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Case Report

Risk Stratification of Short Term Sudden Death after Acute Coronary Syndrome

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Sudden cardiac death is the leading cause of cardiovascular mortality in acute coronary syndrome. Risk stratification scoring tools are available to better identify patients at risk after acute myocardial infarction. In addition plenty of factors and treatment modalities modulate the risk of sudden cardiac death.
A case of in-hospital SCD in a young woman with acute anterior STEMI is presented as a trigger to the importance of risk stratification and treatment according to guidelines in preventing SCD.

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Keywords: sudden cardiac death, risk stratification

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Laporan Kasus

Stratifikasi Risiko Kematian Jantung Mendadak pada Sindrom Koroner Akut

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Kematian jantung mendadak merupakan penyebab penting mortalitas kardiovaskular pada sindrom koroner akut. Terdapat beberapa sistem stratifikasi risiko yang sudah mapan yang dapat mengidentifikasi lebih baik pasien dengan risiko kematian jantung mendadak. Selain itu banyak faktor-faktor lain dan modalitas terapi yang dapat mengubah risiko kematian jantung mendadak.
Ditampilkan suatu kasus kematian jantung mendadak pada perempuan muda dengan STEMI anterior sebagai pencetus diskusi pentingnya stratifikasi risiko dan terapi yang sesuai dengan petunjuk baku untuk mencegah kematian jantung mendadak pasca infark.

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Kata kunci: kematian jantung mendadak, stratifikasi risiko

Introduction

Sudden cardiac death (SCD) is the leading cause of cardiovascular mortality in developed countries. In USA, incidence of SCD up to 310,000 events per year or about 455 - 870 per day. It was one case every 2 - 3 minutes.[1]

Coronary artery disease is present in 80 - 85% of patients who experience SCD.[2] Patients with CAD who have had an acute myocardial infarction (MI) are at increased risk for SCD, most often due to a ventricular tachyarrhythmia. However, not all post-MI patients have the same risk of SCD. Thus, the therapeutic approach to prevent SCD depends upon the identification of those patients who are most likely to have a ventricular tachyarrhythmia and the effectiveness of the available preventive measures.

The process of risk stratification in a patient who has had an acute MI has two components: (1) early in-hospital identification of patients at increased risk for recurrent ischemic events and (2) identification of patients at increased risk for arrhythmic or non-arrhythmic death.[3]

There are a number of diagnostic parameters such as low left ventricular ejection (EF <30 %), myocardial infarction, onset and early phase, low heart rate variability, abnormal signal-averaged electrocardiograms, T wave alternans, and diabetes

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mellitus, which, if present, indicate an increased risk for SCD.

In National Cardiac Center Harapan Kita, there were 18 registered cases of in-hospital SCD from 2006 to 2010. And there were 204 registered cases of death on arrival from 2006 to 2010. This case report is aimed to discuss the risk of in-hospital SCD after acute MI with acute heart failure syndrome and uncontrolled blood glucose in type 2 diabetes mellitus.

Case Illustration

A 42 years old woman referred from Serang Regional General Hospital with acute MI and uncontrolled blood glucose in type 2 diabetes mellitus. She complained of chest pain since 5 days before admission, described the pain as a heavy-pressure and radiated to her back. The pain began right after he was awoke in the morning and lasted for over 30 minutes. She felt nauseous and diaphoresis as well. This feeling lead her to Serang Regional General Hospital which then she was diagnosed to have acute anterior MI and uncontrolled blood glucose in type 2 diabetes mellitus. After one day of treatment in ICU the patient was then referred to National Cardiovascular Center Harapan Kita (NCVCHK). When arrived in the emergency unit, the patient still felt chest pain. Risk factors for coronary artery disease are hypertension, diabetes mellitus and dyslipidemia.

On physical examination, patient was alert and looked moderately ill, her blood pressure was 116/83 mmHg, heart rate of 124 bpm regullar and with adequate volume and respiratory rate of 22 per minute, and peripheral saturation on oxymetri was 96%. His conjunctiva was not pale and sclera was not icteric. His jugular venous pressure was 5+2 cmH_2O, and from the heart examination his first and second heart sound were normal and no murmur or gallop. Rales was heard over the half of the both lung fields without any wheezing. The abdomen was supple, no enlargement of the liver or the spleen, with normal peristaltic sound. His both lower extremities were warm and not swollen with good and equal pulsation on both sides.

ECG (**Figure 1 A**) showed sinus tachycardia, axis LAD, QRS rate 124x/minutes, pathologic Q waves at V1-V3, poor R progression II, III, aVF, V4-V6 with ST elevation V1-V4 and the chest x-ray (**Figure 1 B**) showed enlarged heart with CTR of 60% (not maximal inspiration), normal aortic and pulmonic segment, without any sign of congestion and infiltrate.

The laboratory examination on admission revealed random glucose test of 298 mg/dl, CKMB of 258 U/L, Trop T of 3.59 ng/ml, Total cholesterol 196 mg/dl, HDL Cholesterol 50 mg/dl, LDL Cholesterol 98 mg/dl, Trigliseride 204 mg/dl, Ratio Cholesterol 3.93 mg/dl, uric acid 8.7 mg/dl and other measures were within normal limit.

She was diagnosed as suffered from Acute antero-septal STEMI day 5 Killip II without revascularization, Type 2 Diabetes Mellitus, and Pneumonia. Calculated TIMI risk score was 7/14. Then following treatment were given: fondaparinux 2.5mg sub cutaneously, RI infusion drip start at 2 iu/hour, ASA 80 mg OD, Clopidogrel 75 mg OD, Atorvastatin 20 mg OD, Captopril 6.25 mg tid, ISDN 5 mg tid, NTG infusion

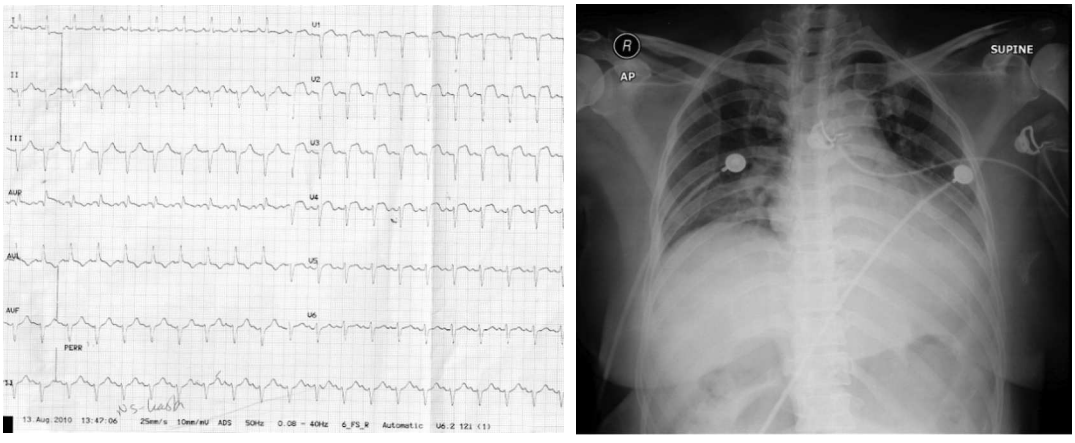

Figure 1 A. 12-leads ECG **B.** Supine AP Chest X'Ray

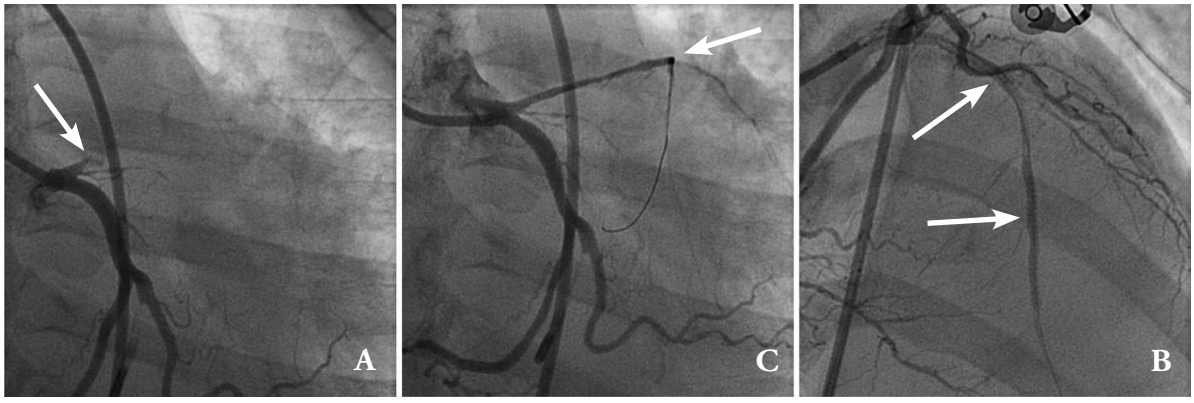

Figure 2 A. coroangiography show total occlusion at proximal LAD, **B**. Flow to LAD after recanalization with wire. **C**. After PCI at proximal and mid part LAD.

start 0.5mg/hour. She was sent to the intensive cardiovascular care unit (CVCU). However, due to refractory heart failure with low EF (20-30%) and signs low cardiac output syndrome then intra aortic balloon counterpulsation (IABP) was inserted and dobutamin was given as well. Lung infection could be controlled with imipenem 1 g IV twice a day. She underwent early revascularization which found normal left main coronary artery, small RCA, total occlusion at proximal LAD and normal LCX. Then a long drug eluting stent 3.0/38mm was implanted at proximal LAD and another bare metal stent 2.5/30 mm at mid LAD with good results. The blood glucose level ranged from 70 to 325 g/dL with syntax score of 29.5.

Four day later the patient's condition improved with stable hemodynamic state and the infection recovered. The inotropic and vasopressor drugs weaned and the IABP was removed. Patient moved to ward and starts mobilization. Her measured body weight was 52 kgs.

Echocardiography reevaluation showed LV dilatatian (EDD of 55 mm), poor LV systolic (EF 22%) and diastolic function with akinetic anteroseptal to apical wall, normokinetic in the lateral and posterior base, while other segment shown hipokinetic motion with increased of LVEDP. The RV function (TAPSE 1.6 cm) was decreased with moderate to severe pulmonary hypertension. Carvedilol was started on day 9 post MI and uptitrated slowly. Unfortunatelly, at 8 pm on day 9 hospitalization, patient suddenly awake and complain of breathlessness and shortly followed by cardiac arrest. Cardio pulmonary resuscitation (CPR) last for 50 minutes was failed and patient died.

Literature Review and Discussion

Definition

SCD is defined as natural death from cardiac causes, heralded by abrupt loss of consciousness within 1 hour of the onset of an acute change in cardiovascular status. Preexisting heart disease may or may not have been known to be present, but the time and mode of death are unexpected.[4]

Sudden Cardiac Death in patients with Acut e MI

Coronary artery disease is present in 80 - 85% of patients who experience SCD.[5] Acute MI can be defined from a number of different perspectives related to clinical, ECG, biochemical, and pathological characteristics. Rapid diagnosis and early risk stratification of patients presenting with acute chest pain are important to identify patients in whom early interventions can improve outcome.

There are several predictor of SCD in patient with MI, such as GRACE risk score, Killip classification, TIMI risk score, heart rate variability, baroreflex sensitivity and uncontrolled blood glucose in type 2 diabetes mellitus .

In-hospital Mortality Risk Scoring Tools

GRACE study[5] showed 1989 patients died in hospital, 1466 died between discharge and six month follow-up, and 2793 suffered a new non-fatal myocardial infarction. Nine factors independently predicted death

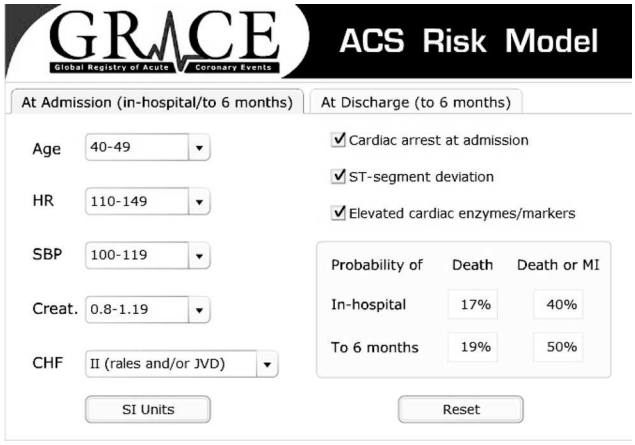

Figure 3 GRACE risk score calculator of the particular patient showed in hospital probability of death was 17%, and probability of composite death and MI was 40%.

and the combined end point of death or MI in the period from admission to six months after discharge are age, development (or history) of heart failure, peripheral vascular disease, systolic blood pressure, Killip class, initial serum creatinine concentration, elevated initial cardiac markers, cardiac arrest on admission, and ST segment deviation. The simplified model was robust, with prospectively validated C-statistics of 0.81 for predicting death and 0.73 for death or myocardial infarction from admission to six months after discharge. The external applicability of the model was validated in the dataset from GUSTO (Global Use of Strategies to Open Occluded Coronary Arteries) IIb trial.[6]

Fox et al. stated that older age, higher Killip class, elevated heart rate, lower systolic blood pressure, and anterior location of the infarct have been identified as the most important independent predictors of early mortality in clinical trials and registries.[7]

The Killip classification is a classification system used in individuals with an acute MI, in order to risk stratify them. Individuals with a low Killip class are less likely to die within the first 30 days after their MI than individuals with a high killip class. High Killip class was independent predictors of mortality in STEMI and non-STEMI patients.[8]

David et al. found that the TIMI risk score for STEMI captures the majority of prognostic information offered by a full logistic regression model but is more readily used at the bedside. This risk assessment tool is likely to be clinically useful in the triage and management of fibrinolytic-eligible patients with STEMI. Careful risk assessment for each patient informs decisions regarding therapeutic interventions, triage among alternative levels of hospital care, and allocation of clinical resources.[9]

Our patient had Killip class II, elevated heart rate, low systolic blood pressure, weight of less than 67 kgs, diabetes, history of hypertension, time to appropriate treatment of 5 days and infarct in the anterior wall, and TIMI risk score of 8/14. GRACE risk score calculation revealed that probability of in hospital mortality of 17% and death or MI of 40% a. TIMI risk score showed the probability of 26.8 mortality rate in 30 days. From these analyses, this patient had a very high risk of SCD.

Out of risk stratification tools, some factors are atributed to SCD in this particular patient. A strong relationship was found between LVEF and SCD which is higher with varying degrees of left ventricular dysfunction.

Beta-blockers in acute MI and LV dysfunction

Because of their initial transient negative inotropic effects, beta-blockers traditionally were considered contraindicated in HF. However, in CAPRICORN study,[10] carvedilol reduced mortality and reinfarction in post-MI patients with left ventricular systolic dysfunction, with or without signs of HF. In our case, carvedilol was initiated at day 5 after MI and uptitrated slowly which is relevant to CAPRICORN subject. As LVEF of patient was only 22%, uptitration

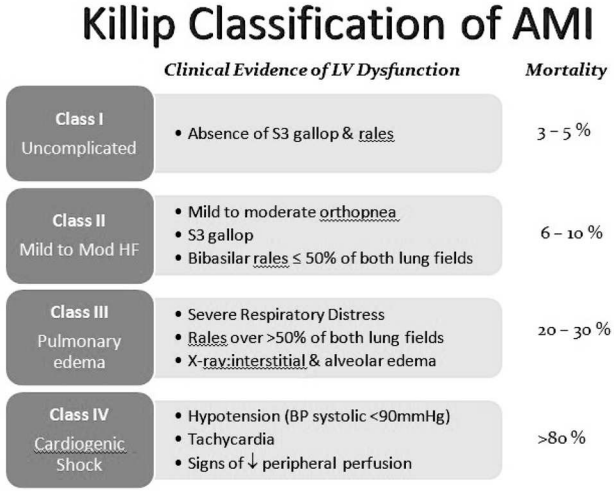

Figure 4. Killip classification of Acute MI predicting 30-day mortality.[8]

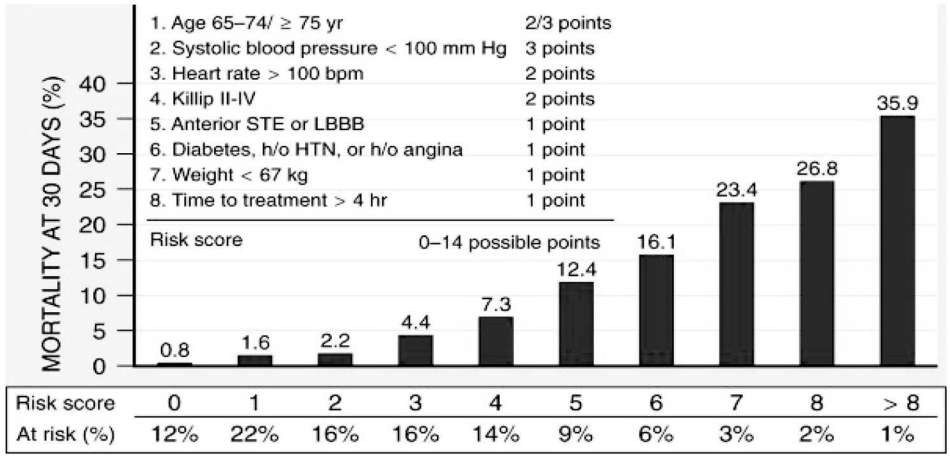

Figure 5 TIMI risk score for STEMI predicting 30-day mortality. h/o = history of; HTN = hypertension; LBBB = left bundle branch block; STE = ST segment elevation; TIMI = Thrombosis In Myocardial Infarction.[9]

of carvedilol was done very cautiosly. Whether the benefit of carvedilol has not yet achieved after 3-4 days administration is not clear. In systematic review of 11 randomized control trial involving more than 5000 patients with acute MI and heart failure, carvedilol significantly reduced all-cause mortality compare to commonly prescribed beta-blocker.[11] Present guidelines advise that in patients with systolic HF, most of whom have coronary artery disease, an ACE inhibitor should be used first, and a beta-blocker such as carvedilol or metoprolol should be started at low doses and gradually increased over 12 weeks. There is now conclusive evidence that long-term beta-blocker use results in improved rates of mortality and morbidity in patients with HF.

Implantable Cardioverter Defibrilator

The Multicenter Autonomic Defibrillator Implantation Trial II (MADIT II) trial[12] confirm a survival benefit from implantable cardioverter defibrillator (ICD) implantation in a vastly enlarged group of patients, those with any history of MI (at least 30 days) and left ventricular dysfunction with an ejection fraction (EF) of 30% or less. Although our patient had low EF (EF 22%), but she only 9 days after MI. That's why she was not yet indicated for ICD.

Diabetes melitus

Diabetes mellitus is a major risk factor for cardiovascular disease in general and for coronary heart disease in particular. Furthermore, the recent National Cholesterol Education Program III guidelines have elevated diabetes to a coronary disease risk equivalent. Among patients with diabetes who survived from MI, less is known about subsequent morbidity and mortality. Up to 20% of all patients with an infarction have diabetes, and this figure is expected to increase.[9] Importantly, patients with diabetes may present with atypical symptoms, and heart failure is a common complication. Diabetic patients who survive from STEMI still have doubled mortality risk as compared with non-diabetic patients. Our patient has uncontrolled blood glucose in type 2 diabetes mellitus despite insulin infusion 2-3 mg/hour. Poor control of blood glucose may increased the event of SCD.

Summary

The risk of SCD is higher in patient with MI with heart failure and low EF, higher GRACE risk score, higher TIMI risk score, higher Killip class, late onset of treatment, and uncontrolled blood glucose in diabetic patient.

We reported a case of SCD on day 5 of acute anterior STEMI with TIMI risk of 8/14 and Killip class II who underwent early PCI. Patient has low ejection fraction, and uncontrolled blood glucose level.

From this case we learn that intensive glucose control with insulin in patients with significant hyperglycaemia (>9.9mmol) is mandatory, regardless of prior diabetes history (level of evidence B). The Scottish

Intercollegiate Guidelines Network state that "Patients with clinical myocardial infarction and diabetes mellitus or marked hyperglycaemia (>11.0mmol/l) should have immediate intensive blood glucose control." This should be continued for at least 24 hours.Target glucose levels 140 mg/dL (7.8 mmol/L) have been suggested. However, care needs to be taken to avoid blood glucose levels below 80–90 mg/dL (4.4–5 mmol/L), as hypoglycaemia-induced ischaemia might also affect outcome in diabetic patients with acute coronary syndromes. Uncontrolled blood glucose in non-diabetic patient gives higher mortality than uncontrolled blood glucose in diabetic patient. Both diabetes mellitus and MI affect the baroreflex sensitivity so that it could increase the rates of SCD.

Another lesson learnt is that in such high risk post MI patient, electrophysiology study should be considered to determine the need of ICD implantation earlier. The analyses from MADIT trial revealed that the sicker patients achieved better benefit from the ICD.

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