 40 having percutaneous closure of an ASD.

Case illustration:
A 51-year-old woman with a history of transcatheter ASD closure by Amplatzer Septal Occluder (ASO) in 2005 presented with intermittent palpitations. One year ago, she was admitted with supraventricular tachycardia (SVT) and was prescribed beta-blockers. She denied several cardiac risk factors but was overweight (BMI 29.4 kg/m²). Vital signs were normal, except for an irregular and elevated heart rate (97 bpm). Holter monitoring showed several SVT episodes. In September 2023, an electrophysiology study (EPS) revealed atrial tachycardia (AT) and dual AV node physiology. Consequently, EPS with 3D activation mapping ablation was performed in February 2024. Initially, reverse typical atrial flutter was observed due to entrainment pacing showing PPI-TCL -2 ms at the CTI area. Ablation was performed, converting the rhythm to sinus. During the observation period, isoproterenol was injected, and atrial burst pacing was administered. Another AT was induced, with the earliest activation at CS 9-10, leading to ablation at the CS ostium. A third atrial tachyarrhythmia was induced by atrial entrainment pacing. Diagnostic maneuvers showed no reset, a VAV pattern, and a VA interval of 18 ms, indicating typical AVNRT; hence, slow pathway ablation was performed.

Conclusion:
Diagnosing and treating arrhythmia in ASD patients is crucial, especially those with multiple tachyarrhythmia mechanisms. Late atrial arrhythmias can develop due to age-related atrial remodeling, with large occluder sizes being a risk factor. Patients with large occluders relative to interatrial septal length and high shunt volumes are prone to these arrhythmias. Lifetime follow-up and screening for atrial tachyarrhythmia are essential after intervention. 3D activation mapping catheter ablation effectively ameliorates symptoms, reduces morbidity, and decreases the need for long-term anti-arrhythmic therapy and associated side effects.

Keywords: Amplatzer Septal Occluder, ASD Closure, Atrial Septal Defect, Triple Atrial Tachyarrhythmias, Catheter ablation

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TALE OF TRIUMPH: CONQUERING CHALLENGES IN SURGICAL ASD CLOSURE FOR A PATIENT WITH COMPLETE REMISSION FROM AIHA

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Background:
Autoimmune hemolytic anemia (AIHA) is a rare disorder in hematology with an incidence of 1-3 per 100,000 per year. It occurs when antibodies attack an individual's red blood cells, leading to their destruction. Haemolysis is a significant complication of cardiopulmonary bypass (CPB), which has been noted to damage the red blood cell membrane and reduce their lifespan. Limited information is available about open-heart procedures for AIHA patients. Despite presenting periprocedural challenges, multidisciplinary efforts enabled the successful performance of surgical ASD closure in a patient with Warm Reactive Autoimmune Haemolytic Anaemia (AIHA).

Case illustration:
A 56-year-old woman with a large elliptical Atrial Septal Defect was planned for surgical ASD closure. The patient has never received a blood transfusion or experienced any previous haematological issues. During the surgical preparation, the patient's immunoglobulin G Coombs test result was positive with the precensce of immunoglobulin G. The patient was diagnosed with a remission state of warm AIHA. A challenge arose when surgical ASD closure needed a cardiopulmonary bypass, which increased the risk of hemolysis. The patient also needed to be hypothermic to reduce metabolism, which may interact with the pathophysiology of AIHA. Several approaches were taken, and the procedure was conducted successfully without noteworthy obstacles.

Conclusion:
In summary, we experienced a successful surgical ASD Closure in a patient with complete remission of warm-reactive AIHA. Considering the different hemolytic mechanisms between Cardio Pulmonary Bypass and AIHA, determining whether AIHA is cold or warm reactive is crucial for managing temperature in the heart-lung machine. Several approaches, such as utilizing a roller pump, a heparin-coated circuit, and administering steroids, can be implemented to prevent hemolysis.

Keywords: Autoimmune Hemolytic Anemia, Atrial Septal Defect, Heart Lung Machine, Cardiovascular Surgery, Cardiopulmonary Bypass

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HOW TO TREAT STABLE PULMONARY EMBOLISM RIGHT

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Background:
Pulmonary embolism (PE) is the third most common cause of death among hospitalized patients Acute pulmonary embolism is a common clinical condition with a variable clinical presentation, making the diagnosis challenging. PE occurs when a thrombus originates elsewhere and disrupts blood flow in the pulmonary artery or its branches. The initial approach to patients with suspected PE depends upon whether the patient is hemodynamically stable or unstable . Among hemodynamically stable patients . For most cases of acute PE without haemodynamic compromise, parenteral or oral anticoagulation (without reperfusion techniques) is adequate treatment

Case illustration:
A 54 year-old woman presents to Emergency Room with Shortness of breath worsened a week before admission, The patient has a known history of DVT in left lower extremity at July 2023. She has just diagnosed with ovarium cancer and but has not been on therapies. Her blood pressure is 125/82mm Hg, and her heart rate is 92 bpm and an oxygen saturation of 97 % on room air. Echocardiography showed there was no RV dysfunction. Chest computed tomography scan showing embolies at right pulmonary arteries. The patient diagnosed with low risk stable PE and got anticoagulant fondaparinux 1x 7.5 mg for 5 days and has been discharged after 1 weeks in admission then continued with rivaroxaban 1x 20 mg for 3 months then followed up and underwent chest computed tomography revealed pulmonary arteries embollies has not been found

Conclusion:
Anticoagulation is the mainstay of PE treatment both in the in-hospital treatment phase and after hospital discharge. For most cases of acute PE without haemodynamic compromise, parenteral or oral anticoagulation (without reperfusion techniques) is adequate treatment . Treating acute PE using fondaparinux followed by rivaroxaban is should be considered for treating VTE and need further study to provide additional evidence that may help to clarify the efficacy of anticoagulant alone in treating acute stable PE

Keywords: Pulmonary Embolism, DVT, Anticoagulant, VTE

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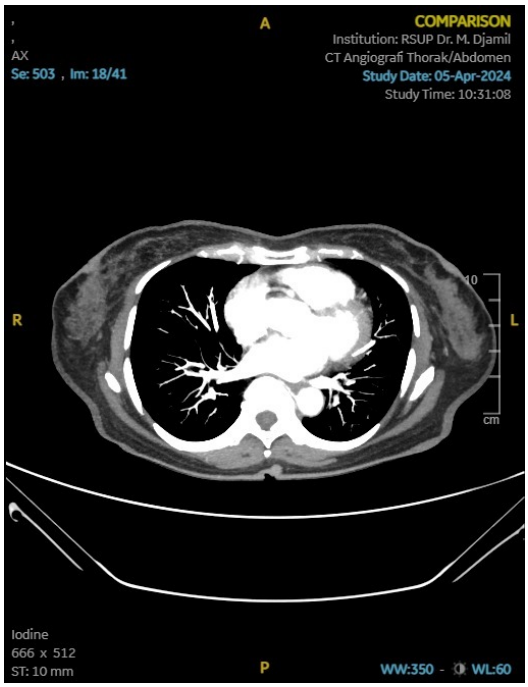

Figure 1. CTPA showing no thrombus or embolies after 3 monts of anticoagulant

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ANOMALOUS ORIGIN OF RIGHT CORONARY ARTERY: A CASE REPORT

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Background:
There are 3 main coronary arteries, Right Coronary Artery (RCA) which arises from the right coronary sinus, Left Anterior Descending Artery (LAD) and Left Circumflex Artery (LCx) which originates from the Left Main (LM) vessel which arises from the left coronary sinus. Anomalies of RCA prevalence estimated from autopsy or imaging studies of around 0.026% - 0.25%. Patients with coronary anomalies may be asymptomatic or may experience angina, infarction, or sudden death. The mechanism of myocardial ischemia is dynamic and not always due to atherosclerotic plaque. Other possible causes include compression of the RCA between the aorta and pulmonary artery or stretching of the RCA with distention of the aortic/pulmonary arteries.

Case illustration:
A 62 years old male patient with complaints of recurrent shortness of breath during activity in the last 6 months accompanied by severe chest pain. On ECG examination, Wellen Syndrome Type A was noted. The patient underwent Coronary Angiography with tubular stenosis 70-80% distal in the left main, multiple diffuse stenosis 70-80% osteal-distal LAD, multiple diffuse stenosis 70-80% osteal-distal LCx, and anomaly of RCA origin adjacent to left coronary sinus as well as diffuse stenosis 70-80% proximal-distal RCA and tubular stenosis 95-99% after Acute Marginal Branch receiving collaterals from LAD and LCx. The patient was sent home with preparation for Bypass Surgery.

Conclusion:
Patient with anomalous of RCA origin from the left coronary sinus had typical symptoms of angina with findings of significant blockage in the major coronary vessels. Echocardiography showed LVEF (38% Simpsons Biplane) with an enlarged left ventricle and global hypokinesis. Although in this patient the symptoms of chest pain and shortness of breath were likely caused by significant blockage of main coronary arteries, surgery to correct anomalies can reduce the risk impact on the patient. Apart from that, other supporting examinations such as CT scan can be carried out to obtain a comprehensive imaging such as the location, position, pathway and flow of RCA anomalies.

Keywords: Coronary Anomaly; Coronary Angiography

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VERY HIGH ATHEROSCLEROTIC BURDEN AT A YOUNG AGE: A CASE REPORT

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[2]Siloam Hospital Lippo Village

Background:
Atherosclerotic cardiovascular disease typically occurs in the fourth decade of life in susceptible individuals, but it has recently been more common in younger age groups. Accelerated atherosclerosis at a younger age may result in severe clinical disease and worse outcomes. This report aimed to present a very young patient with atherosclerotic disease involving multiple vascular regions.

Case illustration:
A 26-year-old woman with an extreme sedentary lifestyle, high carbohydrate diet, and morbid obesity (BMI 36 kg/m2) had uncontrolled diabetes with poor medication compliance since she was 23-year-old (HbA1C 11.8%), along with dyslipidemia (LDL 243 mg/dL) and nephropathy (eGFR 34.9 mL/min/1.73 m2), indicating CKD.She came with peripheral artery disease manifesting as a non-healing ulcer in her right toe. Post-amputation, she experienced worsening dyspnea, desaturation, and bilateral lung rales. Seven days later, she had resting chest pain; ECG showed anterior leads T inversion and increased Trop-T (166 pg/mL). Coronary angiography revealed diffuse atherosclerotic plaque with severe stenosis in LAD, LCx, and RCA, subsequently, PCI was performed. Several days later, her consciousness decreased alongside left hemiparesis. Brain MRI revealed multiple infarcts. A right carotid ultrasound showed high-risk atherosclerotic plaques. To reduce atherosclerotic plaque burden and prevent future adverse cardiovascular events, she receives dual antiplatelet therapy, high-intensity statin and ezetimibe, empagliflozin, semaglutide, ARNI, beta blocker, along with strict lifestyle modifications. Three months later, she lost 20 kg, HbA1C decreased to 5.8%, LDL decreased to 42 mg/dL, and showed functional class improvement.

Conclusion:
We have presented a case of a young female with atherosclerotic disease involving the coronary bed, carotid, peripheral, and renal arteries. Invasive revascularization strategy and optimal medical therapy were done to mitigate clinical complications. Long-term secondary prevention measures including blood glucose control, LDL target <55 mg/dl and 50% reduction from baseline, weight reduction, and appropriate antiplatelet therapy, are essential.

Keywords: young, diabetes mellitus, coronary artery disease

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CARDIAC REVERSE REMODELING IN PPCM: MYTH OR FACT?

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Background:
Peripartum cardiomyopathy (PPCM) is maternal heart failure characterized by dilated cardiomyopathy and LV systolic dysfunction (EF<45%) which develops within the last months of pregnancy or the first 5 months postpartum. It has variable outcomes include complete recovery, persistent heart failure, arrhythmias, thromboembolic events, and death. The recovery rate is high during the first 3 to 6 months, however delayed recovery may also occur up to 2 years following diagnosis.

Case illustration:
A female, 41 years old, complained dyspnea on effort, orthopnea dan paroxysmal nocturnal dyspnea for 2 months before admission. She gave birth 5 months ago. Physical examination revealed widening cardiac border, pansystolic murmur grade 3/6 at apex and LLSB, early diastolic murmur grade 2/4 at URSB, S3 gallop, rales (+/+) and pitting oedema in both legs. CXR showed cardiomegaly with CTR 70%. Echocardiography showed dilated all cardiac chambers with LVEF 16% (Simpson), TAPSE 16 mm, mild-moderate AR, moderate-severe MR and moderate-severe TR with high probability of PH. Patient was diagnosed as PPCM. She was managed as per HFrEF guideline with Furosemide, Spironolactone, Candesartan and Bisoprolol. She then migrated and referred to Boyolali, Central Java. After 12 months of follow-up period, the symptoms were improved and follow-up echocardiography showed improved LVEF 16% to 61% (Simpson), reduced cardiac chambers size and improved cardiac valve abnormalities.

Conclusion:
Complete recovery with cardiac reverse remodeling can occur in patients with PPCM during treatment periods. HFrEF therapy regiment, such as MRA, ARB/ACE-I and BB, may have a role in promoting cardiac reverse remodeling.

Keywords: PPCM, Heart Failure, Cardiac Reverse Remodelling

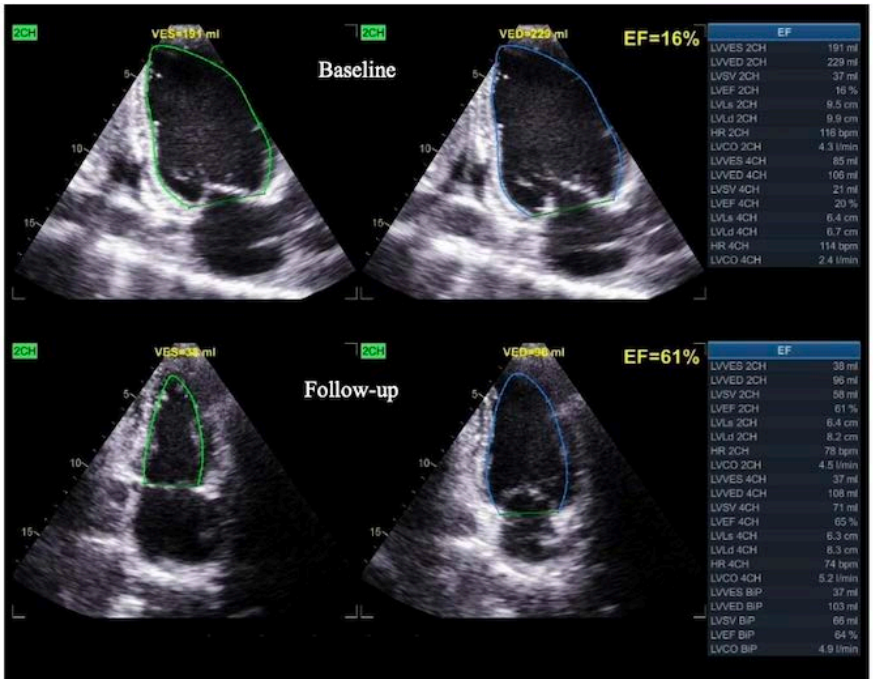

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Figure 1. Baseline and follow-up echocardiography during 12 months of therapy showed significantly increased LVEF 16% to 61% (Simpson) and reduced LA and LV chambers size

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MYOCARDIAL INFARCTION WITH NON-OBSTRUCTIVE CORONARY ARTERIES (MINOCA) PRESENTING WITH HYPOVOLEMIC SHOCK IN A YOUNG WOMAN : A CASE REPORT

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[2]Cardiologist, Undata Regional General Hospital

Background:
Myocardial infarction with non-obstructive coronary arteries (MINOCA) is a clinical infarct syndrome caused by many different pathophysiological conditions without relevant evidence stenosis of coronary artery. Hypovolemic shock due to severe dehydration can be as a potential cause of coronary underfilling leading artery spasm to infarct myocard. The evaluation of MINOCA should be aimed determining the specific cause to achieve optimal targeted therapy.

Case illustration:
A 42-year-old female patient was referred to the emergency room with severe hypotension. The patient complained chest pain (level 6/10) radiating to the back and left arm 8 hours prior to admission, palpitations, cold sweat, dizziness, weakness, and frequent vomiting accompanied by diarrhea. She had no fever, dyspnea, and other significant medical history. The physical examination showed cold extremities with undetectable blood pressure, pulse rate was 136 bpm, and normal oxygen saturation. Cardiopulmonary examination revealed normal heart sounds without murmurs or gallop and clear lung fields. The electrocardiography showed sinus tachycardia with ST depression in anterolateral and inferior leads. The laboratory findings an increase troponin I level of 0,23 ng/ml. However, complete blood count, electrolytes, blood sugar, lipid profiles, kidney and liver function were unremarkable. Patient was treated for non ST-elevation myocardial infarction and hypovolemic shock. At this time, the blood pressure responded to adequate fluid resuscitation but remained hypotensive, and was therefore started on low-dose dobutamine to reach targeted blood pressure. Furthermore, transthoracic echocardiography demonstrated normal ejection fraction (61%) with TAPSE 2 cm, global normokinetic, concentric left ventricular hypertrophy, diastolic disfunction, and mild regurgitation of mitral and tricuspid valve. She underwent urgent coronary angiography which was reported as non significant CAD, right coronary dominant, and no signs of obstructive, coronary dissection, bridging, or embolism. After four days hospitalized in ICVCU, her condition was stable and discharged home. We plan to refer the patient for CMR examination if persistent chest pain occur.

Conclusion:
Clinicians should be vigilant to this possibility in all patients presenting with infarct syndrome when a state of severe dehydration is often present. Although MINOCA had less comorbidity and mortality, hypovolemic shock can worsen the patient's condition.

Keywords: MINOCA, Hypovolemic Shock, Myocardial Infarction

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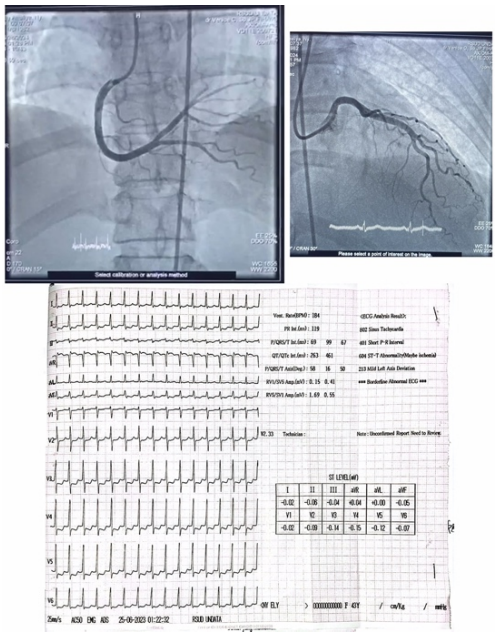

Figure 1. ECG and Coronary Angiography

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RARE CASE OF ARRHYTHMIA: INCESSANT ATRIAL FLUTTER IN TODDLER.

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[1]MYCardia Eka Hospital BSD

Background:
A case of ventricular tachycardia, followed by an atrial flutter, after an episode of fever found in a pediatric patient.

Case illustration:
A 4-year-old male patient presented to the emergency department with complaints of palpitations, with reports of fever from 2 day prior. On examination, the temprature of the patient was 38.1°C, with an electrocardiogram (ECG) which showed findings consistent of ventricular tachycardia (VT) with a heart rate of 232 (Fig. 1). With that finding, a 4 mg bolus of Herbesser was administered, the VT was converted and the palpitations were no longer felt. The anti-arrythmia treatment was continued with 100 mg of Amiodarone. An echocardiogram was also done by a pediatric cardiologist, and found that there was no intra-cardiac abnormalities. The patient was then transferred to pediatric intensive care unit (PICU) for observation and further examination. The next day, the ECG showed atrial flutter, and cardioversion was done once with 20 Joule which successfully converted the ECG to normal sinus rhythm. Later that day the atrial flutter came back, cardioversion with anesthesia was done once again using 20 Joule. The ECG converted to normal sinus rhythm. Throughout the hospitilization, the patient underwent thorough examination to find the cause, including laboratory tests and echocardiogram. Through the laboratory result, the pediatrician concluded that there was a bacterial infection, which causes the fever, which could potentially triggered the arrythmia. Following the last cardioversion the patient remained haemodinamically stable without further episodes of palpitation. The patient was then discharged from inpatient care, with a follow up planned one week after.

Conclusion:
This case presents a rare case of a malignant arrythmia in form of a ventricular tachycardia, coupled with an atrial flutter, following an episode of fever. The prompt recognition of the situation and a comprehensive evaluation involving a pediatrician, pediatric cardiologist and an electrophysiology cardiologist was needed to optimize the management and outcome of this patient. Further research regarding the relationship of fever and arrythmia may be warranted.

Keywords: ventricular tachycardia, arrythmia, pediatric cardiology, atrial flutter

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THE ROLE OF PHLEBOTOMY IN SECONDARY ERYTHROCYTOSIS IN EISENMENGER SYNDROME PATIENT: A CASE REPORT

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Background:
Eisenmenger syndrome (ES) is a highly severe form of pulmonary arterial hypertension that is associated with an untreated ventricular septal defect (VSD). These groups of patients are chronically hypoxemic and might lead to a secondary erythrocytosis, which is a natural adaptive response to persistent hypoxemia. Phlebotomy may temporarily lower haematocrit and blood viscosity while treating the primary underlying cause. We report a case demonstrating the role of phlebotomy in patient with secondary erythrocytosis secondary to ES.

Case illustration:
A 26-year-old female patient was admitted to the ER due to shortness of breath and hemoptysis. The medical history recounts a VSD and on treatment. A general examination revealed a pulse rate of 99 beats/minute, a blood pressure of 141/90 mmHg, a respiratory rate of 30/minute, and a pulse oximetry of 57% on room air. In addition, clubbing fingers on both upper and lower extremities were noted. Rhonchi is positive in both lungs. A systolic murmur (4/6) was discovered during cardiovascular auscultation in the apex. Chest radiograph showed pneumonia and cardiomegaly. On laboratory investigation, hemoglobin was 24.6 g/dl, hematocrit was 83.6%, platelet count was 96.000, and procalcitonin was 32.04 ng/mL. The ECG showed right ventricular hypertrophy, and the echocardiography showed a VSD subarterial doubly committed with a diameter of 7–11 mm, mainly R–L shunt, mild tricuspid regurgitation with a high probability of pulmonary hypertension, and RA–RV dilatation. The patient was transferred to the ICCU for adequate oxygenation therapy and medical therapy with oral furosemide, bisoprolol, captopril, digoxin, sildenafil, broad-spectrum antibiotics, and other pulmonary therapies were given. Phlebotomy was performed with close monitoring. After phlebotomy, symptoms were resolved, saturation gradually improved, and the laboratory results showed a decrease in hemoglobin level (17.68 g/dl) and hematocrit level (61%). The patient was discharged after 6 days with a progressive improvement of functional status with pulse oximetry of 97%.

Conclusion:
The case showed that appropriate medical therapy with phlebotomy may improve symptoms and functional status in patients with secondary erythrocytosis due to ES. However, further follow-up at congenital heart disease centers is essential to prevent and manage the complications of ES.

Keywords: eisenmenger syndrome, ventricular septal defect, secondary erythrocytosis, phlebotomy

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CHEST PAIN TURNED OUT TO BE AORTIC DISSECTION AT REPRODUCTIVE AGE. A CASE SERIES AND LITERATURE REVIEW

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Background:
Aortic dissection is a life-threatening condition characterized by a tear in the inner layer of the aorta. The incidence rate is 2.9 cases per 100,000 person-years. Risk factors include hypertension, connective tissue disorders such as Marfan syndrome. Early diagnosis and treatment are crucial in determining the patient's outcome

Case illustration:
We have two cases of Aortic dissection. A 19-years-old Marfan Syndrome with systemic score 7.0 complaining of sudden tearing pain in the back after accident and a 43-years-old with uncontrolled hypertension complaining sudden tearing pain in the back during activity. Echocardiography on both patients reveal intimal flap in the aorta and dilatation of the aorta. MSCT found the dissection of ascending aorta with exit point at the abdominal aortic bifurcation (Stanford A, DeBakey I) in Patient with Marfan Syndrome, and dissection in the aortic arch with exit point at the abdominal aorta branch (Stanford B, DeBakey III) in patient with hypertension. The target therapy is pain management, reduce blood pressure and heart rate, statin, and other symptomatic drugs. Both patients were planned for the Bentall and TEVAR surgery.

Conclusion:
Not all chest pain is ACS, aortic dissection is one that has clinical of chest pain. Aortic dissection in Marfan syndrome has a pattern that can differentiate it from patients without Marfan syndrome. The overall in-hospital mortality rate of Marfan Syndrome was lower than without Marfan syndrome. Correct diagnosis and treatment can prevent wrong management that have negative outcome for the patient.

Keywords: Aortic dissection, Marfan syndrome, Hypertension

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THE INTERCONNECTION OF MYOCARDIAL INFARCTION PRESENCE IN ACUTE ISCHEMIC STROKE

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Background:
Cardiovascular and cerebrovascular diseases are the leading causes of mortality and morbidity worldwide. Both are interrelated based on Randomized Controlled Trials (RCT) and observational studies reporting involvement in heart damage of about 10–20% in patients with acute ischemic stroke. Neurocardiogenic mechanisms trigger the occurrence of cardiac events after stroke.

Case illustration:
Mrs.M, 63 years old, came to the hospital with a condition of unconsciousness and no history of head trauma. The vital signs show blood pressure 90/70, afebrile, and left hemiparesis. There is an elevation of the ST segment in the inferior leads with an unknown angina pectoris sign. The head CT scan showed a lacunar infarction in the bilateral caudate nuclei and a high troponin I level (>15,000 ng/L). The echocardiography showed kinetic disturbances in the inferior chamber without the presence of thrombus. During hospitalization, the choice of therapy involves anti-thrombotic and anti-coagulant drugs. A significant improvement in response appeared on the second day of treatment at the ICU. On the fifth day, the patient could be returned to the hospital in a state of improvement.The type I stroke-heart syndrome is indicated by the presence of a focal neurological deficit without intracranial bleeding and typical chest pain, accompanied by elevated cardiac enzymes and myocardial infarction. A patient HEART score of 7 points has a high risk of major adverse cardiovascular event (MACE). Pathophysiology can be caused by inflammation and atherosclerosis. Catecholamines release induced platelet aggregation and thrombosis in blood vessels. In addition, the formation of embolism due to plaque rupture and blood stasis in patients with ventricular dysfunction may increase the incidence of myocardial infarction. The elevation of the ST-segment, followed by an increased cardiac enzyme, was associated with a poor prognosis. Consideration of combination therapy without anti-thrombolytic therapy can result in clinical improvement.

Conclusion:
The occurrence of acute ischemic stroke becomes a chronic disorder that can cause high mortality at the onset of myocardial infarction. Both short-term and long-term effects require proper management in the selection of therapies for a better life expectancy.

Keywords: Myocardial infarction, heart stroke syndrome, acute ischemic stroke

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A RARE CASE OF TOTAL AV BLOCK IN HYPERTHYROIDISM

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Background:
Bradyarrhythmias are rare cardiovascular complications of hyperthyroidism compared to hypertension and tachyarrhythmias, including sinus tachycardia and atrial fibrillation. Conduction abnormalities shown by PR prolongation were only reported in 2%-30% of patients with hyperthyroidism. Although rare, 1st degree AV block, Mobitz type 1 and Mobitz type 2 AV block, and 3rd degree heart block, have been all previously reported in Graves' disease, thyrotoxicosis, and subclinical hyperthyroidism.

Case illustration:
A 33-year-old man came to the ER with fever 7 days before admission. He also complained nausea and vomiting around 3 times a day for the last 3 days. There were no chest discomfort or any other complaints. Physical examination showed bradycardia with blood pressure of 180/70 mmHg and fever. Laboratory findings showed slight elevation of leukocytes, low thrombocyte level (146.000) and low natrium level (128). ECG was performed with the result of total AV block. The patient was observed in the ICU and was given dopamine IV. CKMB and troponin levels were normal. Echocardiography showed no vegetation and no microorganism was found so infective endocarditis was rejected based on the duke criteria. Further investigation showed the patient had a history of hyperthyroidism which was supported by elevated T4 level and low TSH level. Patient's ECG kept changing but constantly showed bradyarrhythmia. Patient was diagnosed with bradyarrhythmia in thyroid heart disease.

Conclusion:
Bradyarrhythmia is a rare but important complication of hyperthyroidism. Conduction abnormalities with fever suggesting myocarditis could be caused by increased thyroid level. Further investigation must be performed to conclude the cause of bradyarrhythmia.

Keywords: Thyroid Heart Disease, Bradycardia, Atrioventricular Block, Hyperthyroidism

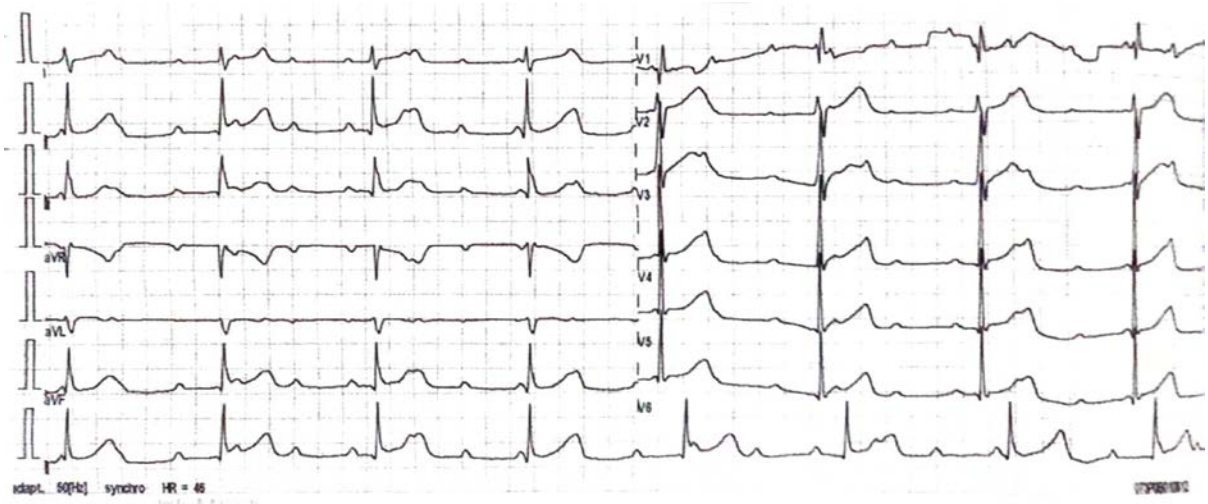

Figure 1. Patient's ECG with TAVB

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PERSISTENT SEVERE BRADYARRHYTHMIA FOLLOWING DONEPEZIL DISCONTINUATION IN DEMENTIA PATIENT: A CASE REPORT

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Background:
Donepezil is an acetylcholinesterase inhibitor used in Alzheimer's management, enhances neurotransmission but carries risks of orthostatic hypotension and bradycardia. Uncommon severe bradyarrhythmia's post-discontinuation merit notice, although underlying mechanisms are unclear. Besides, there have been no previous case reports regarding severe bradyarrhythmia's post donepezil discontinuation

Case illustration:
We report the case of a 33 year old woman with a history of recurrent fainting. She has dementia and regularly takes donepezil. He experienced severe bradyarrhythmia unresponsive to atropine and discontinued donepezil. ECG examination showed sinus arrest with junctional escape rhythm at 51 beats per minute. The patient's potassium level is low. No complaints during treatment. The patient continues to use temporary pacemaker. despite the correction of potassium level and evaluation up to seven days, the ecg is still bradyarrhythmia. we decide to set the permanent pacemaker to the patient.

Conclusion:
This case report highlights donepezil's link to severe bradyarrhythmia in dementia patients. It stresses the challenges in managing adverse effects, suggesting that stopping donepezil and anticholinergic therapy may not always be sufficient. Temporary pacemaker installation may be necessary, but after several days, permanent pacemaker should be the option to emphasizing an approach for optimal care

Keywords: bradyarrhytmia, dementia, donepezil

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BRASH SYNDROME: A STORY OF SUCCESSFUL TREATMENT

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Background:
BRASH syndrome (Bradycardia, Renal Failure, AV Blockade, Shock, and Hyperkalemia) is a vicious cycle precipitated by renal failure, leading to hyperkalemia and accumulation of AV nodal blockers like beta-blockers (BB) or calcium channel blockers (CCB). Both hyperkalemia and AV nodal blockers cause bradycardia and hypoperfusion, which make renal failure worse. This syndrome is an emerging clinical entity that can lead to catastrophic events if left untreated.

Case illustration:
A 55-years old male patient with past medical history of angina pectoris, hypertension, acute kidney injury, and type 2 diabetes mellitus presented to emergency department with the complaint of dyspnea, oliguria, and body swelling. There were notable home medications included atorvastatin 20 mg once daily, bisoprolol 1,25 mg once daily, candesartan 16 mg once daily, clopidogrel 75 mg once daily, nitroglycerin 2.5 mg twice daily, insulin glulisine injection 8 units thrice daily, and insulin glargine injection 8 units once daily. Clinical examination revealed a blood pressure of 92/50 mmHg and bradycardia of 37 bpm. The patient's laboratory results were notable for elevation of urea 89 mg/dL, creatinine at 5.0 mg/dL, and potassium at 8.1 mEq/dL. Initial electrocardiogram showed a junctional escape rhythm (Fig.1), raising concern diagnostic of BRASH syndrome. The patient was admitted to our intensive care unit and his previous medications were discontinued. He was treated with IV calcium gluconate, insulin, and D40 fluid. Dopamine and norepinephrine drip was also initiated to provide ionotropic and vasopressor support. His treatment was followed with emergent dialysis due to severe condition. After the dialysis, the patient's renal function appeared to improve, and potassium levels returned to normal. The bradycardia gradually resolved to the 80s, resulting in conversion to sinus rhythm. The patient continued to improve clinically and was stable for discharge after 10 days completion of treatment.

Conclusion:
BRASH syndrome is a life-threatening condition which clinicians should be timely aware in case to improve the patient outcomes. Aggressive management to the severity of kidney injury and hyperkalemia is a critical measure in the management of BRASH syndrome.

Keywords: Hyperkalemia, BRASH syndrome, Renal failure, Bradicardia

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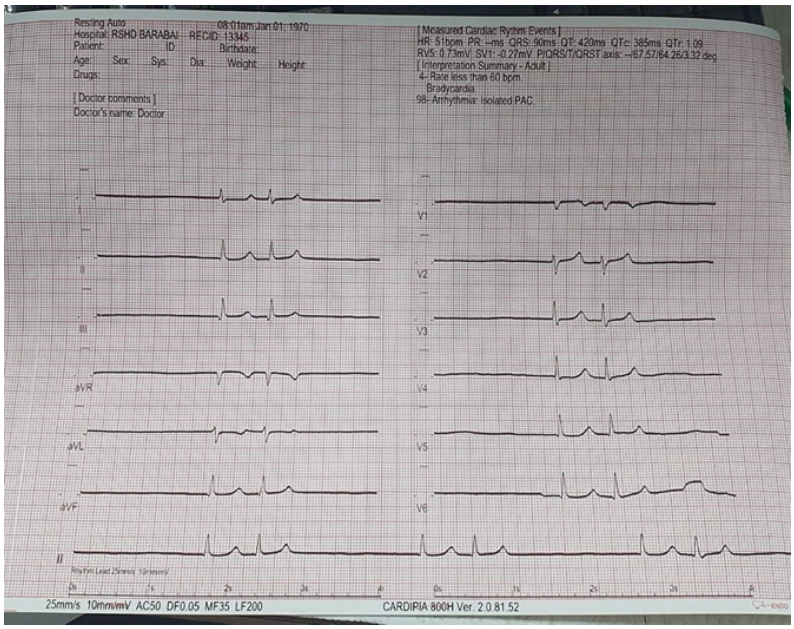

Figure 1. Electrocardiogram on admission

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FIBRINOLYTIC THERAPY IN INFERIOR STEMI WITH CARDIOGENIC SHOCK: A CASE REPORT

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Background:
In the selection of reperfusion strategies, fibrinolytics have the same efficacy as primary PCI in achieving TIMI flow grade 3 in completely occluded coronary arteries when performed in cases with an onset < 3 hours. In this report, we are reporting an inferior STEMI case complicated with cardiogenic shock successfully treated and discharged from hospital with fibrinolytic therapy.

Case illustration:
A 62-year-old male patient admitted to Emergency Department of Hermina Ciputat Hospital with chief complaint of chest pain 2.5 hours before admission, accompanied by cold sweating, shortness of breath, nausea, and vomiting. He had no relevant history of cardiac disease, but possessed risk factor of CAD as he was an active smoker. Upon arrival, he was fully alert with his vital sign: blood pressure 90/60 mmHg, heart rate 57 bpm, respiratory rate 21, saturation 97% room air, and other physical examination revealed no significant findings. ECG revealed sinus bradycardia with ST elevation in lead II, III, aVF, and ST depression in lead I, aVL, V1-V3. The possibility of the RV and posterior infarction can be excluded based on right-sided and posterior ECG. Blood test revealed elevated troponin I level (0.11 ng/mL). CXR showed normal findings. Transthoracic echocardiography revealed borderline LV function with LVEF 52%. Patient was diagnosed as inferior STEMI complicated with cardiogenic shock (blood pressure dropped to 70/47 mmHg while observed in ED). The patient was treated with aspirin 320 mg followed by 80 mg once daily, clopidogrel 300 mg followed by 75 mg once daily, morphine 2 mg iv, atorvastatin 40 mg once daily, enoxaparin 60 mg twice daily, and dopamine started from 5.0 mcg/kg/min (down-titrated), reperfusion strategies applied was fibrinolytic therapy using streptokinase 1.500.000 IU because our hospital do not have cardiac catheterization laboratory and onset of this case < 3 hours. After one-day observation in HCU, dopamine was stopped, patient condition improved and moved to ward and discharged after 3 days of treatment.

Conclusion:
Early reperfusion with fibrinolytic therapy in inferior STEMI with onset of less than 3 hours can treat life-threatening conditions such as cardiogenic shock in this case as effectively as primary PCI.

Keywords: cardiogenic shock, fibrinolytic, reperfusion, inferior STEMI

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MY BABY IS A HERO: CASE REPORT OF PATENT DUCTUS ARTERIOSUS IN CHILDREN

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[1]Undata

Background:
A patent ductus arteriosus (PDA) is a most common congenital disease in preterm infants. Diagnosis and management of patent ductus arteriosus still a challenging game for both neonatologists and pediatric cardiologists. Treatment options had consider due to early recognition, severity and adverse outcome.

Case illustration:
Here, we report a 11 months old male infant patient with “superhero“ or “Ironman”-like presentation which is a bulging on its chest. The patient came up with heart failure like symptoms such as prolonged cough, often cyanosis and frequent fever as parents said it was also low birth weight at 1900 grams and premature gestation at 33-34 week which predicted as congenital disease. The signs and symptoms was persistent since his birth. Echocardiography result shown PDA and the patient was prescribed while waiting on its DA to close by drug’s help.

Conclusion:
This case report emphasize that patent ductus arteriosus could be present with unique appearance to our healthcare society.

Keywords: congenital, heart disease, patent ductus arteriosus, infant

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ACUTE CORONARY SYNDROME EVENTS FOLLOWING CERVICAL DISC REPLACEMENT: A CASE REPORT

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Background:
Acute Coronary Syndrome (ACS) covers various conditions related to sudden decreases in coronary blood flow, including myocardial infarction and unstable angina. Cervical Disc Replacement (CDR) is a surgery to ease symptoms from cervical disc disease by replacing a damaged disc with an artificial one. While generally safe, it can sometimes lead to postoperative complications, including cardiovascular events. The stress of surgery, inflammatory responses, and underlying risk factors may contribute to ACS after such procedures. This case report discusses a patient who experienced ACS events after CDR, emphasizing the potential cardiovascular risks associated with this surgery. Understanding the link between CDR and ACS is important for improving patient care and management for individuals undergoing similar procedures.

Case Illustration:
A 36-year-old male with a history of a motorbike accident resulting in a fractured left clavicle and CDR presented with neck pain spreading to shoulders and arms but was relieved by resting and taking isosorbide dinitrate sublingual. Electrocardiography results showed elevated cardiac enzymes and high cholesterol levels. MRI Cervical showed spondyloarthrosis and bulging discs of the spinal canal. Echocardiography showed ejection fraction 60% and diastolic dysfunction. Coronary Angiography (CAG) revealed CAD 3 VD LM disease, and Coronary Artery Bypass Graft (CABG) was recommended. The patient received intravenous medications including nitroglycerin and heparin drip, as well as oral medications Rosuvastatin, Valsartan, Nebivolol, Amlodipine, Ezetimibe, Atorvastatin, and empagliflozin. Following the CAG, the patient is being closely monitored in the intensive Cardiac Care Unit (ICCU) to prepare for CABG. This case emphasizes the risk of ACS events after CDR procedures due to factors like perioperative stress, inflammation, and hemodynamic changes. There are few reports on this association, so it's important to be vigilant for cardiac symptoms postoperatively.

Conclusions :
This case report emphasizes the need for thorough cardiovascular risk assessment and management during and after CDR surgery. While the procedure is generally effective for cervical disc disease, it's crucial to be aware of the potential for ACS events, especially in patients with existing risk factors. Integrating cardiovascular care into the surgical management can enhance patient safety and outcomes.

Keywords: Postoperative Complications, DDR, ACS

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THE FIRST PERCUTANEOUS CLOSURE OF PATENT DUCTUS ARTERIOSUS UNDER MINIMAL FLUOROSCOPY GUIDING IN PATIENT WITH PREVIOUSLY UNDERWENT DEVICE CLOSURE OF VENTRICULAR SEPTAL DEFECT IN EAST NUSA TENGGARA : A CASE REPORT

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[2]Department of Cardiology and Vascular RSUD Prof. Dr. W. Z. Johannes Kupang

Background:
The ductus arteriosus is a vascular structure that connects the pulmonary artery and aorta which should closed spontaneously during the first 48 hours of life. Persistent patency of the ductus arteriosus beyond a few weeks is considered abnormal. Percutaneous closure of Patent Ductus Arteriosus (PDA) is the standard management in most cases, especially in patient with signs of left ventricular volume overload, left-to-right shunt, and with severe pulmonary arterial hypertension. Cases of defect closure with a previously installed occluder device are still rare and also challenging. Even though, PDA closure still may be performed and considered under specific conditions.

Case illustration:
A 14 years old boy presented with shortness of breath. The patient has a history of Ventricular Septal Defect but he already underwent device closure of VSD 6 month ago. On Physical examination, grade III/VI continuous murmur was heard on auscultation at the upper left sternal border. The ECG showed a LVH and the Chest X ray showed cardiomegaly. Echocardiography revealed a type A PDA with isthmus' diameter is 2.5-3.1 mm (figure 1.a). Patient then underwent percutaenous closure and was successfully performed with a HeartR PDA occluder no. 06/08 mm with antegrade transvenous approach, snarring technique, guided by fluoroscopy zero contrast and transesophageal echocardiography. After the Procedure, the echocardiography of the patient showed the device stowed in place without any residual shunt (figure 1.b). It also showed the VSD occluder device still stowed in place without any residual of VSD (figure 1.c).

Conclusion:
The Patent Ductus Arteriosus Closure with minimal fluoroscopy guiding and with VSD occluder device already installed has never done before in East Nusa Tenggara. The procedure could be a challenging, yet promising because it is reliable treatment for PDA.

Keywords: transesophageal echocardiography, patent ductus arteriosus, catheterization

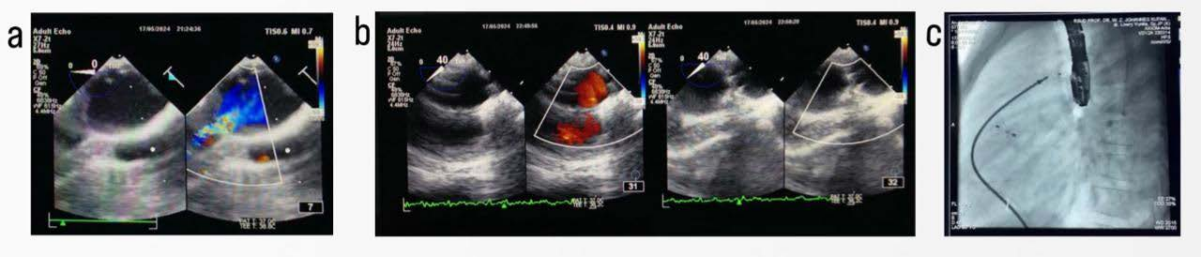

Figure 1 (a) TEE showed the isthmus' diameter of the PDA **(b)** TEE showed the PDA device stowed in place **(c)** Fluoroscopy showed VSD device stowed in place

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MATERNAL CARDIAC ARREST AT 1 WEEK POST PARTUM DUE TO PERIPARTUM CARDIOMYOPATHY: A LESSON IN IGNORANCE

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Background:
Peripartum cardiomyopathy (PPCM) is a rare type of heart failure typically occurring withinthe last month of pregnancy or five months postpartum. Clinical presentation of PPCM may vary and can mimic the typical complaints in a normal pregnancy such as shortness of breath, cough, and tiredness. Thus, this disease can be challenging for clinicians to recognize and diagnose.

Case illustration:
A 32-year-old primigravida woman with 39 weeks of gestation was referred to the ER with complaints of high blood pressure, difficulty of breathing on activity, and cough. Vital signs showed blood pressure of 159/108, heart rate of 121 beats/minute, respiratory rate of 26 breaths per minute, and oxygen saturation of 96%. Physical examination showed a regular heartbeat with a decreased vesicular lung sound on the right side with bilateral soft Ronchi. ECG showed a sinus tachycardia rhythm. Urine protein was found to be positive on blood examination. A chest radiograph was performed and found a pleural effusion on the right side of the lung accompanied by a pneumonia-like appearance and cardiomegaly. An echocardiography examination later revealed diagnostic features of cardiomyopathy and a dilated left ventricle with an ejection fraction of 23%. The patient was scheduled for an emergency C-section and termination was done successfully. In addition, the patient also received oral and intravenous medications to support heart functions. The patient's condition worsened daily and on the 7th day of monitoring in the ICU, the patient went into cardiac arrest and was presumed dead.

Conclusion:
Peripartum Cardiomyopathy (PPCM) is a serious condition that can lead to significant heart failure during or shortly after pregnancy. Appropriate management and a multidiscipline approach can minimize poor prognosis and help reduce morbidity and mortality.

Keywords: Peripartum Cardiomyopathy, Maternal Cardiac Arrest, Cardiomyopathy

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ATRIAL SEPTAL DEFECT - PULMONARY ARTERIAL HYPERTENSION IN THE SETTING OF PREGNANCY : A CASE SERIES

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Background:
Congenital heart disease (CHD) is one of the most congenital malformation found in human. Acyanotic CHD remains problem worldwide and one of its type, atrial septal defect (ASD), ranks second of acyanotic CHD cases with 10 % of total CHD and more common in female than male. We occasionally met ASD pregnant patient with pulmonary hypertension secondary to ASD condition. Physiologically, pregnancy alters cardiovascular system and may worsen the clinical condition of ASD with pulmonary hypertension patients and may subsequently leads to heart failure.

Case illustration:
We reported four interesting cases ASD pregnant patient with pulmonary hypertension. Two of our patients had previous documented medical history and routinely controlled and one of them has undergone ASD closure. All of our patients had dilated right atrial and ventricle and two of them were with right ventricle (RV) systolic dysfunction. All of the patients were with normal left ventricle (LV) systolic function. Despite management of multidisciplinary heart team management and intensive care management, we found that one of our patients died after the termination of pregnancy by sectio caesarian. The patient was with poor RV systolic function, bidirectional shunt lesion, high maximum pressure gradient, without any clear cardiovascular or obstetric documented medical history and poor mWHO class. She suffered from unresolved post operative shock and bleeding.

Conclusion:
Managing pulmonary hypertension patient secondary to congenital heart disease remains challenges among multidisciplinary experts. Intensive perioperative management is mandatory to reduce the mortality and morbidity of pregnant pulmonary hypertension patient. Optimal medical therapy, timing of delivery, method of delivery and post delivery management need to be prepared well for better outcome.

Keywords: pregnancy, heart team, congenital heart disease, pulmonary hypertension

Table 1. Patient's baseline data

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	Case 1	Case 2	Case 3	Case 4
Status of congenital heart disease	Secundum ASD L to R shunt Inlet VSD bidirectional shunt dominant L to R	Primum ASD bidirectional shunt, partial AVSD	Secundum ASD bidirectional shunt dominant L to R	ASD post closure by device
Gestational status	G2P0 38-39 gestational week	G1P0 37-38 gestational week	G1P0 36 – 37 gestational week	G3P1 34-36 gestational week
Modified WHO classification of maternal CV risk	mWHO class II	mWHO class IV	mWHO class II	mWHO class II
Echocardiography finding	Dilated RA and RV, normal LV and RV systolic function, Mild to moderate TR	Dilated RA and RV, normal LV systolic function, poor RV systolic function, Moderate to severe TR	Dilated RA and RV, normal RV and LV systolic function, TR mild	Dilated RA and RV, poor RV systolic function and normal LV systolic function, TR mild
Maxium pressure gradient	88 mmhg	86 mmhg	77 mmhg	58 mmhg
mPAP by Echo	37	42	24	20
TR maximum velocity	4.6 m/s	4.6 m/s	4.4 m/s	3.7 m/s
Anaesthesia technique	Epidural-spinal Ropivacaine, Bupivacaine	General Anaesthesia	Epidural-spinal Ropivacaine, Bupivacaine	Epidural-spinal Ropivacaine, Bupivacaine
Newborn status	Gemeli, one of them died Both newborn were with low birht weight	Alive, with low birht weight	Alive, with low birht weight	Alive, with low birht weight
Maternal complication	No Complication	Shock and bleeding, subsequently death	No Complication	No Complication

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ACUTE MYOCARDIAL INFARCTION FOLLOWING INDUCED VAGINAL DELIVERY WITH UTERINE FUNDAL PRESSURE (KRISTELLER MANEUVER) IN TWIN PREGNANCY PATIENT: A CASE REPORT

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Background:
Acute myocardial infarction (AMI) in pregnancy is rare but has a high maternal mortality rate and neonatal mortality and morbidity. Older maternal age and other cardiovascular risk factors may increase the incidence of AMI in pregnancy. The causes of AMI in pregnancy are diverse, making diagnosis and management challenging.

Case illustration:
A 35-year-old twin-pregnant multigravida woman with 31 weeks gestation came to the ER with complaints of dyspnea, dizziness, and uterine contraction. She had never done antenatal care and had a history of hypertension. Vital signs were BP 190/124 mmHg, HR 140 bpm, RR 40 breaths/min, and SpO2 82% on NRM 15 lpm. Fetal heart rates were 142 bpm and 140 bpm. She was admitted to the ICU and was intubated. Then to facilitate the labor, oxytocin was given, and progressed to full dilation. During vaginal delivery, uterine fundal pressure (Kristeller maneuver) was done, and the babies were delivered. Afterward, on physical examination, we found an increased JVP, bilateral rales in the lungs, S3 gallop, and pitting edema. Electrocardiogram showed Sinus Rhythm with ST-segment elevation in V1-V4 and ST-segment depression in II, III, and aVF. Transthoracic echocardiogram revealed an LVEF of 16%, RWMA (+), akinesia septal and apical, thrombus at LV apex, and mitral regurgitation. Laboratory examination revealed increased Troponin I (553.2 ng/L) and increased D-dimer (>10,000 ng/L) levels. We diagnosed the patient as STEMI Anteroseptal with suspicion of coronary artery dissection. She was given a loading dose of aspirin and ticagrelor followed by a maintenance dose, ISDN, furosemide, bisoprolol, captopril, and amlodipine. Serial troponin I at 6 hours was reduced (383.3 ng/L), and then she was given fondaparinux. The patient was planned for a coronary and thoracal CT scan but unfortunately, it was not performed due to unavailability. Her condition gradually improved, however, the patient refused a PCI referral. She was discharged and planned for further evaluation at the cardiology clinic.

Conclusion:
This case highlights the importance of suspecting AMI in pregnancy patients. Early detection and appropriate management are mandatory to ensure maternal and fetal well-being.

Keywords: Case Report, Induced Vaginal Delivery, STEMI, Acute Myocardial Infarction, Twin Pregnancy

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TETRALOGY OF FALLOT WITH FINGER CLUBBING: A CASE REPORT FROM NUSA WALUYA II FLOATING HOSPITAL

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Background:
Tetralogy of Fallot (ToF) is the most common cyanotic congenital heart disease (CHD) (5-7%), which obstructs the flow of blood from the heart to the lungs and leads to low oxygen levels in the blood. Prolonged decreases in arterial oxygen saturation (more than 6 months) can result in clubbing of the fingers. Currently, there is no data on the incidence of ToF in Indonesia, and the diagnosis is often delayed. Diagnosing and treating patients with limited diagnostic tools and treatment modalities is challenging in remote areas.

Case illustration:
An 8-year-old boy presented to the Nusa Waluya II Floating Hospital Polyclinic with shortness of breath during daily activities, particularly when exercising, which had worsened over the past year. He had never received any treatment. The patient resides in Central Buton, Southwest Sulawesi, where there is no cardiologist. Physical examination revealed a slight difference in oxygen saturation between the right and left extremities, though not significant. Additionally, finger clubbing was observed, indicating prolonged low oxygen levels in the blood. A heart murmur was detected during cardiac auscultation. There were no other congenital anomalies. A mobile echocardiography was subsequently performed, confirming the diagnosis of ToF. These examinations also serve as a modality for the early detection of congenital heart disease. Many children with CHD remain undiagnosed, and some surviving cases experience life-threatening episodes, impaired growth and development, pulmonary hypertension, and infections (such as infective endocarditis and sepsis).

Conclusion:
Early detection of congenital heart disease should be integrated into primary healthcare services to facilitate timely intervention and prevent complications. Early detection is expected to reduce the morbidity and mortality rates associated with congenital heart disease in Indonesia.

Keywords: finger clubbing, diagnosis, remote area, Tetralogy of Fallot

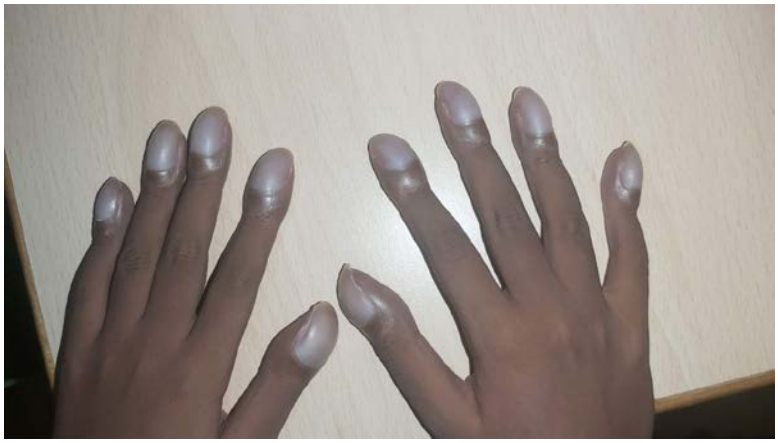

Figure 1. Finger Clubbing

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ACUTE LIMB ISCHEMIA AND SEIZURE IN AN UNCORRECTED TETRALOGY OF FALLOT PATIENT: COMPLICATIONS OR COINCIDENCE?

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Background:
Tetralogy of Fallot (ToF) is the most cosmmon cyanotic heart disease. The prevalence is 5-7% of congenital heart defect. ToF is a combination of four congenital heart defects which consists of; pulmonary stenosis, ventricular septal defect (VSD), overriding aorta, and hypertrophy of the right ventricle. Those defects combination will disrupt the heart to pump effectively, failing to distribute oxygen-rich blood all over the body. The survival rate in uncorrected patients is estimated at 11% at 20 years and 3% at 40 years.

Case illustration:
A 25 year-old woman with a history of uncorrected ToF was hospitalized due to dyspnea, peripheral desaturation (SpO2 40%) and secondary polycythemia (hemoglobin 20.7 g/dL, hematocrit 68%), She underwent phlebotomy and fluid resuscitation. Several hours after the procedure, the patient feel numbness on her left limb. In addition, the saturation of the left hand and leg were undetectable. Doppler vascular ultrasound showed soft trombus and monophasic wave in radial and axillar artery. Diagnosis of acute limb ischemia was established and she got heparinization. The next day, she experienced 3 episodes of seizure. Head CT-scan showed diffuse cerebral oedema and the electroencephalography (EEG) depicted the wave form of encephalopathy. The final diagnosis were ToF, acute limb ischemia, and hypoxic-ischemic encephalopathy. Chronic cerebral ischemia due to uncorrected ToF may cause the condition of encephalopathy.

Conclusion:
Early identification of Tetralogy of Fallot could lead to the better management. Correction of congenital heart defect may diminish the possibility of future complication.

Keywords: Polycytemia, Tetralogy of fallot, Seizure, Congenital heart disease, Acute limb ischemia

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PULMONARY EMBOLISM AFTER UNDERGO SURGERY PROCEDURE: A CASE REPORT

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Background:
Venous thromboembolism, which includes deep vein thrombosis and pulmonary embolism, is an important and common complication of general surgery in the short term. Cancer can lead a high risk for thrombus formation and hence pulmonary embolism. Surgery is defined as a major transient risk factor of venous thromboembolism, particularly beyond 6 weeks, for all types of surgery. The most common symptoms of pulmonary embolism include dyspnea, pleuritic chest pain, cough, hemoptysis, presyncope, or syncope.

Case illustration:
A 69-year-old woman came to the emergency department with increasing shortness of breath that started 4 hours prior to hospital admission. The patient has a history of sigmoid colon cancer surgery performed 1 month ago with a history of prolonged bed rest. Vital signs showed blood pressure: 118/73 mmHg, heart rate: 144 beats per minute, respiratory rate: 35 breaths per minute with SpO2 80%. The electrocardiography examination revealed suspected pulmonary embolism with tachycardia and nonspecific ST-segment, T-wave changes, S1Q3T3 pattern, and also normal chest x-ray. Elevated result of d-dimer for this patient is about > 4.00 ug/mL. And then patient planned for hospitality in high care unit, the patient was given heparin initial dose 4.000 IU and continue with drip 400 IU/hour, and planned to check prothrombin time and activated partial thromboplastin time serial, with target for about 1,5-2,5 times control. The patient that is currently unstable, further examinations were conducted is chest computed tomography scan with contrast that demonstrated extensive bilateral pulmonary emboli.

Conclusion:
The risk of postoperative pulmonary embolism is elevated beyond 6 weeks postsurgery regardless of the type of procedure. So that we must be carefull to diagnose patient with dyspneu after had some surgery procedure for several past months. For established a definitive diagnose that can be done by conducting various supporting examinations such as arterial blood gas, troponin, D-dimer, electrocardiography, chest radiograph and computed tomography pulmonary angiography. Computed tomography pulmonary angiography (CTPA) should be performed emergently.

Keywords: surgery procedure, cancer, pulmonary embolism

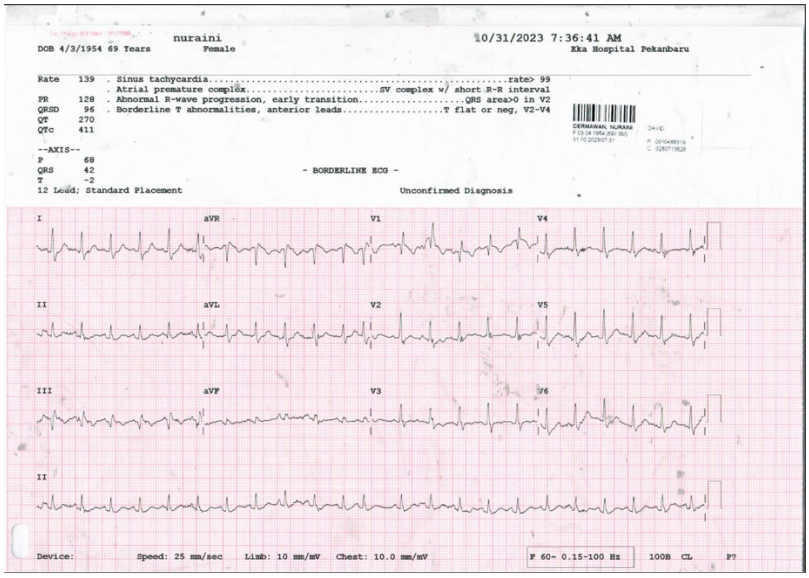

Figure 1. ECG of patient with Pulmonary Embolism

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ATRIAL SEPTAL DEFECT WITH SEVERE PULMONARY HYPERTENSION IN A YOUNG WOMAN: WHEN WE CONSIDER CLOSURE?

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Background:
Atrial septal defect (ASD) is a persistent opening in the interatrial septum after birth that allows direct communication between the left and right atria. Patients with ASD are frequently asymptomatic, but serious symptoms and life-threatening conditions are sometimes found in patients with large ASD who are accompanied by severe pulmonary hypertension (PH). We reported a case of ASD with severe PH in a young woman is there any chance to perform partial or total ASD closure?

Case illustration:
A 33-year-old woman complained of recurrent shortness of breath that had been felt for 1 month and worsened by days. The patient presented with an unstable condition with a respiratory rate of 34 breaths per minute and an oxygen saturation level of 78% room air. The electrocardiogram exhibits sinus tachycardia with P-pulmonal and RV-strain, indicating enlargement of the right atrial and ventricle. The chest x-ray showed an enlargement of the left atria and congestion. The echocardiography showed severe tricuspid regurgitation and mild pulmonary regurgitation with enlargement of all heart chambers, indicating a high PH probability. The patient has a large ASD secundum with a diameter of 36 mm. Five months before admission the patient had performed right heart catheterization (RHC). The RHC exhibits increased mean pulmonary arterial pressure (MPAP) of 73 mmHg and an increased pulmonary vascular resistance (PVR) of 11,2. The patient was diagnosed with large secundum ASD with bi-directional shunt and severe pulmonary hypertension. The patient has been given bosentan and iloprost for 5 months and RHC will be reperformed in the next 2 months. We can consider ASD closure if the patient's PVR improves.

Conclusion:
Patients with ASD and severe PH who come with serious symptoms require immediate treatment to stabilize their condition. ASD closure is the definitive therapy for most ASD patients but ASD closure should not be performed if the patient's PVR > 5. In some cases, the administration of medications such as sildenafil, bosentan, and iloprost for 6 months can reduce the patient's PVR, therefore this is one of the possible choices that can be offered to patients with complicated ASD.

Keywords: Atrial septal defect (ASD), Echocardiography, Pulmonary hypertension (PH), Right heart catheterization (RHC), Pulmonary vascular resistance (PVR)

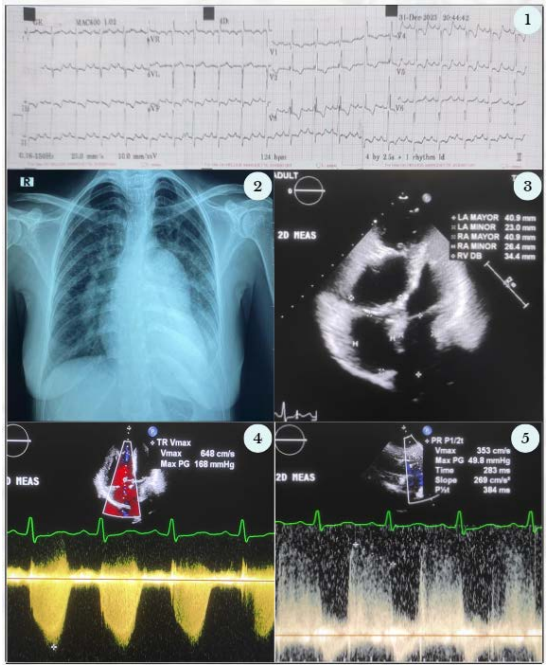

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Figure 1. Abnormality findings from supportive examination: 1. P-Pulmonal, RV and LV strain, 2.Enlargment of Atrium and Congestion, 3. Enlargement of RA and LA, 4. Tricuspid regurgitation, 5. Pulmonary regurgitation

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RARE DISCOVERY OF LEFT VENTRICLE CARDIAC MASSES IN 11-DAY-OLD NEONATE - A CASE REPORT

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Background:
Cardiac tumors are very rare in children with incidence of <0.4% with majority histologically benign. Most common pediatric cardiac tumor is rhabdomyoma 61% of all pediatric cardiac tumors. Echocardiography has high value for diagnosing cardiac tumors and can make a comprehensive, direct, and sensitive assessment of structural imaging, blood flow rate, and cardiac function. Cardiac computed tomography(CT) can display the space-occupying lesions in the heart, which has a definite diagnosis and differential diagnosis significance that show the adjoining relationship between tumor and mediastinum, great blood vessels, extracardiac tissue involvement, and tumor activity.

Case illustration:
An 11-day-old male neonate was referred to emergency department with breathlessnessfor about 3 days. The patient was premature delivery with section caesarean due to placenta previa with history of cyanotic few days after parturition and diminished with oxygen consumption. Physical examination showed minimal subcostal retraction and increased respiratory rates. Chest X-ray showed cardiomegaly. Laboratory tests were within normal limits. Transthoracal echocardiography(TTE) displayed hyperechoic masses in left ventricle(LV) with dilated LV and normal function. CT cardiac revealed multiple masses in LV with diameter 25.4 x 17.1 millimetres and 12.6 x 11.8 millimetres, dilated LV and other findings unremarkable, with conclusion of suspected multiple rhabdomyoma in left ventricle cavity. Patient then consulted to pediatric surgeon for further assessment.

Conclusion:
This case highlighted the rare diagnosis of multiple cardiac masses suspected to be rhabdomyoma in an 11-day-old neonate. The use of multimodality imaging aided in confirming the diagnosis after the patient presented with symptoms of breathlessness and history of cyanosis, emphasizing the complexity and importance of early detection and intervention. Surgery maybe recommended for patient management and histological examination to confirm the masses' pathology.

Keywords: Pediatric, Cardiac Masses, Cardiac Computed Tomography, Imaging, Transthoracal echocardiography

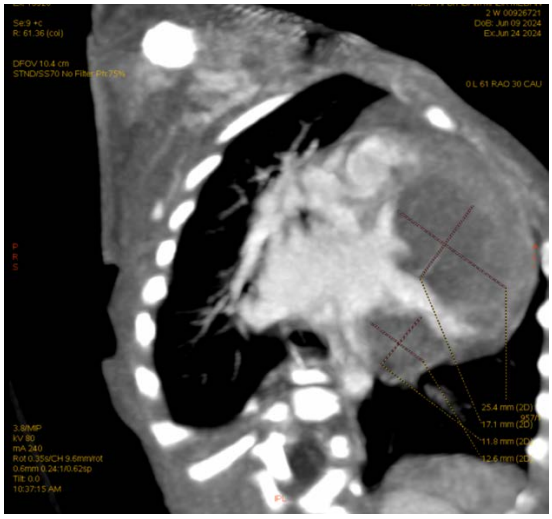

Figure 1. Cardiac CT revealed multiple masses in LV cavity

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NON-SUSTAINED VENTRICULAR TACHYCARDIA DURING FIBRINOLYTIC REPERFUSION THERAPY IN PATIENT WITH ST-ELEVATION MYOCARDIAL INFARCTION : A DIAGNOSTIC AND MANAGEMENT CHALLENGE IN RURAL AREA

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[1]RSUD Lewoleba

Background:
Myocardial ischaemia and infarction can induce life-threatening arrhythmias due to severe metabolic and electrophysiological changes, scar or increased automaticity in the injured myocardium. Arrhythmias can also be occur within the first 20 minutes of fibrinolytics as a reperfusion arrhythmias. However, the diagnosis and management are still challenging, especially in rural area.

Case illustration:
We reported a 40 years old man presented with sudden onset of retrosternal chest pain for 20 minutes. He also has a dyspnea and diaphoresis. His vital sign were stable, no crackles, murmur, gallop, and sign of shock. The ECG showed ST-Elevation in anterolateral leads V1-V5, I, aVL and hyperacute T wave in V4-V6. There is also an ischemic sign in II, III, aVF. Emergency management is focus on reperfusion based on STEMI algorithm. Dual antiplatelet therapy of 320 mg aspirin and 300 mg clopidogrel, sublingual nitrate, fondaparinux, atorvastatin and also fibrinolytic therapy were given as a reperfusion strategy due to >120 minutes for referral to PCI center in Kupang. Fibrinolytic checklist has been carried out and start with strict monitoring sign of bleeding, arrhythmia, shock, and allergy. During the fibrinolytic therapy, the monitor showed frequent PVCs and non-sustained VT. The ECG directly and six hour after fibrinolytic therapy showed no decrease ST-elevation >50%, but there is no pathological Q wave. The chest pain has gone, indicating successful fibrinolytic therapy. However, PVCs still occur frequently after fibrinolytic, hence a 5 mg bisoprolol was given. Amiodarone was about to be given but no longer after that, the PVCs was gone. The patient was referred to RSUD Yohannes Kupang to do early PCI.

Conclusion:
Myocardial infarction can induce PVCs leading to life-threatening ventricular arrhythmia due to automaticity or triggered activity in the region of ischemia or infarction. However, these arrhythmias could be appears as a reperfusion arrhythmia and beneficial sign of reopening of the infarct vessel by solution of the occluding clot. The antiarrhythmic drugs still can be considered wisely with their beneficial and potential harm effect.

Keywords: fibrinolytic, Ventricular Tachycardia, STEMI

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INHERITED STANFORD A DEBAKEY I AORTIC DISSECTION: UNVEILING GENETIC PREDISPOSITIONS AND SURGICAL MANAGEMENT

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Background:
Aortic dissection refers to the disruption of the medial layer of the aorta caused by intramural hemorrhage. This leads to the separation of the layers of the aortic wall, resulting in the establishment of a true lumen and a false lumen, which may or may not be connected. The prevalence of aortic dissection ranges from 0.2% to 0.8%. Additionally, a notable percentage, surpassing 20%, of individuals exhibit a familial inclination towards thoracic aorta dissection

Case illustration:
A male patient in his middle age, 41 years old, with risk factors uncontrolled hypertension, obesity, and a familial propensity to aortic dissection, presented to the emergency department exhibiting symptoms of chest pain. It characterized the sensation as having a tearing quality and indicated that it extended towards the abdomen region. The results of the test revealed several notable findings. A blood pressure reading of 235/133, A wider mediastinum and cardiomegaly on the chest X-ray, slightly increased of Hs-Troponin levels, and ECG showed sinus rhythm with LVH. He underwent a CT scan, which revealed the existence of an aortic dissection that extended from the ascending to descending aorta without any aortic valve involvement. He was assessed with Stanford A DeBakey I Aortic dissection and underwent a successful therapy for aortic arch replacement with the elephant trunk procedure and plans to undertake an endovascular procedure for the descending aorta in the upcoming period.

Conclusion:
Aortic dissection is a pathological disorder that may exhibit hereditary inheritance, and The choice of surgical technique is dependent on the specific underlying disease.

Keywords: Total aortic arch replacement, Dissection flap, Aortic dissection

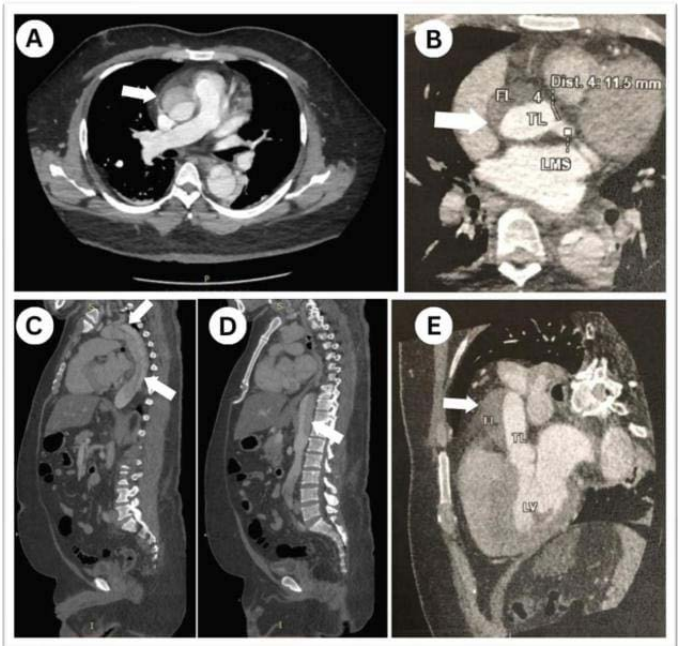

Figure 1. The CT scan reveals the presence of an intimal flap in the ascending aorta, as depicted in images A, B, and C from both axial and coronal perspectives. Image B illustrates the intimal flap extending from the aortic arch

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to the descending thoracic aorta. The intimal flap at the level of the abdominal aorta is demonstrated in image B. Based on these CT scan findings, we can conclude that this is a case of Stanford A DeBakey I aortic dissection.

Indonesian Journal of Cardiology
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SUCCESSFUL AMIODARONE IN A PATIENT WITH AN UNSTABLE VENTRICULAR TACHYCARDIA : A CASE REPORT

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Background:
Sustained monomorphic ventricular tachycardia (VT) can result in hypoperfusion or devolve into more dangerous rhythms such as ventricular fibrillation. In an unstable patient with VT and a pulse, synchronized cardioversion is the first-line treatment. When the VT is refractory to standard cardioversion, the next step is to add an antiarrhythmic, such as amiodarone, that carries with it the risk of lowering blood pressure in the already hypotensive patient. Here we describe a case of successful amiodarone in the treatment of patient with unstable VT, resulting in conversion to sinus rhythm and return to hemodynamic stability, without synchronized cardioversion.

Case illustration:
A 69 years old man came to the emergency department for evaluation of palpitation, chest pain and tightness as well as dyspnea. He developed palpitation two hours prior to presentation. His past medical history included hypertension and coronary artery disease.On examination, he was unconscious with blood pressure 89/59 mmHg, a regular pulse at 180 beats per minute (bpm) and oxygen saturation 98 %. The electrocardiogram showed Monomorphic Ventricular Tachycardia, heart rate 180 bpm and no capture or fusion beats. Therefore our working diagnosis was Unstable Ventricular Tachycardia.Planning for the patient was electrical cardioversion but the family didn't agree. Therefore the patient was treated with Amiodarone 300 mg intravenous in 50 ml D5% for one hour. ECG still revealed Monomorphic Tachycardia. The next treatment was amiodarone 150 mg intravenous every five minutes until 750 mg or five times. ECG still revealed Ventricular Tachycardia. The patient was transferred to intensive care unit for further observation with Amiodarone 600 mg for 6 hours. After 6 hours, ECG confirmed a sinus rhythm (Figure 2), blood pressure 128/64 mmHg, heart rate 65 bpm, and his mental status returned to his baseline.

Conclusion:
We describe a case of successful Amiodarone in a patient with an unstable VT with a pulse. Amiodarone intravenous with dose until 1650 mg was succeeded to return normal heart rhythm. Amiodarone could be solution for the patient that rejected synchronized cardioversion which could also cause adverse event such as skin burn, stroke or cardiac arrest.

Keywords: synchronized cardioversion, ventricular tachycardia, amiodarone

Indonesian Journal of Cardiology
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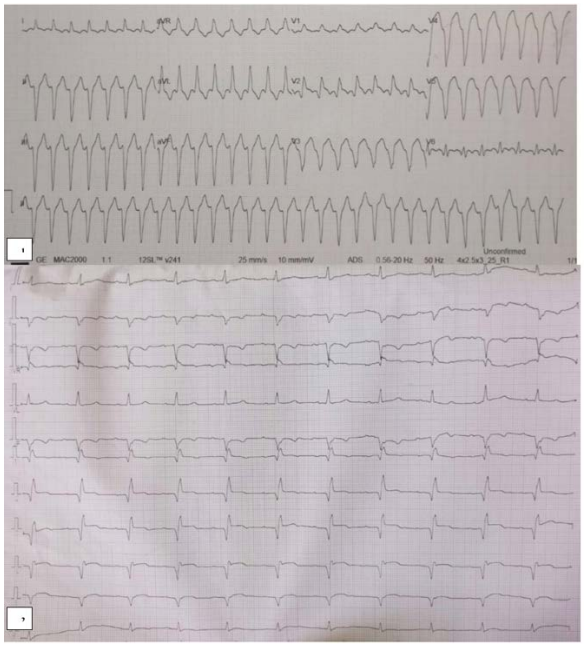

Figure 1. Electrogram before Amiodarone 2. Electrocardioogram after Amiodarone

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STEMI WITH SEVERE ANEMIA ET CAUSA GI BLEEDING

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[1]RSUD H Abu Hanifah

Background:
In patients with acute coronary syndrome, anemia can lead to poor prognosis. Currently, there are no specific recommendations for the management of STEMI in patients at high risk of bleeding due to lack of data.(1)

Case illustration:
A 63-year-old man came to the ER with complaints of weakness (+) since 2 am (4 hours before admission). Shortness of breath (+) since yesterday. Cold sweat (+). Chest pain has been felt intermittently since yesterday. The patient looks pale. Melena with blood clots (+) frequency 3 times today and yesterday. Nausea (+), swelling in the face and legs for the past week. The patient has a history of uncontrolled hypertension, no known history of diabetes. Vital signs: BP 111/80 mmhg, HR 74x/minute weak pulse, RR 24x/minute, T 36.2℃ SpO2 94% room air and Sp02 98%,with oxygen nasal canul 4lpm On significant physical examination found, anemic conjunctiva (+), epigastric tenderness, accompanied by swelling in both legs.Laboratory result : HB: 4.4 mg/dl , WBC : 14.400u/L, RBC 1.5m/uL, PLT 418.000, HCT 14%, Blood glucose Level : 450mg/dl

Conclusion:
Anemia is independently associated with major bleeding, the frequency of which increases with decreasing hemoglobin levels.(1) Primary PCI should be the standard procedure in patients with STEMI who are being considered for reperfusion strategies if they are at high risk for bleeding.(2) Guidelines recommend dual antiplatelet therapy (DAPT) with aspirin and an adenosine diphosphate (ADP) receptor blocker along with parenteral anticoagulation in patients undergoing primary PCI. Patients may be given aspirin and clopidogrel first.(2)Patients undergoing percutaneous coronary intervention (PCI) are at very high risk for bleeding, and they often have anemia secondary to comorbidities. In the management of anemia in patients with coronary artery disease (CAD), the role of blood transfusion therapy remains somewhat controversial, with increasing evidence suggesting that it may be harmful.(3)

Keywords: stemi, severe anemia

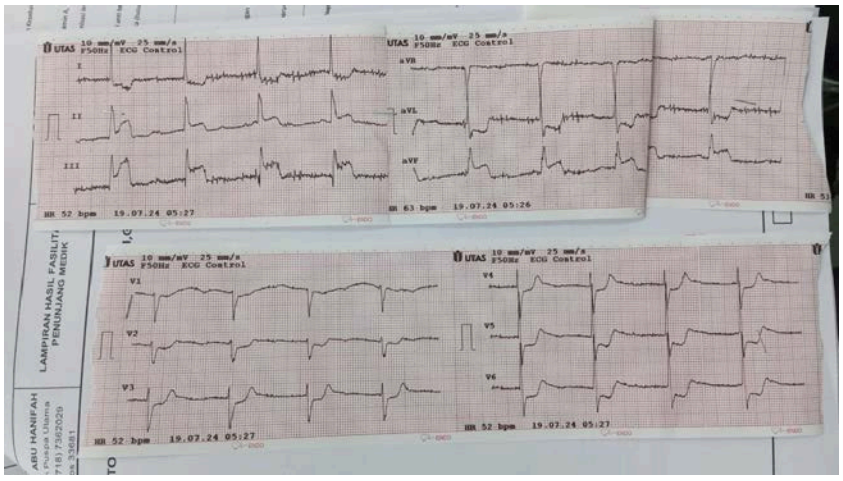

Figure 1. ECG patient

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A SUCCESSFUL LIVE SAVING RESCUE IN ACUTE MYOCARDIAL INFARCTION WITH MALIGNANT ARRHYTHMIA

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Background:
Electrical storm (ES) is a heterogeneous clinical emergency that can present with malignant ventricular arrhythmias such as ventricular fibrillation (VF), ventricular tachycardia (VT), requiring electrical defibrillation or electrical cardioversion. ES is a life-threatening condition with a high mortality rate. Successfully managing ES in the setting of acute myocardial infarction (MI) is expected to be known by physicians on call to reduce in-hospital mortality.

Case illustration:
A 46-year-old patient with cardiovascular risk factors, including pre-diabetes and active smoker, was admitted to our emergency department, complaining of a sudden, severe chest pain, radiating to his arms associated with dispneu and palpitations. Initial vital parameters were as follows: pulse of 253 beats/min, respiration rate of 36 cycles/min, blood pressure of 43/32 mmHg, and oxygen saturation of 70%. Electrocardiogram (ECG) showed a ventricular tachycardia (VT) at a rate of 215 beats/min. Initial laboratory tests were as follows: elevated troponin I, 4.11 µg/mL; aspartate aminotransferase, 166.5 U/L; alanine aminotransferase, 205.4U/L; serum potassium, 4.61 mmol/L. The patient was diagnosed with NSTEMI very high risk. Being hemodynamically unstable, the patient received synchronized direct current (DC) cardioversion with restoration of PVC bigeminy. Bedside echocardiography revealed global hypokinesia with LVEF 44%. Amiodarone protocole was administrated. After consideration, patient was then referred to coronary angiography capable center for immediate invasive strategy.

Conclusion:
ES represents an arrhythmic emergency with a high likelihood of substantial morbidity and mortality. Multiple underlying mechanisms and triggers can be evoked, and acute myocardial infarction should be ruled in depending on the context. Management of intractable arrhythmias requires the teambased approach including specialists in acute cardiac care, interventional cardiology and electrophysiology.

Keywords: Myocardial infarction, Ventricular Tachycardia, Electrical cardioversion

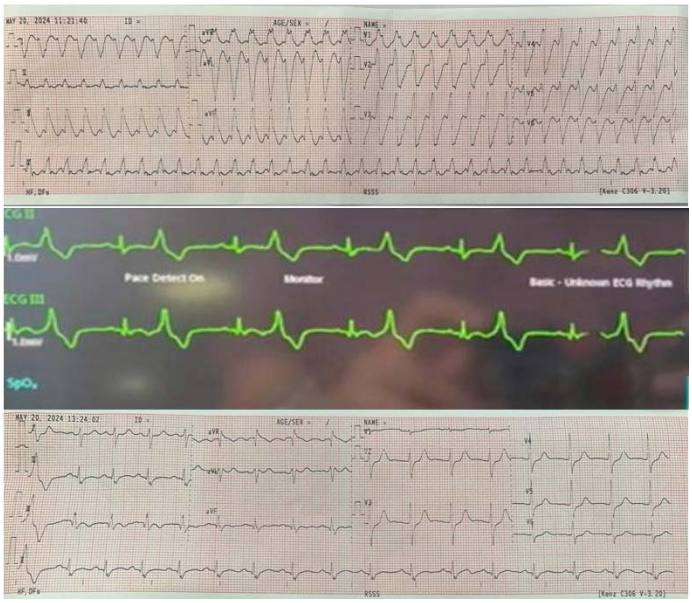

Figure 1. ECG before and after cardioversion

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PERIPARTUM CARDIOMIOPATHY IN A YOUNG INDONESIAN WOMAN : A CASE REPORT

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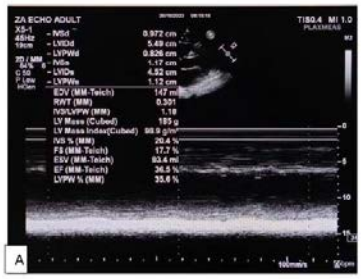
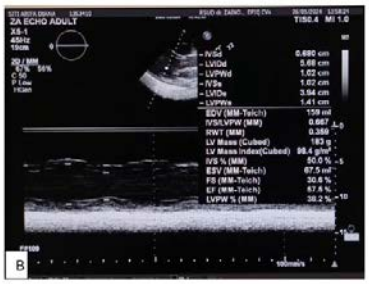
Background:
Peripartum cardiomyopathy is a type of systolic heart failure that affects young women, typically occurring near the end of pregnancy or in the months following childbirth. The symptoms of heart failure in peripartum cardiomyopathy can be similar to those of a typical pregnancy, often leading to delayed diagnosis and preventable complications. The essential diagnostic tool is echocardiography, which reveals a decrease in myocardial function.

Case illustration:
A 29-year-old woman presented at the ER of RSU Zainoel Abidin with a history of worsening shortness of breath over the past 2 weeks, worsened during light physical activity and noted that she became fatigued quickly. These symptoms began approximately 2 months ago, around 1 month after she had given birth to her child via a caesarean section. On admission, patients vital sign mostly in normal limits, other than heart rate of 117 bpm and respiratory rate of 24 breath pe minute. Physical examination reveals edema on the right leg. The chest x-ray showed The heart appears to be enlarged, while the aorta and superior mediastinum are not dilated . The transthoracic echocardiography (TTE) showed an ejection fraction (EF) of 36%, a left ventricular internal diameter in diastole (LVIDd) of 54 mm, and a tricuspid annular plane systolic excursion (TAPSE) of 1.8 cm. After receiving treatment in the cardiac ward, the patient displayed a progressive reduction in symptoms related to shortness of breath. On follow up examination, transthoracic echocardiography of the patient shows an increment of ejection fraction to 57% on her second echocardiography examination and 55% on her third.

Conclusion:
The consideration of peripartum cardiomyopathy (PPCM) should arise in pregnant or postpartum women displaying heart failure-like symptoms. Even when up until now, the pathogenesis is not fully know due to the rarity of this case

Keywords: Heart Failure, Young Woman, Pregnancy, Peripartum Cardiomyopathy





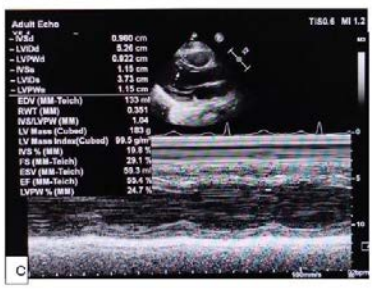


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Figure 1. Following therapy, the patient's ejection fraction increased gradually, rising from 36% to 57% on her second echocardiography but slightly decreased to 55% on her third. A. October 2023, B. January 2024, C. May 2024

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NOT ALL ST-ELEVATION ARE CAUSED BY INFARCTION: THE STORY OF HYPERKALEMIA

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Background:
Hyperkalemia is a life-threatening electrolyte abnormality associated with the increasing prevalence of medical management of hypertension and cardiovascular disease. The ability to evaluate the ECG of hyperkalemia is vital in emergency settings.

Case illustration:
A 60-year-old woman was admitted to the ER with loss of consciousness, vomiting, and weakness for 2 days, and difficulty eating. She had a history of hemorrhagic stroke and hypertension for 1 month. She was on treatment with nimodipine and candesartan 16 mg daily. The patient's consciousness was somnolent. BP was 80/50 with signs of severe dehydration and an unremarkable cardiac exam other than cardiomegaly. ECG showed 1st degree AV block with diffuse ST elevation and peaked T waves in all leads. Laboratory results showed potassium of 8.79 mmol/L. The patient was rehydrated and given serial hyperkalemia correction with IV calcium and insulin. Serial ECGs showed resolution of ST elevation and peaked T waves in line with the normalization of potassium levels. Echocardiography showed concentric LVH with preserved LVEF.ECG changes in hyperkalemia cases occur along with potassium levels. The ECG manifestations of hyperkalemia include peaked T waves, progressive prolongation of the PR interval, widened QRS complexes (sine wave), and decreased amplitude and/or eventual loss of the P waves followed by ventricular fibrillation or asystole. It is necessary to start treatment for hyperkalemia if ECG changes are detected. There are several differential diagnoses of ST elevation on ECG other than hyperkalemia, namely STEMI, early repolarization, pericarditis, and ST elevation secondary to an abnormality of the QRS complex (left bundle branch block, left ventricular hypertrophy, or preexcitation) with their own clinical pearls.

Conclusion:
It’s important to be aware of other possible causes of ST elevations in ECG and match them with the clinical presentation. Evaluate these findings accurately and treat them in a timely manner to prevent potentially life-threatening conditions.

Keywords: ECG

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THE IMPORTANCE OF ECHOCARDIOGRAPHY DIAGNOSTIC APPROACH IN CARDIAC IMPLANTABLE ELECTRONIC DEVICE (CIED) INFECTIVE ENDOCARDITIS (IE)

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[1]PJNHK

Background:
Pacemakers are potentially life-saving treatments for certain cardiac conditions but are not without risk. The most worrisome is the potential of a cardiac implantable electronic device (CIED) infection, which is linked to significant morbidity, increased hospitalizations, reduced survival, and increased healthcare costs. Early aggressive treatment of such infections and lead management could prevent unfavorable outcomes. Imaging is an important tool in diagnosing and managing infective endocarditis (IE). Echocardiography is an essential examination and the first to be performed as soon as IE is suspected.

Case illustration:
A 78-year-old male was admitted to the emergency ward with chief complaint fever and headache. The patient with past medical history of recurrent syncope ec Mobitz II 2nd degree AV block (2:1 ratio) degenerative status-post PPM single chamber placement 2 months ago. A month after PPM placement, the patient began to have fever, headache, breathlessness, pain and oedema in the PPM area. The patient was hospitalized and underwent Transthoracic Echocardiography (TTE) that showed no vegetation in PPM lead but refused to do transesophageal echocardiography (TEE). He was diagnosed with Possible Infective Endocarditis (IE) as the blood cultures were positive Staphylococcus aureus. He got intravenous (IV) antibiotics treatment for 14 days and was discharged with oral antibiotics for another 2 weeks. The patient got rehospitalization 2 weeks after being discharged with the same complaint. The 12-lead electrocardiogram showed ventricular pacing of 62 bpm. A follow-up TTE and TEE revealed the presence of vegetations in the PPM lead.

Conclusion:
This case highlights the importance of echocardiography as the first examination to be performed for suspected CIED-IE. Careful diagnostic and management of CIED-IE is needed to prevent unfavorable outcomes.

Keywords: pacemaker, infective endocarditis (IE), echocardiography, Cardiac Implantable Electronic Device (CIED)

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THE JOURNEY OF YOUNG WOMAN WITH PINK TETRALOGY OF FALLOT

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Background:
Tetralogy of Fallot (TOF), one of the most common congenital heart diseases, has four major components and various minor associations. The four major components being Right Ventricular Hypertrophy, Overriding Aorta, Ventricular Septal Defect (VSD), and Right Ventricular Outflow Tract (RVOT) obstruction. The severity of the RVOT obstruction determines the direction and the shunt through the VSD. If the RVOT obstruction is mild and VSD is in left to right shunt, the patient might acyanotic TOF or Pink TOF. If more severe obstruction, the shunt is right to left, resulting in cyanotic TOF.

Case illustration:
An 18 years old female with clinical shortness of breath. Physical examination showed stable haemodynamics. Based on echocardiography, it showed the Tetralogy of Fallot where pulmonary valvar stenosis/RVOT obstruction, ventricular septal perimembrane defect, overriding aorta, and right ventricular hypertrophy were found. In this patient, a Right Heart Catheterization (RHC) has been performed and the results of RHC obtained the impression of TOF with valvar pulmonary stenosis. In this case this patient was diagnosed with acyanotic TOF or Pink TOF. A surgical conference was performed and the results of the conference planned to do a total correction. After 6 months from RHC, the patient undergo total correction with VSD surgical closure and commisurotomy at pulmonary valve.

Conclusion:
Pink Tetralogy of Fallot is a mild form of TOF with the less obstruction to blood flow results in better oxygenation. Early diagnosis and appropriate surgical treatment are key to ensuring optimal outcomes and quality of life for affected individuals.

Keywords: pink TOF, total correction

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VENTRICULAR SEPTAL RUPTURE FOLLOWING ACUTE MYOCARDIAL INFARCTION: NIGHTMARE CASES AND MANAGEMENT STRATEGIES

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Background:
Ventricular septal rupture (VSR) is a rare mechanical complication following acute myocardial infarction, with very high mortality rate. Hemodynamic deterioration and cardiogenic shock are common in such cases. Using full mechanical circulatory support to delay VSR Closure is an attractive option, but data on success is limited to case reports.

Case illustration:
A 61-year-old man came to our hospital with late presentation of anterior STEMI. Fortunately, patient came with stable hemodynamics. Echocardiography showed apical VSR 9-11 mm L to R shunt and no sign of chronic pulmonary hypertension. Coronary angiography showed CAD three-vessel disease with critical stenosis at mid-distal LAD. We discussed patient in the heart team multidiscipline. Patient was planned to perform VSR closure percutaneously and continue with PCI procedure due to stable hemodynamics and sufficient rim to deploy device. Both procedures were performed successfully. Patient was improved and discharged on 20th day of admission.In the other case, 60-year-old man presented with late presentation of anterior STEMI complicating hemodynamic deterioration. Echocardiography showed apical VSR 11-14 mm L to R shunt. Patient was given adequate fluids, multiple inotropic agents, and also IABP implantation. Primary PCI procedure was performed immediately. IABP was maintained to delay VSR closure. Unfortunately, the patient worsened due to cardiogenic shock accompanied by other comorbidities and passed away on the 5th day of admission. In another case, 58-year-old man came to our hospital also with late presentation of anterior STEMI complicating hemodynamic deterioration. Echocardiography showed apical VSR 10-12 mm L to R shunt. Cardiogenic shock management was performed, but unfortunately, IABP could not be performed because IABP was used by other patients. Patient worsened due to prolonged shock and multi-organ failure. Patient passed away on the 2nd day of admission.

Conclusion:
Rapid diagnosis and prompt treatment are the keys to optimal management of VSR complicating late presentation STEMI. Immediate revascularization, using full mechanical circulatory support, and correction of VSR are required to optimize patient outcomes despite VSR still having a poor prognosis.

Keywords: ventricular septal rupture, STEMI, cardiogenic shock

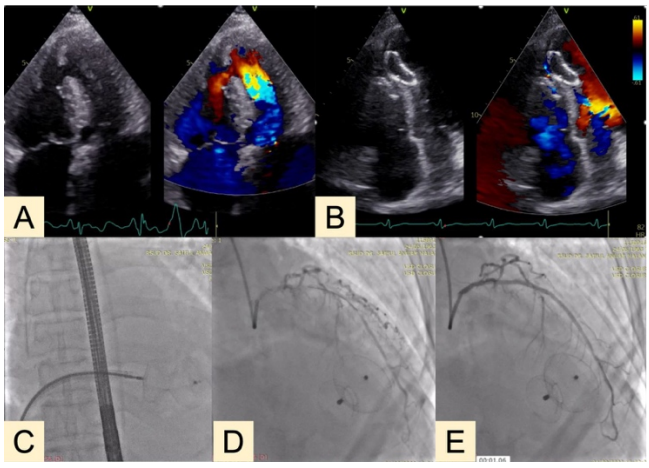

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Figure 1. (A) Echocardiography showed apical VSR. (B) Post-transcatheter VSR closure showed occluder in situ. (C) Fluoroscopy during transcatheter VSR closure. (D) PCI procedure on critical stenosis at LAD. (E) Post PCI showed stenotic lesions improved.

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COMMUNITY-ASSOCIATED METHICILLIN-RESISTANT STAPHYLOCOCCUS AUREUS SEPTICAEMIA IN MASSIVE MANDIBULAR ABSCESS AND ACUTE PERICARDITIS

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Background:
Methicillin-Resistant Staphylococcus Aureus (MRSA) pericarditis is rare. It is associated with nosocomial infections, but has recently become common in the community. MRSA-induced bacterial pericarditis has a higher mortality rate than methicillin-sensitive resistance. We aim to present a rare case of bacterial pericarditis following mandibular abscess in a non-immunocompromised patient.

Case illustration:
A 40-year-old man was consulted to cardiovascular department with sudden short of breath after drainage incision surgery and tooth extraction for a mandibular abscess 2 days prior. He also had cheek pain, fever and chills, without chest pain. He had history of toothache and cavities for a week, and was an active smoker. Examination showed blood pressure 110/72mmHg, heart rate 80 beats/min, oxygen saturation 99% on NRBM, respiratory rate 28x/min, temperature 39°C, VAS 4/10. Right cheek swollen, trismus, pus production on cheek drainage, and normal thorax auscultation were found. Electrocardiography showed diffuse concave ST elevation and reciprocal ST depression in lead avR (Figure 1). The laboratory examination showed elevated white blood cells 50,430/uL, thrombocyte 97,000/uL, serum albumin 2.2 g/dL, normal hs-Troponin I, and elevated C-reactive protein 79 mg/dL. Panoramic x-ray showed perforative pulp dental cavity and periapical abscess on right 3rd molar tooth. Thorax x-ray showed cardiomegaly, left pleural effusion, while blood culture showed MRSA. He received intravenous Ampicillin Sulbactam which was replaced with Meropenem, Albumin 25%, oral ibuprofen, colchicine, captopril, and spironolactone. He was diagnosed with pericarditis, mandibular abscess, and right 3rd molar periapical abscess. Diagnosis of peri-myocarditis could not be made because there was no cardiac MRI. His condition improved and discharged after 10 days of hospitalization.

Conclusion:
Community-acquired MRSA can occur through a local infection with hematogenous spread to the pericardium, burdened by smoking as risk factors for MRSA infection. Colchicine provides an anti-inflammatory effect that halves the risk of recurrent pericarditis. Therefore, prompt and appropriate treatment should be taken to prevent in-hospital mortality and reduce recurrence.

Keywords: sepsis, mandibular abscess, Methicillin-Resistant Staphylococcus Aureus, pericarditis

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TRIPLE THREAT: MANAGING PHLEGMASIA CERULEA DOLENS AND DVT IN MORBID OBESITY

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[1]FK UNAIR, RS DR SOETOMO

Background:
Deep vein thrombosis (DVT) is a significant health concern, especially in people who are morbidly obese. Obesity is a known risk factor for the development of DVT. Phlegmasia is an uncommon, limb-threatening complication of deep vein thrombosis. Although the exact incidence is unclear, phlegmasia cerulea dolens can cause significant morbidity and mortality if not treated promptly. Although no recommendations exist for treating phlegmasia, prompt treatment is required and should focus on reducing venous blockage to prevent development of gangrene. We report the case of a 53-year-old morbidly obese woman who presented with bilateral lower extremities DVT, right lower extremity phlegmasia cerulea dolens and left superior extremity DVT.

Case illustration:
A 53-year-old obese woman (BMI 41.6 kg/m2) presented to the emergency room with swollen, darkened skin on her right arm and left leg, as well as pain and paresis in her right leg. She has a history of untreated hypertension. Vital signs returned normal findings. Upper extremity vascular and oxygen saturation levels were normal. Lower extremity examination revealed a deterioration in vascular status and oxygen saturation on the right leg, as well as partial compression of the femoral and popliteal veins. Echocardiography showed normal results. CT angiography of both lower extremities supports the diagnosis of DVT in bilateral lower extremities. Patient was given anti-coagulant and recommended doing cardiac and physical rehabilitation. Patient then was safely discharged after a 17 days stay.

Conclusion:
Obesity is known to increase the chance of developing DVT. Excess weight and fat in morbidly obese people might cause blood clot development, provided their immobilization state . Phlegmasia cerulea dolens is a rare form of deep vein thrombosis. Recognizing and treating symptoms as soon as possible is critical. Effective prevention and management techniques are required to reduce problems.

Keywords: Deep vein thrombosis, Morbid Obesity, Immobilization, Phlegmasia cerulea dolens

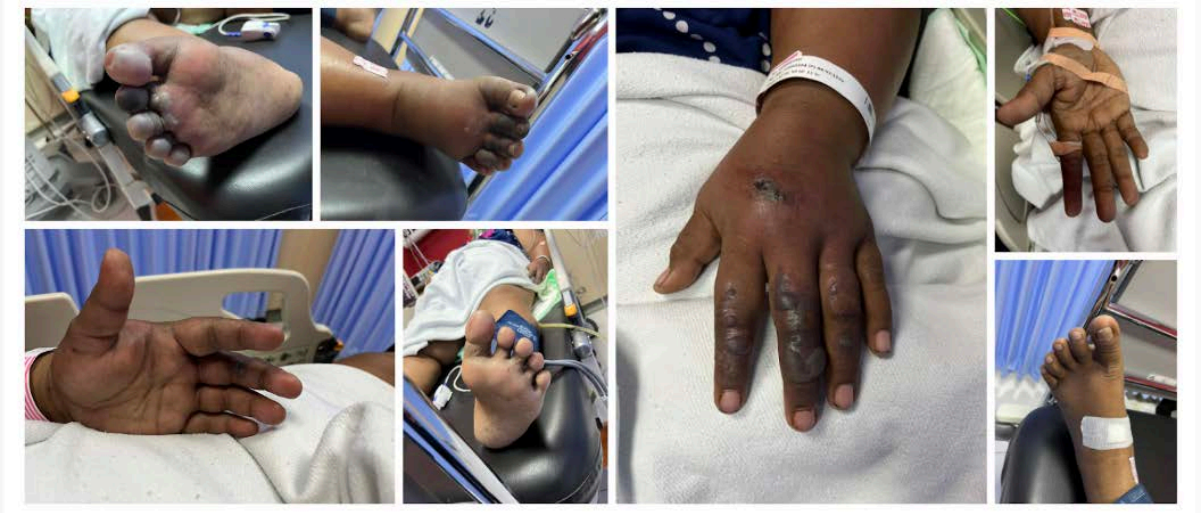

Figure 1. Cyanosis, edema, and blisters on left arm and both legs

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NIGHTMARE IN EMERGENCY ROOM: INFERIOR STEMI WITH BRADYARRHYTMIA – A CASE SERIES

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Background:
Bradyarrhythmia was a common electrical complication in patient with ST Elevation Myocardial Infarction (STEMI). Managing STEMI with electrical complication was challenging in non-PCI capable center. Here we report a case series of two inferior STEMI patients with bradyarrhythmia and how we manage it in our non-PCI capable hospital.

Case illustration:
Case 1: A 66-year-old man with history of type 2 diabetes mellitus came to ER due to general weakness for 15 minutes prior to admission. His vital sign showed BP of 141/74 mmHg with HR 39 bpm. The ECG demonstrated a ventricular rate of 38 beats/min, an atrial rate of 75 beats/min, ST-segment elevation in leads II, III, and aVF, and diagnosed as acute inferoposterior STEMI with Total AV Block.Case 2: A 52-year-old woman with history of type 2 diabetes mellitus came to ER due to nausea and vomiting for 5 hours before admission. Patient were hypotensive with BP of 86/53 with HR 36 bpm. ECG showed ventricular rate 38 bpm, no P wave observed, with ST elevation in II, III, aVF lead and diagnosed as Acute inferior STEMI with junctional bradycardia and hemodynamic instability. Both patients underwent fibrinolytic with Streptokinase and administered 3 mg of Sulphas Atropine, 20 mcg/kgBW/min Dopamine in IVSP, and Salbutamol 8 mg tid. After 3 days in ICU, the first patient's ECG converted to sinus rhythm with normal heart rate without any medical support. As for second patient, the ECG changed to total AV block and still in need of Salbutamol until referred to tertiary hospital. Both patients were referred to PCI capable center.

Conclusion:
Inferior STEMI often present without typical chest pain, and often accompanied with electrical complications. Quick and accurate identification of inferior STEMI and potential electrical complications is necessary to provide appropriate treatment and reduce mortality.

Keywords: Complete Heart Block, ST Elevation Myocardial Infarction, Junctional Bradycardia, Bradyarrhythmia

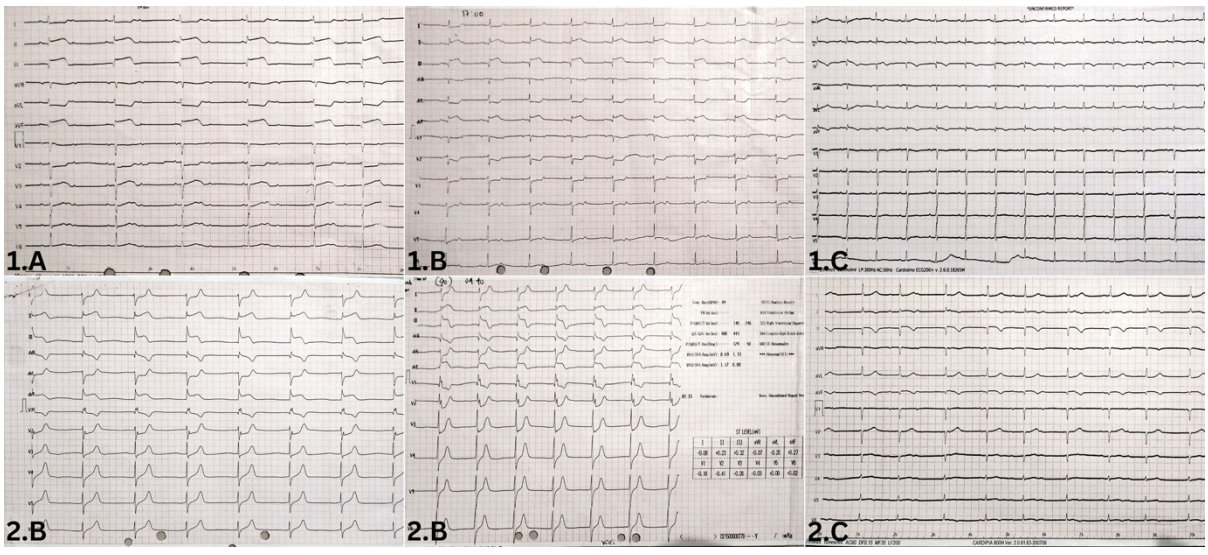

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Figure 1A-1C ECG from patient A showed Inferior STEMI progressed after fibrinolytic from Total AV block to 1st Degree AV Block 2A-2C ECG from patient B showed Inferior STEMI with junctional bradycardia turned to Total AV Block after fibrinolytic

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PERIPHERAL ARTERY THROMBOSIS LEADING TO LOWER LIMB AMPUTATION IN CONGENITAL HEART DISEASE : A COMMONLY FORGOTTEN, YET PREVENTABLE COMPLICATION

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Background:
Peripheral artery thrombosis is a common complication of uncorrected cyanotic congenital heart disease (CHD). Structural lesions in CHD can cause turbulence in blood flow and stasis, leading to intracardiac thrombuses. These thrombuses can escape to peripheral arteries, causing peripheral artery thrombosis, a potentially catastrophic event for the child.

Case illustration:
A 2-year-old male baby came to our emergency ward with chief complaints of left lower limb becoming darker and can't be moved at the level of below the knee since 2 days prior of emergency ward admission. Previously he was diagnosed with cyanotic congenital heart disease (cc-TGA), VSD, severe infundibular PS, and dextrocardia with situs solitus. He was born as an omphalopagus twin in 2020 and separated at 14 months without major complications. An emergency ward physical examination revealed left lower extremities with irreversible ischemic segment below the tibial tuberosity. Doppler ultrasounds revealed a thrombus at the level of the femoral artery with critical occlusion of the arterial lumen. A multidisciplinary team, including pediatric, vascular, orthopedics, plastic surgeons, and pediatric cardiologists, was formed to discuss the appropriate management approach. The decision was for trans-tibial limb salvage of the left lower limb, along with open surgery thrombectomy of the occluded femoral artery. No major complications occurred after surgery, and the patient continued on an increased dose of anti-failure medication and orthotics-prosthetics installation.

Conclusion:
Cyanotic congenital heart disease is a significant risk factor for peripheral artery thrombosis in pediatrics. The difficulties in predicting thrombosis events leading to clinicians' dilemma in prophylaxis anticoagulants treatments due to high bleeding risk and unclear benefits. Thorough, strict, and routine assessment is needed to prevent catastrophic events affecting children's future life stages.

Keywords: limb salvage, peripheral artery thrombosis, Congenital heart disease

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UNEXPLAINABLE HYPOKALEMIA AND RECURRENT CARDIAC ARREST: A CASE REPORT OF A NINE-TIME CARDIAC ARREST SURVIVOR

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Background:
Uncorrected hypokalemia can lead to malignant arrhythmias and is among the most common causes of cardiac arrest. Typically, potassium levels below 3.0 mmol/L require correction to prevent cardiac complications. However, the situation can vary by case.

Case illustration:
A 73-year-old man presented to the ER complaining of headache and was admitted without chest pain, diarrhea, nausea and vomiting, or dyspnea. He had a hypertensive urgency that was controlled after two rounds of oral anti-hypertensive drug. Initial tests showed mild hypokalemia (3.3 mmol/L), mild anemia (Hb 11.3g/dL), and cardiomegaly, otherwise normal results. Within an hour of admission, he had a seizure followed by two cardiac arrests, both successfully resuscitated after multiple rounds of CPR and epinephrine injection. He was intubated and transferred to the ICU.In the ICU, he experienced three more cardiac arrests due to PVCs progressing to pulseless ventricular tachycardia, each restored to sinus rhythm after CPR and defibrillation. Blood tests revealed worsening hypokalemia (2.7 mmol/L), positive troponin I (1758ng/L), and rising d-dimer levels. Brain CT-Scan showed no infarct or signs of increasing intra-cranial pressure. The source of hypokalemia progression was not determined, as there was no fluid loss from the gastrointestinal, and regular urine output from the urinary tract during the four-hour time gap. Patient was assessed with NSTEACS with hypokalemia. Treatment included electrolyte correction, dual antiplatelet therapy, anticoagulation, and amiodarone protocol.Despite these interventions, he had four more cardiac arrests beginning with similar PVC patterns, necessitating additional potassium correction through oral and central venous catheter. Plans for early PCI were delayed due to guardianship concerns. Echocardiography on day four showed anteroseptal hypokinesis and an ejection fraction of 61% (Teicholz). He improved gradually, was extubated by day five, transferred to a regular ward by day six, and discharged on day eleven.One-month follow-up echocardiography showed improved ejection fraction (72%, Teicholz) with normal ventricular function and residual left ventricular hypertrophy. The patient is currently scheduled for follow-up Coronary CT-Angiography.

Conclusion:
We presented a case of nine-time cardiac arrest survivor induced by hypokalemia and NSTEACS, with improved follow-up condition. Rapid electrolyte correction might be beneficial in recurrent cardiac arrest.

Keywords: ACS, Cardiac Arrest, Hypokalemia, NSTEACS

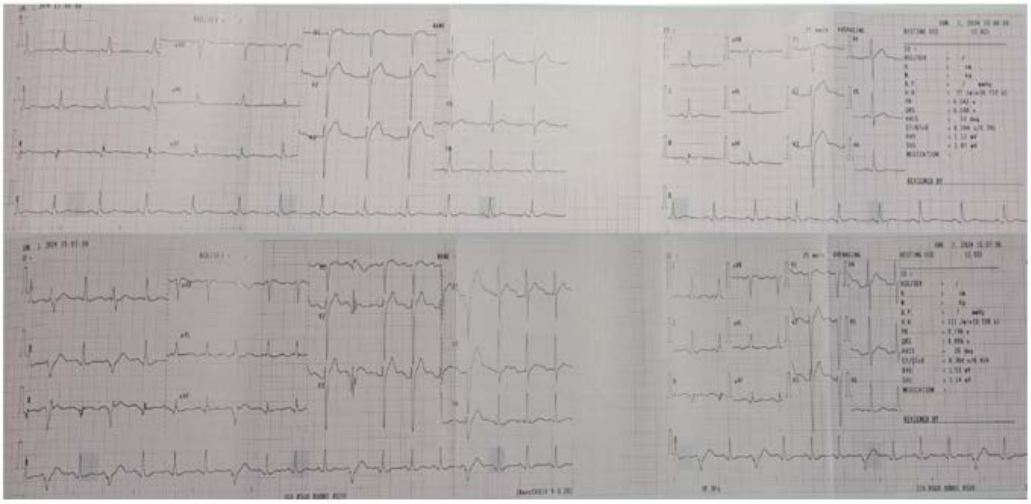

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Figure 1. 7-minute difference ECG which later develop to ventricular tachycardia

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TAILORED STRATEGIES FOR LEFT VENTRICULAR THROMBUS FOLLOWING MYOCARDIAL INFARCTION: A CASE REPORT

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Background:
Left ventricular (LV) thrombus was a serious complication following myocardial infarction (MI), particularly in cases of anterior MI. This condition could lead to life-threatening embolic events, necessitating prompt diagnosis and effective management. Although anticoagulation therapy was recommended, there is no standardized treatment guideline, making individualized patient care and regular monitoring essential.

Case illustration:
A 46-year-old man was admitted to our hospital and diagnosed with Recent STEMI anterior onset 1 week Killip II TIMI 7/14, CHF fc class III ec CAD. A 12-leads Electrocardiogram (ECG) showed Sinus tachycardia, QRS rate of 107 beats per minute, normal heart axis, normal P wave, PR interval 160 ms, QRS duration 10 ms, Q pathologist with elevation ST segment 1-2 mm in V1-V6, LVH (-), RVH (-), QTc 474 ms. Laboratory finding revealed Troponin I 173 ng/L. The full study echocardiography showed reduced systolic LV function with ejection fraction 18% (Simpson), akinetic basal-mid anteroseptal, anterior basal-mid, mid anterolateral, broad apical, basal-mid inferoseptal, basal-mid inferior, other segments are hypokinetic, Sec (+) in LV, thrombus (+) large in apical size 45 x 58 mm (area 23 cm^2). We treated the patient with low molecular weight heparin, double antiplatelet, along with VKA and optimized heart failure therapy. On the sixth day, the patient complained bloody cough with 1-2 tablespoons per cough, as we checked the INR value in this patient was 6,34. We stopped the warfarin, stopped ASA, continued clopidogrel 1x75 mg, and monitored the bleeding. We got the targeted INR value (2-3) along as we gave the warfarin within 1x2 mg and no bleeding occurred. On the thirteenth day, the patient was stable, the patient was then discharged.

Conclusion:
LV thrombus formation post-MI was a critical condition that requires a multifaceted approach for effective management. This case highlights the importance of individualized treatment plans, regular monitoring, and patient education in improving outcomes. Although anticoagulation therapy is a cornerstone of treatment, the lack of standardized guidelines necessitates a tailored approach based on the patient's specific clinical scenario. Ensuring patient compliance with medication and lifestyle modifications is crucial for preventing recurrence and enhancing long-term prognosis.

Keywords: Left ventricular thrombus, myocardial infarction

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DIAGNOSED AS NSTEMI BUT FOUND TO HAD TOTAL OCCLUSION: A SUPPORT FOR A NEW PARADIGM

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Background:
Under the previous STEMI vs. NSTEMI paradigm, 25–30% of NSTEMI had unrecognized acute total occlusion discovered on delayed angiogram and approximately double short and long-term mortality compared to NSTEMI patients with Non-Occlusion MI (NOMI). Conversely, 15–35% of cath lab activations due to perceived STEMI criteria were found to be false positives without even a culprit lesion. The ACS spectrum using the Occlusion MI (OMI) vs NOMI paradigm primarily consisted of STEMI (+) OMI, STEMI (-) OMI, STEMI (+) NOMI, and STEMI (-) NOMI. This paradigm proposed eight OMI ECG findings which indicated a high likelihood of OMI in addition to obvious STE.

Case illustration:
A 50-year-old man with chief complaint of typical chest pain onset 9 hours ago. Physical examination revealed normal blood pressure, normal cardiac and pulmonary exams. ECG showed normal sinus rhythm, normal axis, subtle ST elevation with pathologic Q wave in inferior leads, dominant R wave with ST depression in V1-3 leads, and T wave inversion in V4-5. Laboratory findings showed leukocytosis and significant elevation of troponin I (TnI > 25.00 ug/L). The patient was initially diagnosed with high-risk NSTEMI and planned for invasive strategy (PCI). Follow-up echocardiography showed normal left ventricular geometry, regional wall motion abnormalities, 46% ejection fraction by biplane, grade III LV diastolic dysfunction, and normal right ventricular systolic function. Coronary angiography revealed total occlusion in proximal LCx, 90% stenosis in proximal RCA, multiple significant stenoses in LAD (70% in proximal and 70-80% in mid-distal), and 80% stenosis in Diagonal 1.

Conclusion:
STEMI (-) OMI patients experienced significant delays to cardiac catheterization but exhibited similarly severe clinical, angiographic, and laboratory features as the STEMI (+) OMI group when compared to the No Occlusion group. STEMI (-) OMI patients were an under-identified population with the potential to benefit from emergent intervention, and our results suggested that they could be rapidly and noninvasively identified using OMI ECG findings. This case supported that OMI ECG findings were superior to STEMI criteria.

Keywords: STEMI; NSTEMI; Occlusion MI (OMI); Non-Occlusion MI (NOMI); OMI vs NOMI paradigm

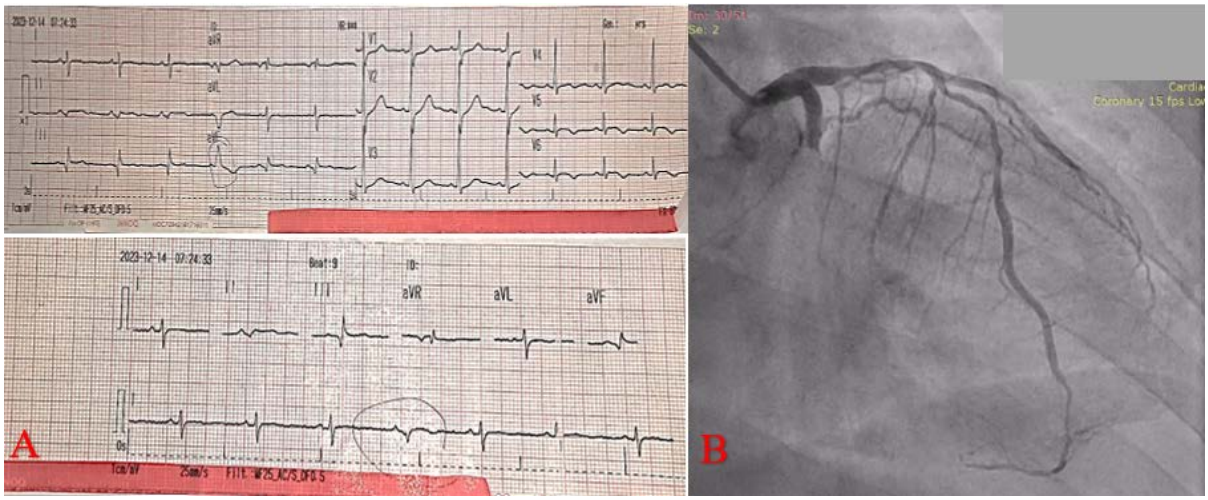

Figure 1. (A) Electrocardiographic finding of onset 9 hours and (B) coronary angiography showed total occlusion in proximal LCx

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A DOUBLE STRIKES AORTIC ENDOCARDITIS WITH ACUTE LIMB ISCHEMIA : TACKLING THE TOUGHEST DISEASE CHALLENGES IN EAST NUSA TENGGARA

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Background:
Aortic endocarditis is a deadly disease, despite optimal care, the mortality approaches 30%, it is heterogenous in etiology, manifestation, and course. Acute Aortic Endocarditis with Heart Failure and vascular phenomenon should be operated immediately. This is challenging in East Nusa Tenggara due to limited cardiac surgery facilities.

Case illustration:
A 37 years old male referred to the emergency room of Ben Mboi General Hospital suspected with ACS due to shorthness of breath, changes in ECG, and positive HS-Troponin value. Presenting in Ben Mboi, we found painful, black discloration on the left hand fingertips, leading to Acute Limb Ischemia, so we suspected other causes. Cardiac Auscultation revealed Austin Flint murmur. Echocardiography examination showed vegetation on the aortic valve, causes Acute Aortic Endocarditis with Heart Failure and vascular phenomenon thus, emergency surgery is mandatory per guideline. Due to lack of facilities, we optimized conservative therapy with heart failure regimen, empirical antibiotics, and supportive therapies. The patient responded well, thus providing preparation time for referral. Patient then referred for surgery, during the period, he experienced slight hemiparesis. Non-contrast Head CT-scan showed subacute cerebral infarct, possibly a complication of endocarditis, so collaboration with neurologist was performed. The patient then underwent Aortic Valve Replacement surgery with mechanical valve SJM, complicate to cardiac tamponade post surgery and handled with pericardiocentesis. Post-operative TEE was performed and obtained good results. Patient then discharged with an improvement of condition and now underwent routine control at Ben Mboi Hospital.

Conclusion:
Aortic Endocarditis is a deadly disease with many clinical manifestation, which has the proclivity to cause complications both at the cardiac and extracardiac site. The preparation for surgery is never easy, optimizing conservative therapy is the key for maintain patient's clinical condition before definitive therapy is initiated. Definitive therapy often followed by catastrophic events such as in this case.

Keywords: Aortic Endocarditis, Aortic Valve Replacement, Acute Limb Ischemic, Stroke

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UNVEILING PROBLEMATIC TRUTH BEHIND RECURRENT NSTEMI: TYPE 2 MYOCARDIAL INFARCTION RELATED TO PULMONARY ARTERIOVENOUS MALFORMATION

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Background:
Type 2 Myocardial Infarction (T2MI) prevalence is about one fourth of Type 1 Myocardial Infarction (T1MI). Studies show that short and long term all cause mortality of T2MI are higher than T1MI due to its provoking factors of supply/demand mismatch and multiple comorbidities. Rising awareness in T2M1 is important in order to treat the precipitating condition accurately.

Case illustration:
A 75-year-old woman who lived in mountain area was referred to outpatient-clinic with history of recurrent NSTEMI (June and October 2023) and hypertension. She had chest discomfort and cough with room air oxygen saturation was 65-70% without any sign of respiratory distress. She had BP 153/88 mmHg, pansystolic murmur 4/6 at left lower sternal border, crackles and wheezing on both lung fields with peripheral sign of congestion, and no clubbing fingers was noted. The ECG was sinus rhythm with ST depressions and T wave inversions at inferior and anterior leads. The CXR showed cardiomegaly with rounded apex, oligemic, and inverted comma sign. There were elevated Troponin I above upper normal limit and slight azotemia. Blood Gas Analysis showed compensatory respiratory acidosis with hypercapnic state. Echocardiography showed RA-RV dilatation with D-shaped concentric LV during systolic and diastolic, LVEF 78.1%, TAPSE 18.8mm, moderate TR and PR, and high probability of pulmonary hypertension. Triple rule out CT Angiography revealed severe calcium plaque burden without any significant stenosis, RA-RV dilatation, and we also found complex Pulmonary Arteriovenous Malformation (PAVM) at inferoposterior right lung field and sign of pulmonary hypertension. Our final diagnoses were complex PAVM with pneumonia that contribute to T2MI, isolated right heart failure, stable CAD, and hypertension. We gave iv loop diuretic, spironolactone, ARB, statin, bronchodilator, and antibiotic. Patient was discharged in stable condition with room air oxygen saturation 82%. Further intervention of PAVM was indicated because of the symptomatic hypoxemic condition. However, geriatric condition can complicate the intervention. We suggested the patient to live in lowland area and to continue conservative therapy with routine medication.

Conclusion:
We should rise awareness that elevated troponin is not always related to coronary artery disease. Multimodality imaging can help us to determine the correct etiology.

Keywords: recurrent NSTEMI, Type 2 Myocardial Infarction, Pulmonary Arteriovenous Malformation

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LEFT MAIN CORONARY ARTERY OCCLUSION WITH CARDIOGENIC SHOCK ON A RELATIVELY YOUNG PATIENT AT A SECONDARY HOSPITAL : A CASE REPORT

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Background:
Left main coronary artery (LMCA) occlusion is a rare but severe condition often leading to acute myocardial infarction (AMI) and cardiogenic shock (CS). This condition is associated with high mortality rates and presents significant challenges in management and treatment especially in secondary hospital settings.

Case illustration:
A relatively young, 41-year-old female without any atherosclerotic heart disease risk factor presented to the emergency department of a secondary hospital with typical chest pain and diaphoresis onset an hour before. Vital signs showed low blood pressure on the 80/palpation, cold extremities, and a weak pulse. ECG results showed ST depression on II, III, aVF, V4, V5, and V6, and ST elevation on aVR lead, and the laboratory results showed an increased CK-MB. The patient was treated with a loading dose of aspilet and clopidogrel, started by loading IV fluid, and then a norepinephrine infusion was started. After vital signs were stable, the patient was immediately referred to a tertiary hospital for primary PCI.

Conclusion:
Left main coronary artery occlusion with cardiogenic shock is a critical condition with high mortality rates. Early and aggressive intervention, including PCI and mechanical circulatory support, can improve immediate survival, but long-term outcomes remain poor. Predictors of poor prognosis include severe left ventricular dysfunction and cardiac arrest during PCI. Further research is needed to refine treatment strategies and improve long-term survival for these high-risk patients.

Keywords: Cardiogenic Shock, LMCA Occlusion, Acute Myocardial Infarction

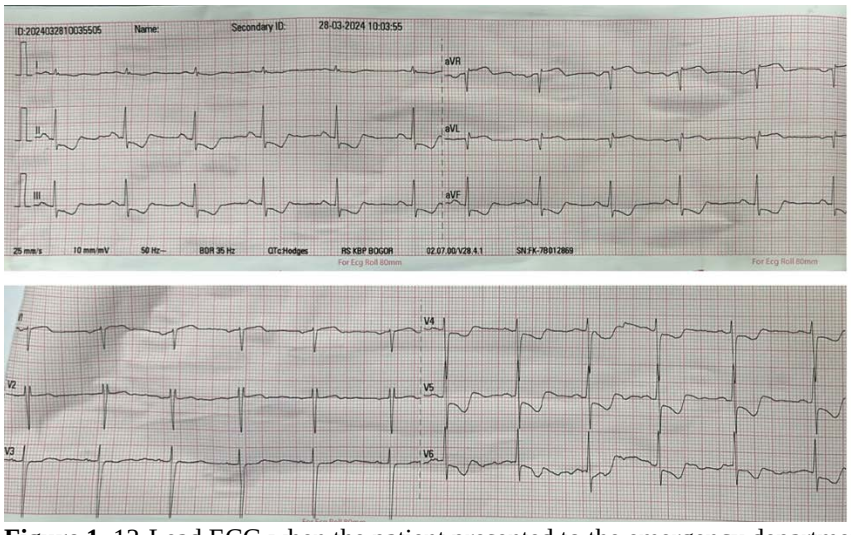

Figure 1. 12-Lead ECG when the patient presented to the emergency department

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SUCCESSFUL VA-ECMO SUPPORT FOR POST-CARDIOTOMY CARDIOGENIC SHOCK IN A PATIENT WITH SEVERE HEART FAILURE

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Background:
Patients with severe heart failure (HF) are considered high-risk candidates for any cardiac surgery. Post-cardiotomy cardiogenic shock (PCCS) can develop perioperatively, resulting in a significant mortality rate. Here we report a case of PCCS that occurred after coronary artery bypass grafting (CABG) in a patient with severe left ventricular (LV) dysfunction, which was successfully managed by peripheral veno-arterial (VA) ECMO.

Case illustration:
A 61-year-old male with known ischemic cardiomyopathy and an LVEF of 20-25% presented with sustained ventricular tachycardia (VT) necessitating urgent cardioversion. Subsequent cardiac catheterization showed severe three-vessel coronary artery disease. Therefore, CABG was planned, and an intra-aortic balloon pump (IABP) was inserted preemptively. Following the surgery, the patient developed intractable cardiogenic shock, supported with triple inotropes and IABP. Consequently, peripheral VA-ECMO was urgently implanted via the right femoral artery and vein to stabilize hemodynamics successfully. On the first day after ECMO implantation, cardiac stunning with minimal arterial pulsatility occurred, although mean arterial pressure was steadily maintained by the machine. Subsequently, he underwent a re-do on the second day because of cardiac tamponade. After these events, VA-ECMO was successfully explanted on day three, followed by the discontinuation of IABP support the next day. The patient was discharged in favorable condition after 27 days of hospitalization. His clinical status continued to improve at the 5-month follow-up in the outpatient clinic.

Conclusion:
VA-ECMO can serve as a crucial intervention for patients with severe heart failure undergoing cardiac surgery, particularly if refractory cardiogenic shock occurs.

Keywords: Cardiogenic Shock, VA-ECMO, Severe Heart Failure, CABG, Cardiac Surgery

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STEMI IN CORONARY ARTERY ANOMALIES: WHERE IS THE CULPRIT?

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Background:
Coronary artery anomalies are found in < 1% of the population. Ischemia up to myocardial infarction in coronary artery anomalies patients can be caused by disruption of coronary flow due to imprecise arterial flow, low coronary artery oxygen content or acceleration of the atherosclerotic process. Primary percutaneous coronary intervention (PPCI) in patients with coronary artery anomalies who experienced acute ST-segment elevation myocardial infarction (STEMI) carries potential technical difficulties, especially in cannulation of the coronary ostium which can result in delays in revascularization of the infarct-related arteries.

Case illustration:
A male patient, 59 years, was admitted to the hospital with typical angina with onset of 27 hours. Other complaints were denied. The patient had history of excessive smoking. Vital signs showed tachycardia and tachypnea. Rales was found. ECG showed ST-segment elevation in anterior lead. The patient then underwent PPCI. When diagnostic coronary angiography was performed, a subtotal occlusion lesion appeared in the proximal-mid left anterior descending artery (LAD) and rudimentary left circumflex artery (LCx) was suspected. After performing aortography, it was found that there was LCx originating directly from the left sinus of Valsalva without passing through the left main coronary artery (LMCA). The final coronary angiography results showed dual ostium of the left coronary artery originating from the left sinus of Valsalva, subcritical diffuse stenosis in proximal-mid LAD, 50–60% stenosis in mid right coronary artery (RCA), and normal LCx. A sirolimus-coated stent 2.25x25 mm was placed in proximal-mid of LAD. Angiographic evaluation showed TIMI 3 flow, without residual thrombus, residual stenosis, dissection, or perforation.

Conclusion:
Normal left circumflex artery (LCx) in this patient can be mistaken for total occlusion of the proximal LCx. Therefore, physicians need to exercise heightened caution when performing PCI with coronary artery anomalies.

Keywords: coronary artery anomaly, acute coronary syndrome

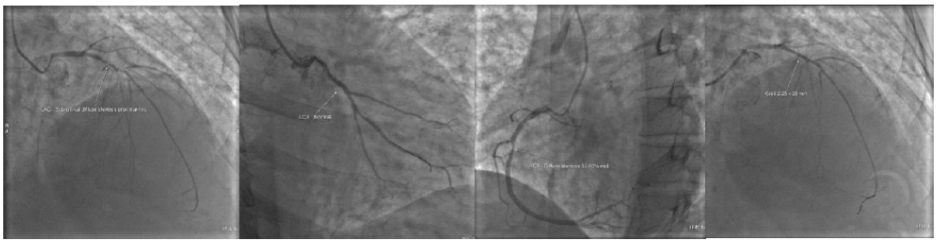

Figure 1. Angiography and PPCI

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CARDIAC AMYLOIDOSIS VS HEYDE'S SYNDROME – A GLIMPSE OF CLUES IN THE MIDST OF DOUBTS

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Background:
Cardiac amyloidosis is not only involving the myocardium but also cardiac valves, particularly aortic stenosis which is frequently found in those > 65 years old. In some circumstances, aortic stenosis can be accompanied by GI bleeding. If it occurs in the elderly, this raises a question whether it is a cardiac amyloidosis with GI involvement or Heyde's syndrome.

Case illustration:
A 87 – year – old woman had engaged in monthly checkups with a cardiologist for the last 2 years because of HFpEF and already received GDMT. In March 2023, she came to the polyclinic with worsening HF symptoms and newly discovered ejection type murmur on URSB. The echo showed LVEF 29% and low flow, low gradient AS. The NT pro BNP at that time was 7522 pg/mL and declined to 1566 pg/mL after having consumed low dose ARNi for a month. The CT – AVC showed severe aortic calcification (2393 AU). Unfortunately, in May 2024, the patient came to the ER with hypovolemic shock with hemoglobin only 6.9 g/dL. Fluid resuscitation and blood transfusion were given immediately. The ECG showed pseudo- infarction pattern with no history of MI. We decided to screen the cardiac amyloidosis possibility because of the ECG finding. The increase of kappa – lambda ratio led us to suspect AL – Cardiac Amyloidosis. The colonoscopy concluded a pan colitis as the bleeding source. After eleven days of hospitalization, the patient is stable and discharged. A definite Von Willebrand Disease was confirmed latter as the Von Willebrand factor level was 0.24 U/mL. Two possibilities now come in mind, Heyde's syndrome or cardiac amyloidosis?

Conclusion:
We should raise our awareness of cardiac amyloidosis as the cause of AS in the elderly, especially if the red flags exist. Aortic stenosis and GI bleeding should be considered as a unity, not different cases accidentally occur at the same time

Keywords: Cardiac Amyloidosis, Heyde's Syndrome, Aortic Stenosis

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ADDRESSING DIAGNOSTIC CHALLENGES OF ACUTE AORTIC DISSECTION IN PATIENTS WITH ATYPICAL CHEST PAIN AFTER BLUNT TRAUMA: FROM SUSPICION TO DIAGNOSIS

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Background:
Up to 80% of patients with blunt thoracic traumatic aortic injuries expire before hospital admission, while in-hospital mortality among survivors up to 19%. High-energy blunt trauma combined with rapid deceleration causes torsion and shearing stresses, resulting in aortic dissection or even rupture. Aortic dissection following blunt trauma is extremely uncommon, accounting for only 1.5% of all thoracic trauma.

Case illustration:
A 19-year-old male presented to the emergency department with severe, stabbing chest pain localised to the mid-left hemithorax after striking his chest on a bicycle accident. The pain was continuous and progressively worsening without radiation to the back or arm. Hemodynamics were stable, and an early diastolic murmur was heard in the Erbs area. The patient's stature appeared tall and thin, with pectus excavatum, long fingers, flat feet, and striae on the axilla and inguinal area. The ECG revealed LVH without ST-T changes, while the chest X-ray showed LVH, aortic dilatation, and scoliosis. Laboratory results revealed an elevated D-dimer of 3290 µg/L. Bedside echocardiography was performed and revealed aortic root dilatation with a flap in the ascending aorta, aortic arch, and abdominal aorta, suggesting suspicion of an Stanford A DeBakey I aortic dissection. In addition, there was severe AR, LV dilatation, and prolapse of the PML. These findings yielded a Marfan Systemic Score of 8 with a Z-Score of 4.72, leading to a diagnosis of definite Marfan Syndrome according to the 2010 Revised Ghent Nosology. We administer continuous morphine, as well as ACE-inhibitors, beta-blockers, and MRAs. Furthermore, we performed a CT angiography, which showed the presence of an aortic dissection including the ascending aorta, aortic arch, left common carotid artery, thoracic aorta, and abdominal aorta, with the exit point at the bifurcation of the abdominal aorta at L4-5. The patient was then scheduled for a staged modified Bentall and TEVAR procedure.

Conclusion:
Aortic dissection after blunt trauma requires a high level of suspicion such as detailed anamnesis, physical examination, D-dimer, and point-of-care ultrasound might quickly determine whether or not an aortic injury existed. This can then be followed by a brief CTA, which is ideal for subsequent procedure planning.

Keywords: Blunt Thoracic Traumatic Aortic Injury, Marfan Syndrome, Aortic Dissection, Traumatic Aortic Injury, Blunt Trauma

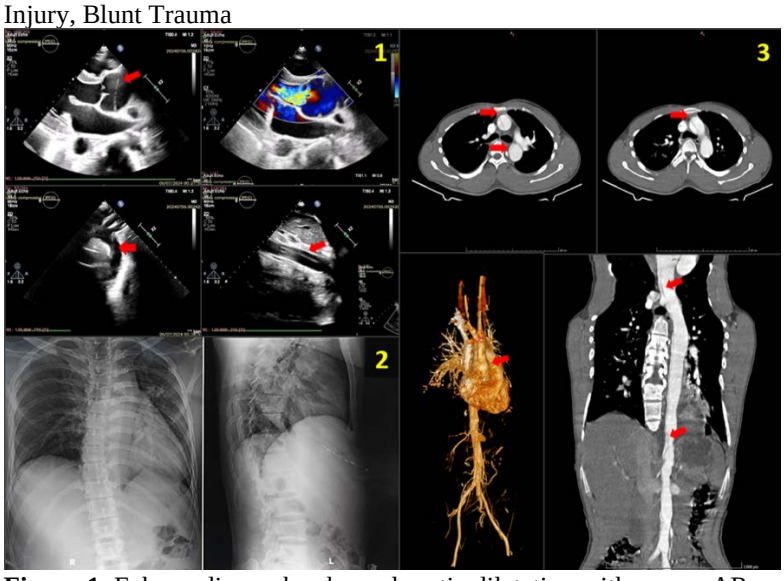

Figure 1. Echocardiography showed aortic dilatation with severe AR and flap at aorta ascenden, aortic arch and abdominal aorta **(1)**. Chest X-Ray showed scoliosis with dilatation of aorta and cardiomegaly LV **(2)**. CTA

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examination showed dilatation at the segmen of aortic root and aortic dissection from aorta ascenden until abdominal aorta **(3)**.

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DIAGNOSIS AND RISK STRATIFICATION OF ACUTE PULMONARY EMBOLISM POST CESAREAN SECTION

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Background:
Pulmonary embolism is a rare but life-threatening complication during pregnancy and postpartum. It is often under-diagnosed as the majority of signs and symptoms are nonspecific. Compared with vaginal delivery, the risk of pulmonary disease is almost twice as high with cesarean section. Risk stratification became one of the cornerstones of acute pulmonary embolism management to determine diagnostic and therapeutic strategies, including in post cesarean section patients.

Case illustration:
A 39 year old woman came to the ER with a sudden onset of dyspnea and palpitation. There was no chest pain. Patient had just gone through the cesarian section 2 days prior to admission. No history of venous thromboembolism and no sign of deep vein thrombosis was found. Physical examination showed no hemodynamic instability and from the ECG, we found Q waves, inverted T on lead III and P pulmonale on lead II and III. Acute pulmonary embolism was suspected and the patient was directly given enoxaparin 2x0,6 mg, furosemide 2x20 mg iv, ramipril 1x2,5 mg, digoxin 1x1,25 mg in ICCU observation. Despite class I PESI score, increasing D-dimer level, echocardiography which showed McConnell's sign, mild tricuspid regurgitation, and negative troponin suggest intermediate-low risk pulmonary embolism. CT pulmonary angiogram was also performed with the result of filling defect on the right pulmonary artery which concluded the diagnosis of pulmonary embolism.

Conclusion:
In patients experiencing sudden onset of dyspnea who have undergone cesarean section, risk stratification of pulmonary embolism could be useful to decide the optimal treatment strategy.

Keywords: Pulmonary thromboembolism, postpartum period, pregnancy complications, Risk Assessment

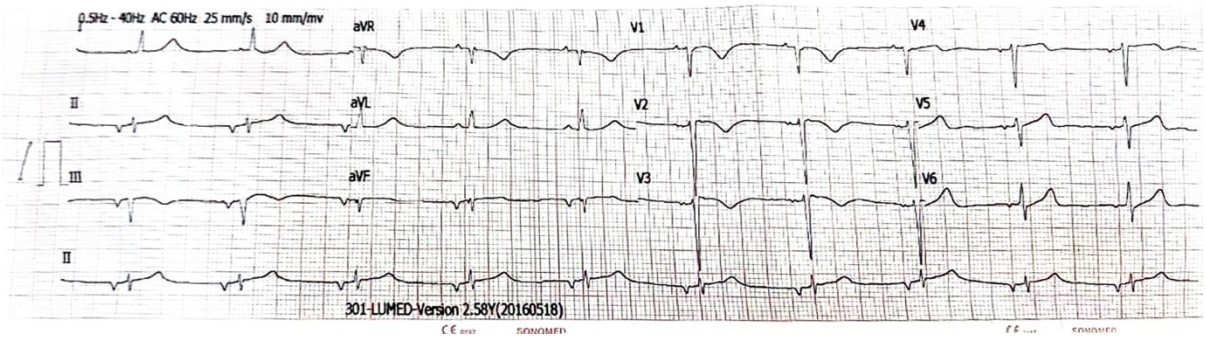

Figure 1. ECG of the patient which showed S wave in lead I, Q wave and inverted T wave in lead III suggesting Pulmonary Embolism.

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DECRYPTING TOTAL OCCLUSION IN NON-ST ELEVATION MYOCARDIAL INFARCTION SCENARIOS: DIAGNOSTIC CHALLENGES IN THE OCCLUSION MYOCARDIAL INFARCTION PARADIGM

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Background:
Occlusion Myocardial Infarction (OMI) is defined as an acute coronary occlusion or near occlusion of a culprit artery with insufficient collateral circulation, leading to transmural myocardial infarction. About 25–30% of Non-ST Elevation Myocardial Infarction (NSTEMI) patients have unrecognised acute total occlusion, resulting in delayed angiography and intervention. These patients can be categorised as OMI and have twice the short- and long-term mortality rates compared to NSTEMI patients without total occlusion.

Case illustration:
A 58-years-old male came to the emergency department complaining of severe chest pain followed by cold sweat. An ECG examination was carried out and found sinus rhythm with slight pathologic q waves in inferior leads without any ST-T changes. Serial ECG examination revealed pathologic q waves with additional t-inversion in V4-V6 and aVL. Laboratory results showed blood glucose was 379 mg/dL and HS-Troponin-I 58.8 ng/L, with serial examinations of >40,000 ng/L. The patient underwent immediate invasive strategy and revealed total occlusion (thrombus type) on the distal LCX artery. PCI 1 DES and further thrombosuction were performed, with the final result being TIMI 3 flow. The patient's chest pain improved greatly and the patient was given guideline directed therapy. The second case was a 50-year-old male referred to our hospital with complaint of sudden left chest pain radiating to his left arm that began 9 hours before. An ECG was performed, showed modest non-significant ST-elevation 0.02 second and pathologic q waves in inferior leads, as well as t-inverted V5-V6. A serial ECG shows a similar pattern to the previous one with additional PVCs, but no significant ST elevation. The troponin-I level exceeded 25.00 U/L. The patient had an early-invasive approach and showed CAD3VD with total occlusion (thrombus type) at the proximal LCX. PCI with 2 DES was completed successfully, resulting TIMI 3 flow. The chest pain was improved with ECG evaluation showed pathological Q-waves in inferior leads.

Conclusion:
ECG diagnosis of OMI without ST-elevation should evaluate for small alterations such as STEMI equivalents ECG (including de Winter or Wellen pattern), subtle ST-elevation <1mm, suspected acute pathologic Q waves, ST depresion in V1-V4 in suspicion of posterior MI, or a positive modified Sgarbossa criteria. Clinical presentation, persistent symptoms, and peak troponin levels should all be regarded as markers of total occlusion to ensure timely revascularization.

Keywords: Acute Coronary Syndrome, Acute Myocardial Infarction, STEMI, NSTEMI, occlusion myocardial infarction

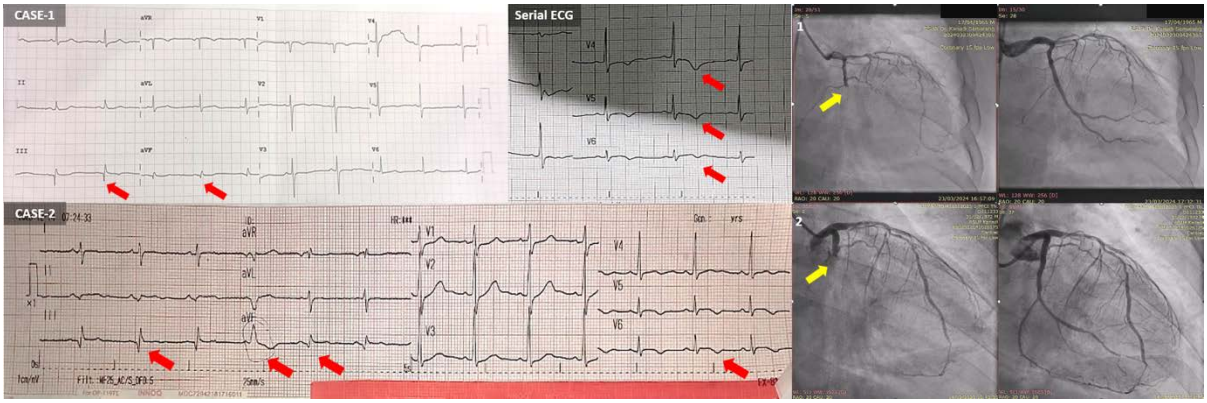

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Figure 1. Case-1 (above) ECG with suspicion of new q pathologic wave at lead II, III with no ST-T changes, and the serial ECG showing additional t inverted at V4-V6. Angiography turns out revealed total occlusion thrombus type at LCX. **Case-2 (below)** ECG with q pathologic wave at inferior leads and biphasic t wave at V4-V6. Angiography revealed CAD3VD with total occlusion thrombus type at proximal LCX.

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THE USEFULNESS OF CARDIAC MAGNETIC RESONANCE IMAGING IN EVALUATING ACUTE MYOCARDITIS: A CASE SERIES

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Background:
Acute myocarditis is characterized by inflammation of the heart muscle, presenting a diagnostic challenge due to its varied symptoms, which can include chest pain, dyspnea, palpitations, exercise intolerance, and syncope. These symptoms often overlap with other cardiac conditions such as acute coronary syndrome, heart failure, and arrhythmias, as well as non-cardiac issues. Cardiac magnetic resonance imaging (MRI) has become a crucial tool for assessing myocarditis, offering detailed visualization of myocardial inflammation and edema. In this case series, we present two cases of acute myocarditis diagnosed through cardiac MRI, highlighting the modality's diagnostic value and its role in guiding clinical management.

Case illustration:
A 73-year-old female and a 42-year-old male both presented with atypical chest pain during exertion for one week. Electrocardiograms (ECGs) in both patients revealed no signs of acute coronary syndrome or arrhythmias. Coronary CT angiography showed normal coronary arteries, and chest x-rays revealed no lung infiltration. Cardiac MRI was performed on both patients using T1, T2 mapping, and late gadolinium enhancement (LGE) sequences. In the first patient, the LGE sequence showed a non-ischemic pattern of increased enhancement in the lateral, anterior, and pericardial regions of the left ventricle, with T1 and T2 mapping revealing increased values in the same areas. In the second patient, the LGE sequence revealed a non-ischemic pattern of increased enhancement in the anterolateral, inferior, and anteroseptal walls of the left ventricle, with T1 and T2 mapping also showing increased values in the same areas. A second cardiac MRI performed one month after treatment revealed improvement in the LGE sequence, T1, and T2 mapping (Figure 1).

Conclusion:
Cardiac MRI is vital in assessing acute myocarditis. LGE sequences, along with T1 and T2 mapping, provide important findings for differentiating normal, ischemic, and non-ischemic inflamed myocardium. These imaging modalities are valuable for clinicians to distinguish between a normal heart, acute coronary syndrome, arrhythmias, and myocarditis.

Keywords: myocarditis, cardiac mri, cardiac magnetic resonance imaging

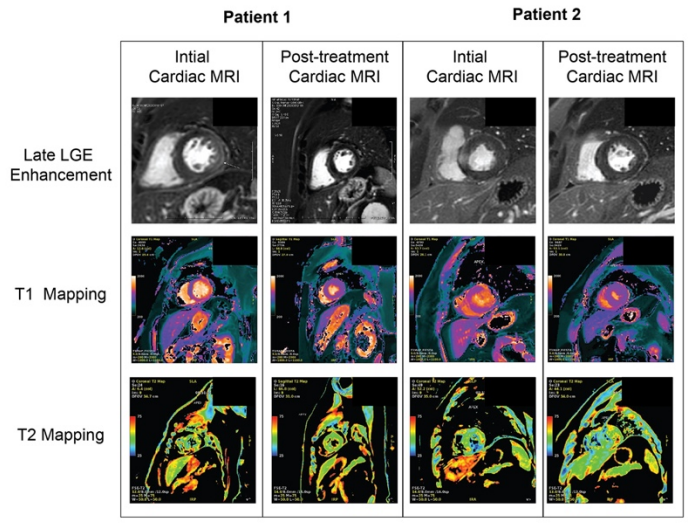

Figure 1. Cardiac MRI findings in patient 1 and 2.

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WHEN TAMPONADE PATIENT DECLINED PERICARDIOCENTESIS: WHAT SHOULD WE DO?

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Background:
One of the common cardiac manifestations in patients infected with Human Immunodeficiency Virus (HIV) was pericardial effusion. One of the causative factors involved in the development of pericardial effusion was Tuberculosis. HIV individuals were more susceptible to TB pericarditis because the infection was more likely to be a part of a disseminated disease, which increases morbidity and mortality. In this report, we described a case of large pericardial effusion in a patient with HIV and tuberculosis.

Case illustration:
In our cardiology outpatient clinic, a 46-year-old man was consulted by pulmonologist due to dyspnea for two weeks prior to admission. This patient was on ARV medication due to HIV and in the 2nd month of Tuberculosis treatment regimen. His vital sign showed BP of 95/58 mmHg, HR of 118 bpm, with oxygen saturation of 97% room air. From physical examination, we found raised JVP and muffled heart sound. The ECG examination only showed sinus tachycardia. Imaging examinations revealed cardiomegaly on a chest X-ray and an 18 mm circumferential pericardial effusion with signs of tamponade on an echocardiography. This patient declined pericardiocentesis and hospitalization due to financial constraints. The patient was then treated with Ibuprofen 600 mg tid, Colchicine 0.5 mg bid, and Digoxin 0.25 mg od. All HIV and Tuberculosis medication was continued. After 2 weeks of medication, the patient came back with clinical improvement and no signs of tamponade. Echocardiography showed reduced in pericardial effusion from 18 mm to 13 mm.

Conclusion:
HIV-associated TB pericarditis is a more aggressive disease with a greater degree of myocardial involvement. HIV alters the natural history and outcomes of TB pericarditis. The presence of an effusion is associated with shortened survival. When the diagnosed of cardiac tamponade was made, pericardiocentesis was mandatory, but sometimes, for socio-economic reasons, we are forced to choose conservative methods. From this case, we found that conservative methods still provide benefits in patients who refused pericardiocentesis.

Keywords: tuberculosis, pericardial effusion, hiv, tamponade

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ATRIAL FIBRILLATION, CEREBRAL ISCHEMIA AND LOW-OUTPUT HEART FAILURE; A CASE DILEMMA IN DOC PATIENT

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Background:
Atrial Fibrillation (AF) and Heart Failure (HF) are common risk factors and coexist in up to 50% of patients and increased risk of stroke. Low-output Heart Failure (Lo-HF) is an uncommon form of HF but one that signals very advanced HF. Lo-HF describes a high-acuity form of HF characterized by decreased forward CO (CI <2.2 L/min/m^2 and SBP <90 mmHg) and end-organ hypoperfusion with little or no evidence of pulmonary congestion and metabolic acidosis. However, Lo-HF remains largely under-researched. In this case, we will discuss case of a patient AF and HF who present in ER with decreased of consciousness (DOC).

Case illustration:
A male patient, 52 yo, came to ER of Urip Sumoharjo Hospital Lampung complaints of sudden decreased awareness at rest. None shortness of breath/chest pain. 1 week previously, patient went to cardiologist the first time and had diagnosed as CHF (EF 55%; EDV 100; ESV 41; CI 1,9), MS and AF. At ER GCS E1V1M3 BP 100/60 mmhg HR 72 bpm RR 24 bpm SO^2 96%. Normal JVP, rales +/+, mid-diastolic murmur and ankle edema, ECG AF NVR and incomplete RBBB. 15 minutes observation, ECG change to AF RVR, BP 130/90 mmhg down continuously until 90/60 mmhg HR 177 bpm RR 32 bpm SO^2 82% and UO 0,3 cc/kgbb/hour. Laboratory results increased creatinine 5.1 mg/dl and metabolic acidosis (pH 7.32; pCO2 19; pO2 214; HCO3 9.6), x-ray CTR >60%, and brain CT-Scan result an ischemic bilateral frontotemporoparietalis lobe.

Conclusion:
The patient in this case had annual AF, presenting with DOC and history of HF. This DOC was suspected due to a stroke induced by annual AF, however the patient's hemodynamic condition during treatment showed symptoms of Lo-HF, so a dilemma arose as to whether this DOC was due to a stroke or due to Lo-HF. After further investigation, we concluded that these two things were related, but specifically in this patient, the DOC was probably caused by Lo-HF.

Keywords: Lo-HF, AF, Stroke

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MANAGED SEVERE MITRAL STENOSIS IN LATE PREGNANCY: A MULTIDISCIPLINARY APPROACH TO OPTIMIZED MATERNAL AND FETAL OUTCOMES

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Background:
Mitral stenosis (MS) was a significant cardiac condition often caused by rheumatic heart disease, posing unique challenges during pregnancy due to the increased hemodynamic burden. Managing MS in pregnant women required careful consideration to balance maternal and fetal health outcomes. Pregnancy in women with MS was particularly challenging and could necessitate interventions such as percutaneous mitral commissurotomy (PMC) or valve replacement surgery.

Case illustration:
A 41-year-old pregnant woman at 35-36 weeks of gestation presented with worsening dyspnea, palpitations, and chest pain. She had a history of rheumatic heart disease, severe mitral stenosis (MS), moderate mitral regurgitation (MR), severe tricuspid regurgitation, and atrial fibrillation. Laboratory findings and physical examination indicated an unstable cardiac condition. The patient was managed with oxygen therapy, fluid restricted, digoxin, furosemide, warfarin, and bisoprolol. PMC was not recommended for her because echocardiography revealed more than mild MR and a multivalve diseases, including severe tricuspid regurgitation. Therefore, valve replacement surgery was advised, but the patient refused the procedure. Consequently, an elective cesarean section with epidural anesthesia was decided upon, and she delivered a low birth weight baby. The patient was advised to discontinue warfarin at least five days before the procedure and switch to heparin. Postpartum, she was advised to follow up regularly at the cardiology clinic and was counseled against future pregnancies unless valve replacement surgery was performed.

Conclusion:
This case underscored the importance of timely diagnosis and appropriate management of mitral stenosis in pregnant patients. Aggressive medical management, including the use of beta-blockers, diuretics, anticoagulants, and rate control, was crucial for patients with severe MS and atrial fibrillation. Close monitoring and careful handling during pregnancy were essential to minimize risks to both mother and fetus. While vaginal delivery was generally preferred, severe cases might have necessitated cesarean section for safety. Guidelines recommended considering PMC after 20 weeks of pregnancy. If PMC was contraindicated, valve replacement surgery should have been considered, tailored to the patient's clinical condition and characteristics. This case highlighted the need for multidisciplinary collaboration in managing complex cardiac conditions in pregnant women to optimize health outcomes for both mother and child.

Keywords: Pregnancy, Mitral stenosis

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PALPITATION IN YOUNG AND HEALTHY MALE : A CASE OF ATRIAL FIBILLATION WITH WOLFF-PARKINSON-WHITE SYNDROME

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Background:
Wolff-Parkinson-White (WPW) syndrome refers to the presence of an overt Accessory Pathways (AP), thus resulting in the so-called pre-excitation, in combination with usually recurrent tachyarrhythmias. An incidence of 4.5 episodes of sudden death from 1000 patient-years was recently reported. The mechanism of sudden cardiac death (SCD) in patients with WPW is thought to be associated with atrial fibrillation (AF).

Case illustration:
A 24-year-old male presented to the emergency room with a chief complaint of palpitation, followed by chest pain and a pre-syncopal event that had occurred in the previous three hours. The Electrocardiogram (ECG) showed Atrial Fibrillation with wide complex QRS, Heart rate 218 bpm. We perform cardioversion 100 joule with sedation, then the ECG convert to sinus rhytm with qrs rate 76 bpm and revealed delta wave consistent with WPW pattern. The patient continued with maintenance Amiodaron drip followed amiodaron oral when the patient discharged. We planned to referred patient for ablation at accessory pathway as the definitive therapy.

Conclusion:
When WPW syndrome is present, AF may have catastrophic outcomes including cardiac arrest. Early detection of ECG abnormalities is crucial for effective therapy. In order to reduce the chance of SCD and AF episodes in patients with WPW, early therapy and classification are crucial.

Keywords: Palpitation, Wolff-Parkinson White Syndrome, Accessory Pathway, Atrial Fibrillation

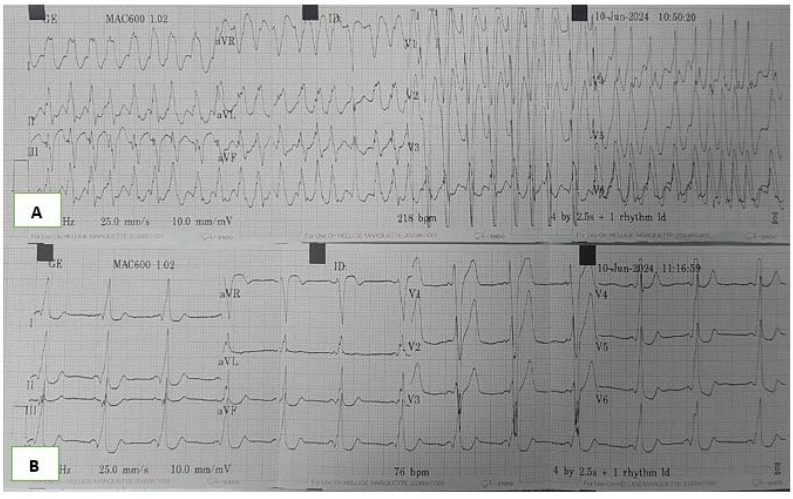

Figure 1. A. ECG during admission showed Atrial Fibrillation with wide complex QRS, QRS rate 218 bpm; B. ECG after cardioversion showed sinus rhythm with QRS rate 76 bpm, revealed delta wave consistent with WPW pattern

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THE ROLE OF CT IN PATIENT WITH MULTIPLE AORTIC DISSECTION UNDERGOING BENTALL PROCEDURE – A CASE REPORT

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[1]RSUP HAM

Background:
Aortic dissection is a life-threatening cardiovascular critical illness mainly caused by the formation of true and false lumens due to the separation of the intima and media after the blood enters the media upon tearing of the aortic intima. The Stanford classification divides dissections into type A and type B based on their location. Common symptoms are chest pain that may mimic a heart attack. Several imaging techniques may help to diagnose this condition. These include computed tomography/computed tomographic angiography.

Case illustration:
A 46-year-old male patient referred to Adam Malik Hospital with chief complain sharp and stabbing chest pain and being easily fatigued with moderate activity. Experienced by the patients since 6 months ago without any history of trauma on the chest. From vital sign, there is no difference between both arm and both lower extremities on blood pressure measurement. From physical examination, early diastolic murmur was found on lower left sternal border. The patient then had echocardiography examination with the result of intimal flap was found in aortic root and descending aorta and AR severe d/t dilated annulus with the conclusions of Aortic dissection Stanford B and AR Severe. The patient then underwent cardiac CT examination with the result aortic dissection found at sinotubular junction and aorta Descendens until Common Iliaca Arteries, and left renal artery. The patient then planned to undergo a bentall procedure operation.

Conclusion:
Although the sensitivity and specify of CT, MRI, and TEE are all high, CT has become the preferred modality for evaluating most patients with suspected Acute aortic Syndrome. CT is recommended for initial diagnostic imaging given its wide availability, accuracy, and speed, as well as the extent of anatomic details. Not only does it diagnose the underlying AAS, it also shows the full extent of the dissection. CT was also can be used to evaluate complications after surgery. The primary goals of open surgical or endovascular stent graft repair for acute aortic dissection are to prevent aortic rupture, prevent retrograde extension of the dissection into the aortic root and prevent antegrade propagation of the dissection into distal yet undissected segments.

Keywords: aortic dissection, computed tomography, bentall procedures

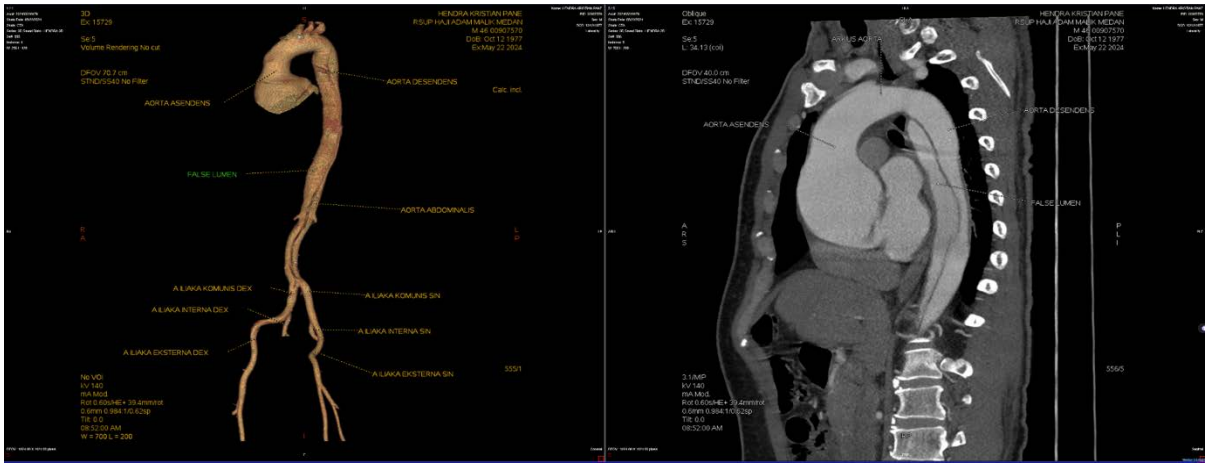

Figure 1. Computed Tomography shows multiple Aortic dissection at sinotubular junction and descending aorta

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BRADICARDIA-INDUCED VENTRICULAR ARRYTHMIA RESOLVED BY PACEMAKER IMPLANTATION: A CASE REPORT

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Background:
Bradycardia-Induced torsade de Pointes (TdP) are linked to Prolonged QTc which can be congenital or acquired. Chronic AV block and miocardial infraction are included in acquired prolonged QT. These could be challenging to decided which approach are necessary for patient with elevated Hs-cTnI, significant stenosis coronary artery and history of bradicardia and intermittent Total AV Block (TAVB)

Case illustration:
a 68 yeas old woman with cardiac syncope due to recurrent VT (Polimorphic and monomorphic) with history of intermintent TAVB, DM tipe 2, hypertension. Baseline ECG was bradicadia, rates in 42 bpm with prolonged QT interval (610 ms). Normal blood electrolite, Hs-cTnI 4533 ng/L without typical angina pectoris in the clinical presentation. Holter monitoring showed episode of monomorphic VT and TdP with short-long-short periode, and multiple episodes of junctional rhythm. 5 defibrillations were performed. A transvenous TPM was emergently performed with overdrive pacing at 70-75 bpm for bradycardia with prolonged QTc concomitant with coronary angiography (CAG) was shown has shown diffuse stenosis 80% proximal-mid LAD and diffuse stenosis 60-70% proximal-mid RCA with TIMI flow 3. TTE showed normal LVEF function in 60% without regional wall motion abnormality, no sign of cardiac infiltration. Due to no induce VT in ICCU monitors after 24 hours of TPM implantation and TIMI flow 3 in CAG without typical angina, we decided to implant Dual chamber PPM with setting mode DDDR 75 bpm, RA/RV 3.92/3.87 V, AVD 200/180ms and no further VT episode was documented

Conclusion:
This case illustrated the importance of basic mechanism of TdP pause dependent which can lead to a necessity management. Overdrive pacing with TPM or PPM implantation was the first choice for life saving in patient with bradicardia induce recurrent TdP even though there was a significant stenosis coronary artery

Keywords: Overdrive Pacing, Torsade de pointes, Prolonged QT, CAD, Bradycardia

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NAVIGATING THE UNCOMMON: A RARE CASE REPORT OF ARRHYTHMOGENIC RIGHT VENTRICULAR CARDIOMYOPATHY (ARVC) SUCCESSFULLY MANAGED WITH IMPLANTABLE CARDIOVERTER DEFIBRILLATOR (ICD) AS SECONDARY PREVENTION OF SUDDEN CARDIAC DEATH (SCD)

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Background:
With a high risk of sudden cardiac mortality, arrhythmogenic right ventricular cardiomyopathy (ARVC) is a rare inherited cardiomyopathy characterized by right ventricular dysfunction and ventricular arrhythmias

Case illustration:
a 35-year-old presented with palpitations and revealed epsilon waves on the right precordial electrocardiogram (ECG) leads, which led to the suspicion of ARVC. The 24-hour Holter monitoring revealed frequent premature ventricular contraction-right ventricular outflow tract (PVC-RVOT) of >500 per 24 hours, and cardiac magnetic resonance imaging (CMRI) revealed RV dyskinesia and transmural late gadolinium enhancement (LGE) in the apical RV. These results validated the diagnosis of ARVC. A further electrophysiologic study (EPS) revealed non-inducibility or arrhythmia; however, right after the EPS procedure was done, a spontaneous ventricular tachycardia (VT) polymorphic was seen. These results validated that ARVC is high-risk and indicated that an implantable cardioverter defibrillator (ICD) is necessary for secondary prevention of sudden cardiac death (SCD).

Conclusion:
The ability to identify potential ARVC's ECG abnormalities, the timing to pursue further studies, and determining necessary treatment execution are critical. The evaluation of cardiac, electrophysiological, and clinical imaging parameters is essential for the risk stratification, diagnosis, and management of patients with ARVC.

Keywords: sudden cardiac death, implantable cardioverter-defibrillator, arrhythmogenic right ventricular cardiomyopathy

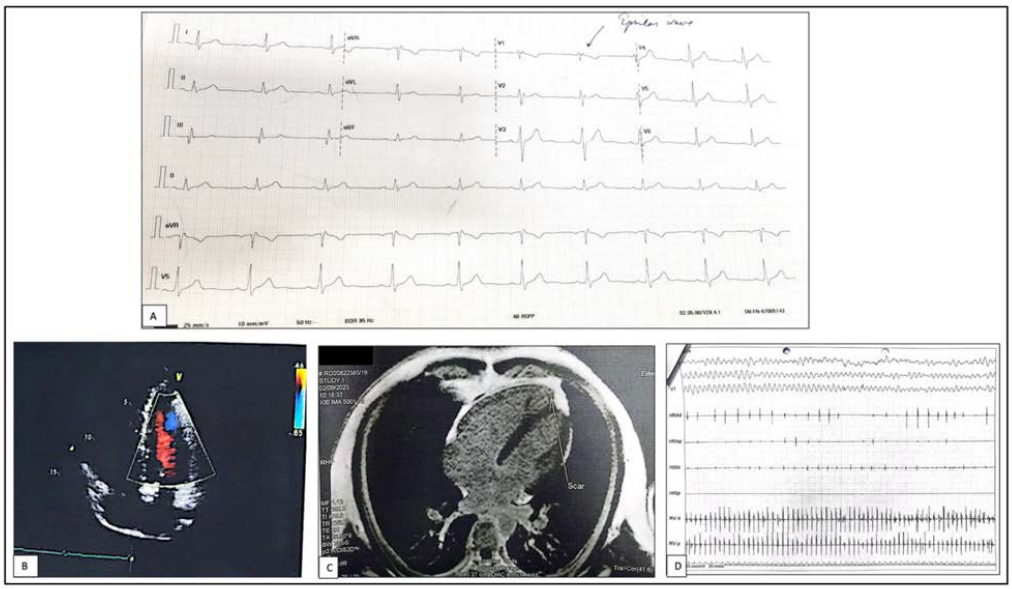

Figure 1. A. The electrocardiogram (ECG) revealed epsilon waves in the right precordial lead B. Echocardiography found a dilated right ventricle (RV) and right atrium (RA) C. Cardiac MRI (CMRI) revealed

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dyskinesia in the RV and transmural late gadolinium enhancement (LGE) in the apical RV. D. Spontaneous Ventricular Tachycardia (VT) polymorphic during electrophysiology study (EPS)

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HOW TO DEAL WITH DOUBLE TROUBLE OF PERIPARTUM CARDIOMYOPATHY COMPLICATED BY ACUTE RENAL FAILURE: FROM PREPARTUM TO CONTINUOUS RENAL REPLACEMENT THERAPY, A-MULTIDISCIPLINARY MANAGEMENT

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Background:
Peripartum cardiomyopathy is a potentially life-threatening cardiomyopathy that occurs in previously healthy women during the peripartum phase. We present a case of severe PPCM complicated with respiratory failure and acute renal failure.

Case illustration:
A 21-year-old female (primigravida) in her 34-week pregnancy was referred from primary health care with a complaint of worsening shortness of breath 1 day before admission and bilateral leg swelling 2 weeks before. She was routinely controlled by a midwife and obstetric doctor with normal blood pressure and normal antenatal care previously. At the emergency room of Saiful Anwar Hospital, she had normal blood pressure, HR 136x/m, RR 26x/m, SpO2 90% on SM 8lpm, and 97 % on NRBM 15lpm. Physical examination showed bilateral basal rales, S3 gallop, 2+ pitting edema of both legs and fetal heart rate 172x/m. Electrocardiography revealed sinus tachycardia, Chest X-ray cardiomegaly, congestive pulmonary, and left pleural effusion. Echocardiography showed global hypokinetic with all chamber dilatation and decreased LV systolic function (28% by Teich). Because of emergency conditions in the form of respiratory distress due to cardiogenic acute lung edema due to peripartum cardiomyopathy and fetal distress, it was decided to terminate the pregnancy by cesarean section under general anesthesia. Post-operatively the patient was still intubated and using mechanical ventilation. She was given bromocriptine therapy and initiated ACE inhibitor, beta-blocker, spironolactone, and anticoagulant. During observation in the ICU, she had hemodynamic instability, with a trend of increasing creatinine, anuria for 12 hours, and hyperkalemia. After a multidisciplinary team discussion, it was decided to do continuous renal replacement therapy. After 3 days the patient had hemodynamic improvement, successfully weaning ventilator, and CRRT was completed. The patient was transferred to a regular treatment room after 6 days of treatment in the ICU, and went home after 12 days of hospitalization

Conclusion:
Severe peripartum cardiomyopathy can cause complications such as acute renal failure. Appropriate management from prepartum to postpartum observation requires good team collaboration to reduce morbidity and mortality.

Keywords: CRRT, Peripartum Cardiomyopathy, PPCM, Severe PPCM, Acute kidney injury

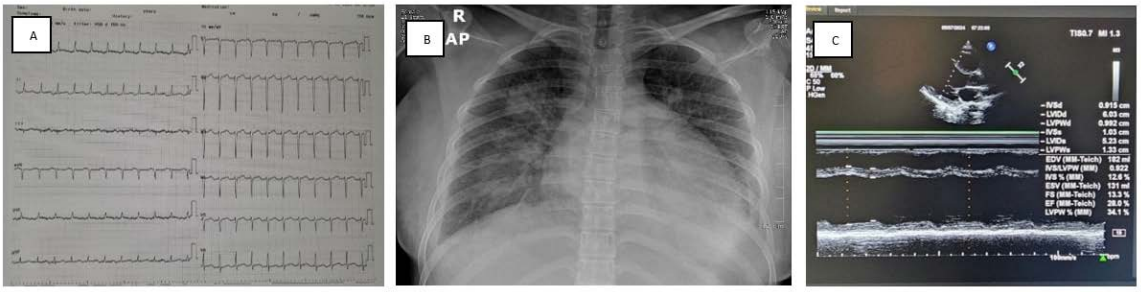

Figure 1. (A) Electrocardiography on admission showed sinus tachycardia at 136 beats per minute. (B) Chest X-ray showed cardiomegaly and congestive pulmonary. (C) Echocardiography LV study showed dilatation of LV with reduced LV Ejection Fraction (28% by Teich)

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DRESSLER'S SYNDROME PRESENTING AS ACUTE DECOMPENSATED HEART FAILURE: A RARE AND OVERLOOKED DIAGNOSIS - A CASE REPORT

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Background:
Dressler's syndrome, also known as late post-myocardial infarction pericarditis, is a type of secondary pericarditis that can be followed with or without pericardial effusion following a myocardial infarction event. Dressler's syndrome has a varied and non-specific presentation, with dyspnea may accounting for 50-60% of the presentation. A recent study revealed the disappearance of Dressler's syndrome, which might have contributed to the advancement of reperfusion strategies making it a rare clinical entity in daily practices.

Case illustration:
A 47-year-old man was admitted to the emergency department with the chief complaint of breathlessness for one week prior to admission. There was dyspnea on exertion and orthopnea without a typical chest pain. The patient had a history of one-vessel coronary artery disease post-stenting on the proximal-mid LAD with complete revascularization 12 days before admission. On physical examination, clinical signs of congestion was observed with elevated JVP, ventricular gallop, bilateral rales, ascites, positive hepatojugular reflux, and bilateral ankle edema. Pericardial friction rub was also found on cardiac auscultation. A chest X-ray revealed cardiomegaly with pulmonary edema. Echocardiography was performed afterwards, revealing akinetic LAD territory with a reduced LVEF of 35.1%, TAPSE 12,6 mm, minimal pericardial effusion, and thickening of the visceral pericardium with no sign of cardiac tamponade. Due to our suspicion of Dressler's syndrome, we ordered HsCRP examination revealing an elevated HsCRP of 5,23 mg/dl, confirming the diagnosis of Dressler's syndrome. Coronary CT angiography was also performed to exclude the diagnosis of stent thrombosis due to worsening of cardiac function. Aspirin 720 mg t.i.d. and colchicine 0,5 mg b.i.d., was added in conjunction with his coronary artery disease and optimal heart failure medication. On the 7th day of hospitalisation, he was discharged with an euvolemic and stable hemodynamic condition. No bleeding or MACE had been observed after one month of follow-up.

Conclusion:
Although Dressler's syndrome is a rare clinical entity, it could share a similar clinical features with ADHF. This case highlights the importance of prompt differentiation of Dressler's syndrome with ADHF; thus, disease-specific therapies could be administered to maximize patient's outcome.

Keywords: Dressler's syndrome, ADHF, Pericardial effusion, Post cardiac injury syndrome

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PULMONARY EMBOLISM MIMICKING ACUTE CORONARY SYNDROME: A CASE REPORT

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Background:
Acute pulmonary embolism (PE) is a potentially life-threatening emergency that needs prompt management to reduce preventable deaths. Symptoms like dyspnoea and chest discomfort, elevated troponin level, and electrocardiogram (ECG) findings often lack specificity and overlap with the acute coronary syndrome (ACS). Although ECG findings, notably the S1Q3T3 pattern, is pathognomonic of right heart strain. Well's criteria for risk assessment revealed an intermediate risk for PE. Elevated D-dimer levels necessitated computed tomography pulmonary angiography (CTPA), confirming thrombus presence resulting in subsequent thrombolysis. This case presents the clinical, electrocardiographic, and radiologic findings alongside appropriate therapeutic strategies.

Case illustration:
A 64-year-old woman presenting with typical angina and diagnosed with NSTEMI was referred from a previous hospital for PCI strategy. ECG showed sinus rhythm, anterior ischemia, and an S1Q3T3 pattern, while laboratory results showed elevated CK-MB. The patient was given Aspirin 160 mg, Clopidogrel 300 mg, and ISDN 5 mg three times, however complaints did not improve. At the Cardiac Center H. Adam Malik Hospital Emergency Department, the patient presented with typical angina, desaturation, and a history of untreated joint dislocation of his right leg one year ago. The physical examination was unremarkable. ECG showed sinus rhythm, anterior ischemia, and S1Q3T3 pattern. Chest X-ray showed cardiomegaly. The laboratory results revealed elevated leukocytes, Troponin I, Creatinine,and D-dimer, other lab results were within normal limits. Trans-thoracic echocardiography showed preserved LV systolic function, LV concentric remodeling, RA-RV dilatation, TR severe d/t functional with a high probability of PH, PR moderate d/t functional, decreased RV function, and signs of pulmonary embolism such as McConnel sign and 60/60 sign was found, RV size > LV size. DVT was found on Doppler ultrasonography of the lower extremity. CTPA showed a thrombus on the right and left pulmonary artery, leading to thrombolysis via catheter-directed intervention with Streptokinase. The patient was discharged with routine oral rivaroxaban anticoagulant.

Conclusion:
We report a case with PE mimicking ACS symptoms. Symptoms like dyspnoea and chest discomfort, elevated troponin level and electrocardiogram's finding often lack specificity and overlap with ACS. This case presents the clinical, electrocardiographic, and radiologic findings, alongside appropriate therapeutic strategies.

Keywords: Thrombolysis, Pulmonary Embolism, CT Pulmonary Angiography, Acute Coronary Syndrome

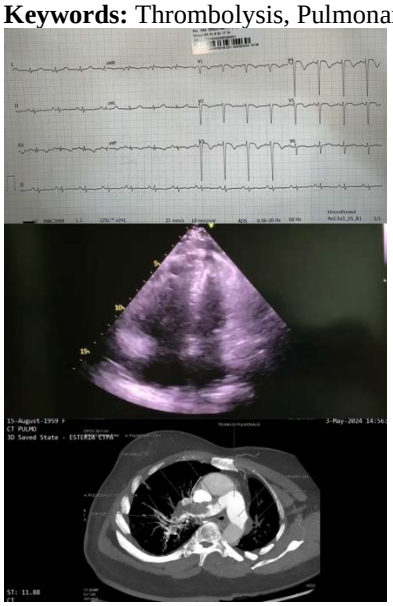

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Figure 1. ECG, Echocardiography and CTPA showed the patient with pulmonary embolism

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INFECTIVE ENDOCARDITIS PRESENTING WITH BACTERIAL MENINGITIS : A CASE REPORT FROM RURAL AREA

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Background:
Infective endocarditis (IE) is a worldwide major health problem with the number incidence of IE estimated at 13.8 cases per 100.000 subjects worldwide per year. The majority of extracardiac complications of IE are neurological in nature, such as ischemic stroke, intracerebral hemorrhage, cerebral abscess, infectious intracranial aneurysm and last meningitis. In the past, IE could not be diagnosed in Flores Island due to unavailability of echocardiography as the gold standard diagnostic tool.

Case illustration:
A 22-year-old presented to the emergency room with altered mental status, seizure, febrile, and throbbing cephalgia. On arrival the patient was somnolent, with blood pressure of 90/60 mmHg, axillary temperature 38.9 Celsius, heart rate 70x/minute, respiratory rate 22x/minute, and oxygen saturation of 98%. Physical examination showed widening of the left heart border, a 4/6 grade holosystolic murmur at apex was heard and electrocardiography showed sinus rhythm. Laboratory tests revealed leukocytosis with neutrophils, anemia but urinalysis test showed normal. The chest x-ray showed cardiomegaly and Trans-Thoracic Echocardiography (TTE) showed severe mitral regurgitation with multiple vegetation on the anterior and posterior leaflet of the mitral valve. Blood culture and brain CT scan were not conducted due to limited resources. “Probable IE” was the concluded diagnosis based on Modified Duke’s Criteria fulfilling only one major and one minor criterion. Intravenous ceftriaxone and gentamicin were given as the empirical antibiotic therapy. After 14 days of antibiotic treatment, the patient developed persistent fever, new onset hemiparesis, and worsening condition even though the next laboratory results showed decreasing leukocytes levels and differential counts and no bacteria found in urinalysis. Prompt changes in therapy were implemented but to no avail as the patient experienced cardiac arrest and was pronounced dead.

Conclusion:
The availability of TTE in Flores Island has played a major role in diagnosing infective endocarditis, however without the availability of blood culture equipment, a specific antibiotic therapy and management could not be carried out to obtain better patient outcome.

Keywords: stroke, vegetation, meningitis, infective endocarditis

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CHRONIC THROMBOEMBOLIC PULMONARY HYPERTENSION IN PATIENT WITH ATRIAL SEPTAL DEFECT : FOCUS ON LUNG PERFUSION SCINTIGRAPHY

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Background:
Chronic thromboembolic pulmonary hypertension (CTEPH) is currently underdiagnosis and consequently undertreatment in the clinical practice. Unrepaired atrial septal defect (ASD) may be associated with pulmonary arterial hypertension (PAH), but furthermore, could also be associated with other conditions, such as or thromboembolism (CTEPH) or left heart disease, contributing to the disease progression. Imaging can be useful in diagnosing patients suspected of having CTEPH. Ventilation/Perfusion (V/Q) lung scintigraphy is the imaging modality of choice for the screening of CTEPH. However, there is no consensus on the criteria to use for interpretation.

Case illustration:
We present a 41-year-old woman, referred from a type C hospital with complaints of shortness of breath since several months ago. Patients began to complain of shortness of breath since pregnancy and after delivery. No history of prior deep vein thrombosis (DVT). The patient was performed echocardiography with result large secundum ASD with L-R shunt, minimal posterior and inferior rim, right atrium and right ventricle dilatation, D-shaped left ventricle, normal right ventricle contractility, mild tricuspid regurgitation with high probability of PH, and normal left ventricle systolic function. Righ heart catheterization revealed with low flow high resistance with non-reactive to oxygen test and normal coronary angiography. Imaging performed to looking for another etiologies of PH. Lung perfusion scintigraphy performed with result “wedge shaped” perfusion defects at apical and upper posterior lobe of the right lung and apico-posterior upper lobe of the left lung with normal lung parenchymal (mismatched defects). Based on Perfusion-only modified PIOPED II Criteria, patient suggestive for CTEPH.

Conclusion:
Invasive catheter pulmonary angiography is the gold standard for CTEPH diagnosing, whereas V/Q scan is currently the gold standard screening method for CTEPH. The infrequent use of V/Q scans, despite recommendations in the guidelines, is assumed to contribute to the underdiagnosis of CTEPH. V/Q mismatch is essential for diagnosis of CTEPH, while some institutions obtain only perfusion scans with alternative use of chest radiography or CT for ventilation, we can use diagnostic criteria based on perfusion-only modified PIOPED II criteria to diagnose of CTEPH.

Keywords: ASD, Pulmonary Hypertension, V/Q Scan, CTEPH

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BEYOND THE OBVIOUS : UNVEILLING AORTIC DISSECTION MIMICKING ACUTE CORONARY SYNDROME PRESENTING WITH CARDIAC ARREST IN EMERGENCY ROOM

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Background:
Aortic dissection is defined as a tear in the intimal layer of the aortic wall. Approximately 30% of the patients die during hospital care. Aortic dissection can lead to severe complications, causing the mortality rate to increase by 1-2% per hour without appropriate management. Although, it is a critical condition, it's often misdiagnosed as acute coronary syndrome (ACS) due to overlapping symptoms. Prompt and accurate diagnosis is essential to manage and improve patient outcomes. This case illustrates a misdiagnosis of ACS, highlighting the importance of comprehensive evaluation in patients presenting with chest pain and hemodynamic instability.

Case illustration:
A 55-year-old woman presented to the emergency department with severe chest pain and was initially diagnosed with non-ST elevation myocardial infarction (NSTEMI) at another hospital. Upon transfer, she suffered cardiac arrest but was successfully resuscitated. Post-resuscitation, her electrocardiogram (ECG) showed no significant changes. Serial cardiac troponins also did not increased nor decreased. Bedsite transthoracic echocardiography was then performed and revealed an intimal flap suggestive of aortic dissection. To establish the diagnosis patient then undergo a cardiac computed tomography (CT) examinations that confirmed a Stanford type A De Bakey Type I aortic dissection with extension from the ascending aorta to the iliac arteries. Initial treatments with medications successfully relieved the symptoms and stabilize the patient's hemodynamic. Patient then subsequently planned to undergo surgery.

Conclusion:
This case underscores the critical need for thorough diagnostic assessment in patients with chest pain, even when initial diagnoses suggest ACS. Echocardiography and cardiac CT are invaluable in identifying aortic dissection, which can mimic ACS but requires distinctly different management strategies. Early detection and appropriate intervention are pivotal in reducing mortality associated with aortic dissection

Keywords: cardiac arrest, acute coronary syndrome, echocardiography, Aortic dissection, computed tomography

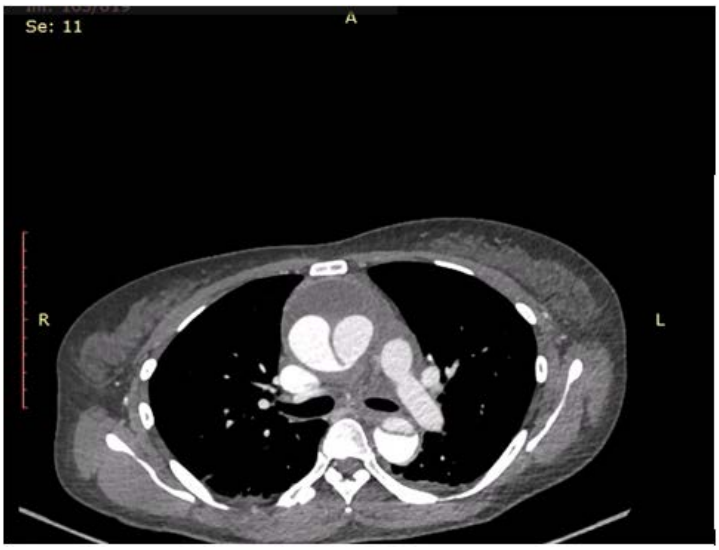

Figure 1. Aortic dissection as seen from axial view of CT

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MULTIGRAVIDA PATIENT WITH MS SEVERE DUE TO RHD: HOW TO DEAL WITH RISK AND COMPLICATION IN PREGNANCY?

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Background:
Rheumatic Mitral Stenosis is a leading cause of acquired cardiovascular disease that complicates pregnancy. Currently, the stenosis is progressing, the left atrium dilates, and the left atrial pressure increases. The risk of maternal death is greatest during labor and during the immediate post-partum period. The hemodynamic disturbance related to pregnancy caused by higher blood volume, high heart rate, mean left atrial pressure, and pulmonary venous pressure inducing pulmonary edema. Palpitation, chest discomfort, thrombosis, hepatomegaly, ascites, and edema are frequent signs of mitral stenosis. It impact to worse maternal outcomes such as arrhythmia, pulmonary edema, and thromboembolism, as well as worse neonatal outcomes such as intrauterine growth retardation, preterm delivery, low birth weight, and neonatal or fetal death.

Case illustration:
Thiry-eight old Female complained shortness of breath, dyspnoe on exertion and orthopnea with lower extremites oedema since 2 years ago. Patients complain chest pain sometimes. On physical examination, rales on basal lung field not found and murmur on lower mid left clavicula ICS 4-5 border. ECG results found sinus rhythm normoaxis in limit normal, on echocardiography found MS severe with thicked and calcification in AML d/t RHD, Wilkin score 9 and suitable for BMV, LV systolic function was decrease, LVEF 46%, LA dilatation and TR mild, PR mild d/t functional. Patient planed to S-Cection procedure and sterilization pomeroy procedure. Patient was planed SC delivery in RSUP HAM, with spinal anesthesia, the baby was born with apgar score 8/9, body weight 3100gr, the patient was discharged from hospital and planned to visite polyclinic Cardiac Centre RS HAM with planning for Ballon Mitral Valvuloplasty

Conclusion:
Induction, management of labour, delivery, and postpartum surveillance require specific expertise and joint management by the obstetrician, cardiologist, and anaesthesiologis. Caesarean delivery could be considered for symptomatic severe mitral stenosis.

Keywords: MS severe, Pregnancy, S-Cection

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DETECTING EBSTEIN ANOMALY FROM THE PRESENCE OF ACCESSORY PATHWAY A CASE FROM RURAL AREA

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Background:
Ebstein Anomaly (EA) is a congenital malformation of the septal leaflet of the Tricuspid valve (TV) that displaces apically in the right ventricle (RV). This causes some of the proximal part of the RV become atrialized. It appears in 1 out of every 20.000 live births, making it a rare occurrence in general, let alone detected clinically in a rural area. It usually appears with Right Bundle Branch Block (RBBB) from electrocardiography (ECG) in up to 95% of patients with this condition, which is not the case on this patient.

Case illustration:
A 19-year old female patient came to the emergency room of a remote hospital with syncope. She has a history of Antidromic Supraventricular Tachycardia one year prior to this admission. The blood pressure was 80/54 mmHg and the heart rate was 83 beats per minute. On physical examination, we found a left parasternal heave grade I, normal 1st and 2nd heart sound and a holosystolic murmur grade III/VI at the lower left parasternal area that accentuated with Rivero-Carvallo maneuver, indicating Tricuspid Regurgitation (TR). The ECG showed tall P waves and normal PR interval in most leads, with delta waves most prominently appearing in leads V1-V2, along with Left Bundle Branch Block indicating a right-sided accessory pathway (AP). Chest radiography showed cardiomegaly with normal pulmonary vasculature. We referred her to the Regency Hospital for echocardiography examination that revealed an apical displacement of the septal leaflet of TV > 8 mm/m^2 of body surface area with right atrium (RA) and RV dilatation, mild to moderate TR, TV gradient of 41 mmHg, mean Pulmonary Arterial Pressure of 35 mmHg, high probability of pulmonary hypertension and normal biventricular function. Thus, confirming EA diagnosis. She was eventually transferred to the Province hospital for further evaluation and management.

Conclusion:
The presence of TR murmur, delta wave with normal PR interval and the absence of typical feature of RBBB on the ECG helped us in diagnosing EA with right-sided AP. It should leads to further diagnostic testing like electrophysiology study to locate the AP and for the purpose of ensuing intervention.

Keywords: Ebstein Anomaly, Rural Area, Accessory Pathway

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SEVERE HYPOKALEMIA-INDUCED ARRHYTHMIA IN YOUNG ADULT PATIENT

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Background:
Hypokalemia is one of the common electrolyte imbalances in clinical practice. Cardiovascular abnormalities may present. It can lead to changes in electrocardiographic (ECG) and become a life-threatening arrhythmia. The common ECG changes are ST segment depression, U wave, flattened T wave, and prolonged QT interval.

Case illustration:
A 22-year-old female patient was admitted to the emergency department with frequent vomiting and nausea three days before. She denied alcohol consumption or diuretic use. She had no history of previous heart disease, hypertension, or kidney disease. Physical examination showed a blood pressure of 115/80 mmHg and heart rate of 52 beats per minute. Chest auscultation revealed regular heart sounds with no murmurs and abdominal examinations were normal. ECG first day showed prominent U wave and premature ventricular complexes. ECG second day showed sinus rhythm with prolonged QT interval and ST depression. Serum electrolyte results showed severe hypokalemia (K = 1.6 mmol/L, normal = 3.5-5.1 mmol/L). She was treated with potassium supplement, both intravenous and orally, but still unchanged. Immediately, she got hypokalemia correction via central venous catheter. After the serum potassium level reached normal, ECG was rechecked and showed normal sinus rhythm, she was discharged. Etiologies of hypokalemia can be caused by increased potassium loss (renal or gastrointestinal), decreased potassium intake, or intracellular shift due to medications or hormonal disorders. Potassium has a significant role in maintaining electrical potential of cell membranes as well as for depolarization and repolarization of heart muscle cells. Hypokalemia can increase resting potential and prolong the action potential, especially in the third phase of repolarization and refractory period. Characteristic ECG changes of hypokalemia include dynamic changes in T wave morphology, prolonged OT interval, ST segment depression, and U wave. It also can present with different patterns of arrhythmia such as premature ventricular complexes, atrial fibrillation, and in worse cases, arrhythmia malignant that lead to cardiac arrest.

Conclusion:
Arrhythmia can be caused by hypokalemia with various different changes in ECG. Clinicians should be familiar with different ECG manifestations of hypokalemia, so it can help to diagnose and give treatment to patients.

Keywords: Arrhythmia, Hypokalemia, ECG

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CARDIO-HEPATO-RENAL DISORDER : UNVEILING THE DISPARITY OF NEUROHORMONAL DYSFUNCTION

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Background:
The co-occurrence of cardiorenal and hepatorenal dysfunction are common in cardiometabolic disease. As such, cardiac disorder is also suggested either as the predisposition factor of kidney disease or merely as the aberrant consequences in hepatorenal syndrome.

Case illustration:
A 38 years old male came to the emergency room with the problem of shortness of breath within a month. This symptom appeared along with the abdominal distension which occurs progressively. Anuria also occurred within 4 days. The patient had a history of smoking about a pack of cigarettes per day in these 20 years. Intermittent alcohol consumption was affirmed about 17 years ago. The patient also declared that he had a history of gallstone about 3 months ago, but he declined it to be discarded. However, abdominal pain has not been declared. Physical examination showed blood pressure 100/70, heart rate 117, respiration 22, normal temperature and normal oxygen saturation. Icteric conjunctiva and shifting dullness was found. On the other hand, abdominal mass could not be palpated. Heart auscultation revealed split S2 during expiration. Electrocardiogram showed sinus arrhythmia with normal axis, slight inverted T-waves, and low voltage QRS at the precordial leads. Bedside echocardiography showed dilated all heart chambers with ejection fraction 50%, mild pericardial effusion, mild bilateral pleural effusion, and liver congestion. Moreover, chest x-ray showed cardiomegaly, lung edema, with bilateral pleural effusion. The patient was assessed as congestive heart failure and suspected intra abdominal tumor. Furthermore, the patient was treated with dobutamine 3 mcg/kg/min, dopamine 2,5 mcg/kg/min, furosemide 5 mg/hr, spironolactone, captopril, digoxin, channa, and clopidogrel. After 2 days, the symptoms were slightly resolved and went out further to be referred to the central hospital.

Conclusion:
The principal treatment of this syndrome is volume control by assessing intravascular volume status. The involvement of the kidney will further deteriorate the disease through the role of neurohormonal and hemodynamic mechanisms that are associated with unfavorable outcomes. Furthermore, liver dysfunction by itself could also be the factor that worsening fluid regulation resulted in heart failure.

Keywords: hemodynamic, neurohormonal, heart failure, hepatorenal, cardiorenal

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BIVENTRICULAR ARRHYTHMOGENIC CARDIOMYOPATHY IN AN ASYMPTOMATIC YOUNG MALE

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Background:
Arrhythmogenic cardiomyopathy (ACM) is a rare hereditary cardiomyopathy with various phenotypes and characterized by fibro-fatty replacement of myocardial tissue, mostly affects on the right ventricle, although biventricular involvement might occur. It significantly correlates with ventricular arrhythmia and sudden cardiac death (SCD) in appeared-healthy young population.

Case illustration:
A completely asymptomatic 30-year-old male was referred to Cardiologist due to an incidental finding on ECG of T inversion at V1-V4 and occasional ventricular ectopics. History of syncope or SCD at family members were denied. Physical examination only revealed gallop S3 at left lower sternal border, otherwise was unremarkable. Echocardiography showed mildly dilated RV including RVOT, global hypokinetic of both ventricles with slightly impaired both ventricles systolic functions, and hypertrabeculation of RV. NT-proBNP level was 292 ng/L, meanwhile 24-hour ambulatory ECG recording captured high risk ventricular ectopics originated from RVOT and LV. Further cardiac MRI confirmed the diagnosis of ACM by the features of fatty infiltration and fibrosis at RV free wall and lateral LV. Bisoprolol 2.5 mg once daily was prescribed and Implantable Cardioverter Defibrillator (ICD) was suggested to prevent SCD.

Conclusion:
Since the majority cases of ACM are asymptomatic in young patients meanwhile SCD could be the first clinical event to emerge, early recognition of this abnormality is pivotal to reduce mortality in this population.

Keywords: sudden cardiac death, hereditary, ICD, Arrhythmogenic cardiomyopathy, arrhythmia

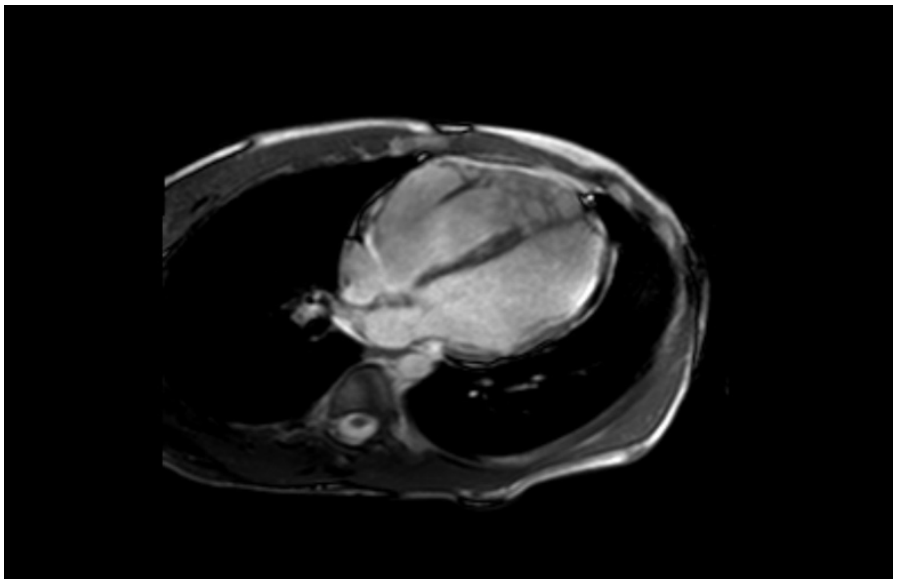

Figure 1. Fatty infiltration captured at right ventricular free wall from cMRI

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A RARE PRESENTATION OF A WIDOW-MAKER HEART ATTACK AND INTUBATION COMPLICATION IN A YOUNG PATIENT WITH HYPOKALAEMIA

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Background:
A widow-maker heart attack refers to a total occlusion of the left anterior descending (LAD) artery that leads to a cardiac arrest. Acute myocardial infarction (AMI) in young adults, though less common, presents unique clinical challenges and underscores the need for prompt, effective management. This case report illustrates a dramatic presentation of AMI in a young adult, highlighting the need of acute and critical management.

Case illustration:
A 33-year-old male presented to the emergency department with acute onset of severe, substernal chest pain and diaphoresis. The patient had a significant smoking history of over a decade but no other cardiovascular risk factors. An initial electrocardiogram (ECG) revealed ST-segment elevation in the anterior and septal leads. Elevated cardiac troponins confirmed the diagnosis of AMI, and blood tests showed a low potassium level (2.4 mmol/L). In the emergency room, the patient experienced a seizure and ventricular fibrillation, necessitating immediate intubation and defibrillation. Return of spontaneous circulation (ROSC) was achieved within minutes. Urgent coronary angiography revealed a 100% occlusion of the LAD artery with a large thrombus, but no significant stenosis in other arteries. Thrombus aspiration and percutaneous coronary intervention (PCI) with a second-generation drug-eluting stent (DES) placement were successfully performed. Three days post-PCI, the patient underwent bronchoscopy to retrieve a tooth fragment dislodged during intubation. Extubation followed two days later, and he was transferred to a regular ward. The patient was initiated on dual antiplatelet therapy, statins, and anticoagulation therapy. Echocardiography showed a left ventricular ejection fraction (LVEF) of 31%. He was discharged in stable condition, started on medication for heart failure, and scheduled for angiography evaluation in three months.

Conclusion:
Accelerated coronary artery disease was the underlying cause of AMI in this patient. Compared to elderly patients, younger individuals lack collateral arteries and ischemic pre-conditioning, potentially leading to severe complications as seen in this case. The presence of hypokalaemia may have aggravated the condition, contributing to cardiac arrest. This case highlights the importance of immediate intervention. The patient was successfully managed with timely coronary revascularisation and potassium correction followed by bronchoscopy.

Keywords: young, hypokalaemia, thrombosis, Acute myocardial infarction

A. B.

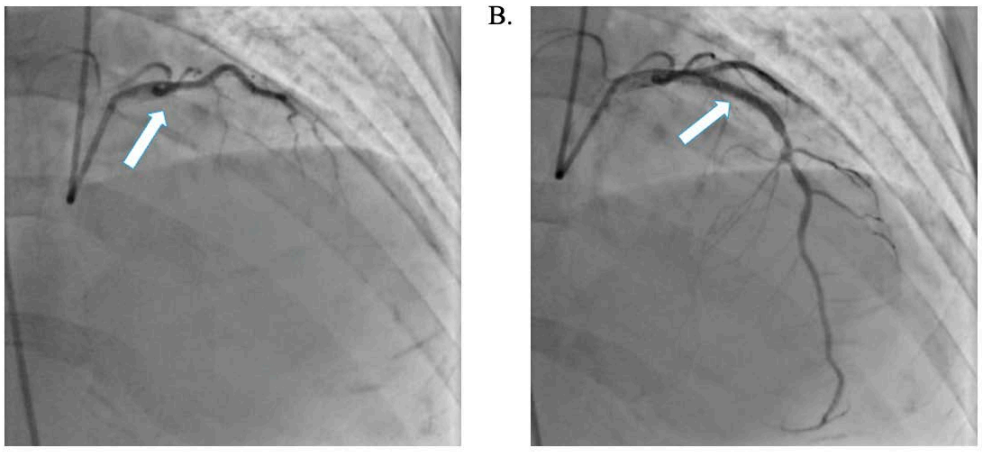

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Figure 1. Coronary angiography. (a) Pre-percutaneous coronary intervention angiogram showed total occlusion of the left anterior descending artery (white arrow). (b) Post stenting showed increased flow to the left anterior descending artery (white arrow)

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A 45 YEARS OLD MAN WITH VERY LATE STENT THROMBOSIS AND NON - ST ELEVATION MIOCARD INFARCTION WHICH HAVE BAD COMPLIANCE DUE TO DUAL ANTI-PLATELET THERAPY

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Background:
Stent thrombosis is defined as a thrombotic occlusion of a coronary stent and major complication associated with stent placement in percutaneous intervention (PCI). Dual antiplatelet therapy (DAPT) has been shown to reduce stent thrombosis and major adverse cardiac event after treatment. The authors of this article describe a case of very late stent thrombosis and non-ST elevation myocardial infarction associated with non-adherence DAPT.

Case illustration:
A 45 years-old man came to the emergency department (ED) presented with chest pain for the last seven and a half hours. Chest pain radiated to the back. But, pain has worsened since the last two hours. He had a history of coronary artery disease and got percutaneous intervention (PCI) stent Drug Eluting Stent (DES) at right coronary artery (RCA) in 2019. Thus, he did not see a doctor and non-adherenced due to therapy for the last two years. On physical examination, his blood pressure was 93/54 mmHg, pulse rate 62 beats per minutes, and no significant gallops and murmur on cardiac auscultation. Rales was not heard on the lung. Hs-troponin was 1859 ng/L, and electrocardiograph (ECG) showed Non - ST segment elevation myocardial infarction. He received acetylsalicyclic acid, ticagrelor in the ED, then admitted to the intensive coronary care unit (ICCU). Computed tomography angiography showed total occlusion in the distal right coronary artery (RCA)

Conclusion:
In our patient, the suspected precipitants of his stent thrombosis were non-compliance to medication. However, he had been taking the medication for approximately 12 months, and continued to see his doctor for up to 2 years post the first PCI. Afterwards, the patient never saw his doctor again. The most common causes of stent thrombosis across multiple studies is premature cessation of DAPT. Other risk factors include patient characteristics likes ; diabetes, acute coronary syndromes and procedural factors. Platelets have a pivotal role in thrombus formation including stent thrombosis and, thus, an optimal anti-platelet therapy is a crucial therapy for prevention of stent thrombosus. Identification of precipitant factors play an essential role in managing very late stent thrombosis, especially in coexisting life-threatening clinical conditions that require immediate treatment.

Keywords: coronary, antiplatelet, bad compliance, precipitant factors, stent thrombosis

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UNSTABLE SUPRAVENTRICULAR TACHYCARDIA AVRT: HOW TO DEAL IN RURAL EMERGENCY ROOM SETTING

Hariadi[1]

[1]RSI AT-TAQWA GUMAWANG

Background:
Supraventricular tachycardia (SVT) is a dysrhythmia originating at or above the AV node, characterized by a narrow QRS complex and a heart rate exceeding 100 bpm. One of SVT is Atrioventricular Re-entry Tachycardia (AVRT) which has abnormal cardiac electrical conduction through accessory pathways (AP). This condition can result in life-threatening arrhythmias, hence requiring immediate cardioversion treatment. However, electrical cardioversion is not always available, therefore, an alternative approach is needed.

Case illustration:
A 45-year-old man came to RS Islam At-Taqwa with suddenly lost consciousness while delivering a lecture. Examination in emergency room revealed fully conscious and the patient complained vomiting, diaphoresis, palpitations, and mild shortness of breath. There is no history of hypertension or diabetes mellitus. The patient did not report any chest pain but did experience epigastric pain radiating to the back. The patient has a history of smoking and had one prior episode of syncope 6 months ago. Vital signs were blood pressure 120/80 mmHg, HR 190 - 200 x/min, RR 25x/min, and oxygen saturation 95% room air. Physical examination revealed no murmur, rales and gallop. Initial ECG revealed regular narrow QRS <120 ms with hidden P–wave and atrial rates of 190 bpm. Laboratory results were within normal limits, however, cardiac enzyme tests were not conducted due to unavailability. We assesed the patient as an Unstable SVT. Due to direct-current (DC) synchronized cardioversion was not availabe, we administrated drip of 150 mg amiodarone in 100 cc ns within 10 minutes. Then, palpitations were relief with BP 125/80 mmHg, HR 115x/min. Surprisingly, the ECG showed sinus with delta waves and shortened PR interval at lead I, aVL, V4, V5, and V6. This phenomenon was later identified as Wolff-Parkinson-White (WPW) syndrome. The patient was admitted to the High Care Unit for observation.

Conclusion:
SVT in WPW Syndrome can be lethal due to rapid conduction into the ventricles. In a rural setting, treating this patient was challenging due to numerous limitations. However, initial management with pharmacological cardioversion is feasible.

Keywords: Cardioversion, Atrioventricular Reentrant Tachycardia, Supraventricular Tachycardia

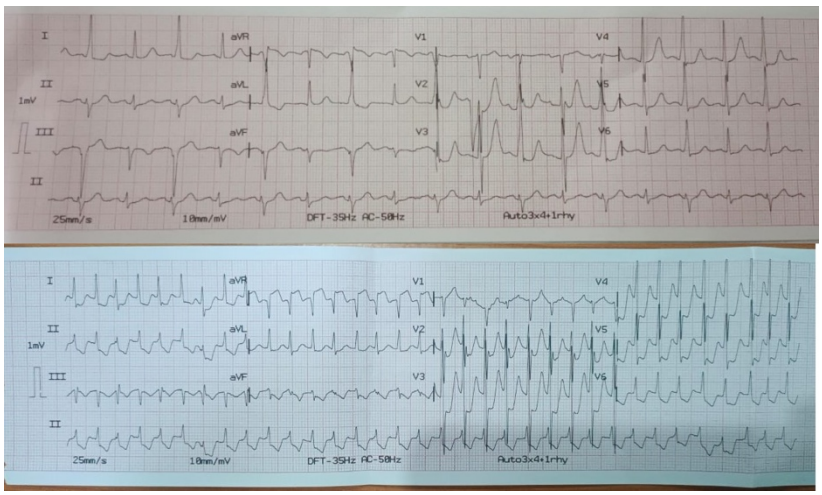

Figure 1. Initial ECG showed (A); ECG after drip amiodarone convert to sinus with WPW syndrome (B).

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THE IMPACT OF CYP2C19 GENOTYPING ON THE GUIDANCE OF ANTIPLATELET THERAPY IN A CASE OF IN-STENT RESTENOSIS

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[1]Universitas Syiah Kuala / RSUD dr Zainoel Abidin

Background:
Clopidogrel-induced platelet inhibition is patient-specific. Current available data show that about 4% to 34% of patients treated with conventional doses of clopidogrel do not display adequate antiplatelet response. It is well-established that patients carrying CYP2C19 loss-of-function (LOF) alleles have a reduced capacity for clopidogrel bioactivation, impaired platelet inhibition, and a significantly higher risk of MACE when treated with clopidogrel compared with patients without a LOF allele.

Case illustration:
A 73-year-old man with a long-standing history of recurrent chest pain presented with progressive chest pain. He denied DM and hypertension. He had a history of PCI since 7 years ago and claimed to have undergone consecutive procedures with a total of three drug-eluting stents due to chest pain. Initial ECG was within normal limits and echocardiogram showed akinetic in apical, inferior basal-mid and inferoseptal segments, other segments were hypokinetic with decreased left ventricular systolic function. Coronary angiography showed 80-95% in-stent restenosis in LM, LAD and LCx stents. CTO was also seen in the proximal RCA. The patient admitted to regularly taking dual antiplatelet drugs (clopidogrel and ASA) but still experienced chest pain several weeks after DES implantation. Resistance to clopidogrel was suspected. The patient underwent clopidogrel genotyping molecular testing. Apparently, the patient had loss-of-function (LOF) CYP2C19 *2/*2 allele and was classified as a Poor Metabolizer of clopidogrel. Antiplatelet was switched to Ticagrelol. There were no further ischemic events during the remainder of the hospitalization and the patient was discharged in stable condition. The ELEVATE-TIMI 56 trial found that a clopidogrel dose of 225 mg daily in patients with one CYP2C19 LOF allele was necessary to obtain platelet inhibition that was similar to that observed with 75 mg daily of clopidogrel in patients without a LOF allele. In patients with 2 LOF alleles, even doses as high as 300 mg daily of clopidogrel did not result in equivalent platelet inhibition. Retrospective analyses of patient registries and clinical trials have consistently shown that Intermediete Metabolizer and Poor Metabolizer treated with clopidogrel have an increased risk of adverse cardiovascular outcomes after PCI compared with those without a LOF allele.

Conclusion:
Our case demonstrates the existence of a subgroup of patients with clopidogrel resistance. As antiplatelet therapy has become the cornerstone of modern cardiovascular treatment, this has led to an increase in stent thrombosis and recurrent ischemic events adding to healthcare costs, as well as increasing periprocedural morbidity and mortality.

Keywords: CYP450, Clopidogrel Resistance, Pharmacogenomics, Poor Metabolizer, CYP2C19

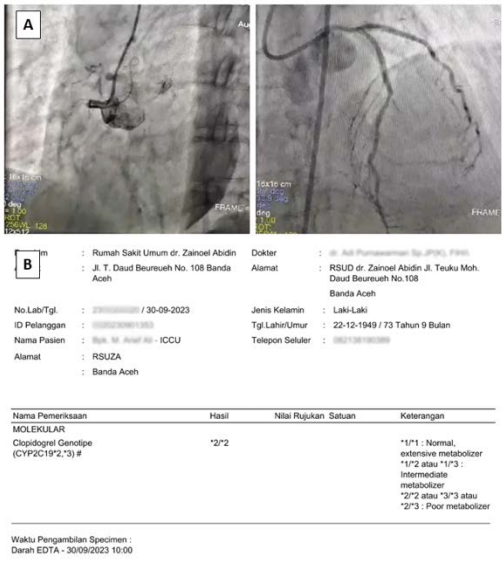

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Figure 1. A. In-stent restenosis at LM, LAD and LCx and CTO on proximal RCA; B. The Clopidogrel genotype (CYP2C19) test

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UNVEILING THE COMPLEXITIES OF DILATED CARDIOMYOPATHY: INSIGHTS FROM A PEDIATRIC CASE STUDY

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Background:
Dilated cardiomyopathy (DCM) is a condition characterized by the enlargement of the heart chambers, leading to decreased cardiac function. This case report discusses a pediatric patient diagnosed with DCM and its correlation with multiple right ventricle thrombi. The patient presented with symptoms of congestive heart failure, prompting further investigations including echocardiography and electrocardiography.

Case illustration:
The echocardiographic findings revealed dilatation of all heart chambers, reduce left ventricular with ejection fraction 38% and reduced right ventricular function with TAPSE 0.9cm, and the presence of thrombi attached to the right ventricular outflow tract and apex, respectively with dimensions of 3.2 cm^2 and 3.3 cm^2. The patient's laboratory results showed mild anemia, normal leukocyte count, and elevated D-dimer levels. Electrocardiography indicated sinus tachycardia with a slightly prolonged QTc interval. The management of the patient included anticoagulation therapy with warfarin to address the thrombi and optimize cardiac function.

Conclusion:
This case highlights the importance of early detection and management of DCM in pediatric patients to prevent complications such as thrombus formation. Echocardiography played a crucial role in diagnosing the condition and guiding treatment decisions. Anticoagulation therapy was effective in resolving the thrombi, emphasizing the significance of a multidisciplinary approach in managing pediatric cardiomyopathies. Further research and clinical studies are warranted to enhance our understanding of DCM in children and improve outcomes in this patient population.

Keywords: Multiple Thrombi, Dilated cardiomyopathy, Right Ventricle Thrombus

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FACING NIGHTMARE AFTER PERCUTANEOUS PATENT DUCTUS ARTERIOSUS CLOSURE IN RURAL AREA: A KEY LESSON

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[1]RSUD Prof. DR. W. Z. Johannes

Background:
Patent Ductus Arteriosus (PDA) comprises 5-10% of congenital heart diseases and is estimated to occur in approximately 1 in 2000 live births. Percutaneous closure of PDA has become the preferred method due to lower complication rates and quicker patient recovery. However, occluder device migration and embolization to the heart chambers or distal vasculature can occur. We present a case of PDA closure with device embolization in a resources-limited setting.

Case illustration:
A 12-year-old female was admitted with chief complaint dyspnea and easily fatigue. On physical examination, a continuous murmur was noted in left infraclavicular area. Transthoracic echocardiography revealed an 8.4 mm PDA. The patient then underwent PDA Closure using no. 14/16 HeartR PDA Occluder with antegrade transvenous method and fluoroscopy guided.
Initially, the procedure appeared successful with no residual shunt observed. Three hours later, it was discovered that the occluder device had dislodged into the distal right pulmonal artery. Device embolization into a pulmonary artery branch is usually well tolerated and rarely causes acute hemodynamic instability. If a device embolizes into the aorta, immediate intervention is necessary due to systemic hypoperfusion, which can affect critical organs such as the gut, kidneys, or lower extremities.
An immediate fluoroscopy guided percutaneous retrieval attempt of the embolized device was performed using no. 20 snare, which is the only size available, but those attempts were failed. Due to the available resources, the patient was referred to the national cardiovascular healthcare centre for retrieval of the embolized device. The device was successfully retrieved with a no. 10 snare. The patient underwent a subsequent PDA closure using the retrieved device.

Conclusion:
Device embolization is a known complication of percutaneous PDA closure, occurring in 0% to 3.1% of cases. These complications can be reduced by proper expertise and optimal sizing of the ductus. Despite efforts to minimize risks, device embolization can occur, and it is important to always be prepared for a possible retrieval. Preparation includes access to a transcatheter snare kit, diagnostic catheters, and retrieval sheath and even a cardiac surgeon.

Keywords: Percutaneous Closure, Patent Ductus Arteriosus, Device Embolization

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BRUGADA SYNDROME MIMICKING ST ELEVATION MYOCARDIAL INFARCTION: NOT ALL ST ELEVATION ARE STEMI

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Background:
Brugada syndrome (BrS) is an inherited cardiac disorder that is frequently associated with mutations in the SCN5A gene.Precipitating factors such as fever, increased vagal tone and other drugs may trigger BrS. Brugada pattern type 1 showed a right bundle branch block (RBBB) with ST elevation in precordial lead (V1-V3). These characteristics are often misdiagnosed as STEMI.

Case illustration:
Case 1A-52- year old male presented to the emergency department with chief complaints of melena and jaundice 2 weeks before admission. Patient mentioned a previous history of near-syncope. There was no family history of syncope and sudden cardiac death. On examination the patient was hypotensive and looked lethargic. Patient was admitted to the ICU. During routine ECG evaluation, it was determined a ST elevation in antero-septal lead (V1-V3) followed by an inverted T wave. There was no complaint of chest pain and dyspnoea. It was initially suspected as a STEMI. Troponin I levels were 52,3. Echocardiograms were normal with global normokinetic. This case was further concluded as a brugada type 1 pattern that mimics an acute coronary syndrome with ST elevation.Case 2A-64- year old male presented to the emergency department with a chief complaint of typical chest pain 1 day prior admission, accompanied with nausea and fever. Neither previous episodes of syncope nor family history of sudden cardiac death were reported. Physical examinations including vital signs and laboratory examinations were all within normal range. ECG on admission showed ST segment elevation in V1-3, suspected for anterior wall infarction. Troponin I levels were within normal range. These findings were further concluded as brugada type-1 patterns. Echocardiography examinations were normal with global normokinetic. CT angiography showed a non-significant stenosis with myocardial bridging at the left anterior descending coronary artery.

Conclusion:
We report a case series with similar ECG findings that mimics STEMI. In this case we want to highlight that not all ST elevation are STEMI, a meticulous history taking and diagnostic tests play an important role in ruling out STEMI diagnosis. EPS and ajmaline test is needed to establish a definite diagnosis and choose appropriate therapy.

Keywords: STEMI, Brugada Syndrome

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ACUTE ONSET STEMI WITH THREE-VESSEL DISEASE IN A YOUNG PATIENT: A CASE FROM A REMOTE AREA

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Background:
ST-Elevation Myocardial Infarction (STEMI) is a critical cardiovascular emergency requiring timely intervention to restore myocardial perfusion. In rural settings, limited access to advanced cardiac care poses significant challenges in managing such cases. STEMI typically affects older individuals with established cardiovascular risk factors. However, the occurrence of STEMI in younger patients, particularly those with three-vessel disease, is relatively rare and presents unique diagnostic and therapeutic challenges.

Case illustration:
A 44-year-old male with no significant past medical history presented with acute onset of severe chest pain, radiating to the left arm, and associated with diaphoresis and dyspnea. An initial electrocardiogram (ECG) revealed pronounced ST-elevation in leads V1-V6 with hyperacute T wave, consistent with acute onset of anterior STEMI. Given the unavailability of primary percutaneous coronary intervention (PCI), the decision was made to initiate fibrinolytic therapy with streptokinase. Adjunctive antiplatelet and anticoagulant therapies were also administered as per standard protocol. The patient showed significant clinical improvement within 90 minutes post-therapy, with resolution of chest pain and marked reduction in ST-segment elevation on follow-up ECG. After that, the patient was referred to coronary angiography which revealed severe triple vessel disease (3VD), characterized by 90% stenosis at proximal LAD, Extended stenosis in the LCx with a maximum stenosis of 80% in the proximal part, and 90% stenosis at mid-RCA. all with TIMI (Thrombolysis In Myocardial Infarction) flow grade 3. The presence of three-vessel disease in a young patient with STEMI is uncommon. This case raises questions about the underlying mechanisms, including pre-existing plaques, genetic predispositions, inflammatory processes, or prinzmetal angina. The hyperacute T-waves and severe stenosis suggest a complex interplay of factors beyond typical STEMI pathology. Despite the fibrinolytic therapy being appropriate given the acute presentation, the extent of coronary artery disease found on angiography was unexpected.

Conclusion:
STEMI in young patients with extensive three-vessel disease poses a significant clinical challenge. Prompt recognition, detailed angiographic assessment, and appropriate interventional strategies are crucial for favorable outcomes. Further research is needed to elucidate the underlying etiologies and optimize management approaches for this unique patient population.

Keywords: Triple Vessel Disease, Anterior Myocardial Infarction, ST-Elevation Myocardial Infarction, Fibrinolytic Therapy

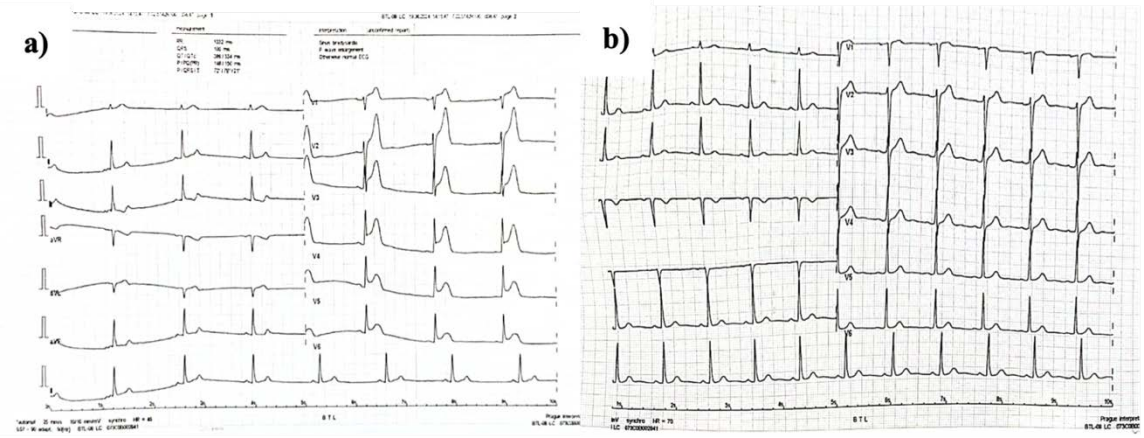

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Figure 1. The ECG before and after fibrinolytic. a) sinus bradycardia with ST-segment elevation in V1-V6, b) normal sinus rhythm.

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ATRIAL FIBRILLATION AND HYPERTHYROIDISM IN INFANTS WITH NORMAL CARDIAC STRUCTURE

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[1]Eka Hospital

Background:
Cardiac arrhythmias in pediatrics is very rare with 1% prevalence. Common arrhythmias occurring in neonates to infants include sinus tachycardia, atrioventricular reentrant tachycardia, and atrial flutter. Atrial fibrillation is quite rare in infants and children. The possibility of atrial fibrillation occurring in pediatric can be attributed to atrial septal defect,cardiomyopathyand hyperthyroidism. Uncontrolled atrial fibrillation can increase the risk of heart failure and thromboembolic events, leading to prolonged hospital stays.

Case illustration:
A 2-months-old boy weight 5.9 kg came to emergency department with history of dyspneu for 1 day prior to admission. The baby was born vaginally without any complications. There were no signs of cyanosis. On physical examination, the pulse was 216/min, blood pressure was 100/55 mmHg, respiratory rate was 35/min, temperature was 36.7°C, and SpO2 was 99-100%. An electrocardiogram revealed atrial fibrillation with a rapid ventricular response of 203 bpm. Chest X-ray showed slight left lung markings. Subsequent echocardiography revealed mild dilation of the left ventricle, 2mm patent foramen ovale with left-to-right shunt, and left ventricular ejection fraction 57%. The patient admitted to Cardio Vascular Critical Care Unit for stabilization. Blood tests showed elevated FT4 (1.65 ng/dL), TSH (6.73 ul/ml), and FT3 (5.3 pg/mL), suggesting subclinical hyperthyroidism.During CVCU treatment, the patient received amiodarone drip at initial dose 25 mcg/kg/min within 4 hours and continue with maintenance dose, and additional propranolol tablets. Then ECG monitor showed sinus rhythm with 146-160 bpm, with systolic 66-110 mmHg and diastolic 40-55 mmHg, SpO2 98-100%. The patient was managed in CVCU for 2 days and then transferred to ward without any symptoms with a stable heart rate <170 bpm.

Conclusion:
The importance of understanding the relationship between disorders of the endocrine system and cardiovascular events cannot be overstated. Thyroid hormones serve both physiological functions and can act as arrhythmogenic agents. Patients with subclinical hyperthyroidism are at significant risk for developing atrial fibrillation. Although rare in pediatric cases, always consider congenital hyperthyroidism as an etiology in infants and the use of amiodaron.

Keywords: atrial fibrillation, paediatrics, hyperthyrroidism, arrythmia

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SUPRAVENTRICULAR TACHYCARDIA WITH ABERRANCY DUE TO BETA-BLOCKER ABRUPT CESSATION IN PAROXYSMAL ATRIAL FIBRILLATION PATIENT: A CASE REPORT

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Background:
Supraventricular tachycardia (SVT) with aberrancy is a dysrhythmia originating at or above the atrioventricular (AV) node with wide QRS complex. Beta-blockers, such as bisoprolol, are commonly used to control heart rate and maintain rhythm stability in atrial fibrillation (AF) patients.

Case illustration:
A 64-year-old male patient presented to the emergency room (ER) due to irregular heartbeat and agitation. Upon examination, the patient was alert with heart rate (HR) of 50 bpm and blood pressure (BP) of 120/67 mmHg. Electrocardiography (ECG) revealed AF with slow ventricular response (AFSVR) and premature ventricular contraction (PVCs). The patient had previous history of percutaneous coronary intervention with stent placement on right coronary artery in December 2023. His prescribed medications included bisoprolol 1.25mg q.d, furosemide 40mg q.d, ramipril 5mg q.d, dual antiplatelet therapy (DAPT), and statin. However, the patient refused to be admitted. Due to AFSVR, bisoprolol was temporarily stopped. Four days later, the patient returned to the ER with palpitation and agitation that had persisted since the previous night. Upon examination, he was alert with HR of 156 bpm and BP of 144/81 mmHg. ECG revealed regular wide QRS complexes tachycardia, RBBB-like morphology, and no AV dissocation; indicative of SVT with aberrancy. After administering 150 mg intravenous bolus of amiodarone, the ECG converted to sinus rhythm. During inpatient care, the patient was given amiodarone maintenance dosage and reinitiated on bisoprolol 1.2 5mg q.d., along with his previous medications. No further episodes of SVT were observed.

Conclusion:
This case underscores the risks associated with the abrupt cessation of beta-blocker therapy in patients with paroxysmal AF, as evidenced by the development of SVT with aberrancy. The initial decision to withhold bisoprolol due to AFSVR led to significant tachyarrhythmia, necessitating acute intervention with amiodarone to restore sinus rhythm. The patient's subsequent stabilization with a maintenance dose of amiodarone and reintroduction of bisoprolol highlights the need for careful and continuous management of antiarrhythmic medications.

Keywords: Atrial Fibrillation, Beta-Blockers, Supraventricular Tachycardia

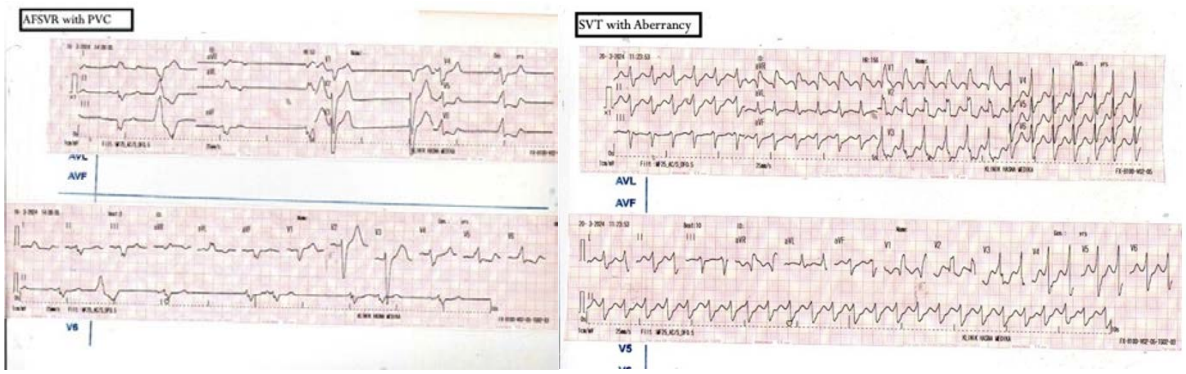

Figure 1. The first ECG is atrial fibrillation with slow ventricular response and premature ventricular contractions during initial patient presentation and the second ECG is supraventricular tachycardia with aberrancy after beta blocker cessation

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UNMASKING SEVERE ANEMIA PRESENTING AS ACUTE CORONARY SYNDROME: CASE REPORT ON CLINICAL MIMICRY

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Background:
Myocardial Infarction (MI) is a leading cause of morbidity and mortality worldwide. While coronary artery occlusion is typically the main cause, other mechanisms can also contribute to MI. In this report, we present a patient with severe anemia that mimics acute coronary syndrome (ACS).

Case illustration:
A 60-year-old man was admitted to the Emergency Department (ED) with severe chest pain (NPRS: 7/10), diaphoresis, and general weakness persisting for 3 days, worsening just before admission. He reported chest pain over the last 7 months, typically relieved by rest, but this time, no position alleviated his pain. The pain duration was over 15 minutes. The patient's heart rate was 60 bpm, blood pressure 153/61 mmHg, and heart sounds were normal. He appeared extremely pale. ECG showed ST segment depression in most leads and ST segment elevation in the aVR lead. Laboratory results revealed severe anemia and thrombocytopenia (Hemoglobin: 4.5 g/dL; Platelet count: 7000/uL) and elevated cardiac troponin I (cTnI: 24.6 pg/mL). Echocardiography showed no abnormalities. After hemoglobin correction, the patient's chest pain resolved. The mismatch between oxygen supply and demand is believed to be the underlying pathophysiology of chest pain in this patient.

Conclusion:
Diagnosing myocardial infarction accurately requires understanding its subtypes. Differentiating between acute coronary syndrome and other types of myocardial infarction is crucial as it determines the diagnostic, management, and prognostic strategies for patients with myocardial infarction.

Keywords: Myocardial infarction, severe anemia, acute coronary syndrome

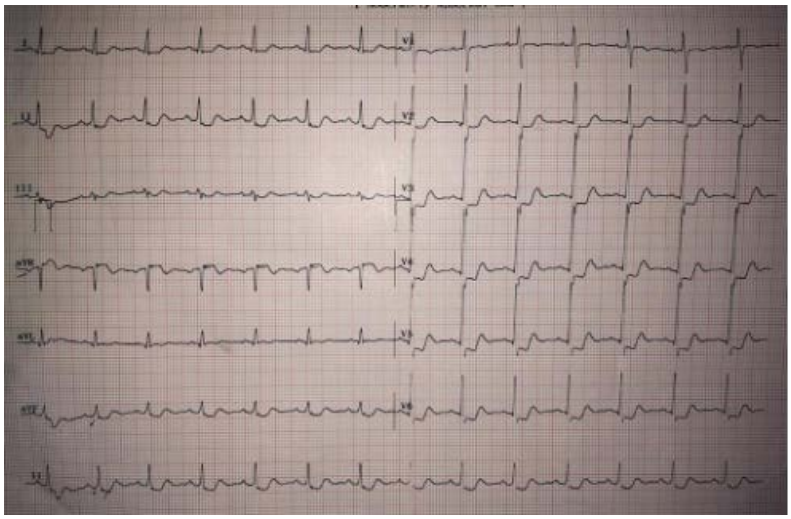

Figure 1. ECG on admission

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MYOCARDIAL STUNNING IN POST-CARDIAC ARREST PATIENTS WITH VERY HIGH-RISK NON-ST ELEVATION ACUTE CORONARY SYNDROME: A CASE REPORT

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Background:
Myocardial stunning is a reversible myocardial dysfunction phenomenon that can be caused by several conditions, including focal ischemia, such as acute coronary syndrome (ACS), and global ischemia, such as cardiac arrest. Two hypotheses underlying the mechanism of myocardial stunning are oxygen radical damage in a few minutes after reperfusion and desensitization of myofilaments by altered calcium inflow.

Case illustration:
A 61-year-old woman with a history of uncontrolled hypertension and obesity presented with loss of consciousness in the past 1 hour. She previously complained of breathlessness and chest pain. The patient experienced cardiac arrest and achieved a return of spontaneous circulation after 10 minutes. Echocardiography revealed left ventricle ejection fraction (LVEF) of 32.9% and global hypokinetic. The patient was diagnosed with very high-risk non-st-elevation ACS, suspected left main coronary artery obstruction with differential diagnosis of three-vessel disease, acute lung edema with respiratory failure, and cardiogenic shock. She was intubated and treated with dual antiplatelet therapy, anticoagulant, atorvastatin, and hemodynamic support with dobutamine and dopamine. After 18 hours, her cardiogenic shock resolved, and she was extubated. On the third day after cardiac arrest, echocardiography revealed LVEF of 69.5%, resolved wall motion abnormalities into global normokinetics. She was discharged after 9 days and still visited the outpatient clinic after 6 months.

Conclusion:
Myocardial stunning is reversible. Diagnosis can be made retrospectively or prospectively in conditions with available facilities. Treatment should address the underlying mechanism; however, in severely altered cardiac function conditions, close monitoring and supportive treatment should be given.

Keywords: acute coronary syndrome, case report, non-st elevation myocardial infarction (NSTEMI), myocardial stunning, cardiac arrest

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INTERVENTION OF RIGHT CORONARY TOTAL OCCLUSION AND SEVERE AORTOILIAC DISEASE: ONE STONE TWO BIRDS

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Background:
Coronary artery disease with severe calcification presents significant challenges for percutaneous coronary intervention (PCI). When complicated by concurrent severe aorto-iliac stenosis, the procedure's complexity increases further. This case study examines the procedural challenges and outcomes associated with simultaneous management of both conditions.

Case illustration:
A 87-year-old male with a history of diabetes mellitus, and smoking presented with chest pain and was diagnosed with an ST-elevation myocardial infarction (STEMI). Coronary angiography revealed a heavily calcified total occlusion of the right coronary artery (RCA). Additionally, severe aorto-iliac stenosis was identified, complicating vascular access for the intervention. The patient underwent PPCI through the femoral approach. Initial attempts were hindered by the aortoiliac stenosis, necessitating the placement of a stent in the iliac artery to facilitate access. Following successful stenting, primary PCI was performed on the RCA occlusion using drug-eluting stents. The GuideZilla guide extension catheter was utilized as for equipment delivery in both the aortoiliac and coronary arteries. The intervention resulted in successful revascularization of both the aortoiliac and coronary arteries. The patient had an uneventful recovery and was discharged on a regimen including antiplatelet therapy, statins, and lifestyle modifications.

Conclusion:
This case underscores the importance of recognizing and addressing peripheral arterial disease in patients undergoing primary PCI. Aortoiliac artery stenting can be a crucial step in ensuring successful coronary intervention, highlighting the need for a comprehensive approach in managing complex cardiovascular diseases.

Keywords: Aorto-iliac stenosis, Percutaneous coronary intervention, STEMI

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A 1-YEAR FOLLOW-UP COVERED ENDOVASCULAR RECONSTRUCTION OF AORTIC BIFURCATION WITH GRAFT OF AORTOILIAC OCCLUSIVE DISEASE WITH REOCCLUSION IN BELOW THE KNEE LESSION AFTER DRUG COATED BALLOON ANGIOPLASTY FOR CHRONIC LIMB THREATENING ISCHEMIA: A CASE REPORT

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Background:
The Covered Endovascular Reconstruction of Aortic Bifurcation (CERAB) is the first line treatment for Aortoiliac Occlusive Disease (AOD) with patency rates up to 90%. Chronic Limb Threatening Ischemia (CLTI) induced by AOD alone is thought to be a rare pathological entity. CLTI patients typically present with complex multi-level lesions and below the knee arteries are frequently involved. Reocclusion rates remain high and present the major limitation of infrapopliteal interventions.

Case illustration:
A 59-years-old female presented with complaint of pain in her left knee. She had no other symptomps such as intermitten claudicatio, rest pain, also ulcer. She had history of Chronic Total Occlussion (CTO) in CLTI femoropoplitea sinistra with post percutaneous transluminal balloon angioplasty (PTA) and AOD Trans Atlantic Inter-Society Consensus (TASC) D post CERAB with graft in June 2023. After those procedures, vascularization from the Computed Tomography Angiography (CTA) were good, the wound healing was improved and she had no complaints. She regularly visited the cardiovascular inpatient clinic once a month for follow-ups. Another CTA was performed, showed a CTO in the popliteal artery sinistra, but the patency of CERAB remained good with no endoleak, stent deformity, in-stent restenosis, or thrombus. Repeat PTA with drug coated balloon was performed in July 2024, injection contras showed TIMI Flow 3 from aorta to distal bilateral pedis, no dissection and no residual stenosis. Our patient was given atorvastatin, anti-platelet, and rivaroxaban for medications. One week follow-up after the procedure, she had no complaint.

Conclusion:
The CERAB technique is associated with favorable short-term outcomes. Recent evidence indicates a primary patency rate of 90% at one year for TASC D lesions. Despite advancements in interventional techniques and devices, the rates of restenosis and reocclusion after infrapopliteal interventions remain high. This limits long-term success and requires frequent repeat procedures, which often become increasingly complex. As a result, the need for repeat interventions presents a significant challenge for both healthcare providers and patients.

Keywords: chronic total occlusion, aortoilliac disease, reocclusion, chronic limb threatening ischemia, drug coated balloon angioplasty

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SPONTANEOUS CORONARY ARTERY DISESSECTION: A CLINICAL CHALLENGES TO UNMASKING THE UNDERDIAGNOSIS OF SCAD IN YOUNG FEMALE

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Background:
Spontaneous coronary artery dissection (SCAD) is a form of ACS that is rarely diagnosed which estimated to account for 10-25% of ACS presentations in women aged < 50 years. Some reasons for lack of diagnosis of SCAD are presentation at sudden death, widespread belief that young women are at low risk for ACS, resulting in less frequent coronary angiography. In this case report, we report SCAD in a young woman confirmed by coronary angiography.

Case illustration:
A 37 years old female patient came to the Dr. Wahidin Sudirohusodo Integrated Heart Center Hospital with complaints of typical chest pain since 5 hours. No previous history of similar complaints. The patient had no significant coronary risk factors. Clinical Examination was unmarkable with BP 107/75 mmHg. An ECG at non-PCI center revealed ST Elevation in leads I, aVL, V1-V3, and serial ECG at PCI center with only T inverted at V1-V3. Hs-Troponin I were elevated. She was diagnosed with STEMI anterior wall KILLIP I with spontaneous reperfusion. Echocardiography bedside showed normal LV systolic function and Global Normokinetic. The patient underwent Primary PCI revealed that Left Main Normal, Diffused stenosis from mid to distal LAD, reduced intracoronary diameter compared to proximal segment, LCx Normal, RCA dominant vessel with conclusion CAD 1 vessel disease suspect SCAD dd coronary spasm at LAD. The patient was advised to undergo MSCTA Cardiac but the patient refused due to her financial reason.

Conclusion:
Diagnosis of SCAD is ordinarily made by coronary angiography, but in this case, separating between intramural dissection, atherosclerotic stenosis, or spasm may be challenging. Patient was recommended MSCTA Cardiac for assist diagnostics, but unfortunately, she denied due to budgetary reasons. For this challenging circumstance when managing with chest pain, especially in youthful healthy females who don't have any hazard factors for atherosclerosis, doubt of SCAD is vital, so we chosen to treat the patient using medical treatment rather than percutaneous coronary intervention.

Keywords: Spontaneous reperfusion, Spontaneous Coronary Artery Dissection, Coronary Spasm

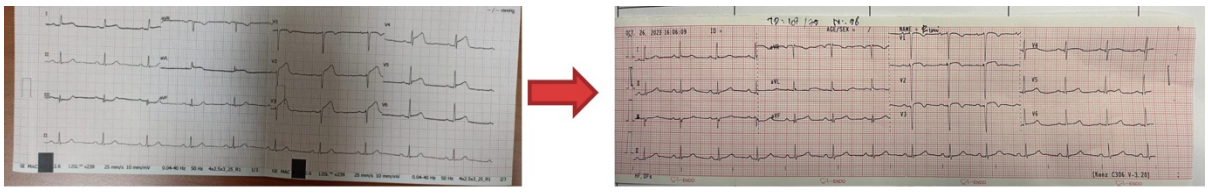

Figure 1. ECG at Non-PCI center with STEMI Extensive Anterior onset 30 minutes and at PCI center onset 5 hours with Inverted T wave at V1-V3

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IVIG AS THERAPY IN PATIENTS WITH LUPUS MYOCARDITIS: A CASE REPORT

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[1]RSAI

Background:
Systemic Lupus Erythematosus (SLE) is an autoimmune disorder that affects various organs. Myocarditis is a uncommon cardiac manifestation of SLE. Here we would like to report a case of SLE that manifested symptoms of myocarditis and was given IVIG therapy.

Case illustration:
A 59-year-old woman presented with a history of intermittent fevers for the past month. She had no history of cardiac disease. Patient admitted to ICU during treatment with clinical manifestations including dyspnea, orthopnea, and hypotension. Clinical examination revealed pulmonary crackles.Chest x-ray showed mild cardiomegaly with pulmonary edema and bilateral pleural effusions. Her platelet count decreased from 129,000 cells/uL to 28,000 cells/uL. COVID-19 infection was initially suspected, but PCR testing was negative. Echocardiography revealed reduced left ventricular systolic function with global hypokinesis at rest. The ejection fraction (EF) was 30.9%, with grade III diastolic dysfunction, moderate mitral regurgitation, pulmonary hypertension, and reduced right ventricular contractility (TAPSE: 11.4 mm) suggestive of myocarditis. Laboratory results showed ANA test was positive with a speckled pattern, antibody titer 1/100. Based on these results, the patient was diagnosed with lupus-induced myocarditis. Treatment included high-dose corticosteroids (methylprednisolone; 250-500 mg/day), inotropic support (dobutamine, dopamine), vasopressors (norepinephrine), and diuretic therapy (furosemide; 80 mg/day). On day 6 of treatment, the patient experienced worsening respiratory distress and cardiac arrhythmias. Invasive mechanical ventilator is placed on the patient. The patient went into cardiac arrest and cardiopulmonary resuscitation with defibrillation was performed (270 J). Return of spontaneous circulation (ROSC) was subsequently achieved. The electrocardiogram showed ventricular tachycardia. The patient was given Amiodarone 150 mg, then 1 mg/minute for 6 hours, 0.5 mg/minute for 18 hours. Considering the patient's condition, intravenous immunoglobulin (IVIG) therapy was initiated at 18 g/day for 3 days. On the 10th day of ICU care, the patient showed improvement and was successfully weaned off the ventilator. Chest X-ray showed resolution of pulmonary edema. Echocardiography demonstrated improved left ventricular function with an ejection fraction of 49.6% and normal right ventricular contractility (TAPSE: 17.8 mm)

Conclusion:
IVIG therapy in a case of lupus myocarditis, resulting in good cardiac outcomes

Keywords: systemic lupus erythematosus, lupus myocarditis

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INFERIOR ST-ELEVATION MYOCARDIAL INFARCTION WITH SPONTANEOUS CONVERSION OF NEW-ONSET ATRIAL FIBRILLATION: A CASE REPORT

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Background:
Myocardial Infarction (MI) and Atrial Fibrillation (AF) contribute significantly to the global burden of cardiovascular disease. Managing atrial fibrillation in patients hospitalized for Acute Myocardial Infarction (AMI) is one of the biggest challenges. Previously, few studies have observed New-onset Atrial Fibrillation in patients with ST-Elevation Myocardial Infarction.

Case illustration:
A 40-year-old man presented to the emergency department with typical chest pain for 30 minutes. He had no history of chest pain. Prior history included hypertension and long-term excessive smoking. The patient was alert and the initial vital signs were: blood pressure 140/97 mmHg, heart rate 105 beats/min irregular, respiratory rate 24 breaths/min, oxygen saturation 97 %, and pain score 9 out of 10. First ECG showed ST-Elevation in II, III, AVF and Atrial Fibrillation pattern. He was diagnosed with Inferior ST-Elevation Myocardial Infarction (Inferior STEMI) with New-onset Atrial Fibrillation. The patient received initial treatment for STEMI and immediate fibrinolytic therapy as part of a reperfusion strategy. Post-fibrinolysis evaluation showed signs of successful reperfusion and spontaneous conversion of atrial fibrillation to sinus rhythm. The patient was observed in the Cardiac Intensive Care Unit (CICU) for 2 days with stable hemodynamic and no recurrence of arrhythmias. On the fifth day of hospitalization, the symptoms improved and he was discharged from the general ward.

Conclusion:
STEMI patients who develop New-onset Atrial Fibrillation (NOAF) during hospitalization have poorer short- and long-term outcomes. Successful invasive treatment may have significantly shortened the duration of NOAF and reduced its adverse effect on prognosis since its duration correlates with mortality.

Keywords: Inferior STEMI, New-onset Atrial Fibrilation, Spontaneous Conversion

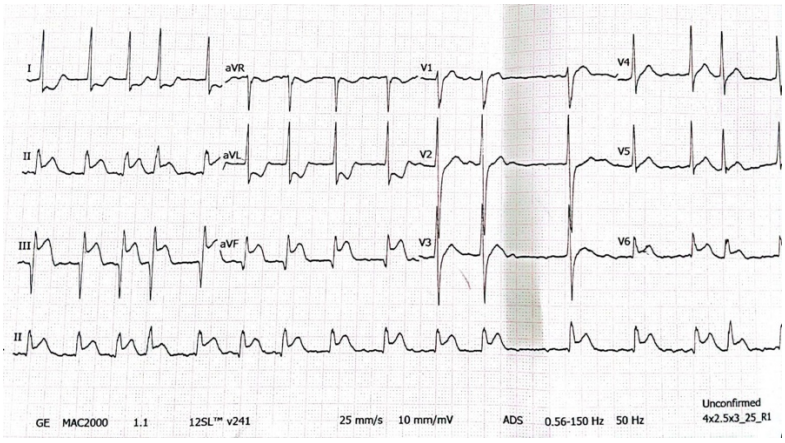

Figure 1. First ECG in emergency department showed ST-Elevation in leads II, III, AVF and Atrial Fibrillation pattern.

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PERICARDIAL EFFUSION LEADING TO CARDIAC TAMPONADE IN A PATIENT WITH MALIGNANCY: A CASE REPORT

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Background:
Pericardial effusion, the accumulation of fluid around the heart, can progress to cardiac tamponade and be fatal. This case report aims to study the patient's overall therapy and diagnosis in order to improve patient outcomes.

Case Illustration:
A 41-year-old woman went to the ER complaining of severe chest pain that radiated to her back, accompanied by shortness of breath, difficulty lying down, a dry cough, cold sweats, and weight loss over the past month. The patient has never had hypertension or diabetes, but her parents died young from cancer. The patient had hypotension, dyspnea with 8 litre of oxygen and cold extremities. The cardiovascular exam revealed jugular venous distention, but no murmurs were audible. Chest x-ray showed cardiomegaly, which was confirmed by an echocardiogram showing massive pericardial effusion. The electrocardiogram indicated electrical alternans. Leukocytosis, anemia, and a negative BTA are all relevant laboratory tests. Emergency pericardiocentesis removed 500 cc of pericardial fluid. Fluid analysis identified malignant cells with metastatic adenocarcinoma. Despite initial hemodynamic improvement with pericardial drainage and supportive care, the patient passed away four days post-admission. Pericardial effusion is often caused by neoplasms, which can lead to cardiac tamponade a medical emergency that can be deadly if not treated. Beck's triad, consisting of hypotension, muffled heart sounds, and increased jugular venous pressure, is simple to diagnose in cardiac tamponade. Initial management focuses on oxygen supplementation, volume expansion, and ensuring the patient's rest. Exercise caution when using mechanical ventilation to avoid exacerbating the venous return. To relieve cardiac compression, pericardial fluid drainage via needle pericardiocentesis is the primary treatment. While preparing for definitive treatment, volume resuscitation and pressor support are critical supportive measures.

Conclusion:
Malignancy is the primary cause of most cardiac tamponades, and patients with this specific cause typically experience a poorer outcome, likely as a manifestation of an advanced illness. The intervention’s timing is the key component. The longer the delay, the more unfavorable the outcomes become. Malignant illness-induced tamponade patients have a mortality rate exceeding 75% within a year.

Keywords: Pericardiosintesis, Malignancy, Cardiac Tamponade, Pericardial Effusion

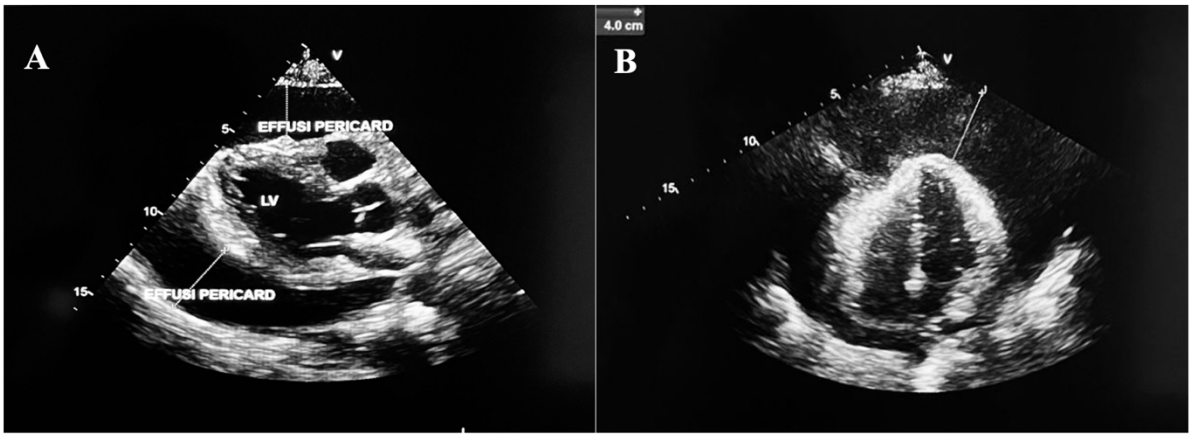

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Figure 1. A. TTE showed massive pericardial effusion posterior (+/- 2.9 cm), anterior (+/- 2.9cm) and lateral (+/- 4.0 cm). **B.** TTE showed pericardial effusion with swinging heart.

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MANAGING THE UNUSUAL ACUTE DECOMPENSATED PULMONARY HYPERTENSION WITH COMPLICATIONS FOLLOWING ATRIAL SEPTAL DEFECT – A CASE REPORT

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[1]Universitas Gadjah Mada

Background:
Pulmonary hypertension (PH) is a rare disease with diverse causes and high mortality rates, affecting about 97 per million people. Of these, 25% have a 3-year mortality rate. This report details a case of acute decompensated pulmonary hypertension (ADPH) in a patient with atrial septal defect (ASD) and related conditions.

Case illustration:
A 36-year-old man experienced sudden, worsening shortness of breath and palpitations five hours before admission. Diagnosed with ASD-PH in 2014, he had bilateral partial thrombi in the pulmonary artery and right atrial and ventricle dilatation by 2022. His medications included beraprost, sildenafil, bisoprolol, digoxin, bosentan, and warfarin. He had no familial or lifestyle comorbidities. Upon admission, his respiratory rate was 26 breaths per minute, and his peripheral oxygen saturation was 91%. Physical examination revealed a fixed-splitting heart sound with systolic murmurs at Erb's point, bilateral bronchovesicular breath sounds, and clubbing finger. Laboratory results showed polycythemia and compensated respiratory alkalosis. MSCT angiography revealed partial thrombi in both pulmonary arteries, a large ASD, cardiomegaly, and an infarction in the left lung's inferior lobe. Diagnosed with acute decompensated PH, he received ventilation with iloprost nebulization and continued his routine oral treatment without bisoprolol. He showed improvement and was discharged after seven days. Untreated ASD can lead to moderate to severe PH in 9–22% of cases. It may lead to various cardiac and pulmonary complications, including fatal ADPH, and can be triggered by thromboembolic events. ADPH treatment focuses on fluid management and pharmacological support to improve cardiac function and coronary perfusion pressure.

Conclusion:
Treating PH in adult ASD is complex due to the variability based on etiology and clinical status. Acute decompensated PH is a life-threatening complication requiring immediate management and close monitoring.

Keywords: atrial septal defect, acute decompensated pulmonary hypertension, pulmonary hypertension

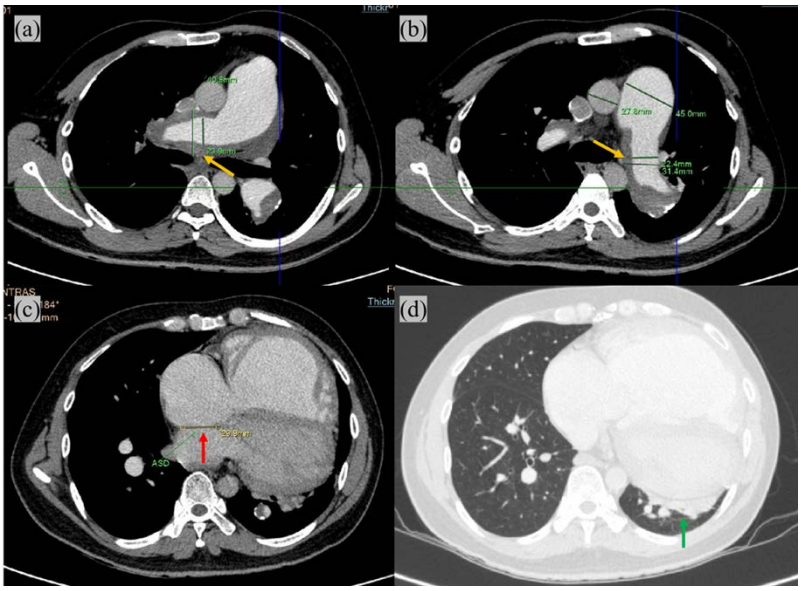

Figure 1. MSCT Angiography during admission showed partial thrombus (yellow arrow) in right pulmonary artery (a) and left pulmonary artery (b); cardiomegaly (c) with large ASD (red arrow); and pulmonary infarction at posterobasal segment of the inferior lobe (green arrow).

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A RARE ENCOUNTER: STAPHYLOCOCCUS AUREUS-INDUCED PERICARDIAL EFFUSION IN A 66-YEAR-OLD MALE

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[1]Rumah Sakit Umum Daerah dr. R. Koesma Tuban

Background:
Pericardial effusion, the accumulation of fluid in the pericardial cavity, can lead to life-threatening cardiac tamponade if untreated. Infectious pericarditis is a known etiology of pericardial effusion, with Staphylococcus aureus being a rare but significant causative agent. This case report highlights the clinical presentation, diagnosis, and management of a 66-year-old male diagnosed with pericardial effusion with a positive culture for Staphylococcus aureus.

Case illustration:
A 66-year-old male presented to the emergency department with symptoms of chest pain, dyspnea, and general malaise. Physical examination revealed muffled heart sounds and jugular venous distension. An echocardiogram confirmed the presence of a significant pericardial effusion. Diagnostic pericardiocentesis was performed, and the pericardial fluid culture grew Staphylococcus aureus. The patient was treated with Clindamycin antibiotic therapy based on sensitivity testing. Serial echocardiograms and taping production observation monitored the resolution of the effusion. The patient's symptoms improved significantly however the production stopped at the 8th day.

Conclusion:
This case showed the importance of considering bacterial infections, including Staphylococcus aureus, as potential causes of pericardial effusion. Early identification and targeted antibiotic are crucial for a good outcome. This case also highlights the necessity to always consider infectious etiologies in patients with pericardial effusion.

Keywords: Pericard effusion, Staphylococcus aureus

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YOUNG MALE WITH OBSTRUCTIVE HYPERTHROPIC CARDIOMYOPATHY AND SYSTOLIC ANTERIOR MOTION : EARLY DETECTION IS CRUCIAL

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Background:
Hypertrophic cardiomyopathy (HCM) is a genetic disorder of the myocardium, characterized by marked myocardial hypertrophy (>15 mm) that cannot be explained by any other diseases that may cause secondary hypertrophy. Its prevalence is 0.2–0.6% with an overall annual mortality rate of 1%.. HOCM is a significant cause of sudden cardiac death in young people, including well-trained athletes, and affects men and women equally across all races. The hypertrophy can occur in any segment of the left ventricle but is most common in the interventricular septum. 2D-echocardiography is the primary imaging modality to make the diagnosis of HCM. The echo has an 80% diagnostic accuracy for HOCM.

Case illustration:
A 30-year-old man arrived at the Zainoel Abidin Hospital's cardiovascular Polyclinic with the chief complaints of being easily fatique that was felt from since 6 month. Initially, the patient felt had no complaints, but in the last month he felt easily fatique when doing activities. The patient was denied of chest pain. Initial ECG was found of LV strain and LVH and the echocardiogram was found Obstructive Hyperthropy Cardiomyopathy, with Preassure Gradient 50 mmHg,LVOT Diameter 13 mm, SAM (+),Moderate of Mitral Regurgitation ,Systolic Function LV normal (LVEF 84%),RV function normal (TAPSE 2.4),LVH Concentric with Grade II Diastolic Dysfunction,Global normokinetic.

Conclusion:
Hypertrophic cardiomyopathy is an important cause of disability and death in patients of all ages, although sudden and unexpected death in young people is perhaps the most devastating component of its natural history. Because of marked heterogeneity in clinical expression, natural history, and prognosis.

Keywords: HCM, SAM, Echocardiography, HOCM, LVOT Pressure Gradient

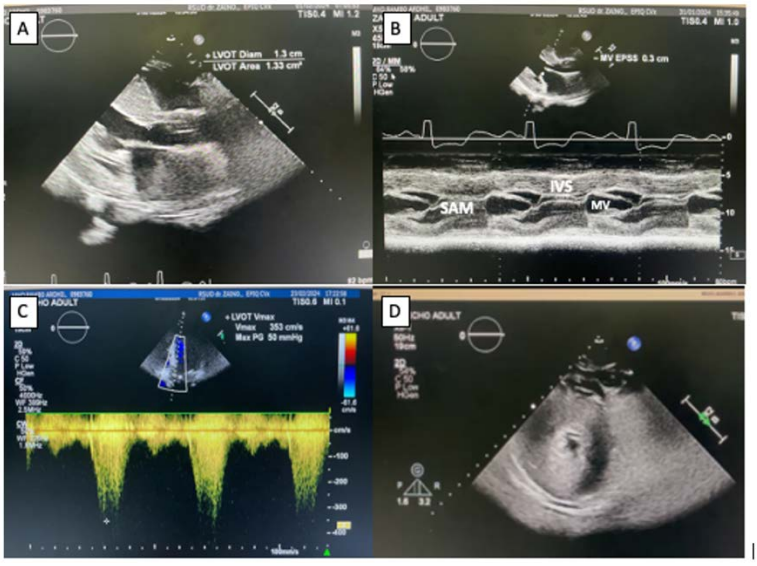

Figure 1. A. Narrowing of the LVOT, B. Intracardiac M-mode in a patient with hypertrophic obstructive cardiomyopathy demonstrating systolic anterior motion (SAM), C. LVOT Pressure Gradient 50 mmHg, D. LV wall thickened (28 mm)

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THE HEART AND MIND IN CRISIS: PERIPARTUM CARDIOMYOPATHY AND ACUTE POSTPARTUM PSYCHOSIS ACCOMPANIED BY MASSIVE THROMBUS, AND HFREF – A COMPLEX CASE REPORT

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Background:
Peripartum cardiomyopathy (PPCM) is a rare, life-threatening condition affecting 1 in 1,000 to 4,000 live births, characterized by heart failure and left ventricular dysfunction late in pregnancy or postpartum. The development of acute psychotic disorders occurring concurrently with PPCM, for which very limited data are available, presents a complex clinical challenge. We report a 27-year-old woman who developed severe heart failure due to PPCM and an acute psychotic disorder postpartum, underscoring the need for a multidisciplinary treatment approach.

Case illustration:
A 27-year-old woman presented to the emergency room with a three-day history of worsening shortness of breath, cough, fever, and weakness, accompanied by transient hemiparesis. Her vital signs included a blood pressure of 127/103 mmHg, heart rate of 132 bpm, respiratory rate of 32 breaths/min, and SpO2 of 74%. The patient reported similar episodes since giving birth a year prior, leading to multiple emergency admissions. Physical examination revealed rales at the lung bases and elevated JVP. During hospitalization, the patient was said to have a history of hysterics and unnatural behavior several months after giving birth, so she was consulted by a psychiatrist and the patient was diagnosed with acute psychotic disorder and given therapy with aripiprazole 5mg, clozapine 25mg, and fluoxetine 10mg and the response to the given therapy showed significant improvement. Echocardiography revealed severe reduce LV function (LVEF 20%) with massive intracardiac thrombus. She had good therapeutic response to heart failure therapy. Neurological consultation and brain CT revealed cerebral atrophy but no acute lesions.

Conclusion:
This case highlights the severe consequences of PPCM, including significant psychological impact. The coexistence of acute psychotic disorders complicates management, underscoring the need for an integrated multidisciplinary approach. This is also compounded by the risk of other co-morbidities such as this patient's emerging which will worsen the prognosis.

Keywords: acute psychotic disorder, thrombus, heart failure, Peripartum cardiomyopathy

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SUCCESFUL TRANSJUGULAR RVOT STENTING PROCEDURE IN REDUCING RV PRESSURE OVERLOAD: A CHALLENGING CASE IN 20 YEARS OLD MAN WITH VSD, SEVERE PS, SEVERE TR AND GIANT RA

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Background:
RV outflow tract obstruction (RVOTO) can lead to elevated RA pressure and RV dysfunction. Severe PS resulting RVOTO concomitant with VSD responsible for the right to the left shunt. A sudden decrease in systemic vascular resistance can lead to cyanotic spells. RVOT stenting is necessary for achieving critical balance between systemic and pulmonary vascular resistance

Case illustration:
A 20-years-old male was admitted with a chief complaint of shortness of breath and fatigue since he was a kid. He often experienced swollen stomach and cough during sleep. His functional class was NYHA III with several histories of rehospitalization despite optimal treatment, he took medication as follows; ramipril 1.25 mg od, hydrochlorothiazide 12.5 mg od, spironolactone 25 mg od, propranolol 40 mg tid, and furosemide 80 mg bid. At admission, vital sign showed 92/50 mmHg in BP with HR 93 bpm, respiratory rate 16 tpm and SpO2 was 76% measured by pulse oximetry in left upper extremity. Physical examination revealed a loud and long ejection-type systolic murmur (grade 4/6) at ULSB transmits to the back. ECG showed a RAD and RVH with QRS rate 90 bpm. At catheterization lab, the preprocedural TEE found RV peak systolic gradient was 90 mmHg, RVOT diameter was 5-6 mm, PV annulus diameter was 18 mm, giant RA, massive TR, a bidirectional shunt of peri membranous VSD (PG trans-VSD 28 mmHg). We decided to perform direct RVOT stenting using 10.0x38 mm dynamic vascular stent, double stenting technique via jugular vein access. A jugular approach will allow the delivery system to enter the RVOT stent more centrally with less friction on the lateral walls of the RVOT. After procedure, we found an increased aortic saturation from 78% to 99% with improving RV peak systolic gradient from 90 to 44 mmHg. In ward, there were improving symptom of shortness of breath and hemodynamic in stable state with saturation 99%.

Conclusion:
RVOT stenting can be choice to be performed in patients with PS for relieving the obstruction to the pulmonary circulation. Certain anatomic conditions may necessitate a trans-jugular approach if crossing RVOT is difficult.

Keywords: pulmonary stenosis, RVOT stenting

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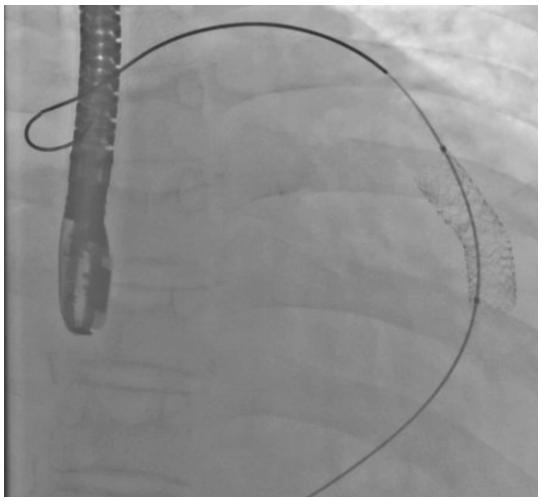

Figure 1. Post RVOT stenting

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SUBACUTE STENT THROMBOSIS AFTER PRIMARY PCI IN STEMI PATIENT WITHOUT CLOPIDOGREL RESISTANCE: A CASE REPORT

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Background:
Stent thrombosis (ST) is a critical complication following PCI. Multiple factors, including patient conditions, lesion characteristics, stent types, and antiplatelet therapy, contribute to early ST risk. This case report explores a subacute ST incident in a STEMI patient following primary PCI, emphasizing the multifaceted risk factors involved.

Case illustration:
A 59-year-old male presented to the emergency department with chest pain and tightness radiating to his left arm for over 20 minutes. He had a history of anterior STEMI three days prior and had undergone primary PCI with a sirolimus-eluting stent (SES) (2.75 mm x 48 mm) in the proximal LAD. Risk factors included chronic hypertension, obesity, and heavy smoking. ECG showed anterior STEMI. Elevated creatinine (1.86 mg/dl), high-sensitivity troponin T (3865 ng/L), and CK-MB (>40 IU/L) levels were noted, along with hypokalemia (3.2 mEq/L). He was diagnosed with acute reinfarction anterior STEMI, KILLIP class I, mild hypokalemia, and acute renal failure. Coronary angiography revealed total occlusion within the stent at the proximal LAD and subtotal stenosis in the RCA. A drug-eluting stent was deployed, restoring TIMI 3 flow. Echocardiography showed reduced EF (38%). Clopidogrel resistance tests indicated a low risk, and the patient tested positive for type 2 DM (HbA1C 6.9%). Several risk factors in this patient contributed to the subacute ST. He presented with STEMI, heart failure, DM, and had a long stent implanted in a bifurcation lesion of the LAD. These factors align with known predictors of early ST. The implantation of a SES, especially in long lesions, is associated with higher ST rates compared to newer-generation stents. Despite no evidence of clopidogrel resistance, the patient's complex clinical profile necessitated close monitoring and aggressive management to prevent further thrombotic events.

Conclusion:
This case underscores the complexity of managing subacute stent thrombosis in STEMI patients, highlighting the interplay of various risk factors. The patient's successful revascularization and stable clinical course post-intervention demonstrate the critical importance of timely and appropriate therapeutic strategies. This report contributes valuable insights into the risk stratification and management of subacute ST, advocating for tailored approaches in high-risk patients to optimize outcomes and prevent recurrence.

Keywords: subacute stent thrombosis, case report, sirolimus-eluting stent, clopidogrel resistance, STEMI

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LATE-ONSET TYPE I LEFT VENTRICULAR RUPTURE FOLLOWING DOUBLE VALVE REPLACEMENT: AN UNEXPECTED CAUSE OF CARDIAC TAMPONADE

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Background:
Cardiac tamponade is a medical emergency caused by any significant accumulation of pericardial fluid (effusion), blood, or pus in the pericardium, compressing the heart chambers and leading to hemodynamic compromise, circulatory shock, and potentially fatal. The incidence of post-operative cardiac tamponade was estimated to be 0.1 – 6%. We present a case of late-onset type I left ventricular (LV) rupture following double valve replacement with a consequence of cardiac tamponade during presentation.

Case illustration:
A 58-year-old woman was referred to our hospital with a history of progressive breathlessness and orthopnea over 1 month. The patient has a history of double valve replacement (aorta and mitral) 3 months before admission. Physical examination revealed a hemodynamic compromise with support of norepinephrine, irregular heart sound, and metallic sound at the 5th intercostal space (ICS) linea midclavicular sinistra and 2nd ICS right parasternal border also muffled heart sound. Transthoracic echocardiography (TTE) from the previous hospital revealed a loculated mass suspected of hematoma intra pericardial which compresses left ventricle structure. Further imaging with cardiac computed tomography (CT) revealed contrast extravasation at the atrioventricular groove near the prosthetic mitral valve which confirmed type I LV rupture. Pericardiocentesis was performed and the total accumulation of fluid which have been removed was approximately 3,000 mL with hemorrhagic characteristics. Later, after careful consideration with the Heart Team, the patient was performed for open thoracotomy and confirmed the rupture, approximately 1,000 mL hematoma was evacuated. The surgeon decided to repair at the rupture site and created pericardial window, and Cardiac CT evaluation revealed a reduced volume of pericardial hematoma and no contrast extravasation from the left ventricle to the pericardial space. Combining multimodality of non-invasive cardiac imaging can characterize the source of post-operative cardiac tamponade.

Conclusion:
Multimodality cardiac imaging using echocardiography, and cardiac CT are often required for complete characterization the source of post-operative cardiac tamponade and confirmed type I LV rupture following double-valve replacement.

Keywords: Cardiac tamponade, echocardiography, pericardial window, cardiac CT, left ventricular rupture

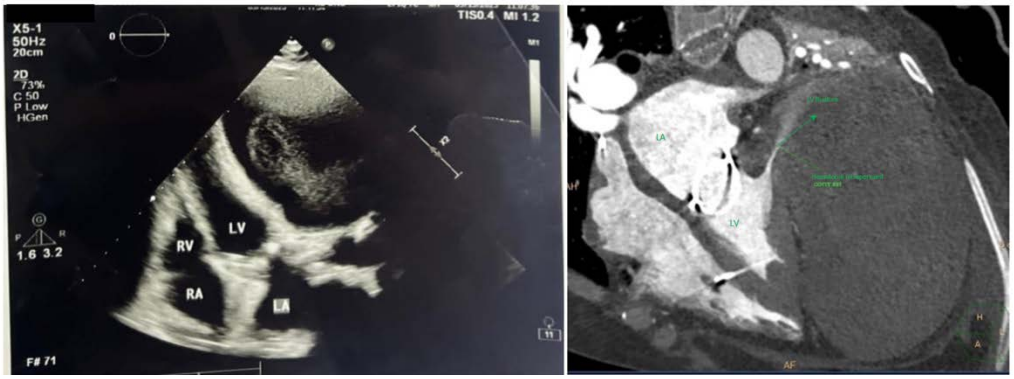

Figure 1. (Left) TTE findings loculated mass suspected of hematoma intra pericardial which compresses left ventricle structure (Right) Contrast extravasation from left ventricle to pericardial. A large hematoma was observed with estimated size as large as the heart (1:1 ratio).

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LEFT VENTRICLE PERFORATION AS A COMPLICATION OF PERICARDIOCENTESIS PROCEDURE IN RECURRENCE PERICARDIAL EFFUION": A CASE REPORT

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Background:
Iatrogenic perforation of the Left Ventricle (LV) is a rare but recognized complication of pericardiocentesis. Treatment strategy for LV perforation in this case report is surgical approach to evacuate the corpus alienum (catheter) and to repair the iatrogenic LV perforation.

Case illustration:
A 57-year-old man who had undergone pericardiocentesis in rural hospital and refer to our hospital caused of LV perforation. Chief complaint was shortness of breath within 4 weeks and followed by cough. The etiology was suspected malignancy because of recurrence pericardial effusion and this was the second pericardiocentesis after a week of discharge from the first pericardiocentesis. Echocardiography was performed and revealed there was a massive circumferential pericardial effusion with early sign of cardiac tamponade. A catheter was found in cardiac intra-chamber of LV and suspected the tip of catheter in ascending aorta. A CT cardiac showed an improper catheter placement with apical LV perforation through chamber of LV and the presence of the catheter tip in the ascending aorta.Urgent sternotomy was performed to evacuate the catheter and to repair the LV. We found a malignant-likely mass in mediastinum space and suspected this was the etiology of recurrence pericardial effusion.The patient's hemodynamic condition improved hours after surgery, and was discharged 7 days later.

Conclusion:
Pericardiocentesis should be performed guided by ultrasonography, and even so, it carries risks of complications. Cardiac injury after pericardiocentesis is a rare but serious complication that must be identified quickly and should be treated by a multidisciplinary team.

Keywords: Pericardian Effusion, LV Perforation, Pericardiocentesis

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MANAGEMENT OF DILATED CARDIOMYOPATHY IN LATE TRIMESTER OF PREGNANCY: A CASE REPORT

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Background:
Dilated Cardiomyopathy (DCM) is a disease of the heart muscle characterized by enlargement and dilation of one or both ventricles along with impaired contractility, defined as left ventricular ejection fraction (LVEF) less than 40%. Pregnancy can exacerbate these conditions, posing serious risks to both mother and fetus.

Case illustration:
A 23-year-old woman in her second pregnancy at 33-34 weeks of gestation came to the emergency room with progressively worsening shortness of breath. Her vital signs showed BP 98/55 mmHg, HR 112 bpm, RR 28 bpm, with SpO2 91% on room air. Physical examination revealed a pansystolic murmur at the apex grade IV/VI, bilateral rales at the lungs, anemia, and pretibial edema. Chest X-ray showed cardiomegaly and bilateral infiltrates and cranialization. She had a history of peripartum cardiomyopathy during her first pregnancy 4 years ago but did not regularly seek a cardiologist post-delivery. Echocardiography examination indicated a dilated left atrium and left ventricle, reduced left ventricular systolic function with ejection fraction (biplane) at 34% and GLS -13.1%, global hypokinesia. There was severe eccentric functional mitral regurgitation and moderate tricuspid regurgitation. The patient was diagnosed with CHF NYHA FC III due to DCM in late trimester of pregnancy. She was treated initially with oxygen therapy, furosemide continuous infusion, and spironolactone. This patient was classified as class III of maternal risk in the modified WHO classification for pregnant women with heart disease. Given this condition, a caesarean section under spinal anesthesia was urgently performed. Patient and the baby was stable post delivery. She was then discharged on furosemide, spironolactone, bisoprolol, ramipril, and digoxin therapy, with detailed instructions on the high risks of future pregnancies and the need for effective contraception such as IUD insertion or irreversible options such as tubal occlusion in the future.

Conclusion:
This case highlights the complexities of managing pregnancy in patients with DCM. Pregnancy can significantly worsen the hemodynamic status in these patients, leading to severe maternal and fetal complications. Multidisciplinary management involving cardiology, obstetrics, and neonatology is crucial for optimizing outcomes. Additionally, counseling on contraception is vital to prevent high-risk pregnancies in women with significant cardiomyopathy.

Keywords: Dilated Cardiomyopathy, Pregnancy

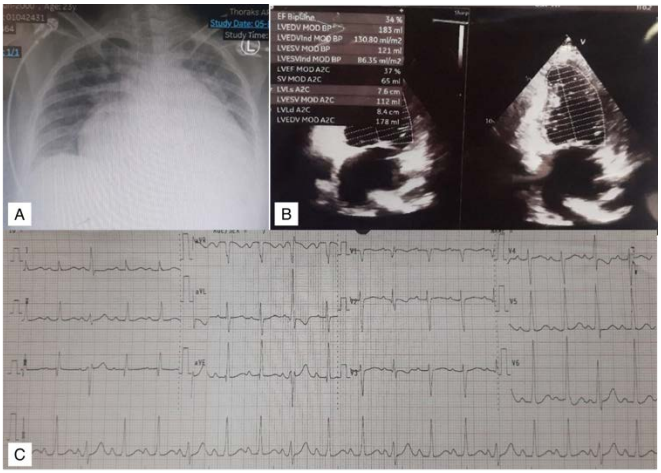

Figure 1. A. Chest X-Ray, B. Echocardiography, C. Electrocardiogram

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USE OF ANTICOAGULANT IN 67 YEAR OLD MALE WITH MECHANICAL AORTIC VALVE : BENEFIT, RISK AND DRUG OPTION

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Background:
Mechanical heart valve in one of definite therapy for patients with valvular heart disease. Patients with mechanical valve are recommended to have anticoagulant therapy to reduce the risk of clotting around the mechanical valve. However anticoagulant therapy isn't without any side effect, as bleeding is one of the most adverse event found in patient with anticoagulant therapy. This case tries to highlight a patient with mechanical valve who has history of bleeding with anticoagulant therapy

Case illustration:
A 68 year-old man presented to the Emergency Department (ED) with shortness of breath that feels worse since 1 day before admission. Patient has a history of valvular heart disease and undergoes valve replacement surgery with mechanical valve 5 years before current admission. Patient had a history of bleeding gum and bleeding sputum when coughing when patient consume direct oral anticoagulant (rivaroxaban). Vital sign in normal limit. A 12 lead ECG showed atrial fibrillation rapid ventricular respond with QRS rate of 117 beats per minute. Laboratory finding in admission shows elevated INR. Echocardiography examination shows mechanical prosthetic aortic valve with good functional movement, no leakage or pannus detected, decreased LV systolic function with EF of 42%. We gave the patient warfarin to help increase patient INR to therapeutic range of 2.5 to 3.0. Every patient with mechanical heart valve must consume anticoagulant to control the risk of thromboembolic complications, while also considering about bleeding effect on the patient. Unlike atrial fibrillation, currently there is only one type of anticoagulant that has been approved by ESC guidelines as therapy for mechanical heart valves, which is Vit K antagonist or heparin. Currently direct oral anticoagulant (DOAC) such as apixaban and rivaroxaban is not recommended in patient with mechanical heart valve since there is no study that manage to prove the superiority of DOAC to heparin in controlling thromboembolic and bleeding event in patients with mechanical heart valves

Conclusion:
Mechanical heart valves requires lifelong anticoagulant therapy. As of now, only Vit K antagonist is recommended to be used to treat patients with mechanical heart valves

Keywords: mechanical heart valves, anticoagulant

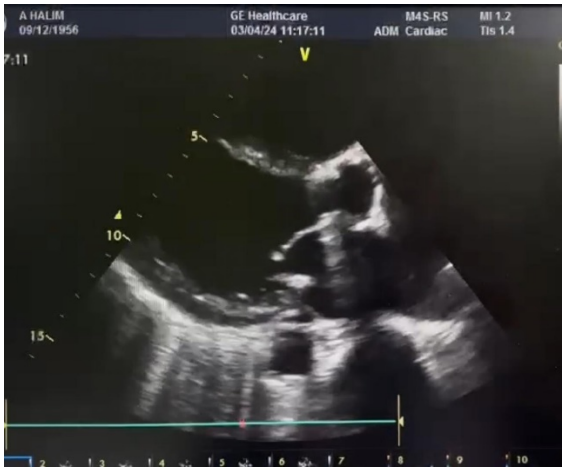

Figure 1. Accostic shadow showing presence of functional mechanical aortic valve in patient

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CHALLENGES IN THE MANAGEMENT OF ACUTE PULMONARY EMBOLISM ON CHRONIC THROMBOEMBOLIC PULMONARY HYPERTENSION IN A RESOURCE-LIMITED SETTING: A CASE REPORT

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[1]RSUD Berkah Pandeglang

Background:
Acute pulmonary embolism (PE) is a potentially life-threatening cardiovascular disorder. Patients presenting acutely to the emergency room (ER) with signs of PE may already have unresolved prior clots leading to chronic thromboembolic pulmonary hypertension (CTEPH), decompensated by the new PE episode resulting in fatal right ventricular (RV) failure. Identifying this condition is a formidable clinical challenge in resource-limited contexts due to constrained access to computed tomographic pulmonary angiography (CTPA) as the imaging method of choice.

Case illustration:
A 39-year-old woman with a history of contraceptive implant use referred to our ER due to worsening dyspnea for 7 hours before admission. The patient had previous exertional dyspnea and orthopnea for months but had no known history of prior PE or deep venous thrombosis. Physical examination showed tachycardia (103 beats per minute), tachypnea (28 breaths per minute), desaturation (SpO2 64-74% with NRM at 15 lpm) with clear lungs and holosystolic murmur best heard at lower left sternal border, and elevated jugular venous pressure. No signs of hemodynamic instability were found. The ECG showed the S1Q3T3 pattern. A simple bedside echocardiography was performed by the ER doctor in charge, revealing RV enlargement with D-shape, indicating RV dysfunction. We initiated anticoagulation treatment with IV UFH without delay, while awaiting the result of D-dimer testing. The result confirmed an increased D-dimer level, that was 2990 ng/ml. During intensive observation in ICU, the patient's oxygen saturation level remained dropping despite heparinization, raising suspicion of acute PE on CTEPH. She was planned to be referred for further evaluation. CTEPH should be considered in all patients with sudden onset of PE, because the treatment for acute PE and CTEPH differs significantly: while anticoagulation therapy and in some cases, fibrinolysis are effective for acute PE, however in CTEPH, urgent pulmonary thromboendarterectomy (PTE) may be needed to relieve RV pressure if medical therapy fails.

Conclusion:
Identification of acute PE on CTEPH, while challenging, is an important consideration as it will determine the patients' acute and long-term management. In resource-limited settings, we encourage ER doctors to be able to perform a simple bedside echocardiography for early detection of RV dysfunction.

Keywords: bedside echocardiography, right ventricular dysfunction, Pulmonary embolism, chronic thromboembolic pulmonary hypertension

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MODIFIED VALSALVA MANEUVER AS ACUTE MANAGEMENT UNSTABLE HEMODYNAMIC SUPRAVENTRICULAR TACHYCARDIA (SVT) IN RURAL AREAS WITH LIMITED FACILITIES: A CASE REPORT

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[1]RSUD H. Sahudin

Background:
Unstable hemodynamic supraventricular tachycardia (SVT) is a condition of tachycardia that could lead to death if not treated immediately. Acute management of SVT, especially synchronized electrical cardioversion has proven effective in terminating SVT with unstable hemodynamics. Unfortunately, not all areas have complete equipment and medication for acute management therapy. This case report describes how one of the noninvasive methods can be used in SVT patients with unstable hemodynamics in rural areas with limited facilities.

Case illustration:
A 54-year-old man came to H. Sahudin Hospital in Kutacane Southeast Aceh complaining of palpitations that began 2 hours ago. The vital signs showed blood pressure (BP): 89/51mmHg, pulse: 219 beats per minute (bpm), saturation: 79% room air, and respiratory rate: 26 times per minute (tpm). The ECG showed regular narrow complex tachycardia with no visible p waves that suggest Typical AV nodal re-entry (AVNRT). The patient's diagnosis was confirmed as SVT with unstable hemodynamics. In this situation, the patient should preferably receive acute management with synchronized electrical cardioversion as the first choice of therapy. Unfortunately, the equipment is damaged followed by inadequate pharmacotherapy. We decided to perform a modified Valsalva maneuver with the result returned to sinus rhythm. After the procedure, vital signs become 100/60 mmHg for BP, pulse: 80 bpm, saturation: 94% room air, and respiratory rate: 22 tpm. The ECG showed normal sinus rhythm with inferior old myocardial infarction (OMI). The laboratory showed normal electrolytes. After 48 hours of monitoring with consistently normal sinus rhythm, the patient was discharged and prescribed bisoprolol 1.25 mg, aspirin 80 mg, and simvastatin 40 mg once daily. We inform and advise patients to undergo catheter ablation if recurrence occurs frequently.

Conclusion:
Acute management of unstable hemodynamics SVT was challenging in rural areas with limited facilities. However, we managed to carry out acute management according to symptoms with existing facilities to improve the patient's condition. In this case, the modified Valsalva maneuver could convert unstable hemodynamics SVT to sinus rhythm.

Keywords: AVNRT, Supraventricular tachycardia, Modified Valsalva maneuver

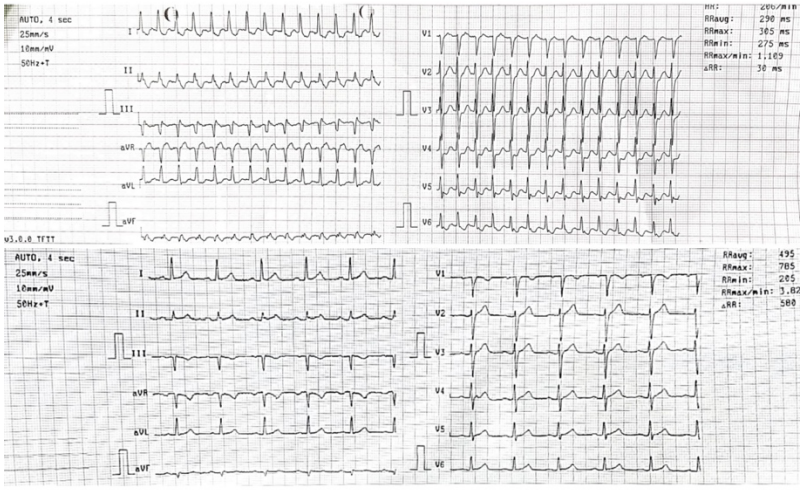

Figure.1 ECG when SVT convert to sinus rhythm

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ACUTE ST ELEVATION MYOCARDIAL INFARCTION IN PATIENT WITH SEVERE MITRAL STENOSIS AND LONG STANDING ATRIAL FIBRILLATION: A RARE CASE OF LETHAL COMBINATION

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Background:
Rheumatic heart disease is widely know as the most common cause of mitral stenosis. In almost half of the cases, atrial fibrillation occurs during the evolution of mitral stenosis, thereby exposing to an increased risk of cardio-thrombo-embolic events. Whereas the most frequent site for embolism is the cerebral circulation, any organ may be involved, including to the coronary circulation causing acute myocardial infarction (MI), a rarely found complication of mitral stenosis.

Case illustration:
A 74-year-old female patient, with history of severe mitral stenosis and long standing atrial fibrillation, referred to emergency room of Mohammad Hoesin General Hospital with acute extensive anterior ST elevation myocardial infarction (STEMI). Her previous echocardiogram showed severe mitral stenosis due to rheumatic heart disease (RHD) with giant left atrium, pulmonary hypertension and left atrial spontaneous echo contrast. The patient got loading of dual anti-platelet (aspirin and clopidogrel) and subcutaneous 40 mg IV of enoxaparin before referred to Mohammad Hoesin general hospital. Immediate coronary angiogram assessment revealed thromboembolic material in the distal of left anterior descending artery without significant coronary artery stenosis there. We did not proceed the coronary stenting and preferred to continue the heparinization in intensive care unit. Unfortunately, the next day, the patient experienced severe gastrointestinal bleeding and did not survive after 6 days of total in-hospital care.

Conclusion:
Atrial fibrillation with valvular heart disease represent the most common cause of cardio-embolic event. The cause of acute MI event in this case was most likely due to cardio-thrombo-embolism suspected originating from left atrial thrombus which triggered by giant left atrium and AF. Long term outcomes of STEMI patients related to cardio-embolism are worst compared to STEMI patients non related to cardio-embolism.

Keywords: ST Elevation Myocardial Infarction, Mitral Stenosis, Atrial Fibrillation, Rheumatic Heart Disease

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DIVERSE CHALLENGES IN MANAGING THE UNPREDICTABLE CLINICAL COURSE OF PERIPARTUM CARDIOMYOPATHY: A CASE SERIES

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Background:
Peripartum cardiomyopathy (PPCM) is a rare but serious condition resulting in high morbidity and mortality. Initial presentation ranges from mild to severe, often resembling normal pregnancy changes, which complicates early detection. It can potentially result in complications, rapid progression to end-stage heart failure, or spontaneous and complete recovery of ventricular function.

Case illustration:
Case 1 : A 30-year-old woman with a history of asthma, multiparous with twin pregnancy, and gestational hypertension presented with dyspnea and cough four weeks after delivery. Initially diagnosed with right pleural effusion and suspected tuberculosis, she was directed to outpatient follow-up at pulmonology clinic. Twelve days later, she was referred to a cardiologist due to cardiomegaly. Echocardiography revealed PPCM (EF 25.01%). Treatment included ramipril, furosemide, spironolactone, aspirin, nitroglycerin, and eventually bisoprolol. One year later, echocardiography showed recovered LV function (EF 65.57%) and medications were gradually discontinued. Subsequent high-risk pregnancy was terminated, followed intrauterine device as contraception.Case 2 : A 24-year-old primiparous woman presented with shortness of breath and fever three days postpartum. Echocardiography showed LV dilatation, EF of 16.94%, and LV thrombus. Diagnosis of PPCM, AKI, and pneumonia was established. She was hospitalized for six days with treatment involving ramipril, bisoprolol, spironolactone, furosemide, digoxin, isosorbide dinitrate, anticoagulant, and antibiotic. Eleven days after discharge, she was readmitted for five days of hospitalization due to acute lung oedema. A follow-up echocardiography six months later showed improved cardiac function (EF 64%) and resolution of LV thrombus.Both cases involved previously healthy young postpartum women with different clinical presentations of PPCM. Case 1 initially presented with mild symptoms (NYHA Class II), was initially diagnosed as a pulmonary case, was never hospitalized, and later faced subsequent pregnancy. Case 2 presented with more severe symptoms (NYHA Class IV), complicated with LV thrombus and pneumonia, requiring intensive care and hospital readmission.

Conclusion:
Key components in addressing various challenges toward successful recovery of PPCM patients include increasing awareness of PPCM among non-cardiologists, early echocardiography, appropriate medication, sufficient patient education, and counseling for subsequent pregnancies. Interdisciplinary collaboration is essential from disease detection to long-term patient management.

Keywords: peripartum cardiomyopathy, interdisciplinary collaboration, management

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PERSISTENT PULMONARY HYPERTENSION ON NEWBORN IN DISTRICT HOSPITAL: A CASE REPORT

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Background:
Persistent pulmonary hypertension in newborns is a potentially life-threatening condition in the early neonatal phase which presents with significant hypoxemia and respiratory failure in the first 12 hours of life. This condition must be recognized early and treated aggressively. In this report, we present a premature newborn with a diagnosis of Persistent Pulmonary Hypertension of Newborn (PPHN).

Case illustration:
A newborn delivered from a cesarian section at 35 weeks of pregnancy (adjusted Ballard Score), large for gestational age status (birth weight 3110 grams), and a mother with a history of gestational hypertension. The patient appeared cyanotic after birth with respiratory distress (Downes score of 3), however, the patient seems to have no improvement from continuous positive airway pressure (CPAP) nor noninvasive positive pressure ventilation (NIPPV). The Thorax X-ray showed suggestive hyaline membrane disorder grade I. The echocardiography showed dilation of the right atrioventricular, IAS gap diameter 0.23 cm, R-L shunt, with severe tricuspid regurgitation (TVG 98,69 mmHg) and moderate pulmonic regurgitation (PG 31,91 mmHg), calculation of mean pulmonary arterial pressure (mPAP) using tricuspid regurgitation peak velocity (VmaxTR) approach was approximated about 65,32 mmHg. No PDA flow and pericardial effusion were found. This patient received non-invasive positive pressure ventilation (NIPPV) for 10 days, a phosphodiesterase-5 (PDE-5) inhibitor, an inotropic agent, and a broad-spectrum antibiotic. This patient's condition was improved and echocardiography evaluation on day 7 showed improved RV contractility with no TR found. The patient was then discharged after 12 days of hospitalization. Echocardiography plays a critical role in defining cardiac function and congenital abnormalities. It also serves as a non-invasive assessment of pulmonary hypertension, differentiating whether it is caused by maladaptation of pulmonary vasculature or cardiac abnormality. Timely diagnosis and prompt therapy are critical to prevent worsening conditions and mortality of this patient.

Conclusion:
In conclusion, we describe a newborn with persistent pulmonary hypertension. This case report emphasizes the recognition, diagnostic tools, and management of PPHN.

Keywords: Persistent Pulmonary Hypertension of Newborn, Pulmonary Arterial Hypertension

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CARDIAC CONUNDRUM: TREATING RECURRENT SUPRAVENTRICULAR TACHYCARDIA IN THE SHADOW OF PROGRESSIVE CORONARY ARTERY DISEASE

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Background:
Coronary artery disease (CAD) often presents with chest pain and palpitations. Arrhythmias in patients are frequently attributed to CAD. When supraventricular tachycardia (SVT) is detected on an ECG, it is often misdiagnosed as a CAD-related issue, leading to overdiagnosis in patients with pre-existing SVT disorders. Therefore, an in-depth understanding of pathophysiology, diagnosis, and treatment is essential. This case report explores the challenges of diagnosing and managing SVT in a patient with concomitant CAD.

Case illustration:
A 63-year-old male with a history of palpitations, hypertension, and smoking experienced seven cardiac events in the past 14 years and was treated with stent implantations on three occasions. Despite complete revascularization, the patient continued to experience palpitations. Further evaluation, including multiple diagnostic coronary angiographies (DCA), revealed no additional issues. However, the patient's electrocardiography (ECG) showed atrioventricular nodal re-entrant tachycardia (AVNRT), a rare arrhythmia in CAD patients. Various treatments, including carotid sinus massage, electric shocks, and medication, were attempted to manage the SVT, which temporarily alleviated the symptoms. Ultimately, the patient underwent successful catheter ablation, which terminated the AVNRT without recurrence.

Conclusion:
This case emphasizes the importance of recognizing atypical presentations of arrhythmias in patients with CAD and the need for a holistic approach to patient care. By remaining vigilant and employing advanced diagnostic and treatment techniques, clinicians can significantly improve patient outcomes, particularly in those with complex cardiovascular histories.

Keywords: arrhythmia, supraventricular tachycardia, coronary artery disease, cardiac ablation, atrioventricular nodal re-entrant tachycardia

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DE NOVO HEART FAILURE IN YOUNG ADULT: AN EASILY MISSED DIAGNOSIS

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Background:
Although rare, heart failure (HF) can occur in young adults due to various causes, such as congenital heart disease, ischemic heart disease, herediter or acquired cardiomyopathy, arrhythmia, valve disease, inflammation, infection, etc. The biggest challenge in diagnosing HF in these patients is identifying its signs and symptoms. Young adults with HF represent a unique characteristic that differs from the older patients. Prompt recognition and treatment of HF in young adults are crucial.

Case illustration:
A 35-year-old male was admitted to the emergency department with dyspnea, cough, and swelling of both legs. These conditions had been worsening for a month after he was diagnosed with GERD in public health center because of nausea and epigastric pain. No chest pain was reported. He has history of hypertension without medication since 3 years ago. No history of cardiac disease, diabetes, smoking, or infection. His father suffered sudden cardiac death and hypertension. His sister has hypertension too. Physical examination showed obesity (BMI 31,7 kg/m^2), hypertension (150/110 mmHg), tachycardia (125 bpm, regular heart rate), tachypnea (24 x/min), normal temperature, and crackles in both lungs. Chest X-ray showed pulmonary edema and cardiomegaly. TTE examinations revealed eccentric LVH, severe global hypokinesis with LVEF 14%, moderate MR due to annulus dilatation, mild TR and PR. Laboratory evaluation presented high blood urea (2 mg/dL), increase FT4 (35.57 pmol/L) and normal TSH (0.42 uUI/ml). Other laboratory findings were normal. He was treated with diuretics (furosemide and spironolactone), b-blocker (bisoprolol), ACE inhibitor (ramipril), ARB (candesartan), and simvastatin. Thiamazole was also used to treat the hyperthyroidism.

Conclusion:
Heart failure among young adults is becoming more common, but the underlying causes are not completely understood. Sometimes it is forgotten because the symptoms are similar to other conditions. This patient had HF classified as NYHA class III, along with hypertension and hyperthyroidism, but we cannot yet rule out coronary artery disease due to a lack of facilities. Further investigations with CT or MRI need to be carried out to determine the exact cause. Therapy needs to be given immediately to avoid fatal events.

Keywords: hypertension, young adult, heart failure, hyperthyroidism

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ATRIAL FIBRILATION IN HYPERTROPHIC CARDIOMYOPATHY WITH LEFT VENTRICULAR OUTFLOW TRACT OBSTRUCTION (LVOTO) AND WOLF PARKINSON WHITE (WPW) SYNDROME PATIENT

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Background:
Hypertrophic Cardiomyopathy (HCM) is a common genetic heart disease inherited in an autosomal dominant pattern. HCM prevalence ranges from 0.2% - 0.5% of the total population worldwide. Left Ventricular Outflow Obstruction (LVOTO), is present in about 75% of patients with HCM. HCM patients will experience adverse events, including sudden death events or progressive symptoms because of LVOTO or diastolic dysfunction. HCM is commonly and associated with an increased incidence of WPW syndrome and 10–40% of AF. Combination of HCM and AF is associated with a markedly increased risk of stroke, overall mortality, and heart failure.

Case illustration:
A 41 year old male came to the emergency room with a chief complaint of left chest pain and irregular wide QRS tachycardia with unstable hemodynamic. Echocardiography showed Hypertrophic Cardiomyopathy with Left Ventricular Outflow Obstruction (LVOTO). Cardioversion was performed to stop the tachycardia. The patient was treated for two days in the ICU with stable hemodynamic condition and no complaints. The patient was then transferred to regular room and then sent home after the fourth day of treatment. The patient was refused to be reffered for further management.

Conclusion:
We presented a case of 41 year old male with atrial fibrillation in HCM with LVOTO and WPW syndrome. Because high risk of sudden cardiac death, the diagnosis of AF WPW with HCM must be carried out appropriately and managed in a health facility with adequate resources.

Keywords: Left Ventricular Outflow Obstruction, Atrial Fibrillation, Hypertrophic Cardiomyopathy, WPW Syndrome

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OPTIMIZING NON-SURGICAL TREATMENT OF LUTEMBACHER'S SYNDROME IN RESOURCE-LIMITED SETTING: A CASE REPORT FROM INDONESIA

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Background:
Lutembacher's syndrome (LS) is a rare condition characterized by the coexistence of an atrial septal defect (ASD) and mitral stenosis (MS), which may be congenital or acquired, presenting at any age. The exact prevalence of LS remains uncertain, though it is thought to be higher in regions with a high incidence of rheumatic heart disease (RHD). Traditionally, the gold-standard treatment for LS has been open-heart surgery. However, advances in percutaneous interventional techniques have made percutaneous transcatheter therapy the preferred treatment approach, particularly balloon mitral valvuloplasty (BMV) for MS followed by device closure for ASD. Despite these advancements, many remote areas in Indonesia lack the necessary facilities and cardiac surgeons to manage LS cases effectively.

Case illustration:
A previously asymptomatic 41-year-old male from West Nusa Tenggara, Indonesia, presented with a one-year history of fatigue, cough, and progressively worsening dyspnea, severely limiting his physical activity. Upon admission, he was classified as Stage C Heart Failure and NYHA Class III-IV. Echocardiography confirmed Lutembacher's syndrome, revealing moderate to severe mitral stenosis, a 4-cm interatrial septal defect with a predominant left-to-right shunt, biatrial and right ventricular enlargement, severe tricuspid and pulmonary regurgitation indicative of probable pulmonary hypertension, and mild aortic stenosis and regurgitation. His medical history was notable only for recurrent childhood tonsillitis, likely related to rheumatic heart disease leading to MS. Initial medical management included an ACE inhibitor, beta-blocker, loop diuretic, and aldosterone receptor antagonist, resulting in significant symptomatic relief within two weeks. The subsequent addition of a phosphodiesterase inhibitor further improved his condition, reclassifying him to NYHA Class I-II with minimal symptoms.

Conclusion:
This case highlights the essential role of medical therapy in managing LS, particularly in remote and resource-limited settings where surgical options are unavailable. It emphasizes the need for guidelines to manage LS medically in peripheral hospitals while patients await referral to tertiary centers. This approach is critical for early diagnosis and management of LS in regions with high RHD prevalence, alleviating severe symptoms and improving patient outcomes in the absence of immediate surgical intervention.

Keywords: Heart Failure, Medical Therapy, Mitral Stenosis, Lutembacher's syndrome, Atrial Septal Defect

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ACUTE CARDIO-CEREBRAL INFARCTION SYNDROME TYPE 1: EXPLORING THE INVISIBLE DILEMMA BETWEEN THE HEART AND THE CEREBRAL

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Background:
Cardio-cerebral infarction (CCI) syndrome type 1 is an extremely uncommon disorder characterized by the simultaneous occurrence of acute myocardial infarction (AMI) and acute ischemic stroke (AIS). The optimal method for diagnosing and treating AMI in patients with AIS remains uncertain. This publication aims to delineate the obstacles associated with the treatment of CCI syndrome type 1.

Case illustration:
A 69-year-old man with the risk factors of hypertension, active smoking, and history of cerebral infarction presented with a new onset of typical angina at rest and left hemiparesis. The patient's electrocardiography revealed sinus rhythm and ST elevation with pathological Q waves in the inferoposterior and RV region. The cardiac enzyme level increased while hs-troponin I was 23.091 ng/L and CKMB was 387 U/L. The brain CT scan demonstrated acute infarcts at lentiform nucleus dextra and corona radiata dextra. Patient was decided to get loaded with dual anti-platelets and anticoagulant and performed Primary PCI after bleeding risk assessment. Coronary angiography showed CAD three-vessel disease and left main coronary artery disease with acute total occlusion at mid-RCA with thrombus appearance. The patient underwent coronary stenting in the proximal-mid RCA using DES Cre8 3.0 x 46 mm. Cineangiography evaluation showed TIMI Flow 3 and no residual stenosis. Patient also was joined care with Neurology department to treat AIS simultaneously. There were no bleeding signs during admission. Patient improved and was discharged on the 7th day of hospitalization.

Conclusion:
Effectively managing CCI syndrome type 1 poses a significant challenge for cardiologists and neurologists, respectively. PCI strategy for AMI as the first strategy due to high mortality remains the preferred choice despite the risk of hemorrhagic transformation in AIS.

Keywords: acute ischemic stroke, acute myocardial infarction, cardio-cerebral infarction syndrome

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FALSE ALARMS AND HIDDEN PERILS: THE UNEXPECTED TWIST OF FALSE NEGATIVE TREADMILL TEST IN A 65-YEAR-OLD WITH ACUTE CORONARY ACUTE SYNDROME

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Background:
Treadmill stress testing involves cardiovascular assessment through exercise combined with electrocardiography (ECG) and blood pressure monitoring. This type of ECG stress test frequently acts as an early disease detection method that can lead to early treatments and help prevent or lessen future health issues. However, like all medical tests, the treadmill stress ECG test has limitations in accurately detecting coronary artery disease (CAD). In asymptomatic individuals with few or no risk factors, there is a high probability of false negatives, which can cause misleading diagnoses that lead to inadequate prompt treatment.

Case illustration:
A 65-year-old man presented with typical symptoms of angina infarction that occurred 4 hours before entering the emergency room and was diagnosed with an anterior ST elevation myocardial infarction. He reported experiencing shortness of breath during moderate exertion, without any symptoms occurring at rest. Surprisingly, two weeks earlier, the patient had undergone a treadmill ECG stress test at another hospital with a negative ischemic response result and was categorized as low-risk for cardiac events based on the Duke score, which estimates an annual mortality rate of less than 1%. Despite this, our patient had a 44% probability of obstructive CAD and would have been advised to undergo non-invasive imaging at that time. In this STEMI setting, the patient was treated with fibrinolytic therapy and underwent DES stent implantation in the LAD artery.

Conclusion:
This case exemplifies that when dealing with patients with suspected CAD, the exercise test should be used simultaneously with other methods to determine the likelihood of subsequent cardiac events. Negative treadmill test results should not be used to rule out significant CAD. In this case, the patient should not rely solely on the treadmill test results. Given the PTP (pre-test probability) of obstructive CAD is 44%, a non-invasive imaging test, such as CT coronary angiography, should also be performed. Additionally, medication should not be discontinued based solely on the negative TMT result.

Keywords: Coronary Artery Disease, Treadmill Test, STEMI

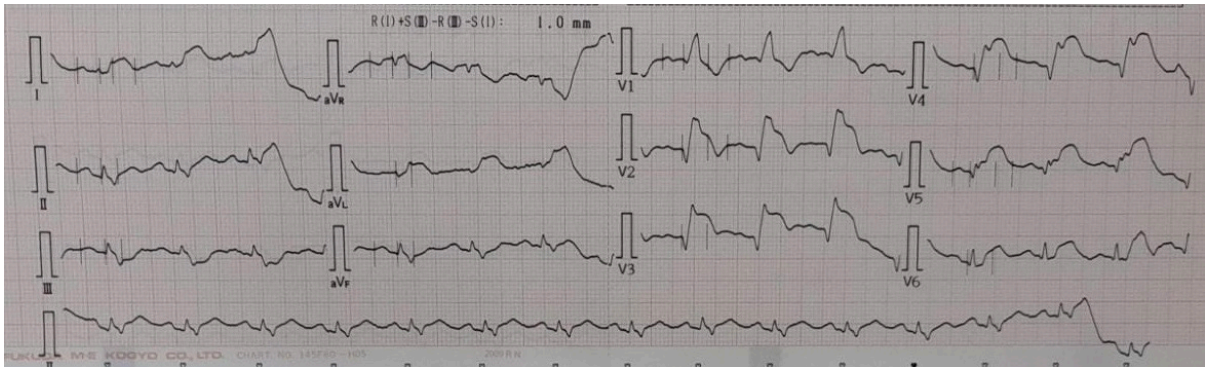

Figure 1. ECG Demonstrating Acute Anterior ST-Segment Elevation Myocardial Infarction (STEMI) in a 65-Year-Old Patient with Acute Coronary Syndrome Following a False Negative Treadmill Test

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BEWARE OF PERSISTENT HICCUPS IN HIGH CARDIOVASCULAR RISKS PATIENT!

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Background:
This case report aims to spread awareness amongst the emergency department physicians and primary care physicians who often encounter patients with symptoms which seem trivial but have a grave underlying pathology. Hiccups, although a seemingly trivial symptom, has been rarely associated with myocardial ischemia. During these rare instances, hiccup has been a manifestation of both non-ST-segment elevation myocardial infarction (NSTEMI) and ST-segment elevation myocardial infarction often requiring percutaneous coronary intervention. Persistent hiccups in the elderly with risk factors for coronary artery disease should be investigated further.

Case illustration:
A 69-year-old male patient with a past medical history of type 2 diabetes mellitus, ischemic stroke and actively smoking come with complaint of persistent hiccups for 2 days and no other complaints. On admission, the patient was afebrile with a blood pressure of 100/57 mmHg and a heart rate of 75 beats per minute. Laboratory tests which included a routine blood count, electrolyte and renal function were significant for haemoglobin count 9.4 gr/dL, creatinine of 1.93 mg/dL and slightly decreased natrium electrolyte serum 132 mmol/L. Chest X-ray show normal size of heart but with a calcification of aortic knob.A routine 12-lead ECG was performed which showed ST elevation in V2 – V4 leads with T inverted in V2 – V6 with sinus rhythm and normal axis, with cardiac troponin-T levels at 55 ng/dl with cut off normal value < 30 ng/dl suggestive of a recent indeterminate anterior wall ST elevation myocardial infarction. A serial 12-lead ECG was performed a hour later showed a dynamic ST segment and T wave changes with a deeper T inverted in V2 – V6. He was given fondaparinux subcutaneous, aspirin and clopidogrel. He was administered to intensive cardiovascular care unit for intensively monitoring before the patient being referred to PCI-center hospital for coronary angiography and coronary intervention.

Conclusion:
Whilst there are more common cause of acute onset of persistent hiccups, if there are no other obvious causes, acute myocardial ischemia / infarction should be considered as a potential differential especially in elderly with high cardiovascular risks.

Keywords: myocardial ischemia, persistent hiccups, myocardial infarction

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SUPRAVENTRICULAR TACHYCARDIA IN ADULT WITH ATRIAL SEPTAL DEFECT BEFORE AND AFTER SURGICAL CLOSURE: A CASE REPORT

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[1]RSUD Panglima Sebaya

Background:
Supraventricular tachycardia (SVT) is a common complication in adult patients with congenital heart disease, including atrial septal defect (ASD). The incidence of arrhythmias can be reduced after ASD closure. However, the procedure may cause myocardial scarring and trigger macro-reentrant atrial tachycardias.

Case illustration:
A 27 years old male patient presented to the emergency room with complaints of palpitations for one day before presentation. Based on the patient's medical history, a year ago, before his cardiac valve surgery, the patient was admitted with previous complaint of fainting and was performed synchronized cardioversion as the electrocardiogram (ECG) showed SVT with unstable hemodynamic. Echocardiography examination results showed that the patient had a large ASD heart defect with a left-to-right shunt and then scheduled for heart valve surgery. Upon the present arrival, ECG examination showed supraventricular rhythm of 160x/minute and wide QRS with rsR pattern V1-V5. This condition could be triggered by additional intraatrial macro-reentrant circuit around the atriotomy site and the edge of the ASD patch resulting intraartrial macro-reentrant tachycardia. The patient was improved after receiving arrhythmia therapy, and he was referred for catheter ablation.

Conclusion:
In conclusion, supraventricular tachycardia is a complication that can occur in adult patients with ASD both before and after undergoing ASD closure surgery. Macro-reentrant atrial tachycardias are a common complication in patients with a previous history of ASD closure surgery. A proper diagnosis and appropriate therapy can reduce mortality.

Keywords: atrial septal defect, supraventricular tachycardia, surgical closure

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THE "DANCING AORTA TECHNIQUE" FOR ABDOMINAL AORTIC ANEURYSM FUSIFORM INFRA RENAL WITH TORTUOSITY: A CASE REPORT

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Background:
An abdominal aortic aneurysm (AAA) is characterized by abnormal aorta enlargement in the abdomen. It develops slowly and asymptomatically but can become life-threatening. Endovascular Aneurysm Repair (EVAR) using a Stent Graft (SG) is a major treatment modality. In large and tortuous infra-renal AAA cases, the Crossed Limb (CL) graft technique, also known as the "Ballerina Technique," has been employed as an alternative therapy.

Case illustration:
A 59-year-old man presented with abdominal discomfort over the past year. He has a history of smoking and hypertension. The patient appeared mildly unwell with a BP of 122/67 mmHg, a HR of 75 bpm, and a Body Mass Index (BMI) of 19.5 kg/m2. A physical examination revealed an end-diastolic murmur in the second right intercostal space. A Computed Tomography (CT) scan showed a dilated and irregularly abdominal aorta with a double-layered wall at the distal to the bifurcation and the proximal end of both common iliac arteries. EVAR was performed using a body SG 22 x 50 mm and a limb SG 18 x 120 mm, extending from the abdominal aorta to both renal arteries. The "Ballerina technique" was employed using a giant S-AT balloon catheter 10x46 mm on both sides. The patient was discharged the following day in good condition.

Conclusion:
The CL graft technique is a valuable addition for addressing complex infra-renal AAA, allowing guidewire passage from one side to the opposite gate and positioning the limb graft like "a ballerina.". This approach aims to potentially reduce complications such as graft disconnection and endoleaks.

Keywords: Ballerina Technique, EVAR, CL graft Technique, AAA

Figure 1. Post EVAR

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PULMONARY VEIN ISOLATION IN YOUNG MALE WITH PERSISTENT ATRIAL FIBRILLATION: ROLE OF ABLATION TO REVERSE LV DYSFUNCTION

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Background:
Atrial fibrillation (AF) is a type of cardiac arrythmia commonly found worldwide, and its incidence kept rising because of extended longevity in general population. Catheter ablation of AF is advised in symptomatic persistent AF patient that is resistant or intolerant to previous treatment, and AF and left ventricular dysfunction suspected to be related to arrythmia mediated cardiomyopathy.

Case illustration:
A 48 years old male presented with palpitation and shortness of breath in the last three weeks. Any cardiac risk factor was denied. On physical examination, blood pressure was 125/75 mmHg, heart rate 150bpm, respiratory rate 21 times/min. Electrocardiogram revealed AF rapid ventricular response and chest x-ray with cardiomegaly and aortic dilatation. Transthoracic echocardiography findings with left ventricle ejection fraction was 42% and global hypokinetic. Transesophageal echocardiography showed no visualized thrombus in left atrium and left atrial appendage. CT coronary angiogram was done and an accessory right middle pulmonary vein was found. Patient then undergone pulmonary vein isolation. Three-dimensional mapping was done around all pulmonary veins and left atrium, followed by ablation of both pulmonary vein antrums with impedance drop above 20 ohms as the target parameters. After 20 minutes of waiting period, electrical isolation was confirmed with findings of entrance block and exit block. Patient then discharged with normal sinus rhythm and continuing heart failure medications, antiarrythmic drug, and anticoagulant. After 1 month evaluation, patient remained asymptomatic and sinus rhythm. From echocardiography evaluation, marked improvement of ejection fraction was observed, improving to LVEF 62%.

Conclusion:
Pulmonary vein isolation is an excellent approach to achieve reversal of LV dysfunction related to AF. Outcomes after procedure needs to be evaluated after blanking period for 8 weeks. The procedure itself preceded by many preparations, in order to gain optimal results after ablation.

Keywords: Pulmonary vein isolation, atrial fibrillation, arrythmia mediated cardiomyopathy

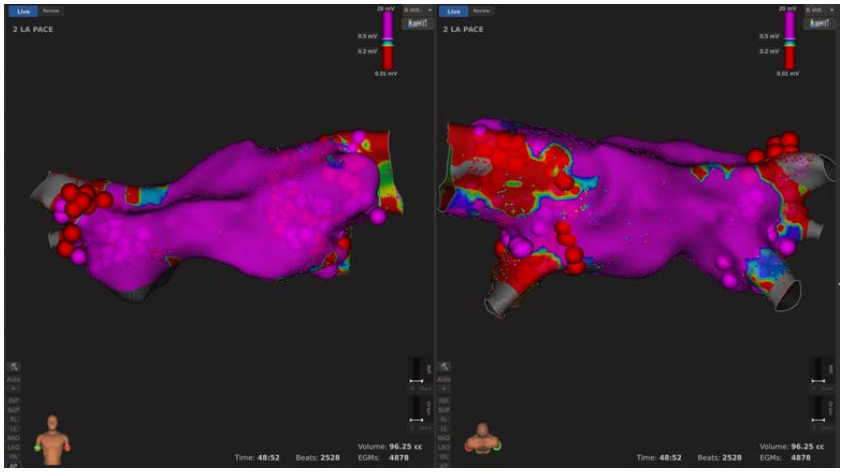

Figure 1. Ablation procedure of both pulmonary vein antrums in patient with persistent atrial fibrillation with heart failure symptoms.

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RARE AND CRITICAL DE WINTER SIGN MORPHOLOGY: A PRECEDING INDICATOR OF STEMI EQUIVALENT

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Background:
The De Winter sign is an infrequently observed ECG pattern typically recorded about 1.5 hours after symptoms begin. Previous research indicates that this pattern includes junctional ST-segment depression (STD) followed by prominent, symmetrical T waves in the precordial leads. This constellation of findings suggests an equivalent of ST Elevation Myocardial Infarction (STEMI) in around 2% of patients experiencing acute anterior myocardial infarction.

Case illustration:
A 60-year-old man presented to the emergency room with chest pain and diaphoresis lasting 3 hours. His medical history included hypertension, atrial fibrillation, and chronic heart failure managed with warfarin, spironolactone, bisoprolol, candesartan, and furosemide. Physical examination revealed irregular heart rates (120-132 bpm), normal blood pressure, and cardiomegaly. The ECG showed atrial fibrillation with rapid ventricular rhythm (AFRVR), ST-segment depressions with symmetrical T waves in leads V2-V6, and Non-Sustained Ventricular Tachycardia (NSVT) in leads V8-V9. Elevated cardiac markers (CKMB) and blood glucose levels were noted. He was diagnosed with anterior ST-elevation myocardial infarction (STEMI) of the De Winter Type, Killip Class I, AFRVR, and NSVT likely due to ischemia and underlying type 2 diabetes mellitus. Pain relief was achieved after taking aspirin and clopidogrel followed by heparinization. Within 24 hours, the ECG returned to baseline, and the patient's condition improved. The De Winter ECG pattern is a sign of critical occlusion of the proximal segment of the LAD artery which typically shows upsloping ST segment depression (>1 mm), peaked T waves in precordial leads (especially V2 and V3), and a slight ST-segment elevation (0.5-1 mm) in lead aVR. Anatomical variations in Purkinje fibers causing endocardial conduction delay and ischemic ATP depletion hindering potassium channel activation may explain these ECG findings. Due to its non-classical STEMI presentation, the de Winter ECG pattern can be mistaken for hyperkalemia. It necessitates urgent management similar to STEMI, including prompt activation of the catheter lab for coronary angiography and potential stenting.

Conclusion:
Primary care physicians must recognize the distinctive features of the De Winter ECG pattern and differentiate it from similar patterns. Misinterpreting this atypical STEMI presentation and lack of awareness among medical professionals could lead to misdiagnosis, delays in treatment, and higher mortality rates.

Keywords: De Winter Sign, STEMI Equivalent, Acute Coronary Syndrome

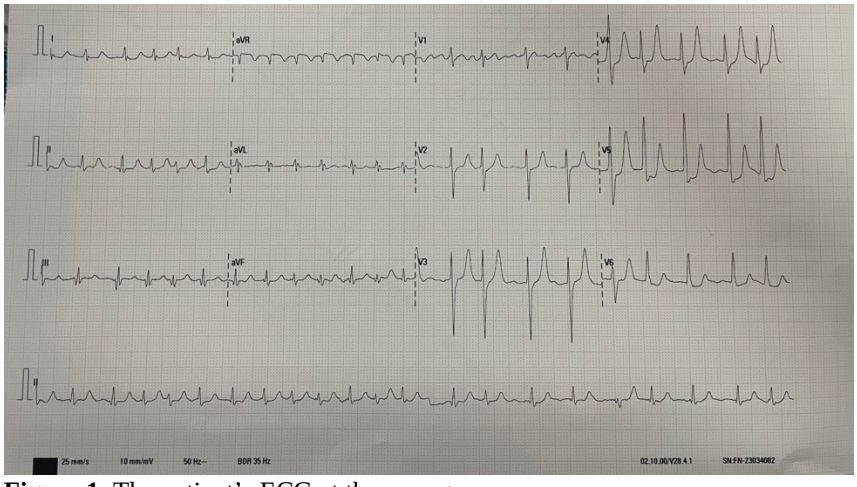

Figure 1. The patient's ECG at the emergency room

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TRANSCATHETER PATENT FORAMEN OVALE CLOSURE FOR SECONDARY PREVENTION OF STROKE IN YOUNG ADULTS : A CASE REPORT

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Background:
Patent Foramen Ovale, a common congenital structure between the cardiac atria, has emerged as being causally involved in approximately 5-10% of all strokes in young adult patients. Through the PFO, paradoxical thromboembolism can occur, and thrombus formation within the PFO can result in systemic or cerebral embolization. Treatment options for PFO may be treated with a percutaneous procedure to close the PFO or with blood thinning medication alone.

Case illustration:
A 32-year-old female with a chief complaint of recurrent migraine and a history of paraesthesia on the right side was referred to the Cardiology outpatient clinic from Neurology Departement for evaluation of cardiac abnormalities.Physical examination, laboratory findings, and radiology findings were within normal limits. ECG showed normal sinus rhythm. The MSCT and MRI also performed, the result were within normal limits.A transthoracic echocardiography examination revealed a positive defect in IAS and mild aortic regurgitation. The bubble test was positive, and transesophageal echocardiography was done to find PFO with a diameter of 3 mm and tunnel length of 11 mm. Transcatheter PFO Closure was done using a PFO Occluder size 23/25 with guiding using TTE and Fluoroscopy. One month later, the transthoracic echocardiography revealed the device was well seated and functioning well with mild aortic regurgitation and the patient had no neurological complaints anymore.

Conclusion:
Transcatheter Closure can be a reasonable option for patients with Patent Foramen Ovale accompanied by neurological conditions.

Keywords: PFO occluder, transcatheter for closure, patent foramen ovale

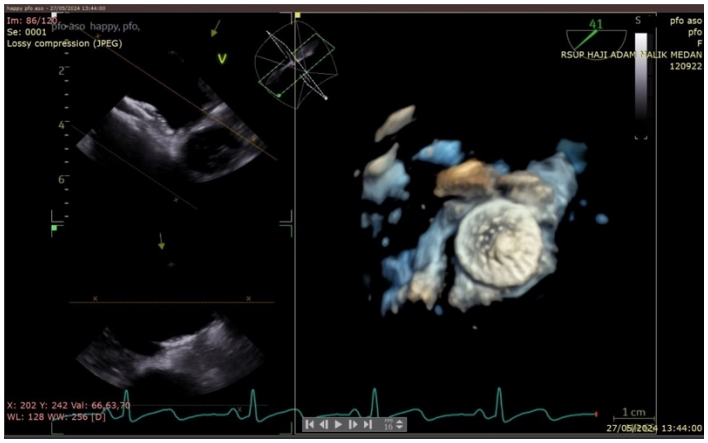

Figure 1. Trans Esophageal Echocardiography findings showed PFO closure using a PFO Occluder size 23/25

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IS IT VT OR NOT VT? (THE IMPLEMENTATION OF VERECKEI AND RWPT ALGORITHM IN RURAL AREA): A CASE REPORT

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Background:
Approximately 61% of patients with ventricular tachycardia (VT) are confused as supraventricular tachycardia. VT diagnosis using one algorithm is uncommon due to its poor sensitivity and specificity. This case reports a combination of algorithms to differentiate electrocardiogram from wide-complex tachycardia (WCT) correctly.

Case illustration:
A male, 31-years-old, 3-hours before entering the hospital, came to the emergency room with palpitations, dyspnoea, and diaphoresis. One day earlier, he experienced left cardiac chest pain radiating to the back and left arm. He went to primary healthcare and was given sublingual nitrate, afterward, the chest pain was relieved.He has a history of smoking 20 cigarettes/day and has been consuming alcohol 1.6 L/day since 18 years ago. He denied any history of heart disease and routine medication. On physical examination, carotid pulse was palpable, blood pressure 110/70 mmHg, respiratory rate 21 times/minute, body temperature 36.5ºC, oxygen saturation 98%, jugular venous pressure 5+2cm. On inspection and palpation of the heart, ictus cordis was neither visible nor palpable. Percussion showed an unwidened border of the heart. Regular S1S2 heart sounds, with no gallops or murmurs, were heard on auscultation. Electrocardiogram (Figure 1) showed tachycardia with wide QRS, rate 188 bpm, QRS duration 140ms, QTc 460ms, superior axis with left bundle branch block. The electrocardiogram applied to several algorithms: Vereckei (R wave dominant in aVR), RWPT (R wave peak time ≥ 50ms in lead 2), Brugada (R-S interval >100ms), ACC (QRS duration > 120 ms regular, V rate faster than A rate); hence the diagnosis of VT with stable hemodynamics was made. Due to limited antiarrhythmic drugs, he was given amiodarone drip 150 mg IV in 30 minutes, followed by amiodarone 360 mg IV in 6 hours, and 540 mg IV in the next 18 hours.

Conclusion:
Proper use of VT algorithms can help establish 90% of VT cases. The two algorithms chosen to distinguish VT from other similar arrhythmias are Vereckei, with the highest sensitivity (94.8%), and the RWPT algorithm, with the highest specificity (88.3%).

Keywords: vereckei, ventricular tachycardia, diagnosis, algorithm, rwpt

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TACKLING HEART FAILURE WITH REDUCED EJECTION FRACTION (HFREF) AND LOW BLOOD PRESSURE IN AMBULATORY SETTING: HOW TO FIND THE SWEET SPOT

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Background:
Heart failure imposes a significant global health burden characterized by high morbidity, mortality, and reduced quality of life. Despite clear guidelines advocating for quadruple therapy in HFrEF, there remains widespread underutilization of guideline-directed medical therapies (GDMT) in clinical practice. Clinical inertia and concerns over side effects, particularly related to low blood pressure, contribute to suboptimal treatment implementation.

Case illustration:
Two patients with different clinical phenotypes of HFrEF and low blood pressure (SBP <90-100 mmHg) routinely visited our heart failure clinic. The first patient, a 63-year-old female with HFrEF (LVEF 19%) and CAD2VD, presented with worsening dyspnea and peripheral edema, with a blood pressure of 71/50 mmHg but no signs of hypoperfusion. She was managed with all four pillars of GDMT during follow-up. Due to symptomatic hypotension, she could only tolerate sacubitril-valsartan 50 mg b.i.d. and bisoprolol 2.5 mg q.d., while achieving optimal doses of spironolactone and empagliflozin. Furosemide was administered as needed only to prevent worsening symptomatic hypotension. The second patient, a 56-year-old male with HFrEF (LVEF 16%) and calcified CAD3VD, frequently experienced dyspnea and fatigue. During follow-up, his mean BP was 91/61 mmHg. Sacubitril-valsartan and bisoprolol were up-titrated to 200 mg b.i.d. and 5 mg q.d., respectively, at the last follow-up, without symptomatic hypotension. However, he was unable to tolerate spironolactone due to gynecomastia and could not afford SGLT-2i. Both patients were also prescribed ivabradine to achieve effective heart rate control around 60 bpm. Monitoring of renal function showed no significant decline and stable potassium levels. Both reported improved symptoms and left ventricular ejection fraction, and neither required further hospitalization.

Conclusion:
Low blood pressure should not hinder GDMT implementation in HFrEF. Despite low blood pressure, the first patient received all four GDMT pillars, albeit at suboptimal doses of ARNI and beta-blockers. The second patient received only optimal doses of sacubitril-valsartan and beta blockers due to MRA tolerability and SGLT2i coverage issues. If dose adjustments are necessary, maintaining low doses of each therapeutic class is preferable to ensure a beneficial effect on prognosis by engaging all neurohormonal blocking pathways. Re-introduction or dose escalation should always be considered.

Keywords: hypotension, ambulatory, heart failure with reduced ejection fraction

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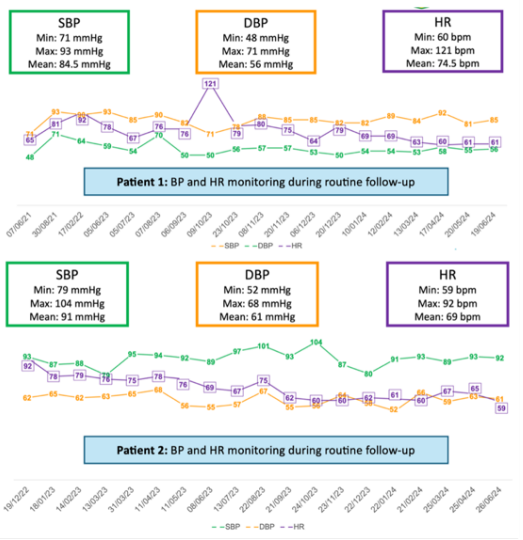

Figure 1. Blood pressure and heart rate measurement during patient's follow-up at clinic

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PERIPARTUM CARDIOMYOPATHY WITH ACUTE ISCHEMIC STROKE: A RARE CASE PRESENTATION

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Background:
Heart failure and pregnancy are both procoagulant conditions and the incidence of acute ischemic stroke is three times higher during the peripartum period compared to non-pregnant women. However, acute cardioembolic stroke is an unusual presentation of peripartum cardiomyopathy (PPCM).

Case illustration:
A 22-year-old female came with a chief complaint of shortness of breath seven months after delivering her baby which worsened over the last two weeks. She also had difficulty speaking, right-sided weakness, and bilateral ankle swelling since a week ago. Her BP was 82/61 mmHg at admission, HR 100 bpm, RR 27, and oxygen saturation 90% room air. On physical examination, the patient looked jaundiced, motoric aphasia, right hemiplegia, positive doll's eye, and pitting edema. Previous medical history was denied. Chest x-ray showed cardiomegaly and bilateral pleural effusion. Brain CT scan revealed right cerebellum infarct and brain atrophy. An echocardiogram was remarkable for left ventricle (LV) dilatation, decreased LV & RV contractility, global hypokinetic with LV ejection fraction (LVEF) of 21%, and mild tricuspid and pulmonary regurgitation. There was a 1,16 x 2,42 cm thrombus at LV. She was diagnosed with cardiogenic shock et causa PPCM and acute ischemic stroke. She was treated with inotropes, bromocriptine, oral HF drugs such as ramipril, furosemide, spironolactone, diuretics, and LMWH overlapping with warfarin. PPCM is diagnosed from the last month of pregnancy until five months after delivery. Our case is unique because the initial symptoms of both heart failure and stroke were presented seven months after delivery with the absence of complete LV recovery. The incidence of ischemic stroke in patients with PPCM is only 5% and LV thrombus presence has been associated with low LVEF.

Conclusion:
PPCM with complications of acute cardioembolic stroke is a rare presentation and the presence of LV thrombus suggests worse outcomes. Diagnostic tests and drug availability in rural areas will pose another challenge at initial diagnosis and monitoring therapy.

Keywords: left ventricular thrombus, peripartum cardiomyopathy, cardioembolic stroke

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DIAGNOSIS OF CORONARY ARTERY-TO-PULMONARY ARTERY FISTULA (CPAF) IN A 54-YEAR-OLD MAN WITHOUT MAJOR RISK FACTORS: A CASE REPORT

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Background:
A Coronary artery fistula (CAF) is an abnormal connection between a coronary artery and a major vascular structure or heart chamber. It is usually congenital, but in some cases, it may be acquired. A rare subtype, coronary artery-to-pulmonary artery fistula (CPAF), typically remains asymptomatic. However, larger or multiple fistulas with severe left-to-right shunting can cause myocardial ischemia, heart failure, pulmonary hypertension, or sudden cardiac death. These symptomatic or large fistulas usually require closure. The choice of intervention depends on the fistula's size, location, and complexity.

Case illustration:
A 54-year-old man reported chest pain worsening with exertion over the past few years and occasional palpitations during activity. He denied shortness of breath and had no history of hypertension, diabetes, or stroke, but had a 10-year smoking history. In 2014, coronary angiography showed non-significant obstruction. Echocardiography and treadmill test results were normal. A recent CT Coronary Angiogram revealed non-significant stenosis in the left main coronary artery, left circumflex artery (LCx), and right coronary artery (RCA), along with a possible coronary branch anomaly from the RCA. Diagnostic coronary angiography confirmed a large fistula from the proximal RCA and a small fistula from the high first diagonal branch (D1) connected to the pulmonary artery, without coronary artery stenosis. The optimal treatment strategy remains unclear and there are no established therapeutic guidelines available. After discussing the case, percutaneous closure with coil embolization and transarterial approach was recommended, considering the proximal origin of the fistula and the fistula's morphology to minimize procedural risks and complications while surgical intervention is more favorable for multiple drainage sites, distal origin, large branches, vessel's tortuosity, and concomitant cardiac disorders.

Conclusion:
CPAF, a rare CAF subtype, shows the high correlation between symptom severity and the number, size, and severity of left-to-right shunts of the fistulas. Management options include conservative approaches, percutaneous transcatheter closure, or surgery. The main indications for closure are the presence of symptoms, complications, and significant shunting. The feasibility of the procedure should account for procedural risks and potential complications.

Keywords: coil embolization, percutaneous closure, coronary artery to pulmonary artery fistula, Coronary artery fistula

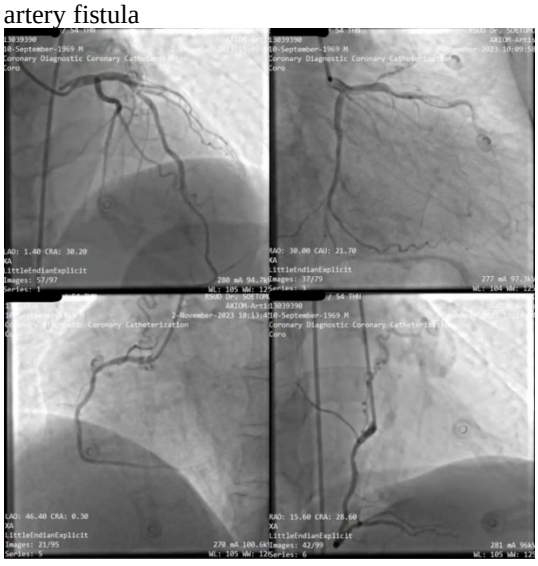

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Figure 1. The coronary angiography reveals a small fistula from the high first diagonal branch (D1) to the pulmonary artery (Upper figure) and a large fistula at proximal RCA connected to the pulmonary artery (Lower figure)

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SEVERE MITRAL REGURGITATION WITH POSTERIOR MITRAL LEAFLET PROLAPSE: A CASE REPORT AND MANAGEMENT IN AN ELDERLY PATIENT

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Background:
Mitral regurgitation is a condition characterized by the incomplete closure of the mitral valve, causing blood to leak backward into the left atrium. This can result in inadequate blood flow through the heart and to the rest of the body, leading to symptoms like fatigue and shortness of breath. Mitral valve prolapse involves the bulging of the valve leaflets into the left atrium during heart contraction, sometimes causing regurgitation.

Case illustration:
A 77-year-old male patient presented with chest discomfort that began one day prior to hospital admission. The discomfort, associated with moderate physical activity (furniture making), was accompanied by shortness of breath. The patient reported that the chest discomfort did not radiate and was not associated with nausea or vomiting. Symptoms improved with rest but recurred the following morning, prompting an emergency visit to Bangil Hospital. Previous echocardiography had indicated the need for heart valve surgery, which the patient declined. The patient had been under routine treatment at Bangil Hospital, receiving Candesartan (4mg once daily), Digoxin (0.25mg once daily), Warfarin (2mg once daily), Simvastatin (20mg once daily), and Lansoprazole (30mg once daily). On admission, the patient had a heart rate of 40-45 bpm and was administered Sulfas Atropine (1mg), a dopamine drip (5mcg/kg/min), and Furosemide (60mg). He was referred for further management.

Conclusion:
The patient was treated in the Cardiovascular Care Unit (CVCU) for six days. His chest discomfort gradually decreased, and efforts were made to stabilize his hemodynamic condition. A mitral valve replacement was recommended due to posterior mitral leaflet prolapse. Following treatment, the patient showed significant improvement in hemodynamic stability. Plans were made for the insertion of a permanent pacemaker (PPM) to address his bradycardia. This case illustrates the clinical management of mitral valve prolapse and severe mitral regurgitation in an elderly patient, highlighting the importance of timely surgical intervention and the potential need for pacemaker insertion in cases of symptomatic bradycardia.

Keywords: Mitral Regurgitation

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RECURRENT NOCTURNAL CHEST PAIN IN PATIENT WITH BRUGADA SYNDROME AND MYOCARDIAL BRIDGE: A CASE REPORT

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Background:
Brugada syndrome (BrS) is an autosomal-dominant inherited disorder without significant structural heart abnormalities, characterized by coved ST-segment elevation ≥2 mm with T-wave inversion at precordial leads (V1-V3). Brugada Syndrome is associated with sudden cardiac death (SCD) and ventricular tachyarrhythmias, such as sustained ventricular tachycardia (VT) and ventricular fibrillation (VF).[1-3,9] Patients with BrS often complain chest pain that must be differentiated from coronary artery disease (CAD). In this case report, we present a unique case of BrS patient with recurrent nocturnal chest pain.

Case illustration:
A 37-year-old male complaining recurrent chest pain during night time with type 1 Brugada Syndrome electrocardiogram (ECG) pattern. Angiography was done to exclude coronary artery disease. Insignificant coronary artery occlusion and myocardial bridging in mid left anterior descending (LAD) artery was found during angiography. Patient then underwent electrophysiology study (EPS), inducible polymorphic VT with normal sinoatrial (SA) and atrioventricular (AV) nodes function was found. Patient was diagnosed with Brugada Syndrome and implantable cardioverter defibrillator (ICD) was implanted after risk stratification. Despite this, he continues to experience occasional nighttime chest pain. Implantable cardioverter defibrillator reprogramming was done revealing no inducible VT after implantation, thereby we suggest recurrent chest pain is caused by myocardial bridging.

Conclusion:
Recurrent nocturnal chest pain in BrS patients might be related to myocardial ischemia. In this case report, it might be associated with myocardial bridging found in mid LAD artery. This condition requires further treatment in order to terminate chest pain symptom.

Keywords: Case report, Recurrent nocturnal chest pain, Brugada syndrome, Myocardial bridging

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HIGH PROBABILITY OF PULMONARY HYPERTENSION AS A RARE PRESENTATION AS A COMPLICATION OF SYSTEMIC LUPUS ERYTHEMATOSUS: A CASE REPORT FROM IN RURAL HOSPITAL

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[1]RSUD SUNGAI DAREH

Background:
Pulmonary hypertension can be caused by connective tissue diseases including systemic lupus erythematosus (SLE). SLE is an autoimmune disease that attacks multiple organs, one of which is the heart. Pulmonary hypertension is a severe and life-threatening complication of systemic lupus erythematosus. The underlying inflammatory component suggests an association of PH with SLE in the pathogenesis of this case. The prognosis of SLE patients with PH is reported to be very poor and is the third most common cause of death in SLE, after infection and organ failure. We present a case report of pulmonary hypertension in a patient with SLE.

Case illustration:
A 16 years old Female, came to the cardiology clinic with the main complaint of shortness of breath, shortness of breath especially during activities, easily tired, no chest pain. She has been known to have systemic lupus erythematosus since 4 years ago, with an ANA > 1:1000. Clinical examination showed pulse 113/minute, BP 100/87 mmHg, RR 25/minute SpO2 94-97%. On physical examination, there were pansystolic murmurs , no rales and pretibial edema. On the electrocardiogram, a sinus tachycardic rhythm was found with a rate of 113/minute, the axis was normal, there was no enlargement of the heart chambers. Echocardiography found an ejection fraction of 85%, and TR Vmax 4.40 m/s, severe tricuspid regurgitation, and mild pericardial effusion. The patient was diagnosed with pulmonary hypertension with systemic lupus erythematosus. We treated the patient with sildenafil by a cardiologist, while the pediatrician was given prednisone, catopril, calcium, zinc and vitamin D tablets.

Conclusion:
In this patient there was a presentation of pulmonary hypertension with a previous history of SLE, this indicates that the cause of pulmonary hypertension in this patient is most likely an autoimmune disease, especially SLE. Clinical manifestations of cardiac involvement in SLE are often found at a severe stage, especially in rural areas, thus requiring further investigation and immediate treatment.

Keywords: Systemic Lupus Erythematosus, Pulmonary Hypertension

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SUDDEN SHORTNESS OF BREATH AND LEUKOCYTOSIS: IS IT ACS? CASE REPORT OF ATYPICAL ACS PRESENTATION FROM SECONDARY HOSPITAL

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Background:
Acute coronary syndrome (ACS) remains a leading cause of mortality worldwide. The typical presentation includes crushing substernal pain, often radiating to the neck or jaw. However, ACS can also present atypically, and the absence of classical symptoms should not exclude the diagnosis. This report presents a case of a patient with sudden onset of shortness of breath, initially misdiagnosed as pneumonia, subsequently revealed to be acute coronary syndrome during hospitalization.

Case illustration:
A 73-year-old woman presented to the emergency room with sudden shortness of breath. She was hypotensive (BP 90/68), with other vital signs within normal limits. Physical examination revealed bilateral lung rales, normal jugular venous pressure, and no peripheral edema. Initial electrocardiogram showed slight ST elevation (1-3 mm) in leads V2 and V3. Laboratory findings indicated leukocytosis (15,760). The patient was initially diagnosed with pneumonia and septic shock and was treated with antibiotics. Despite an ECG evaluation and cardiology consultation, no chest pain was noted, and the ECG pattern remained unchanged. The following day, the patient experienced worsening shortness of breath, increased lung rales, and hypotension. A repeat ECG revealed significant ST elevation in leads V2-V4. The diagnosis was revised to anterior STEMI, cardiogenic shock, and acute pulmonary edema. Fibrinolysis was performed, along with the administration of antiplatelets, statins, diuretics, and inotropes. Post-fibrinolysis, the patient's symptoms improved, and ST elevation decreased by more than 50%. The patient was stabilized and transferred to a higher facility for rescue PCI.

Conclusion:
This case highlights the importance of recognizing atypical presentations of acute coronary syndrome (ACS). General practitioners should be aware that shortness of breath is not exclusively a lung issue but can also indicate a cardiac problem. By paying close attention to the patient's condition and medical history, healthcare providers can improve patient care and prevent potentially harmful situations.

Keywords: Acute Coronary Syndrome, Fibrinolysis, STEMI

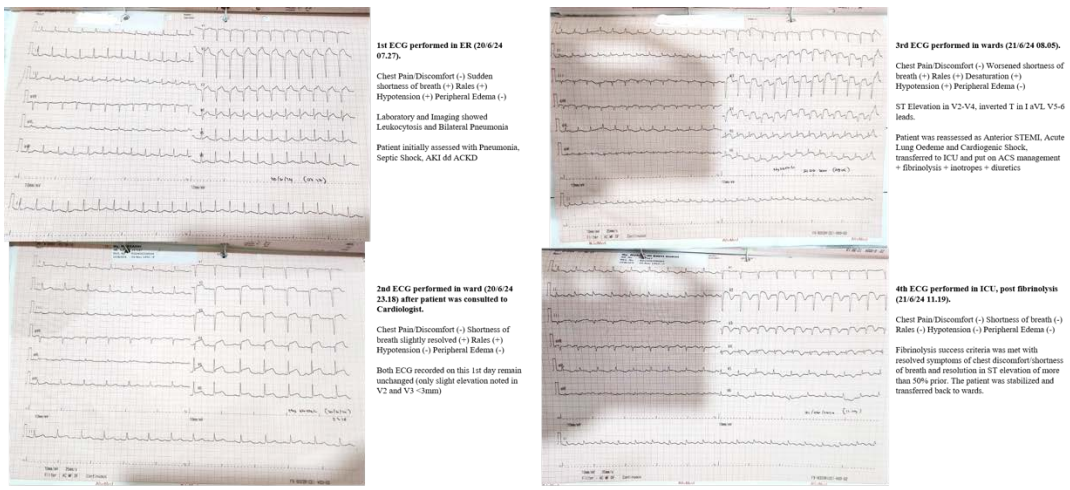

Figure 1. Patient's ECG changes during admission-hospitalization (including pre and post fibrinolysis)

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HEARTFELT CHALLENGES: UNRAVELING THE COMPLEXITY OF PERIPARTUM CARDIOMYOPATHY IN MATERNAL HEALTH CARE

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Background:
Peripartum cardiomyopathy (PPCM) presents a challenging scenario in maternal health, characterized by the development of heart failure in the last month of pregnancy or within five months postpartum. This condition, though rare, carries significant risks for both the mother and the newborn. The etiology of PPCM remains elusive, with various factors such as hormonal, genetic, and immune mechanisms implicated in its pathogenesis. Early diagnosis and prompt management are crucial in improving outcomes for affected individuals.

Case illustration:
of a 29-year-old woman admitted to the Obstetrics and Gynecology Emergency Room with symptoms of dyspnea, fatigue, and edema highlights the clinical manifestations of PPCM. Diagnostic modalities including electrocardiography, cardiac biomarkers, and echocardiography played a pivotal role in confirming the diagnosis. The patient received a multidisciplinary approach to treatment, involving cardiology consultation, pharmacological therapy, and close monitoring of maternal and fetal well-being. Despite the challenges posed by PPCM, the patient showed gradual improvement in cardiac function and symptom resolution over time.

Conclusion:
the management of PPCM requires a comprehensive and coordinated effort from healthcare providers to optimize maternal outcomes and ensure the well-being of both mother and child. Regular follow-up visits, adherence to medication regimens, and lifestyle modifications are essential components of long-term care for individuals with PPCM. Further research is needed to elucidate the underlying mechanisms of this condition and to develop targeted therapies that can improve prognosis and quality of life for affected women. By raising awareness, enhancing early detection, and implementing evidence-based interventions, healthcare professionals can make a significant impact in the management of PPCM.

Keywords: multidisciplinary care., heart failure, Peripartum cardiomyopathy

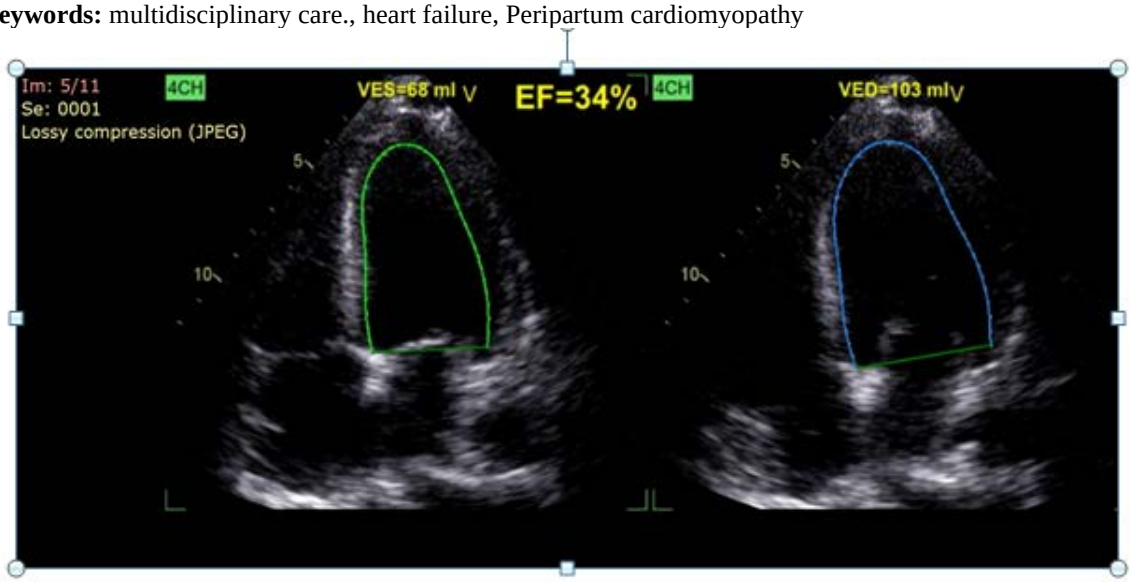

Figure 1. EF biplane of Echocardiography

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LAUBRY-PEZZI SYNDROME IN 35 YEARS OLD WOMAN WITH A CONCOMITANT MITRAL REGURGITATION : A CASE REPORT & A SHORT-TERM OUTCOME AFTER SURGERY

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Background:
Laubry–Pezzi syndrome is a rare congenital heart disease characterized by ventricular septal defect (VSD) accompanied by aortic regurgitation (AR) due to prolapse of the aortic valve cusps or leaflets. Echocardiography is a key examination to diagnose and determine the type of VSD and the severity of aortic regurgitation. *Mitral regurgitation* (MR) is caused by retrograde blood flow from the left ventricle (LV) to the left atrium (LA) through the mitral valve (MV), causing a systolic murmur best heard at the apex of the heart with spread to the left axilla. The management of Laubry-Pezzi syndrome is not yet known with certainty, but early VSD closure should be indicated to prevent the onset or worsening of AR.

Case illustration:
We report a case of a 35 year old woman with *Subarterial Doubly Committed* (SADC) VSD with moderate aortic regurgitation accompanied by moderate mitral regurgitation. The patient was diagnosed with a VSD at the age of 5 years, but was only willing to undergo VSD closure after her symptoms of shortness of breath during activities had increased in the last year. This patient underwent three surgical procedures including: surgical VSD closure, Aortic Valve Replacement, and Mitral Valve Repair. Within 70 – 90 days after surgery, a gradual evaluation is carried out to assess the short-term outcomes of this patient.

Conclusion:
The relationship between congenital defects such as VSD, AR and MR needs to be identified and corrected early in life to obtain better outcomes.

Keywords: AR, MR, VSD, Laubry-Pezzi syndrome, Echocardiography

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INFECTIVE ENDOCARDITIS : A COMPLICATION OF RUPTURED SINUS OF VALSALVA ANEURYSM OR VICE VERSA ?

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Background:
Infective endocarditis is an infectious disease with high levels of morbidity and mortality. Individuals with structural heart disease, such as a ruptured sinus of Valsalva aneurysm, are included in the population at risk of infective endocarditis. The diagnosis of infective endocarditis should be suspected in any at-risk patient who has a complaint of fever of unknown origin.

Case illustration:
A 20-year-old man with complaints of nausea and vomiting 2 days before being admitted to Dr. Moewardi Hospital. He had a history of fever and was treated for 6 days at another hospital (2 weeks before) and untreated dental caries since 1 year ago. The patient had a history of ruptured sinus of Valsalva aneurysm right coronary cusp (RCC) to the right ventricle, with unknown etiology, accompanied by moderate aortic regurgitation diagnosed 3 months previously. On transesophageal echocardiography (TEE), mobile vegetation was found in the right sinus of Valsalva with a size of 1.1 cm x 0.4 cm, and 2 separate blood culture samples were positive for *Streptococcus viridans*. On the 4th day of treatment, the patient experienced a fever again with a body temperature of 38.2^0 C and he also complained of swelling in the face, hands, and legs. Immunological phenomena such as symptoms and signs of glomerulonephritis was found, with a positive rheumatoid factor test. The transthoracic echocardiography (TTE) evaluation after 4 weeks of treatment at Dr. Moewardi Hospital showed an increase in vegetation size of 1.3 x 0.9 cm. The patient refused surgery.
Conclusion:
This case is a sub-acute bacterial endocarditis caused by *Streptococcus viridans* infection and characterized by a slow clinical course with symptoms of not very high fever. The oral cavity may be the port of entry for bacteremia in the patient. The patient's prognosis is dubia et malam because of uncontrolled infection and vegetation formations (>1 cm) which increase in size during evaluation with TTE examination after 4 weeks.

Keywords: infective endocarditis, Streptpcoccus viridans, Ruptured sinus of Valsalva aneurysm, glomerulonephritis

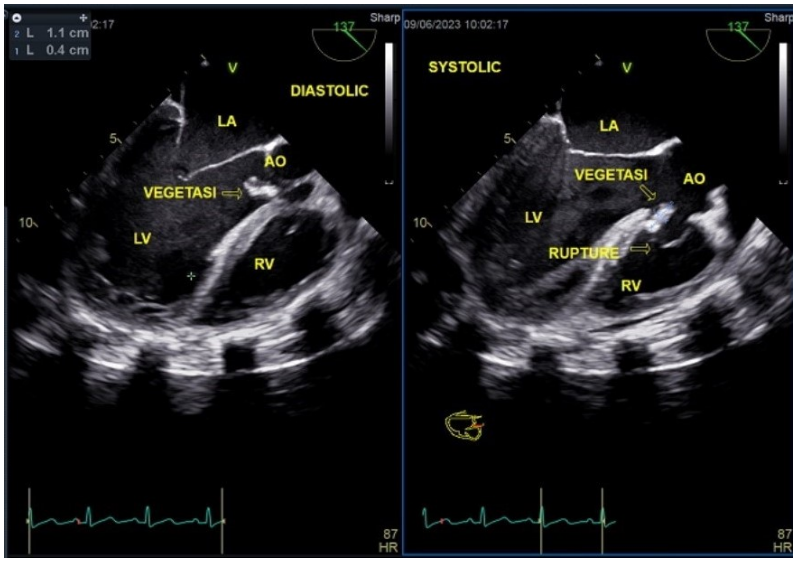

Figure 1. TEE, mid-esophageal, long axis view 137°: Rupture of the sinus of Valsalva in the RCC region, windsock appearance (+). Mobile vegetation was found in the right sinus of Valsalva with a size of 1.1 cm x 0.4 cm.

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COMPLEX INTERPLAY OF CARDIORENAL AND SEPTIC SHOCK: EFFECTIVE STRATEGIES IN CRITICAL CARE

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Background:
Refractory cardiorenal syndrome (CRS) in the context of mixed cardiogenic septic shock is a complex clinical scenario involving the intricate interplay between cardiac, renal, and immune dysfunctions. Effective management requires a multidisciplinary approach and timely interventions.

Case illustration:
A 53-year-old male presented with acute chest pain and was diagnosed with inferior STEMI and right ventricular infarction. He underwent primary percutaneous coronary intervention (PCI) with initial stabilization. Post-PCI, the patient developed contrast-induced nephropathy (CIN) evidenced by rising serum creatinine and oliguria, coupled with urinary tract infection and pneumonia that progressed to sepsis. These complications exacerbated his cardiac and renal impairments, leading to mixed cardiogenic septic shock. Continuous renal replacement therapy (CRRT) and vasopressors were pivotal in managing his condition, maintaining fluid balance, and supporting hemodynamics.

Conclusion:
Managing refractory CRS in mixed cardiogenic septic shock demands a comprehensive understanding of the pathophysiological mechanisms and an integrated treatment strategy. The successful outcome in this case underscores the importance of fluid management, appropriate vasopressor use, aggressive sepsis management, and continuous clinical reassessment in achieving favorable outcomes.

Keywords: Cardiorenal Syndrome, Contrast-Induced Nephropathy, Cardiogenic Shock, Septic Shock, Renal Replacement Therapy

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NT-PRO BNP IN ITS CORRELATION IN PATIENT WITH HEART FAILURE: A CASE REPORT

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Background:
Acute Lung Oedema is one of the Acute Heart Failure Spectrum. Risk factors of Heart Failure (HF) include ischemic heart disease, myocardial infarction, myocarditis, valvular heart disease, tachycardia, diabetes mellitus, structural heart disease related to congenital heart disease, sleep apnea, excessive use of drugs or alcohol, and obesity. A significant percentage of around 30% to 40% of nonischemic heart failure is thought to originate from genetic factors.Natriuretic peptides, commonly B-type natriuretic peptide (BNP) and NT- proBNP are useful biomarker for the diagnosis, estimation of its severity and prognosis, and possible management of heart failure. While a single measurement is very useful in determining the prognostic value, serial measurements will gradually add important prognostic information. patients with acute heart failure, those who do not show significant reductions in BNP or NT-proBNP at the time of hospital discharge, tend to have higher rates of mortality and morbidity.

Case illustration:
A 76 yo man was brought to Emergency Room (ER) with shortness of breath and decreased of consciousness. before going to the hospital, he had shortness of breath and chest pain. Initial Vital signs show Somnolence, Blood Pressure 99/72 mmHg, tachycardia, tachypnea, SpO2 70-80%, on examination bronchial breath sounds and rhonci were found. Patient was treated in the Intensive Care Unit by several specialist doctors with complex diseases including ALO and NSTEMI in the field of cardiologist and was given comprehensive multidisciplinary management.Lab results showed increased NT pro BNP (35,000 pg/mL) and troponin I (6.38 ng/mL), and many more abnormal findings of laboratory tests. ECG showed Sinus tachycardia with ST depression and T inversion in lateral leads and septal OMI. Echocardiography showed mildly reduced CHF. Chest X Ray showed cardiomegaly and pneumonia. Head CT scan showed extensive infarction in the left basal ganglia and frontotemporoparietal. At first, patient was planned to be referred for PCI but the family refused, and the condition was not yet stable. Patient was treated medically for approximately 16 days and being discharged.

Conclusion:
NT-Pro BNP Biomarker is important to evaluate dyspnea origins also in diagnosis of Heart Failure so that appropriate management can be given.

Keywords: heart failure, NT-Pro BNP

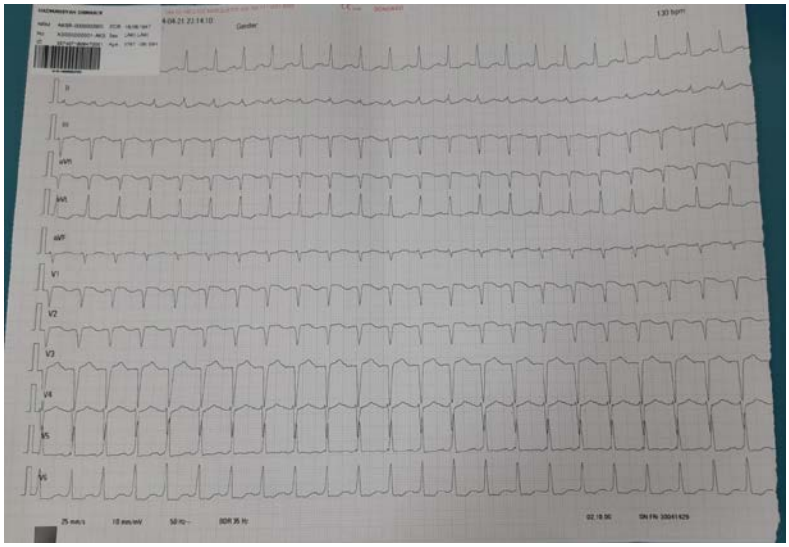

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Figure 1. ECG of patient in Emergency Room

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LUTEMBACHER SYNDROME AN ECHOCARDIOGRAPHY ASSESMENT: A RARE CASE

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[1]RSUD Balaraja

Background:
Lutembacher Syndrome (LS) is a rare heart defect, characterized by the presence of Atrial Septal Defect (ASD) (congenital or acquired) and Mitral Stenosis (MS) (congenital or acquired). The prevalence of Lutembacher syndrome is reported to be 0.001 per million population with a predominance of women. this disease is more frequently reported in countries where developing countries such as Southeast Asia and have a previous history of rheumatic fever.

Case illustration:
A 56 years old woman came to the cardiovascular clinic at Balaraja Hospital with complaints of shortness of breath has been felt since the last 3 months. shortness of breath has been aggravated by activity and relieved by rest. Shortness of breath is also felt when lying down, if she sleeps, she has to use 2-3 pillows or an elevated position, Shortness of breath is also accompanied by chest pounding and swelling in both legs. The patient also complained of feeling full in the stomach and coughing up phlegm for 2 weeks, the phlegm was white and foamy. On physical examination there was an increase in jugular venous pressure (JVP) 5+4 cm, irregular 1st and 2nd heart sounds, and a grade 2/4 diastolic murmur at the apex and a pansystolic murmur were found. grade 3/6 in the tricuspid area. During auscultation, wide fixed splitting was also found. On examination of the extremities, edema was found in both legs. An electrocardiogram (ECG) examination revealed atrial fibrillation (AF), bigemini ventricular extrasystole (VES) and echocardiography conclusion were found Rheumatic Heart Disease, MS severe, mild mitral regurgitation, severe tricuspid regurgitation, pulmonary hypertension, ASD secundum left to right shunt.

Conclusion:
Lutembacher Syndrome is still a rare disease, including in Indonesia. If diagnosed early and appropriate therapy is carried out, it will provide a good prognosis, if the diagnosis is late, it will increase mortality due to heart failure, arrhytmia and embolic stroke.

Keywords: rare case, heart failure, case report, Lutembacher syndrome

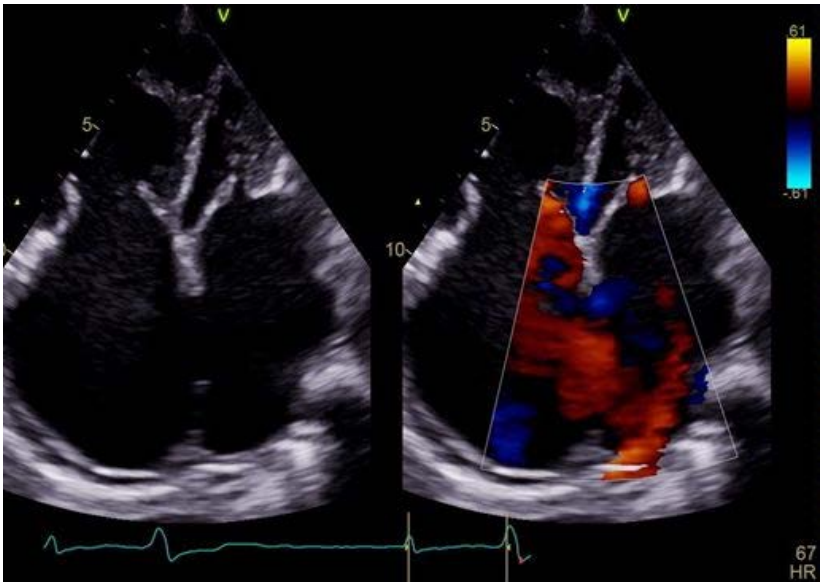

Figure 1. Apical Four Chamber Echocardiogram showing Dilated Right Heart Chambers with Atrial Septal Defect

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SUSTAINED MONOMORPHIC VENTRICULAR TACHYCARDIA IN PATIENT WITH IMPLANTED CARDIOVERTER-DEFIBRILLATOR DUE TO DETECTION FAILURE

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Background:
Ventricular tachycardia (VT) is a potentially life-threatening arrhythmia, and it is responsible for the majority of sudden cardiac deaths. Implantable cardioverter-defibrillators (ICDs) are now widely used in patients who survive sustained VT or VF that is not attributable to a transient correctable cause, or who are at high risk for recurrent arrhythmia.

Case illustration:
A 65-year-old female was admitted with a chief complaint of palpitation and fatigue since 5 hours before admission. The episodes increased in frequency since last month, and worsened. Palpitation onset was sudden. She was fully conscious and cooperative. Chest pain and shortness of breath were denied. She had frequently experienced similar symptoms since 7 years ago. She had history of diabetes mellitus, percutaneous coronary angiography, radiofrequency ablation (2017 and 2022), and dual chamber ICD implantation (2022). No history previous trauma. Her hemodynamic was unstable, with ECG showing wide complex tachycardia (RBBB-type, superior axis), with QRS rate 175 bpm. Magnet was applied to rule out pacemaker mediated tachycardia. Cardioversion with 100 Joule energy was performed, started with 100 Joule, which was successful. Chest x-ray showed no abnormality of ICD system. She was discharged later and referred for an ICD interrogation. The interrogation showed that her sustained VT was due to detection failure. It was programmed to detect VT at 187 bpm, based on recorded episodes in the internal memory, which obviously recorded VTs of more than 200 bpm. The actual VT was about 175 bpm. She was scheduled for ICD reinterrogation.

Conclusion:
Higher programmed VT rate detection is a possible mechanism underlying detection failure in patients with ICD. External cardioversion in patients with implanted rhythm devices may be safe and effective. Biphasic shocks required less energy for VT termination and may thus be preferable in these patients.

Keywords: Ventricular Tachycardia, Cardioversion, Detection Failure, Dual Chamber, ICD

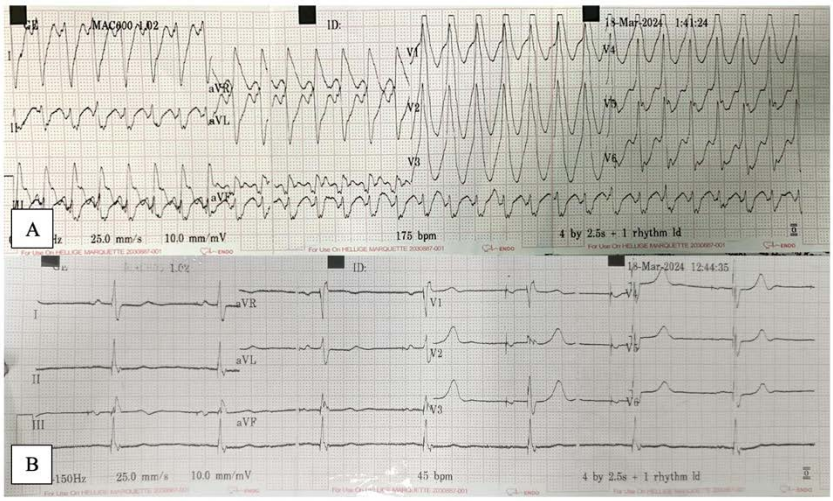

Figure 1. A. ECG during admission showed wide complex tachycardia (RBBB-type, superior axis), QRS rate 175 bpm; B. ECG after cardioversion showed atrial pacing ventricular pacing with 45 beats per minute.

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BIVENTRICULAR THROMBI IN A 23-YEAR-OLD MALE WITH CARDIOMYOPATHY AND LUNG TUBERCULOSIS: A COMPLEX INTERPLAY

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Background:
Intracardiac thrombi more commonly occur in the right (RV) or left ventricle (LV) and are associated with ventricular dysfunction. Nevertheless, concurrent biventricular thrombi formation is rare, but its presence potentially causes life-threatening complications.

Case illustration:
A 23-year-old male in intensive phase treatment for active lung tuberculosis (TB) was consulted by the attending pulmonologist to our department for heart failure management. The patient appeared severely ill and tachypneic. Physical findings were suggestive of acute pulmonary edema. Initial electrocardiogram showed sinus tachycardia, incomplete right branch bundle block, and left ventricle hypertrophy. Thorax x-ray reported LV cardiomegaly and pneumonia with underlying TB. Blood gas analysis revealed that the patient was in an uncompensated metabolic acidosis state, resulting in impending respiratory failure. In response to the acute congestive state, the patient was given a high-dose intravenous vasodilator, loop diuretic, oral angiotensin-receptor blocker, and digoxin. Acid-base correction with sodium bicarbonate was also performed. After 3 days of treatment, the patient's symptoms, diuretic status, and general condition were improved. Follow-up echocardiography demonstrated concentric LV hypertrophy, echo-dense masses with distinctive margins from the LV and RV endocardium suggestive of biventricular thrombi, 32% ejection fraction, grade III LV diastolic dysfunction, and TAPSE 17 mm, however, cardiac valves were normal. Thrombi were observed in LV and RV apex with the approximate area of 1.65 cm and 4.81 cm2 respectively. D-dimer was measured at 9930, indicating hypercoagulability. The discovery of biventricular thrombi and hypercoagulability prompted an immediate anticoagulation. We selected novel oral anticoagulation as treatment-of-choice in the prevention of pulmonary thromboembolism in this patient, due to less drug-drug interaction to antituberculosis compared to warfarin. From the regimen of anticoagulation and heart failure pillars, the patient's outcome was advancing, thus later discharged after 3 weeks of hospitalization. Outpatient care is programmed for the continuation of anticoagulation, thrombus evaluation, and heart failure management.

Conclusion:
Biventricular thrombi is a rare phenomenon, occurring in patients with hypercoagulable states and ventricular dysfunction. Routine echocardiography is a reproducible tool for early detection of intracardiac thrombi. Furthermore, patient-based treatment and prompt anticoagulation are appropriate to prevent cardioembolic events.

Keywords: cardiomyopathy, lung tuberculosis, biventricular thrombi

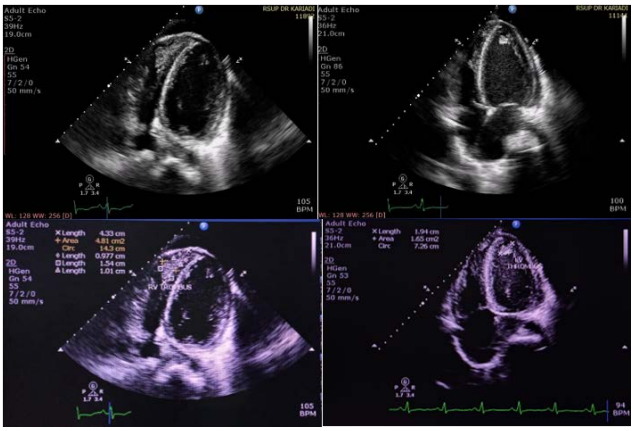

Figure 1. Echocardiography Finding of Biventricular Thrombi

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PALLIATIVE CARE IN ADVANCED HEART FAILURE: THE BEST APPROACH TO FULFILL PATIENT NEEDS DESPITE THE COMPLEXITY OF MEDICAL CARE

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Background:
Chronic Heart Failure (CHF) is a major global health problem with a significant increase in prevalence. CHF patients in the world are estimated to reach 64.3 million in 2017 and about 1-10% of the Heart Failure (HF) population suffers from Advanced Heart Failure. CHF can be characterized by clinical deterioration and may lead to advanced HF, which can be recognized by severe symptoms, recurring episodes of decompensation, and a reduced quality of life. The clinical challenge when treating a patient with Advanced HF is related to a poor prognosis and difficulties in treatment due to persistent symptoms and unresponsiveness to traditional HF therapy. To maintain the patient's quality of life, it is important to implement a holistic management plan that includes palliative care.

Case illustration:
A 59 years-old woman with a history of anterior STEMI after PCI 1 DES at LAD in 2013 and persistent left ventricular dysfunction came to the emergency room with complaints of shortness of breath at rest and chronic leg edema, along with hypotension and oliguria, making hemodialysis therapy failed at the time. The patient experienced symptoms of HF that continued to worsen during the last 3 months before admission. In the hospital, after receiving the best possible medication and extensive hemodynamic therapy, the patient's care shifted to palliative care, which is symptom management and comfort-oriented. Cardiac home palliative care with morphine administration was chosen after family engagement and decision-making with a team of clinicians, in order to prioritize quality of life and patient comfort over aggressive intervention.

Conclusion:
Management of Advanced HF is complex and involves a multidisciplinary approach to improve symptoms and quality of life. Several studies show palliative care impacts better quality of life, improved symptoms, less anxiety and depression, better spiritual well-being, and reduced readmissions compared with standard care. Other evidence supports the role of early palliative interventions in improving patient outcomes and satisfaction. This case highlights the importance of a holistic approach and management including palliative care, especially in the context of Advanced HF.

Keywords: Advanced Heart Failure, Cardiac home palliative care

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ASYMPTOMATIC SEVERE MITRAL REGURGITATION (MR) AFTER BLUNT CHEST TRAUMA: A CASE REPORT

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Background:
Mitral regurgitation (MR) is often caused by degenerative changes, rheumatic heart disease, infective endocarditis, and ischemic heart disease. MR secondary to blunt chest trauma (BCT) is rare and often presents as an acute emergency with severe symptoms. This case illustrates an example of asymptomatic MR following BCT.

Case illustration:
A 57-year-old male presented with left lower chest tenderness following a motorcycle accident three days prior, involving a forceful anterior chest impact. He was hemodynamically stable, with a grade III holosystolic murmur at the cardiac apex, and no visible chest trauma. He denied chest pain, dyspnea, or signs of heart failure. Electrocardiography showed normal sinus rhythm, and chest x-ray revealed isolated fractures of the 8th and 9th left ribs. Given the newly detected murmur, a cardiology consultation was sought. Transthoracic echocardiography (TTE) revealed a flail posterior mitral leaflet (PML) with severe mitral regurgitation (MR). The left ventricular (LV) wall thickness and motion were normal, with mildly dilated LV, and normal left atrial (LA) dimension (38 mm). Both right and left ventricular functions were normal, with an ejection fraction of 75%, and no evidence of valve vegetation. The patient opted for conservative management and remained asymptomatic, with close outpatient monitoring to evaluate disease progression and determine the need for potential intervention. The presence of a flail mitral leaflet due to chordae tendineae rupture appears to be linked to the BCT. The lack of pronounced clinical symptoms is consistent with the subtle and prolonged nature of chordae tendineae rupture, which often remaining subclinical for extended periods. This is in contrast to the rapid onset of symptoms typically seen in in mitral valve prolapse (MVP) resulting from papillary muscle rupture.

Conclusion:
This case underscores the importance of echocardiography in early diagnosis of traumatic MR. It also emphasizes the necessity for long-term follow-up, particularly for MR resulting from chordae tendineae rupture, which can remain asymptomatic for several months.

Keywords: mitral regurgitation, mitral valve prolapse, chordae tendineae rupture, blunt chest trauma

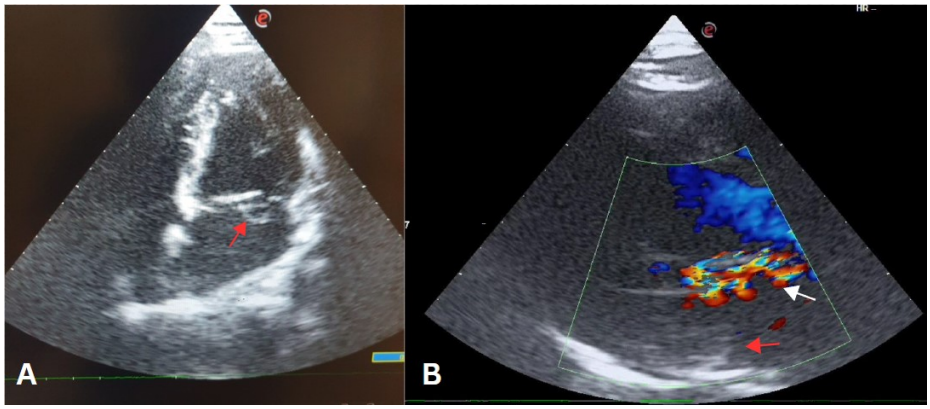

Figure 1. (A) TTE apical four chamber view shows a flail PML (red arrow). (B) Color-flow Doppler TTE in parasternal long-axis view shows severe MR (white arrow) and prolapsed PML to LA, consistent with chordae tendineae rupture (red arrow).

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PERIPARTUM CARDIOMYOPATHY PRESENTING WITH SUPRAVENTRICULAR TACHYCARDIA IN A DEVELOPING COUNTRY: A CASE REPORT FROM INDONESIA

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[1]RSUD Bali Mandara

Background:
Despite advances in medical management, peripartum cardiomyopathy (PPCM) remains a leading cause of maternal mortality. Supraventricular tachycardia (SVT) in patients with PPCM contributes to the complexity and severity of the condition. This case report aims to describe a case of supraventricular arrhythmia in peripartum cardiomyopathy.

Case illustration:
A 35-year-old female, gestational age 35-36 weeks, was admitted to the emergency unit of our hospital due to palpitation and shortness of breath. The heart rate was 191 beats per minute. Electrocardiogram (ECG) showed supraventricular tachycardia. She was injected with 0.5 mg digoxin. Four hours after the injection, the ECG converts to sinus rhythm. She was subsequently admitted to the Intensive Cardiology Care Unit (ICCU). Initial 2D transthoracic echocardiography (TTE) showed global hypokinetic, decreased right and left ventricular failure with poor left ventricular ejection fraction (LVEF 27%), and severe mitral regurgitation function. She was diagnosed with congestive heart failure due to PPCM + SVT. The treatment includes 4 liter/minute oxygen therapy through a nasal cannula, 1.25 mg bisoprolol once a day, and 2.5 mg lisinopril once a day post delivery.

Conclusion:
The main symptoms of PPCM often resemble normal pregnancy or other pregnancy comorbid. Therefore, it is essential to have a high suspicion of cardiac comorbidities in patients with pregnancy to avoid postponed diagnosis.

Keywords: PPCM

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STRIKING A BALANCE: ANTITHROMBOTIC THERAPY IN THE SETTING OF CARDIO-CEREBRAL INFARCTION (CCI) A CASE REPORT

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Background:
Acute ischemic stroke (AIS) and Acute Coronary Syndrome (ACS) both share atherosclerosis as common vascular risk factors. Antithrombotic medications are frequently prescribed for these conditions to reduce the risk of recurrent thrombosis and subsequent damage. However, the use of antithrombotic can also increase the risk of bleeding in patients, necessitating the use of standardized scoring systems to assess each patient's risk and prevent complications.

Case illustration:
A 56-year-old female presented to the emergency room with sudden weakness on the right side of her body four hours before admission. Patients' blood pressure slightly increased (BP 144/70) with other vital sign within normal range. The patient has remarkable physical examination, with the exception of neurological deficits right hemiparesis and central type right cranialis and hypoglossal nerve paresis. CT Scan confirmed the diagnosis of ischemic stroke with NIHSS scoring calculation is 5 and the patient was treated with SAPT (Miniaspi). After four days of care, the patient complained of burning chest pain. ECG was performed and showed Sinus tachycardia (HR 110 bpm) and inverted T on I,III,aVF,aVL,v1-v6 lead. Troponin I level increased to 13.5 ng/ml. The patient was diagnosed as NSTEMI with TIMI score 3, GRACE score 97, and CRUSADE Score 44. After meticulous assessment of bleeding risk in patient, DAPT (Miniaspi and Clopidogrel) and Fondaparinux were administered. By the end of hospital stay, patient's symptom of weakness and chest pain had improved and no bleeding occurred.

Conclusion:
In this case report, we describe a patient with stroke who simultaneously developed ACS during treatment. The choice of antithrombotic regimen in patients with coronary artery disease and ischemic stroke requires attention due to the high risk of bleeding and ischemic events. Thus, an assessment of the risk of bleeding and ischemic events is necessary for each patient as part of the treatment plan.

Keywords: Acute Coronary Syndrome, Antithrombotic, Stroke

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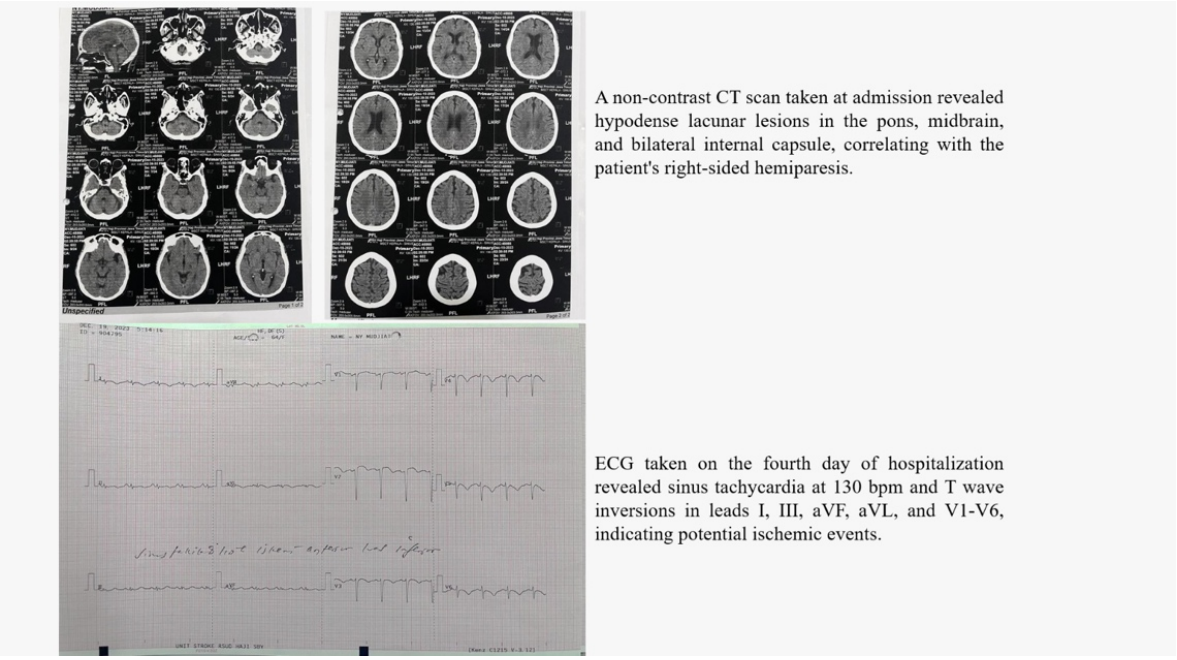

Figure 1. Patient's CT Scan and ECG showed concomitant Cardio Cerebral Infarction (CCI)

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ROLE OF CHEMOTHERAPEUTIC AGENT AS AN ETIOLOGICAL TREATMENT IN MANAGEMENT OF IVC TROMBUS RELATED TO SLE: A CASE REPORT

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Background:
Inferior vena cava (IVC) thrombosis is a condition that often goes unnoticed and associated with significant illness even death. It is estimated about 2.6% - 4.0% of patients with deep vein thrombosis (DVT) have IVC thrombosis. Systemic lupus erythematosus (SLE) is an autoimmune condition involving multiple organs with varied and heterogeneous clinical presentations with varying severity and clinical significance. Various data have shown that SLE is an independent factor of the occurrence of thrombosis, both venous and arterial. Cytotoxic agents such as cyclophosphamide are the main therapy in patients with SLE flare up. We are reporting the effect of cyclophosphamide as an etiological therapy accompanied by optimal anticoagulant therapy for thrombus management in IVC in patients with SLE.

Case illustration:
A 62-year-old female referred from private hospital with chief complaint abdominal pain for 1 week prior to admission. Abdominal pain is mainly felt at epigastric area, radiated to lower part of abdomen. The patient had a history of SLE, on medication with Mycophenolate mofetil and a history of DVT extremity inferior dextra for 1 month prior to admission with medication Rivaroxaban. Abdominal CT-scan examination on referrer hospital showing filling defect on IVC with impression diffuse thrombosis throughout IVC. Anticoagulation was given, catheter directed thrombolysis (CDT) was planned, and the patient was consulted to internal medicine for evaluation of SLE. The patient was diagnosed with diffuse DVT at IVC with severe SLE. Chemotherapeutic agents: Hydroxychloroquine and Mycophenolate mofetil was given due to SLE flare up. On CT evaluation intrahospital, dilated IVC was obtained, thrombus evaluation showing improvement with partial thrombus. Patient was stable, CDT was postponed and discharged. During control, CT scan showing thrombus was no longer appear. CT reevaluation 2 months after, showing no remaining thrombus was seen at IVC.

Conclusion:
Definitive therapy of thrombosis is with anticoagulants, besides underlying etiology management. Cyclophosphamide as an immunomodulatory agent, is an effective therapy to reduce the severity of and improve the prognosis of SLE patients. Optimization and combination therapy is essential to improve patient outcomes, morbidity to mortality.

Keywords: IVC Thrombus, Chemotherapeutic thrombus, Thrombus SLE

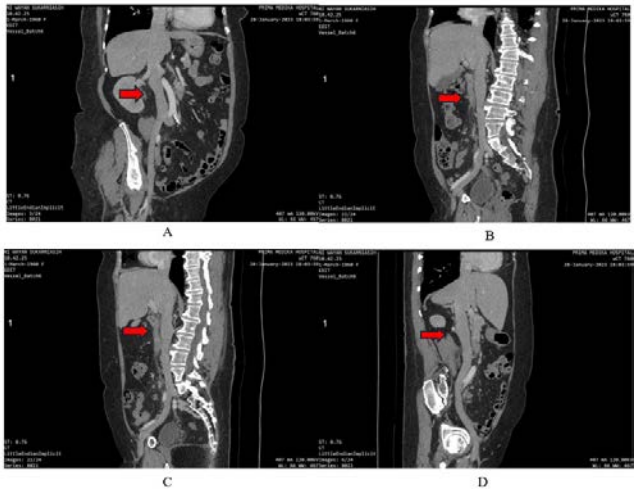

Figure 1. CT scan thorax showing filling defect along IVC consistent with thrombosis

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CARDIAC MANIFESTATIONS OF SYSTEMIC LUPUS ERYTHEMATOSUS: ROLE OF ECHOCARDIOGRAPHY

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Background:
Systemic Lupus Erythematosus (SLE) is associated with higher risk of cardiovascular disease. It may affect all structures of the heart. Most cases are asymptomatic, but the presence of cardiac symptoms is linked with poor prognosis. Echocardiography may help differentiate SLE from other causes of cardiac abnormalities. In this report, we presented a case series to highlight the role of echocardiography in identifying cardiac manifestations of SLE.

Case illustration:
The first case is a 12-year-old girl presented with symptoms of heart failure accompanied by polyarthralgia and constitutional symptoms. She had prior diagnosis of SLE and elevated inflammatory markers (ESR 101 mm/h, CRP 14 mg/dL). Her transthoracic echocardiography (TTE) showed massive pericardial effusion with normal cardiac function and valves. Medical management with steroid, furosemide, ramipril, bisoprolol and spironolactone failed to control her symptoms. She eventually underwent successful pericardiectomy. The second case is a 52-year-old woman who came to clinic with symptoms of heart failure (NYHA FC II), she had history of SLE for 8 years. Her TTE showed mild pericardial effusion, mild MR with thickened leaflets, and mild AR. Her symptoms were well-controlled with optimal medical therapy, corticosteroid and immunosuppressant. The third case is a 17-year-old girl referred to emergency unit with worsened dyspnea, fever, polyarthralgia, recurrent anaemia, and constitutional symptoms. Her TTE showed LV dilation, markedly reduced LV systolic function (LVEF 28%), moderate MR, and thick leaflets with small nodules at the tip of AML. She was initially diagnosed with rheumatic fever and infective endocarditis due to the presence of nodules in AML, however her blood culture was negative. She tested positive for ANA and antidsDNA, thus diagnosis of lupus myocarditis and valvulitis were made. She was given high-dose corticosteroid along with furosemide, ramipril and carvedilol.

Conclusion:
We presented three cases with different cardiac manifestations of SLE. Echocardiography has an important role in detecting structural abnormalities. Combined with comprehensive analysis of anamnesis, physical examination and various supporting data, echocardiography may help us to diagnose cardiac involvement in SLE.

Keywords: lupus valvulitis, lupus pericarditis, systemic lupus erythematosus, echocardiography, lupus myocarditis

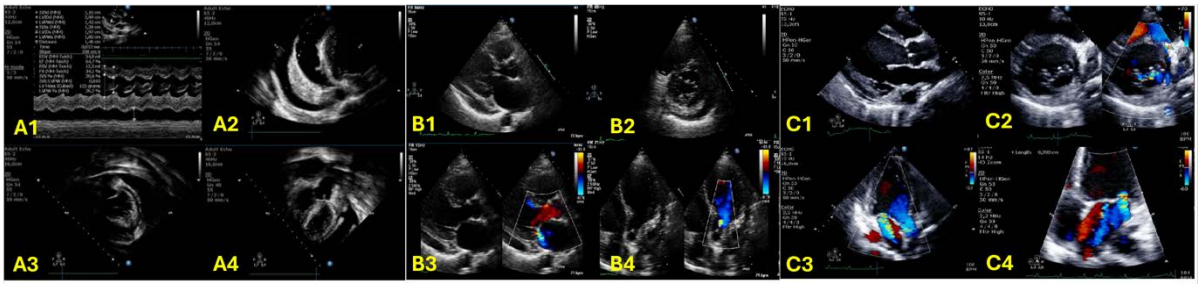

Figure 1. Transthoracic echocardiography showed three different cardiac manifestations in SLE: Fig A1-4 showed large pericardial effusions with normal cardiac functions and valves; Fig B1-4 showed mild pericardial effusion, thickened leaflets causing mild MR, and mild AR; Fig C1-4 showed LV dilation, markedly reduced LV systolic function (LVEF 28%), moderate MR, and thick leaflets with small nodules at the tip of AML

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INFECTION OR NON INFECTION? : DIAGNOSTIC CHALLENGING A RARE CASE ISCHEMIC HEPATITIS MIMICKING VIRAL HEPATITIS

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[1]RS Dr Bratanata Jambi

Background:
The heart is a pump that contract to support systemic metabolism with adequate perfusion. Heart dysfunction can diminish its function and cause tissue hypoxia. Including the liver, It turns to condition called Ischemic Hepatitis. Rapid identification affects prognosis of the patient. Meanwhile, This condition can mimic other condition and rarely happening that make lack of awareness to this condition.

Case illustration:
A 62-year-old female presented to the hospital feeling unwell with fever notably a week before. Patient was felt shortness of breath worsening on lying down and upper abdominal pain. Her medical history included uncontrolled Type 2 Diabetes Mellitus and Hypertension with no history of liver infection. The patient said that she did not take any drug now. On examination She was tachycardia 168 bpm and subfebrile temperature of 37,5 C. Her eyes turn yellow and abdominal examination revealed hepatomegaly. ECG showed sinus tachycardia and chest x-ray showed cardiomegaly and clear lungs.Supported by laboratory result, it showed leukocytosis of 13.300 and liver function test extremely high as AST and ALT level were 6400 U/l and 3420 U/l, respectively. But, on serology testing showed negative for hepatitis A, B, C and HIV. As mismatch on clinical data, The physician re-check and revealed crackles in basal of the lungs. Then, echocardiography was showed global hypokinetic movement with 28% EF. It turned out that the patient had Ischemic Hepatitis secondary Heart Failure which was clinically similar to Viral Hepatitis.The patient was admitted to the ICU and treated with continuous infusion of furosemide 5 mg/hour, 0,5 mg digoxin intravenous and perfusion stabilization. The patient was responsive to therapy on day 3 and liver function follow up showed significant declining. Then two weeks after hospitalization, The patient was sent home in a stable condition.

Conclusion:
Holisitic examination was important to get clue about patient's condition including organ damage. By recognizing it, treatment with a focus on correcting organ hypoxia was given immediately. So, Organ failure that is difficult to treat prevented and the patient gets better prognosis.

Keywords: ischemic hepatitis, viral hepatitis, heart failure

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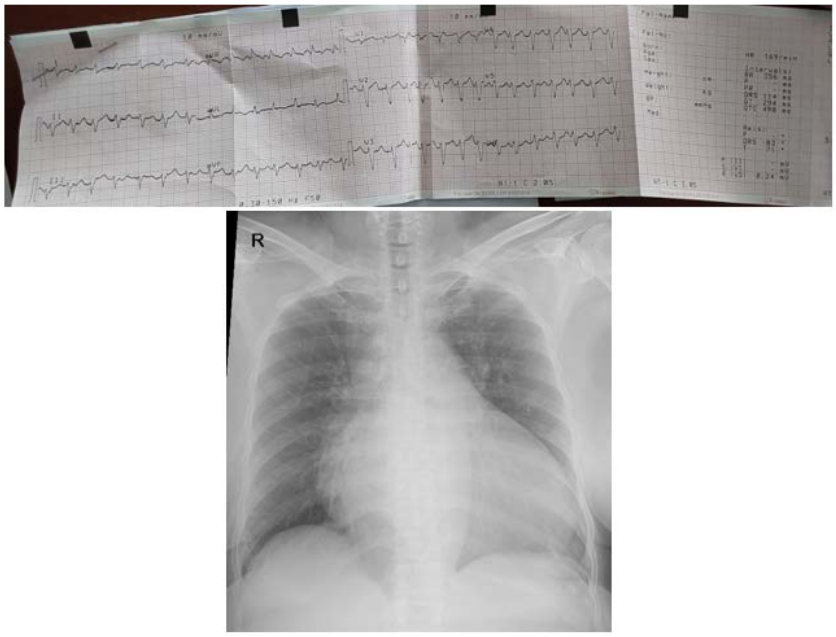

Figure 1. ECG demonstrating sinus tachycardia (Above), chest x-ray showing cardiomegaly and clear lungs in patient (below)

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WELLEN SYNDROME IN ANOMALY ORIGIN OF LEFT ANTERIOR DESCENDING ARTERY, WHERE IS THE CULPRIT?

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Background:
Acute coronary syndrome (ACS) and a coronary artery abnormality together create a rare clinical situation that needs to be carefully considered and managed. Coronary abnormalities might present diagnostic and treatment problems in the context of ACS. Even with the assistance of other testing, the presentation of symptoms associated with ACS may be unusual or deceptive.

Case illustration:
A 60-year-old man was rushed into our emergency department with cardiac chest pain lasting 6 hours. He presented with stable hemodynamics, relieved with nitrates. His past medical history included hypertension and a cerebral infarction six years prior. The ECG revealed sinus rhythm with biphasic T-wave inversion in V2-V4 (Wellen type A), along with a normal ejection fraction observed during bedside echo. Initial Hs-Troponin levels were very low (3.3 mmol/L, rule-in: ≥64). Non-ST elevation (NSTE) ACS with proximal left anterior descending artery (LAD) occlusion was suggested. Unluckily, the angiography could not visualise the left coronary ostia and failed to engage the right coronary ostia, even after several trials. Non-invasive Cardiac Computed Tomography Angiography (CCTA) was performed after ensuring normal renal function. Absence of the left main coronary artery, and the ostia was confirmed with critical stenosis in the proximal LAD originating from the right coronary ostia. The patient then scheduled for a team-based approach to redo the coronary intervention thereafter.

Conclusion:
The coexistence of an anomaly in the origin of the coronary artery and ACS can be a challenging case to face. The help of cardiovascular imaging like CCTA could provide better visualisation of cardiac anatomy, guiding interventional cardiologists to perform the optimal of coronary intervention.

Keywords: acute coronary syndrome, cardiovascular imaging, anomaly coronary artery, wellen syndrome

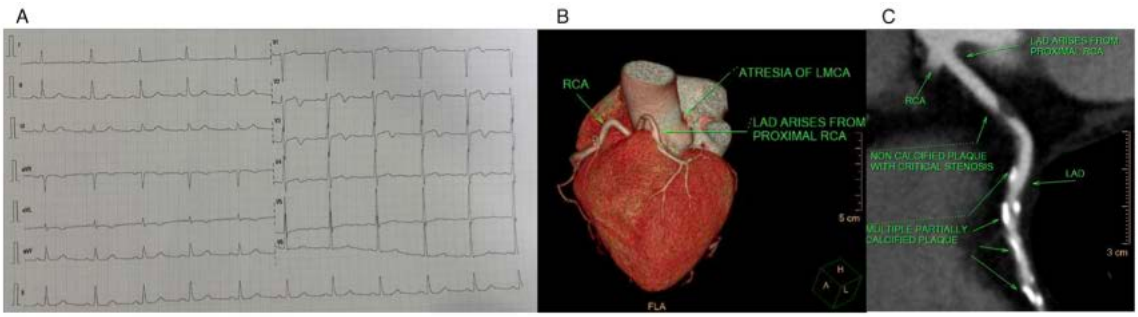

Figure 1. A. ECG shown biphasic T Wave inversion in lead V1-V3, suggest Wellen type A. **B.** CCTA shown atresia of LMCA with anomaly origin of proximal LAD arising from ostium of RCC. **C.** Critical stenosis at proximal of LAD

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A CASE REPORT OF A FIGHTER PILOT WITH ASYMPTOMATIC POSITIVE ISCHAEMIC RESPONSE ON EXERCISE STRESS TEST: IS IT A BIG DEAL?

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Background:
Cardiovascular diseases are the most common cause of flying disqualification globally. Fighter pilots often operate within a demanding physiological environment, that potentially includes exposure to sustained acceleration (G-force) and hypoxia. Aeromedical recommendation for fighter pilots who have coronary artery disease (CAD) should be considered properly and wisely due to high G-force exposure in them which may lead to fatal complications.

Case illustration:
A 45-years-old male fighter pilot was performed for routine medical check-up. He had no hypertension or dyslipidemia. He is an active smoker. The physical examination was unremarkable. Blood test and chest x-ray were between normal range. Due to asymptomatic positive ischaemic response during the exercise stress test (EST), coronary angiography (CAG) was performed and it showed 70-80 % at mid left anterior descending (LAD). Then fractional flow reserve (FFR) was also performed, we found mid stenosis 50% (eccentric plaque) at LAD and functional test FFR using adenosine was 1.0 (> 0.8). It was evaluated as coronary artery disease (CAD) non-significant. Then, he was released to fly and treated with antihypertensive and statin. Several months later, he conducted for EST and the result was normal. Abnormal finding in cardiovascular examinations among aircrew needs to be distinguished with general population due to the consideration for continuing flight duties of aircrew with known CAD that requires a detailed aeromedical review by a cardiologist with aviation medicine experience. Fighter pilot is a special population who work in high altitude and highly manoeuvrable aircraft which lead them to exposure of G-force and hypoxia. Those condition will affect cardiovascular system by stimulating sympathetic tone higher than before at a certain altitude. Sympathetic tone causes an increase in systemic and pulmonary arterial pressure. This will increase left ventricular diastolic pressure and ultimately causes symptoms and even leads to fatal complications.

Conclusion:
Established CAD of any degree in aircrew especially fighter pilot needs to be evaluated as to make aeromedical recommendation, whether it is fit for flying or not.

Keywords: exercise stress test, coronary artery disease, fighter pilot, fractional flow reserve

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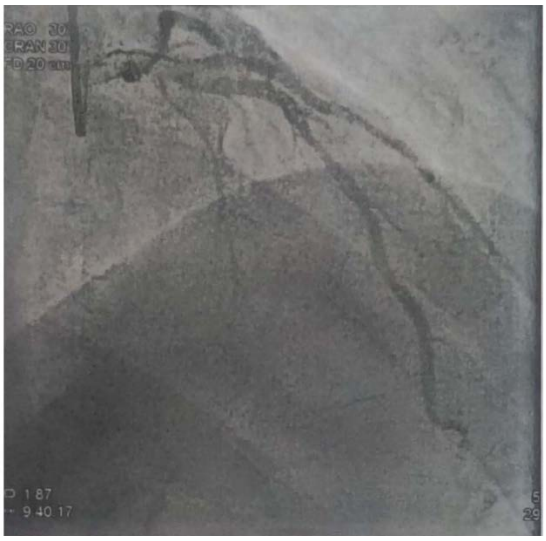

Figure 1. Angiogram image showing stenosis of the mid LAD

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INVERTED LEFT MAIN BIFURCATION STENTING IN HARD-ROCK LESION

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Background:
Coronary angiography may underestimate calcified lesion. In heavy calcified lesion case, lesion preparation is key of success. Inverted Left Main Provisional Stenting is reasonable for Medina 0-0-1 lesion.

Case illustration:
A 76 year old male presented with unstable angina and history of previous percutaneous coronary intervention (PCI) in left anterior descending artery (LAD) and left circumflex artery (LCX) 3 years ago. The patient co-morbidities were hypertension and dyslipidemia. The laboratory result showed Creatinine 1.4 mg/dl (eGFR 52 ml/min/1.73 m2). Electrocardiogram showed LVH strain pattern. Echocardiogram showed LVEF 62% with mildly hypokinetic anterolateral and posterior wall. Coronary angiography showed 90% stenosis in ostial LCX (Medina 0,0,1) and patent stent in distal LCX, mild ISR in LAD, small and non dominant right coronary artery (RCA). PCI was performed by Right radial access with slender sheath 6/7 F. A GC Heartrail BL 3.5/7 F was used to engage left main (LM) ostium. LCX lesion was crossed with a 0.014" Runthrough hypercoat guide wire. Wolverine 2.5/10 mm cutting balloon was deployed up to 20 Atm, but unable to crack the calcified lesion. IVUS confirmed 3 quadrant calcified lesion with calcified nodule. 1.75 Rotaburr was used to ablate the calcified lesion for 7 runs, 164.000-182.000 rpm, followed by predilatation with NC Euphora 4.0/15 mm at 16-18 Atm from LCX to LM. DES Resolute Integrity 4.0/30 mm was delivered to cover LM to proximal LCX segment, inflate at 11 Atm. POT was performed with a 5.0/10 mm NC Thonic at 12 Atm. Post dilatation with NC Euphora 4.0/15 mm was performed in proximal LCX up to 18 Atm. IVUS Volcano Refinity was used for post procedure evaluation, showed the stent was well apposed and well deployed, without medial edge stent dissection. Finally PCI using Inverted Left Main Provisional Stenting technique from LCX to LM was successfully done after ablation the calcified lesion with 1.75 Rotaburr.

Conclusion:
For single stent strategy, Final kissing balloon is not mandatory, unless unsatisfactory angiographic and functional result. After good lesion preparation and debulking, guide wire or jailed balloon protection in main branch during stenting is not necessary.

Keywords: Provisional Strategy, Left Main Disease

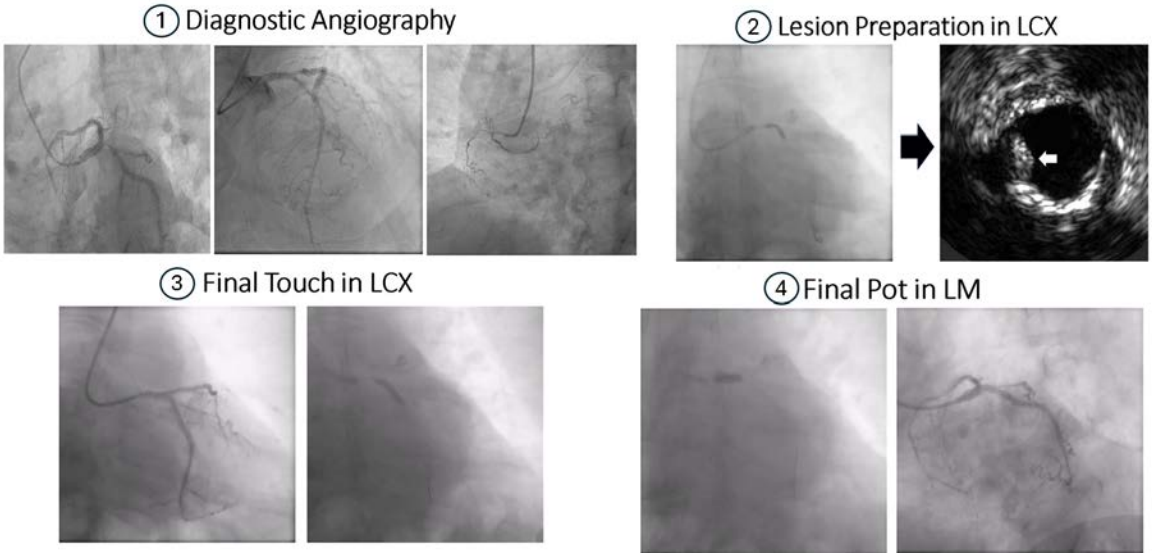

Figure 1. 1) Diagnostic angiography showed 90% stenosis in ostial LCX (Medina 0,0,1) and patent stented segment in distal LCX, mild ISR in LAD, small and non dominant RCA. 2) Lesion preparation in LCX with Wolverin 2.5/15mm at 20 Atm with dog-bone appearance followed by IVUS that showed 3 quadrant of calcified lesion with calcified nodule. 3) Final touch in LCX with DES Resolute Integrity 4.0/30mm deployed at 12 Atm

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followed by postdilatation with NC Euphora 4.0/15mm at 14 Atm. 4) Final POT in LM with NC Thonic 5.0/10mm 18 atm followed by Final coronary angiography that showed TIMI 3 flow to distal LCX.

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MANAGEMENT OF UNREPAIRED ASD II WITH PAH IN PREGNANCY: CASE REPORT OF A SUCCESSFUL OUTCOME

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Background:
Atrial septal defect (ASD), particularly the secundum type (ASD II), is a common congenital heart anomaly. Larger ASDs can lead to pulmonary arterial hypertension (PAH), a life-threatening condition. Pregnancy in patients with PAH, classified as modified WHO grade IV, poses an extremely high risk of maternal mortality or severe morbidity. Despite the contraindication for pregnancy, some patients may refuse termination. We present a case of a sustained pregnancy in ASD II with PAH, demonstrating positive outcomes for both mother and baby through effective interdisciplinary management.

Case illustration:
A 26-year-old primigravida at 30 weeks gestation with a history of ASD, came with complaints of shortness of breath. She reported intermittent dyspnea since the 20th week of her pregnancy, which had worsened the day before admission. Diagnosed with PAH from an uncorrected ASD, her oxygen saturation was 83%, with physical findings including jugular venous distention, a widely fixed split 2nd heart sound (S2), a grade 3/6 systolic murmur at the LLSB, cardiomegaly, and lung base rales. An ECG showed sinus tachycardia with PVC bigeminy, which converted to sinus rhythm after lidocaine injection. Echocardiography revealed a 2.5 cm bidirectional ASD, LVEF of 61%, TAPSE of 2.5 cm, and severe tricuspid regurgitation. The patient refused to terminate her pregnancy and decided to continue the pregnancy. During admission to improve the patient's condition and sustain the pregnancy, she received oxygen supplementation as well as iloprost nebulizer, sildenafil, furosemide, and other supportive medications including dexamethasone and neuroprotector drugs. With close monitoring and multidisciplinary support, she continued her pregnancy to 36 weeks. A successful cesarean section was performed, and both mother and baby were discharged without complications on postoperative day five.

Conclusion:
Terminating the pregnancy early on is the safer choice for pregnant women with PAH. Nevertheless, when the decision is made to continue the pregnancy, a specialized multidisciplinary team is assembled to provide comprehensive support to the patient. Daily clinical evaluation, close monitoring and follow-up, and well-planned medical regimen are crucial for achieving a successful delivery.

Keywords: Pregnancy, ASD, CHD, PAH

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BREAKING THE STEREOTYPE: STEMI IN A YOUNG WOMAN WITH A SMOKING HISTORY

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[1]UNHAS

Background:
Acute coronary syndrome is the leading cause of death worldwide. According to World Health Organization (WHO) data, acute coronary syndrome accounts for 12% of disability worldwide each year. Acute coronary syndrome is generally found more in the male gender than the female gender. This is because traditional risk factors for coronary heart disease, such as smoking, obesity, hypertension, and dyslipidemia, are more common in the male gender group.

Case illustration:
A 34-year-old woman came to the emergency room of Cardiac Center Dr. Wahidin Sudirohusodo Hospital with complaints of chest pain 62 hours ago. Duration of pain >20 minutes, accompanied by cold sweat and nausea. Pain scale: 8/10 (NRS). History of hypertension since 10 years ago. Smoking history since 15 years ago, as much as half a pack per day. On physical examination, vital signs are within normal limits. Results ECG examination in the emergency room: pathological Q in leads II, III, avF, and ST segment elevation in leads V1-V4, Right posterior ECG examination obtained pathological Q in leads II, III, avF, and ST segment elevation in leads V2R-V4R. Echocardiography examination of left ventricular systolic function decreased with an ejection fraction (EF) of 39% (BIPLANE) and grade I diastolic dysfunction. A coronary angiography examination accessed through the dextra femoral artery found the results of coronary artery disease 1 vessel disease. A percutaneous coronary intervention was performed to install 1 DES stent in LAD. On the 4th day of treatment, the patient's general condition was improved.

Conclusion:
High estrogen levels in young women have a cardioprotective effect so cases of Coronary Heart Disease are rare. However, in young women smoking leads to higher nicotine metabolism and counteracts the cardioprotective effect of estrogen. Therefore, despite smoking at the same intensity and frequency, the risk of coronary heart disease will be higher in women than men.

Keywords: Coronary Heart Disease, Smoking, Young Woman

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CANCER THERAPY-RELATED CARDIAC DYSFUNCTION A SERIAL CASE REPORT OF CARDIO-ONCOLOGY CLINIC REGISTRY

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[1]Rumah sakit umum Pusat Mohammad hoesin palembang

Background:
Cancer therapy-related cardiac dysfunction (CTRCD) is defined as left ventricular ejection fraction (LVEF) decrease more than 10% to a value below the lower limit of normal or relative reduction in global longitudinal strain (GLS) >15% from baseline after cancer treatment. While cancer patient survival rate increased because of the recent development of cancer therapy, this also increased the incidence of CTRCD.

Case illustration:
Following the opening of cardio-oncology clinic in Mohammad Hoesin General Hospital, we had 5 patients presented with CTRCD under our registry data. The patient #1 was a 51-year-old woman who came to our institution for an echocardiography examination prior to chemotherapy, presented with orthopnoea after administration of the anti-human epidermal growth factor receptor 2 (HER-2) antibody, trastuzumab, for HER-2-positive breast cancer. Transthoracic echocardiography (TTE) showed a reduced LVEF of 24% with a significant reduction in GLS compared to her baseline echocardiography result before anti-HER-2 targeted therapy. Her cummulative doses are 1150 mg of paclitaxel and 1600 mg of herzumab.
The patient #2, #3, #4 and #5 were all the patients with preserved ejection fraction, but reduced GLS upon the 2nd TTE examinations, with the exception of the patient #3 that developed CTRCD during the 3rd examination. The patient #2, #4 and #5 were all have reduced 2nd GLS examination more than 15% than their respective baseline (35%, 34%, and 17%, consecutively), while the #3 had a 42% reduction on the 3rd TTE examination. All of them were undergoing anthracycline (4AC+4T) based therapy and overall asymptomatic CTRCD. They were cathegorized as mild asymptomatic patients according to HFA-ICOS risk stratification and was allowed to continue their chemotherapy regiments.

Conclusion:
In the management of CTRCD, includes pretreatment screening, cardiotoxicity monitoring, follow-up after anti-cancer agents, and evaluation of the effectiveness of the therapy. Tyrosine kinase receptor erbB2 or HER-2 signaling in the heart is an essential factor for dilated cardiomyopathy prevention, thus HER-2 inhibitor chemotherapy could cause cardiotoxicity in the heart. Cardiotoxicity caused by herzumab was not dose dependent, so that a more frequent follow-up is required for patients undergoing HER-2 Inhibitor chemoteraphy regiments compared to patients with anthracycline therapy.

Keywords: ,HER-2 Inhibitor, GLS, Echocardiography, CTRCD, Anthracycline

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TORRENTIAL TRICUSPID REGURGITATION IN TYPE 3 HYPERTENSION PULMONAL: ONE STEP TO MEET THE GRIM REAPER

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Background:
Torrential tricuspid regurgitation in type 3 pulmonary hypertension (PH) increases mortality risk in individuals with acute heart failure. Moderate-severe tricuspid regurgitation is linked to reduced exercise capacity and survival rates in pulmonary arterial hypertension (PAH). Pulmonary hypertension can lead to expansion of the right ventricle and tricuspid annulus, worsening tricuspid regurgitation. Understanding its underlying mechanisms and exploring potential therapeutic approaches is crucial as prolonged pulmonary hypertension can lead to impaired function and tricuspid valve leakage.

Case illustration:
A 41-year-old woman with persistent shortness of breath, orthopnea, presented to the ED. She had been diagnosed with congestive heart failure, tuberculosis on antituberculosis therapy for three months. Physical examination revealed compos mentis, blood pressure 69/48 mmHg, heart rate 90 bpm, respiratory rate 32 bpm, intercostal retraction (+), SpO2 56% on room air (100% with NRM 15 L/min), amphoric sound (+) at both lung apices, hardened S2 sound, gallop (+), holosystolic murmur at left lower sternal border increased on inspiration, pitting edema on extremities (+/-). The ECG showed sinus rhythm, right axis deviation, RVH, incomplete RBBB. Echocardiography results showed RA and RV dilatation, severe tricuspid regurgitation with torrential, decreased RV contractility, the patient assessed with right heart failure in type III pulmonary hypertension. On the third day the patient was gasping, indicating impending respiratory failure. Blood gas analysis showed partial compensated respiratory acidosis, and the patient was intubated. On the fifth day, haematuria was found, and assessed for acute interstitial nephritis and DIC. On the seventh day, the patient developed respiratory failure and cardiac arrest, and was pronounced dead.

Conclusion:
Torrential tricuspid regurgitation (TR) causes hemodynamic changes in the right ventricle, remodelling of the right side of the heart, and an increased risk of rehospitalisation and mortality. The clinical course of right heart failure with significant tricuspid regurgitation might be further complicated by right ventricular dysfunction and hemodynamic instability. This condition is typically made worse by pulmonary hypertension. A self-sustaining loop of increasing heart failure, increased morbidity, and enhanced death rates results from the co-occurrence of several illnesses.

Keywords: mortality, right heart failure, torrential tricuspid, pulmonary hypertension

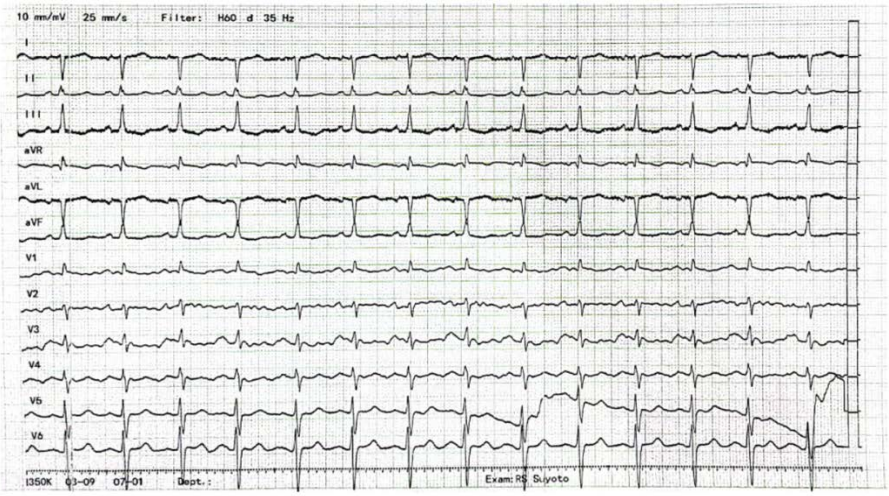

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Figure I. The ECG showed sinus rhythm, HR 88 beats/min, right axis deviation, RVH, incomplete RBBB

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ANTERIOR CHEST ECCHYMOSIS IN AN UNCONTROLLED HYPERTENSIVE PATIENT : COULD IT BE AN ATYPICAL PRESENTATION OF AORTIC DISSECTION?

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Background:
Aortic dissection (AD) is a rare yet critical condition, typically presenting as chest or back pain. The occurrence of chest ecchymosis as a manifestation of AD is scarcely documented. We present a case of suspected AD with chest ecchymosis and the management challenges in a rural setting.

Case illustration:
A 58-year-old man with uncontrolled hypertension presented with chest pain radiating to his back. Physical examination revealed BP of 194/118 mmHg, a grade 3/6 decrescendo blowing diastolic murmur best heard at lower left sternal border, and marked ecchymosis on the anterior chest wall. The patient was anemic with Hb of 7.9 g/dL. A widened mediastinum was seen on chest X-ray. Echocardiography revealed aortic regurgitation and dilatation of aortic root with intimal flap, suggesting Stanford type A AD. Referral for CT aortography was planned with blood pressure and heart rate control. Hypertension is a known risk factor for AD due to increased aortic wall stress. Aortic bleeding into the mediastinum might manifest as subcutaneous bleeding in the front of the neck due to connections between the retropharyngeal and parapharyngeal spaces. The ecchymosis also seemed to be caused by dissection of the arteries supplying the chest wall.

Conclusion:
Diagnosis of AD is challenging due to varied presentations. Chest wall ecchymosis along with diastolic murmur, widened pulse pressure and mediastinum should raise suspicion of AD.

Keywords: rural area, aortic dissection, ecchymosis

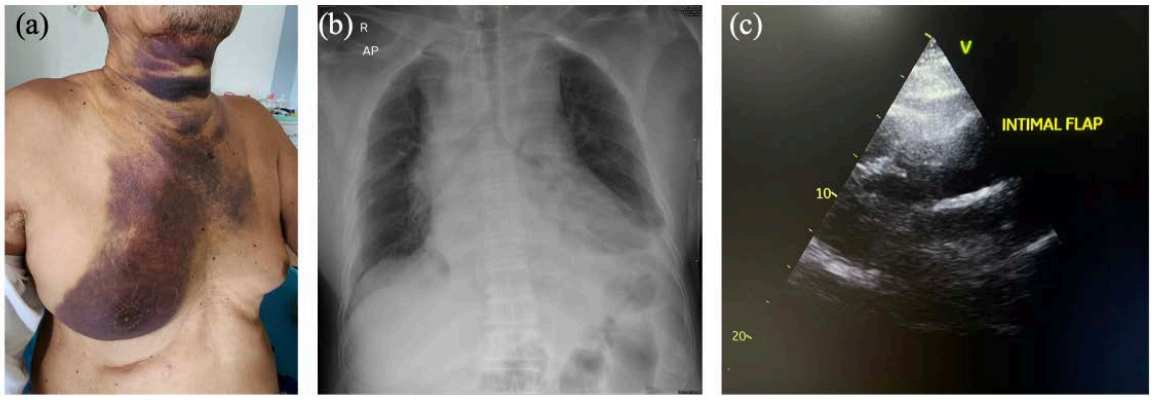

Figure 1. (a) Physical findings of anterior chest ecchymosis extending to the neck area. **(b)** Chest x-ray showed mediastinal widening and double aortic contour, as it is suggestive of aortic dissection. **(c)** Transthoracic echocardiography showed an intimal flap in the aortic root.

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IDENTIFYING HIGH BLEEDING RISK IN PATIENT UNDERGOING PCI : WHAT SHOULD WE DO?

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Background:
Treatments for patients with coronary artery disease has evolved in the last few decades with more advanced reperfusion strategies, including percutaneous coronary intervention (PCI) and more potent antiplatelets. However, among patients undergoing PCI, it is estimated more than 15% are at high bleeding risk and affect its prognostic implications. Recently, the Academic Research Consortium for High Bleeding Risk (ARC-HBR) has released the definition of high bleeding risk with list of criteria to minimize bleeding complications and outcome for patients undergoing PCI. This article reports cases of patients with high bleeding risk and their management of the patient before, during and after the procedure of PCI.

Case illustration:
This study reports 3 cases of high bleeding risk patient based on definition of ARC-HBR (2 cases of ACS and 1 case of CCS). We prepared the patients before the procedure including bleeding stratification and appropriate criteria for revascularization. Procedure was done with radial access, recommended anticoagulation dose, and latest stent generation implantation. After the procedure we considered choice of antiplatelet and de-escalation plan of double antiplatelet therapy (DAPT) and added proton pump inhibitor administration for the patient. All patients were discharged without bleeding complication and followed up in the outpatient clinic for routine antiplatelet therapy.

Conclusion:
Patients undergoing PCI with high risk of bleeding has prognostic implications after the procedure. Identifying high bleeding risk patients and appropriate strategies before, during and after the procedure is important to a better clinical outcome.

Keywords: Acute Coronary Syndrome, High bleeding Risk, Percutaneous Coronary Intervention

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THE SMILING DEATH: A CASE REPORT OF VENTRICULAR ARRHYTHMIA DUE TO CRUSH SYNDROME

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[1]Rumah Sakit Muhammadiyah Lamongan

Background:
Crush syndrome is a systemic complication resulting from extensive muscular cell destruction following severe crush injury. The concurrent pathophysiological processes in crush syndrome can trigger life-threatening complications with rapid onset, a phenomenon known as "smiling death". This case report presents a patient with severe crush injury who suddenly developed ventricular tachycardia and cardiac arrest.

Case illustration:
A 52-year-old male was brought to our hospital 10 minutes after experiencing a major traffic collision. During the primary survey, we observed a severe crush injury to the left lower limb, with extensive damage to its soft tissue, bone, and vasculature. Initial examination revealed no compromises in ABCD. The patient was initially alert, oriented, and "smiling", with vital signs as follows: BP 113/63mmHg, HR 96bpm, RR 18x/min, temperature 36°C, CRT <2 seconds, and warm, dry, but slightly pale extremities.The patient's medical history was hypertension and a previous PCI with stent placement in 2019. We promptly initiated management, including fluid resuscitation, hemorrhage control, antibiotic prophylaxis, and anti-tetanus administration. An initial ECG performed 5 minutes after admission showed a sinus rhythm with aberrancy of wide QRS complexes across all leads, high-lateral and anterior ST-elevation, and R-on-T phenomenon in the last part of the ECG. However, approximately 20 minutes later, the cardiac monitor displayed a ventricular tachycardia rhythm, which rapidly progressing to cardiac arrest. The rhythm remained unshockable throughout the life support attempt, and the patient was pronounced dead after 30 minutes of CPR.

Conclusion:
This case emphasizes the importance of a holistic understanding and prompt, appropriate management in treating crush injuries. The onset of malignant arrhythmias within a very short period (<30 minutes) post-trauma in this patient can be attributed to the complex interaction of various arrhythmogenic factors, including: sympathetic nervous system overactivation; hyperkalemia, hypocalcemia, and acute kidney injury (AKI) due to rhabdomyolysis, acidosis with myocytes ischemia caused by AKI and shock (distributive, hypovolemic, hemorrhagic), and lastly; suspected acute type 2 STEMI. This cascade of pathological processes synergistically alters cardiac electrophysiology through diverse mechanisms, including changes in transmembrane ion gradients, QT interval prolongation leading to early afterdepolarizations, increased automaticity, and reentry phenomena.

Keywords: cardiac arrest, crush syndrome, ventricular tachycardia

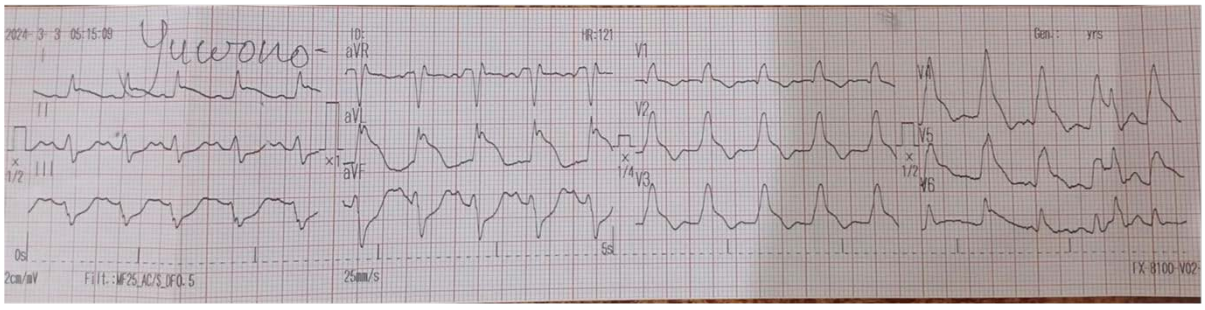

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Figure 1. ECG recorded 10 minutes after the patient's arrival (top), and monitor ECG recorded 30 minutes after the first ECG (bottom)

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INSIGHT FOR REVASCULARIZATION FOR CULPRIT LESION IN EARLY ONSET OF STEMI; THROUGH INTRACORONARY FIBRINOLYTIC

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Background:
Cardiovascular disease (CVD) is the most common cause of mortality and morbidity worldwide with its most common first clinical manifestation is Acute coronary syndrome (ACS). Immediate revascularization in STEMI patients is a must with respect to time to restore blood flow in the IRA (infarct-related artery), with PCI being the cornerstone that has endured to date. Although PCI can meet some obstacle like high burden thrombus. Meanwhile fibrinolytic agent can destroy thrombus if given under certain onset, preferably early as possible. Thus, option of using fibrinolytic agent, while still has no stronghold in current guidelines, can be option when high burden thrombus is encountered in PCI

Case illustration:
A 52 years old male patient was admitted to the hospital with chief complaint of typical chest pain since 6 hours ago. From ECG and cardiac enzymes patient was diagnosed STEMI Inferior Wall and RV Infarction. The patient underwent pPCI, but had difficulty due large thrombus that persisted even after balloon predilatation. It was decided to do intracoronary fibrinolytic with alteplase 2 mg. The results was TIMI Flow 3 with disappearance of thrombus. Since revascularization was achieved, deferred stenting was decided with heparinization for 5 days. At next angiography, the IRA flow was still good and no thrombus seen. The stent was successfully planted without complications, and the patient was discharged home in good condition.

Conclusion:
The intracoronary fibrinolytic in this case gives satisfactory results. Our suggestion is, this successful fibrinolytic is due to early onset of STEMI, under 24 hours where the thrombus is still “fresh” with loose fibrins and are sensitive to fibrinolytic agents, in addition to the fact that the agent is given directly to the lesion that ensure all of the agents reach the lesion. Thus, intracoronary fibrinolytic can be adjuvant in PCI with high burden thrombus and early STEMI.

Keywords: percutaneous coronary intervention, STEMI, early onset, intracoronary fibrinolytic

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APPROACH FOR ATRIAL FIBRILLATION WITH SLOW VENTRICULAR RESPONSE IN LIMITED-RESOURCE SETTING: A CASE REPORT

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Background:
Atrial fibrillation with slow ventricular response(AFSVR) is an arrhythmia with uncoordinated atrial electrical activation with ventricular rate <60 bpm. AFSVR may lead to intracardiac hemodynamic changes and thrombus formation. AFSVR presents unique challenges in management, especially in settings with limited resources.

Case illustration:
A 63-year-old female patient presented to the emergency room with a chief complaint of left-sided chest pain that started 10 hours prior. Upon examination, blood pressure(BP) was 181/77mmHg. Initial ECG revealed AF and an anterior old myocardial infarction(OMI). The troponin T level was <50. The patient was diagnosed with unstable angina pectoris and urgent hypertension. During inpatient care; subcutaneous fondaparinux, intravenous(IV) nitroglycerin drip, dual antiplatelet therapy(DAPT), IV furosemide, ramipril, and statin were administered. On the second day, the patient reported hematuria. Consequently; fondaparinux, DAPT, and nitroglycerine were discontinued. By the third day, she complained of continued hematuria and feeling agitated. Upon examination, the BP was 84/52mmHg with a heart rate(HR) of 42bpm. An ECG revealed atrial AFSVR, a superior axis, and an anteroseptal OMI. A crystalloid bolus of 200cc was administered, resulting in an increase in BP to 106/60mmHg and HR to 49bpm. An IV dobutamine drip was initiated to raise BP and HR. With dobutamine at 6 mcg/kg/min, BP was 81/47mmHg with HR of 42bpm. Subsequently, IV dobutamine, IV atropine sulfate, IV tranexamic acid, and IV vitamin K were administered. Despite these interventions, BP and HR remained low at 82/60mmHg and 61bpm. Because of that, the patient was referred to a tertiary hospital for pacemaker placement.

Conclusion:
This case underscores the challenges in managing patients with complex cardiovascular conditions. The progression to significant bradycardia and hypotension required aggressive intervention with the initial use of crystalloids, followed by inotropic support with dobutamine and atropine. Despite our efforts, the patient's persistent hemodynamic instability necessitated transfer to a tertiary centre for pacemaker placement.

Keywords: Myocardial Infarction, Atrial Fibrillation Slow Ventricular Response, Pacemaker

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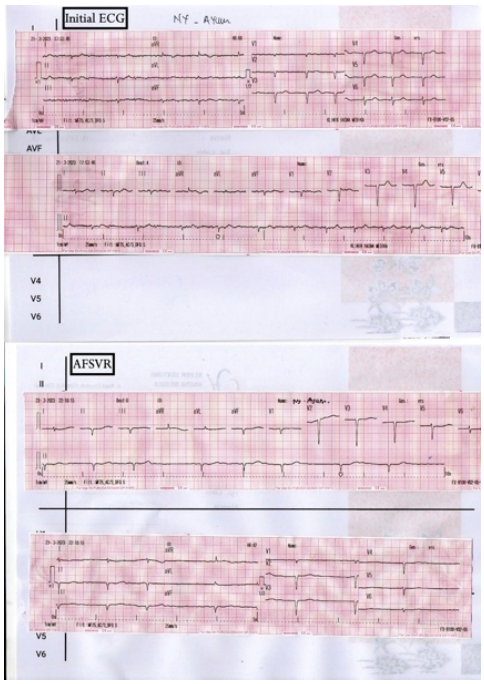

Figure 1. The first ECG is the initial ECG and the second ECG is atrial fibrillation wih slow ventricular response (on the third day of admission)

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EXERCISE ELECTROCARDIOGRAPHIC STRESS TEST IN DETECTING CORONARY MICROVASCULAR DYSFUNCTION: A CASE REPORT

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Background:
When evaluating patients with new-onset angina, electrocardiographic stress testing is a common, non-invasive, and affordable functional test. In many clinical settings, the prevalence of coronarymicrovascular dysfunction (CMD) is higher than previously believed, and its presence is linked to worseclinical outcomes, particularly when myocardial ischemia or nonsignificant coronary artery disease(CAD) are present. This case report describes a patient who showed a positive ischemia response on anexercise stress test but had a normal coronary arteries result when a coronary angiography wasconducted.

Case illustration:
A 19-year-old male came to the cardiovascular clinic with complaints of palpitations, fatigue, and chestdiscomfort during activities. Smoking, asthma, and other respiratory histories were denied. Vital signs inthe clinic were within normal limits. A type B Wolff-Parkinson-White syndrome from the anteroseptalwas discovered during the patient's ECG evaluation and 24-hour Holter monitoring. EPS and 3Dablation were performed successfully. After the procedures, the patient underwent a stress test toevaluate his exercise capacity. The results revealed ST depression in the inferior and lower lateral leads.The patient then underwent a coronary angiography, which showed normal coronary arteries.

Conclusion:
CMD is recognized as the cause of positive stress tests that indicate ischaemia without obstructive coronary arterial disease. Near-normal angiograms may be seen in patients with diffuse coronary atherosclerosis. On angiography, diffuse coronary atherosclerosis without localized stenosis results in a graded, continuous pressure fall, which contributes to myocardial ischaemia. A thorough history-taking and physical examination, followed by appropriate supportive examinations, can help clinicians diagnose CMD. Exercise stress testing should remain the primary method for clinicians to detect CMD.

Keywords: coronary artery disease, coronary microvascular dysfunction, electrocardiographic stress test

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A COMPLEX RARE CASE OF ATRIAL SEPTAL DEFECT: UNRAVELING THE MYSTERY OF VARIABLE AV BLOCK CONDUCTION IN ADULTHOOD

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Background:
Variable atrioventricular (AV) block conduction in Atrial Septal Defect (ASD) and pulmonary hypertension (PH) is a rare phenomenon, highlighting the need for early detection and comprehensive management.

Case illustration:
A 57-year-old male was referred to RSUD Dr. Soetomo Surabaya from Gresik with right-sided chest pain, cold sweats, near-fainting episodes, and dyspnea. He had a history of fainting seven years ago but did not seek further treatment.In the Soetomo ER, the patient was in fair condition with a blood pressure of 136/61 mmHg without support, a heart rate of 59 bpm, a respiratory rate of 20 breaths per minute, an SpO2 of 95% on room air, and systolic murmur at the left parasternal line in the second intercostal space, along with a wide fixed splitting of S2, indicating an ASD.The ECG at the referring hospital showed a total AV block (TAVB) with a ventricular escape rhythm of 24 bpm (Figure-A). At Soetomo, the ECG showed TAVB with a junctional escape rhythm of 59 bpm (Figure-B). After 1 mg IV injection of Sulfas Atropin, the ECG revealed a sinus rhythm of 75 bpm with first-degree AV block (Figure-C).Laboratory results indicated hypokalemia (3.4 mEq/L), which improved to 4.6 mEq/L, and a negative Hs-troponin. The chest X-ray showed cardiomegaly with an inverted comma sign indicative of PH (Figure-D). Echocardiography with a bubble test confirmed an ASD Secundum with left-to-right shunt flow (Figure-E).The patient was diagnosed with variable AV block conduction, ASD Secundum with left-to-right shunt, and a high probability of PH. Treatment involved continuous intravenous infusion of Dopamine at 5 mcg/kg/min during hospitalization and Revatio 20 mg taken three times daily.He was discharged on the fourth day with no complaints and his ECG showed first-degree AV block with sinus bradycardia at 57 bpm and a Crochetage sign in leads II and V2. He was scheduled to visit the cardiology clinic to consider ASD closure, with Revatio as discharge therapy.

Conclusion:
This case underscores the potential for adults with ASD and PH to develop variable AV block conduction due to increased right atrial pressure and volume overload.

Keywords: Variable AV Block Conduction, Pulmonary Hypertension, Atrial Septal Defect

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CASE-REPORT : A 14 BOY YEARS OLD PRESENTED WITH SUPRAVENTRICULAR TACHYCARDIA IN CARDIOMYOPATHY

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Background:
Supraventricular tachycardia (SVT) is one of the children's most common heart rhythm abnormalities. Episodes of SVT attacks in children are often recurring and if not properly recognized can be life threatening.

Case illustration:
A 14 boy years old come to the emergency room with complaining chest pain, palpitation, fever since 1 week ago, he also complaining about abdominal pain, vomiting and dizziness. Family and patient deny any previous similar complaining. From physical examination blood pressure 90/60, heart rate 170 per minutes, respiratory rate 21 per minutes, temperature 36,7 Celsius, oxygen saturation 98%. Electrocardiogram (ECG) showed supraventricular tachycardia 250 beat per minutes. Chest X-ray showed lung oedema with cardiomegaly. From laboratory examination leukosit leukosit 8,0 10^3/µl, trombosit 360 10^3/µl, eritrosit 5,21 10^6/µl, haematocrit 41,5%, Hemoglobin 14,6g/dL, liver function test was increased from normal 88U/I, electrolyte serum was normal Natrium 139.13 mmol/L, Kalium 4,57 mmol/L, Calcium 9,14mg/dL, Clorida 102,58 mmol/L. From echocardiography showed dilated cardiomyopathy and heart failure with reduce of ejection fraction (35%). After 1 week discharged from hospital echocardiography showed normally echocardiography.
Conclusion:
Diagnosis of SVT is based on detailed history and physical examination. Electrocardiogram and electrocardiography should be performed in children with suspected of having SVT. In patients with SVT arising due to a structural defect in the heart, the prognosis depends on the severity of the defect, but in healthy people with no structural defects, prognosis is excellent.

Keywords: cardiomyopathy, Supraventricular tachycardia

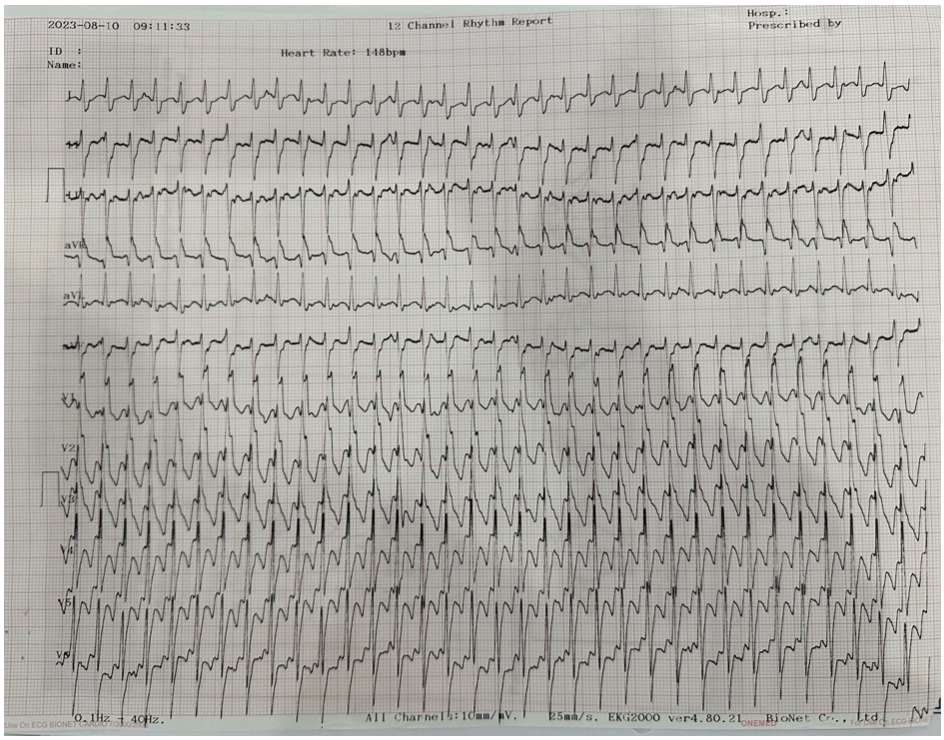

Figure 1. Electrocardiogram showed Supraventricular tachycardia 250 bpm

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MALIGNANT VENTRICULAR ARRHYTHMIA IN MYOCARDIAL INFARCTION WITH NON-OBSTRUCTIVE CORONARY ARTERIES (MINOCA) : A CASE REPORT

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Background:
Myocardial infarction with non-obstructive coronary arteries (MINOCA) is a myocardial infarction condition caused by various etiologies characterized by clinical evidence of myocardial infarction and normal or minimal obstruction (<50% stenosis) of the coronary arteries on angiography. MINOCA is the cause of 10% of acute coronary syndrome (ACS) cases. The etiology of MINOCA is distinguished into epicardial causes (rupture of plaque, coronary dissection, coronary spasm) and microvascular (microvascular coronary spasm, Takotsubo cardiomyopathy, coronary thromboembolism, and myocarditis). Myocardial infarction could be a trigger of malignant ventricular arrhythmias. This case report aims to describe the clinical features of MINOCA patient with ventricular arrhythmia.

Case illustration:
A 48 year old woman came with non-specific complaints of cardiac problems, such as weakness, headache, and nausea. There were no complaints of chest pain or tightness, but the patient had felt unspecific backpain for 1 month. Risk factors were hypertension controlled with one pill combination of valsartan dan amlodipine. On physical examination there was hypotension (systolic 78, Mean Arterial Pressure 60). No rales found in lung examination and no oedema on extremities. On electrocardiogram we found ST segment depression in anterolateral and inferior leads with occasional Ventricular Extrasystole (VES). The troponin was high (91826.70 pg/mL). Electrolyte panel within normal limit. Patient then transferred to the catheterization lab and undergo coronary angiography. During angiography, the patient underwent ventricular tachycardia with pulse (VTp), so the cardioversion was performed. On angiography we found normal coronary artery tree. No stenting was required. The patient then transferred to the ICCU. During hospitalization, recurrent VTp was found then changed to VT without a pulse and then ventricular fibrillation that didn't respond with defibrillation. High quality cardiopulmonary rescucitation was performed but at the end the patient didn't survive.

Conclusion:
MINOCA can lead to malignant ventricular arrhythmias through myocardial ischemic mechanisms. The diagnosis of MINOCA in this case is indicated by the presence of clinical evidence from myocardial infarction, evidence of coronary angiography that does not indicate the presence of obstruction or stenosis of coronary artery lumen, and the absence of other specific causes that explain the patient's clinical condition.

Keywords: angiography, MINOCA, Ventricular arrhythmia, Myocardial infarction

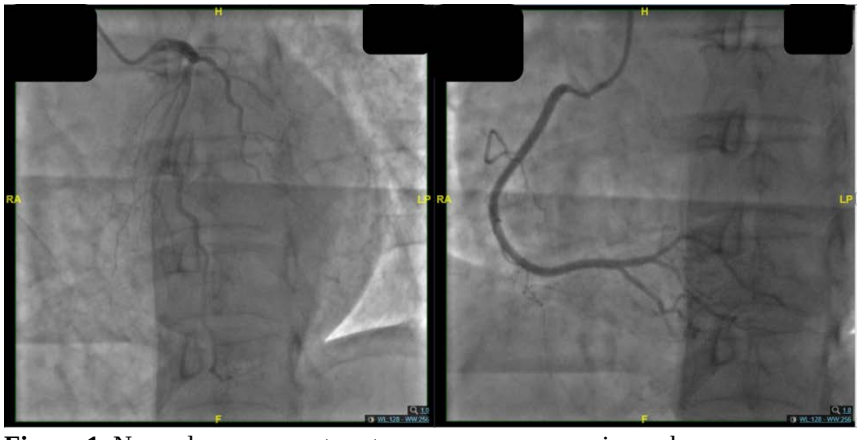

Figure 1. Normal coronary artery trees on coronary angiography

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ACUTE LUPUS MYOCARDITIS WITH SEVERE BRADYARRHYTHMIA : CHAOTIC COMPLICATIONS OF A YOUNG MAN WITH SYTHEMIC LUPUS ERYTHEMATOSUS

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Background:
Systemic lupus erythematosus (SLE), one of the most common systemic autoimmune diseases, is more often seen in females, especially at a young age. Our case report presents an uncommon severe cardiac manifestation of cardiogenic shock and severe bradyarrhythmia in a newly diagnosed SLE patient. Moreover, this 'great imitator' disease makes diagnosis difficult and challenging, especially in low-resource areas.

Case illustration:
A 21-year-old male patient was admitted to the emergency room with complaints of chronic fever, vomiting, weakness, and weight loss. The physical examination showed normal blood pressure and a slow, regular heart rate with no other abnormalities. The electrocardiogram (ECG) revealed a junctional rhythm with a rate of 40-50 beats per minute (bpm). Blood sampling showed severe pancytopenia. The patient rapidly developed severe hypotension and cardiogenic shock. We administered sulfas atropine, followed by continuous dopamine infusion and adequate fluid therapy, but the vital signs remained deteriorated during our observation. The echocardiography revealed severe hypokinesis of the left ventricle with a reduced ejection fraction (EF) of 36% and grade III diastolic dysfunction. Follow-up laboratory examinations showed positive antinuclear antibody-indirect immunofluorescence (ANA-IF), elevated C-reactive protein (CRP), and elevated Troponin I. A diagnosis of systemic lupus erythematosus (SLE) was made and confirmed using the 2019 European League Against Rheumatism (EULAR)/American College of Rheumatology (ACR) criteria. The patient showed significant improvement after administration of corticosteroids concomitantly with inotropic agents to support his failing heart. After a week of hospitalization, he was discharged and continued his medication as an outpatient.

Conclusion:
In cases of young patients with unexplained signs of acute heart failure, the presence of autoimmune myocarditis must be considered as a differential diagnosis. The complexity of diagnosing SLE was simplified using the 2019 ACR/EULAR criteria, especially in facilities with limited resources.

Keywords: Autoimmune myocarditis, Bradyarrhythmia, Systemic lupus erythematosus (SLE), Cardiogenic shock

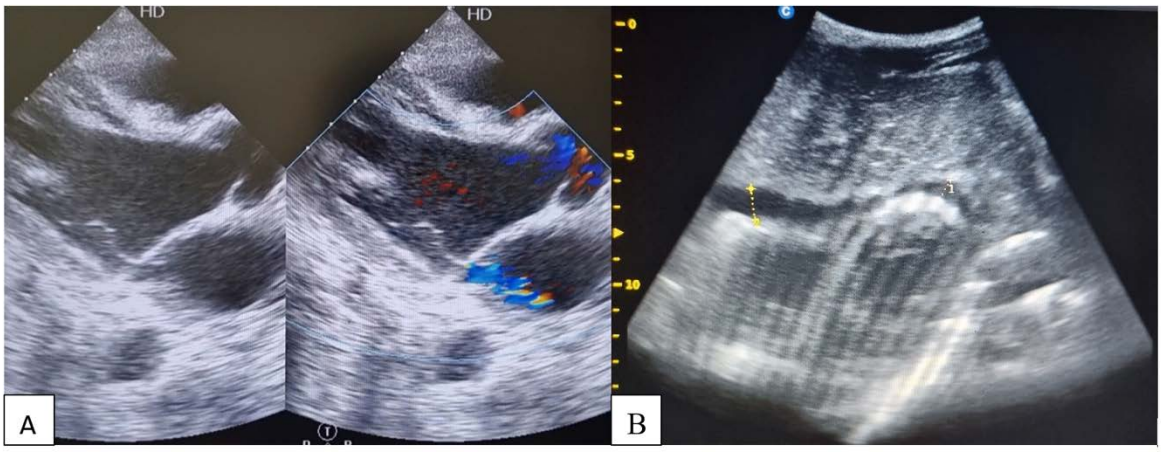

Figure 1. Echocardiography showed A. severely enlarged left ventricle with mitral regurgitation. B. Pericardial effusion

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UNVEILING SILENT THREATS: HEMOLYSIS AND INFECTIVE ENDOCARDITIS IN MYOCARDIAL INFARCTION VENTRICULAR SEPTAL RUPTURE POST TRANSCATHETER CLOSURE

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Background:
Myocardial infarction can lead to complications such as ventricular septal rupture (VSR), which significantly increases morbidity and mortality. While transcatheter closure has become a viable treatment option for some cases of VSR, it introduces its own set of challenges and risks.

Case illustration:
A 61-year-old male patient with uncontrolled hypertension and a history of smoking experienced VSR as a complication of STEMI. Transcatheter closure techniques were then performed on the patient, with a positive outcome, the device securely seated, no evidence of pericardial effusion, and stable hemodynamics. One week after the procedure, the patient had hemolysis, a decrease in eGFR, and an elevation of urea and creatinine. Two months after the procedure, the patient developed infective endocarditis. Nine months after the procedure, the patient developed community-acquired pneumonia. TTE was performed on the patient six and nine months post-transcatheter closure, with the results being device in situ, residual shunt negative, and shunt intradevice negative.

Conclusion:
The risk factors for readmission after myocardial infarction with ventricular septal rupture following transcatheter closure can include several factors: (1) device-related complications, such us device malposition, migration, or failure, can necessitate re-intervention or lead to complications like thrombosis; (2) post-MI VSR often precipitates or exacerbates heart failure due to volume overload and impaired cardiac function; (3) infective endocarditis, patients with implanted devices are at increased risk of infective endocarditis; (4) renal impairment, acute kidney injury or chronic kidney disease, often exacerbated by the hemodynamic instability associated with VSR. Infective endocarditis and renal impairment as a result of hemolysis occur in this patient.

Keywords: ventricular septal rupture, transcatheter closure, short-term follow-up

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NON-ISCHEMIC DILATED CARDIOMYOPATHY AND LONG-STANDING PSORIASIS: A COINCIDENCE OR INTRIGUINGLY ASSOCIATED DISEASES?

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Background:
Dilated cardiomyopathy is a disease with many causes, including genetic factors and immune factors. Psoriasis is a severe, chronic proliferative and inflammatory disease. Idiopathic dilated cardiomyopathy, as a cause of heart failure, and psoriasis have been well established as separate entities, however there is uncertainty with the association between these two.

Case illustration:
A 33-year-old female presented to our clinic with an insidious onset of shortness of breath, fatigue, and palpitations. She reported experiencing moderate limitations in physical activity and denied any prior medical history, medication use, or family medical history. She continued to have regular menstruation with a monthly cycle. Additionally, she had been dealing with skin lesions for years, characterized by erythematous scales and plaques on her upper and lower extremities. These lesions were further clinically and histopathologically identified as psoriasis vulgaris and seborrheic dermatitis by a dermatologist. On physical examination, her joints were normal. Laboratory tests showed normal results for hematology, electrolytes, kidney function, and liver function. Her chest radiograph revealed an enlarged heart with bilateral pulmonary congestion. An electrocardiogram indicated atrial fibrillation with normal ventricular response. Echocardiography revealed general hypokinesis, dilatation of the left atrium and left ventricle, reduced LVEF (38%), and mild mitral regurgitation. A cardiac catheterization confirmed normal coronary vasculature but severe left ventricular systolic dysfunction. She was diagnosed with heart failure with reduced ejection fraction due to non-ischemic dilated cardiomyopathy (NICM) associated with psoriasis. We collaborated with a dermatologist to manage her condition. The patient responded well to heart failure medications and so did she to dermatological treatments. She continued outpatient follow-up with both a cardiologist and a dermatologist, and her condition gradually improved.

Conclusion:
In line with our case, we suggest that genetic risk factors for NICM may overlap with those for psoriasis. The immune reaction triggered in dilated cardiomyopathy can contribute to disease progression, potentially exacerbated in patients with psoriasis. Chronic inflammation and sustained secretion of proinflammatory cytokines may represent a plausible pathway. Further investigation into the genetic and immune risk factors associated with both dilated cardiomyopathy and psoriasis is necessary for a comprehensive understanding of their pathogenesis and treatment.

Keywords: Autoimmune Disease, Psoriasis, Dilated Cardiomyopathy, Heart Failure, Echocardiography

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THE MYSTERIOUS PRIMARY PULMONARY HYPERTENSION WITH PULMONARY THROMBUS IN YOUNG LADY: DEVASTATING COMPLICATION OR CONCEALED CTPEH CONSEQUENT?

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Background:
Chronic Thromboembolic Pulmonary Hypertension (CTEPH) is a rare but serious condition characterized by persistent obstruction of pulmonary arteries by thrombus, leading to pulmonary hypertension and right heart failure, occurs in patients due to chronic pulmonary artery obstruction and is estimated to occur in 3.8% of patients following acute pulmonary embolism. If untreated progressive right ventricular dysfunction and ultimately right heart failure can result in significant morbidity and mortality.

Case Illustration:
A 21-year-old young woman presented with complaints of shortness of breath without chest pain, accompanied by nausea and vomiting three times. Vital sign revealed a blood pressure of 100/72 mmHg and a heart rate of 89 bpm, a respiratory rate of 27 bpm, her saturation was 81% with nrm at 15 lpm. Physical examination reveled fine rales, with elevated jvp > 3 cm, a body mass index of 28 kg/m², and the presence of systolic murmurs in the pulmonary valve, accompanied by regurgitation in the tricuspid valve. The patient's homan sign examination was negative. The electrocardiogram revealed the presence of an inferior ST-segment depression, an inverted T wave anterior extensive leads, which indicated left main artery coronary occlusion, and a right ventricular hypertrophy. A chest X-ray was performed, which revealed cardiomegaly (LV, LA). The patient's APTT and PT were 55.6 and 20 seconds respectively. Subsequently, the patient underwent CT angiography, which showed thrombus formation in the right pulmonary artery, a pulmonary trunk ratio of 2 with ascenden aortic and calcification of the pulmonary trunk. Aortic diameter was 5 mm, with LVH and diastolic dysfunction. The patient was diagnosed with primary pulmonary hypertension with pulmonary thrombus. The patient was administered a treatment regimen comprising sildenafil 10 mg three times daily, digoxin 0,25 mg daily, warfarin 2 mg daily, fondaparinux sc 2.5 mg daily, and spironolactone 25 mg daily, while awaiting the result of D-dimer testing. The result confirmed an increased D-dimer level, which was 778.08 ng/ml.

Conclusion:
CTEPH, though uncommon in young adults, should be considered in patients with unexplained primary pulmonary hypertension. Early diagnosis and appropriate management are crucial to improve outcomes

Keywords: CTEPH, primary pulmonary hypertension, pulmonary thrombus

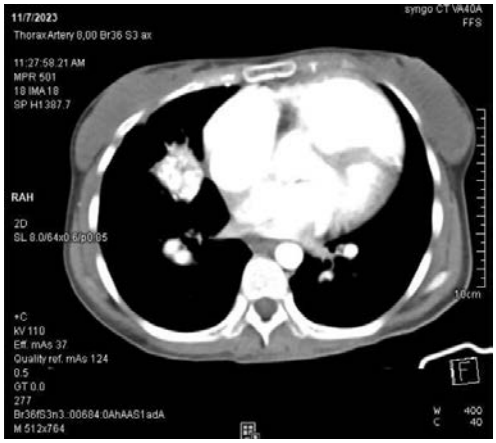

Figure 1. Axial images show proximal filling failure in the Pulmonary Artery of the right middle lobe as well as in the Pulmonary Artery of the anterior segment of the left lower lobe.