Case Series

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Pharmacological Management of Supraventricular Tachyarrhythmia in Right Atrial Enlargement Due to Pulmonary Hypertension

Mochamad Rizky Hendiperdana.[1]

Abstract

Background: Right atrial (RA) enlargement is a common finding in patients with pulmonary hypertension (PH). Supraventricular arrhythmia (SVA) is common in PH patients with RA enlargement. Treatment of SVA should be aggressive since it can cause hemodynamic worsening consequences, because RA function plays an important role in right heart function.

Case Illustration: Three cases of SVA in underlying right atrial enlargement with preserved ventricular function that successfully managed by pharmacological cardioversion according to the guidelines. The first case describes atrial flutter with right bundle branch block (RBBB) morphology with vital sign: heart rate (HR) 170 beats per minute with regular pulse, blood pressure (BP) 110/70 mmHg, respiratory rate (RR) 28 per minute, and peripheral oxygen saturation was 89 % which successfully converted to sinus rhythm by amiodarone (class III antiarrhythmic drug) administration, meanwhile the second and third cases demonstrate paroxysmal SVA with admission vital sign: HR 155 beats per minute, regular pulse, BP 152/115 mmHg, RR 27 per minute, peripheral oxygen saturation was 80 % in the second case and in the third case on admission vital sign: oxygen saturation was 70 %, BP 110/71 mmHg, RR 40 per minute, and the HR were 160 bpm, regular that converted to sinus rhythm by diltiazem (class IV antiarrhythmic drug) administration.

Conclusion: Supraventricular arrhythmia is a frequent arrhythmia that occurs in pulmonary hypertensive and right atrial dilation patients. The tachyarrhythmia in this patient population was tolerated poorly and led to hemodynamic perturbation. Pharmacological cardioversion is one of the effective approaches to alleviate patient symptoms with significant clinical improvement.

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Keywords: *supraventricular arrhythmia, pulmonary hypertension, right atrial enlargement, pharmacological cardioversion.*

[1] Department of Cardiology and Vascular medicine, Pandan Arang General Hospital, Boyolali, Central Java, indonesia.

Correspondence:
Mochamad Rizky Hendiperdana, Department of Cardiology and Vascular medicine, Pandan Arang General Hospital, Boyolali, Central Java, indonesia.
email : mhendiperdana@gmail.com

Introduction

Right atrial (RA) enlargement is a common finding in patients with pulmonary hypertension (PH). Long-standing atrial wall stretch is mediated by chamber dilation that will cause atrial electrical remodeling and arrhythmogenic substrate.[1,2] Supraventricular arrhythmia (SVA) is common in PH patients with RA enlargement. Treatment of SVA should be aggressive since it can cause hemodynamic worsening consequences, because RA function plays an important role in right heart function.[3] We reported a case series of three patients with SVA in pre-existed right atrial (RA) enlargement that resulted in hemodynamic insult leading to emergency ward visits. There was immediate clinical improvement after rhythm control by pharmacological cardioversion.

Case 1

A 57-year-old man presented to the emergency ward at our local community hospital with palpitations and shortness of breath for 3 hours. The patient also had abdominal pain for 4 days. Patient denied any prior chest pain, fever, cough, or other constitutional symptoms. Past medical history was unremarkable for significant prior illness. On admission, vital signs were: Patient was alert with heart rate 170 beats per minute, regular pulse, blood pressure (BP) 110/70 mmHg, respiratory rate 28 per minute, peripheral oxygen saturation was 89 %, and body surface area (BSA) of 1.41 m^2.

Electrocardiography showed wide-complex tachycardia with QRS rate 170x/minute with right bundle branch block morphology (RBBB) (Figure 1A). Chest x-ray revealed increased cardiothoracic ratio with upward apex and increased pulmonary vascular marking in the hilus area (Figure 2A). Initial laboratory examination results showed an increased D-dimer assay of 1330 ng/mL. The routine hematology, renal function, troponin I level, and serum electrolytes were normal.

The patient was diagnosed with unstable wide-complex tachycardia with differential diagnosis of monomorphic ventricular tachycardia, aberrant supraventricular tachycardia or supraventricular arrhythmia in underlying RBBB. Brugada algorithm analysis of patient ECG concluded that the rhythm was atrial flutter 2:1 conduction with RBBB. The absence of AV dissociation, fusion, and captured beat accompanied by the presence of RS complex in V3-V6 ECG leads to the conclusion to atrial flutter with RBBB.

The patient was planned for chemical cardioversion in the emergency ward with antiarrhythmic agent class III, amiodarone 150 mg intravenously administered. Immediately after i.v. amiodarone administration, the rhythm conversion to sinus takes place. Repeat 12-lead ECG showed sinus rhythm with QRS rate 75x per minute with complete RBBB morphology (Figure 1B). The patient's dyspnea and palpitation were instantaneously relieved after rhythm control management, then the patient was transferred to the intensive care unit (ICU) for further monitoring and management.

In the ICU ward, transthoracic echocardiography (TTE) was performed and revealed right atrial (RA) and right ventricular (RV) dilation (RV basal diameter 56 mm, RA area index 20.7 cm^2/m^2) (Figure 2C) with severe tricuspid regurgitation (TR), TR Vmax 3.79 m/s and high probability of PH with pulmonary artery systolic pressure (PASP) 70 mmHg (Figure 2D). Other valves were normal. Left ventricular (LV) appeared D-shaped at the systolic phase (Figure 2B). LV and RV systolic functions were preserved (EF 56 %, TAPSE 1.8 cm, RV S' 9 cm/s).

The patient was diagnosed with pulmonary hypertension (PH) with the suspicion of pulmonary vascular disease etiology (group 4) due to an increase in D-dimer value. The differential diagnosis is group 1 PH due to pulmonary arterial hypertension. The group 2 dan 3 PH were excluded from the differential diagnosis because of normal left heart function and relatively normal chest X-ray appearance. The lack of right heart catheterization (RHC) examination and contrast CT pulmonary angiography was the limitation of this case diagnostic work-up due to limited facilities at our local community hospital.

On admission, medical management was i.v furosemide 40 mg b.i.d, enoxaparin 0.6 cc subcutaneously twice daily for three days followed by warfarin 2 mg o.d, bisoprolol 2.5 mg o.d, and ramipril 5 mg o.d. There was no arrhythmia episode observed during 24-hour monitoring and the patient was discharged on third admission uneventfully with bisoprolol 2.5 mg o.d, ramipril 5 mg o.d, furosemide 40 mg o.d, and warfarin 2 mg o.d. The subsequent monthly outpatient visit was regularly attended by the patient. After 3 months

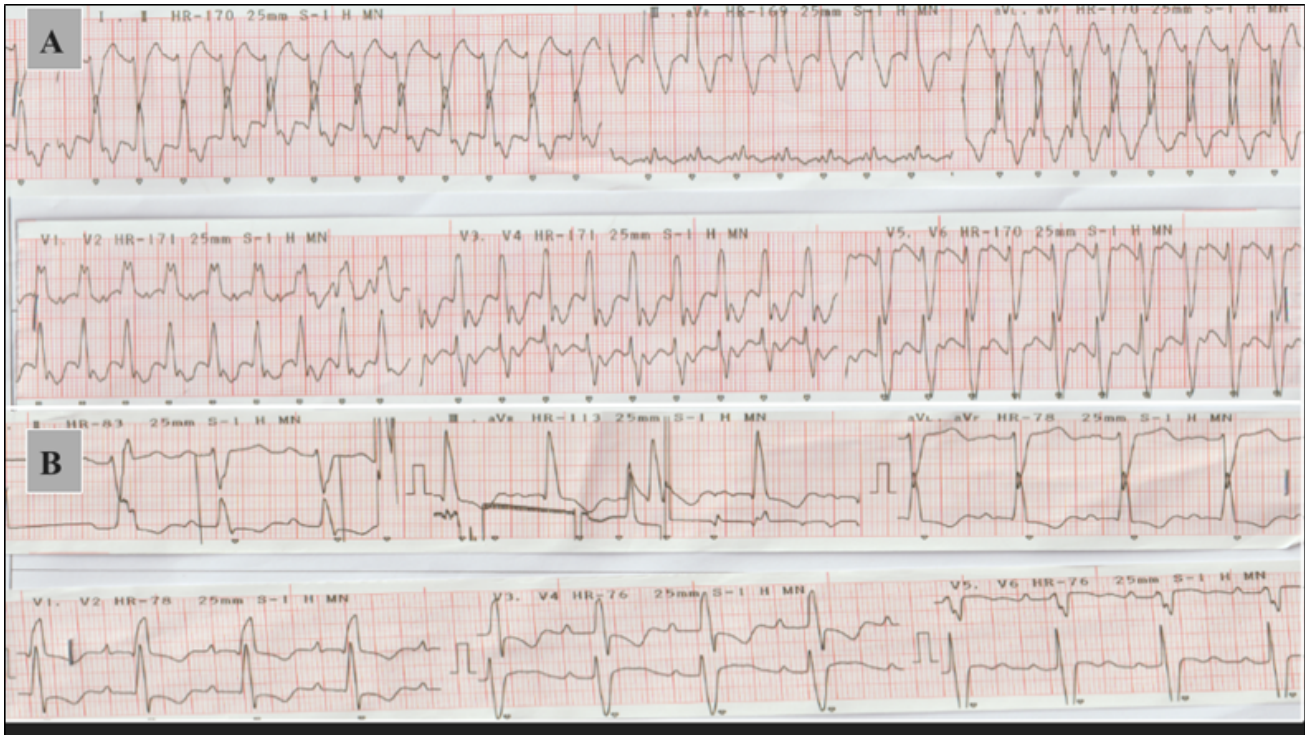

Figure 1. Electrocardiography in case 1. A: Initial ECG presentation at emergency ward showed wide-complex tachycardia with QRS rate 170x/minute with RBBB morphology; B: Post pharmacological cardioversion ECG showed sinus rhythm with QRS rate 75x per minute with complete RBBB morphology. ECG: electrocardiography; RBBB: right bundle branch block.

of follow-up, we added sildenafil up to a titrated dose of 50 mg t.i.d along with warfarin and bisoprolol. The patient was well without a hospitalization episode for the subsequent 8 months.

Case 2

A 44-year-old woman came to the emergency department at our local community hospital with restlessness and palpitation, chest discomfort, and shortness of breath for 1 hour prior to hospital admission. The symptom leads to difficulty of sleep that is caused by chest palpitations. There was no history of cough and fever, chest pain, and chest trauma prior to symptom development. Past medical history was history non-specific 'heart enlargement' without routine follow-up from other hospitals. The medical record files were unavailable. On admission, vital signs were: Patient was alert with heart rate 155 beats per minute, regular pulse, BP 152/115 mmHg, respiratory rate 27 per minute, peripheral oxygen saturation was 80 % and BSA of 1.74 m^2.

Electrocardiography showed narrow-complex tachycardia with QRS rate 155 x/minute with RBBB morphology with absence of P wave (Figure 3A). Thorax x-ray showed increased cardiothoracic ratio with upward apex, prominence of the pulmonary arteries segment and pruning pulmonary vascular marking in the hilus area (Figure 4A). Laboratory examination results were unremarkable. Bedside emergency transthoracic echocardiography showed preserved systolic function.

The patient was diagnosed as unstable narrow-complex tachycardia with the differential diagnosis of typical atrioventricular-node reentrant tachycardia (AVNRT) or atrioventricular reentrant tachycardia (AVRT) or atrial tachycardia (AT) in a pulmonary hypertension patient based on the patient's ECG and chest x-ray. The patient was planned for pharmacological cardioversion with antiarrhythmic agent class IV. Diltiazem 20 mg intravenously was administered. Rhythm conversion to sinus occurred immediately

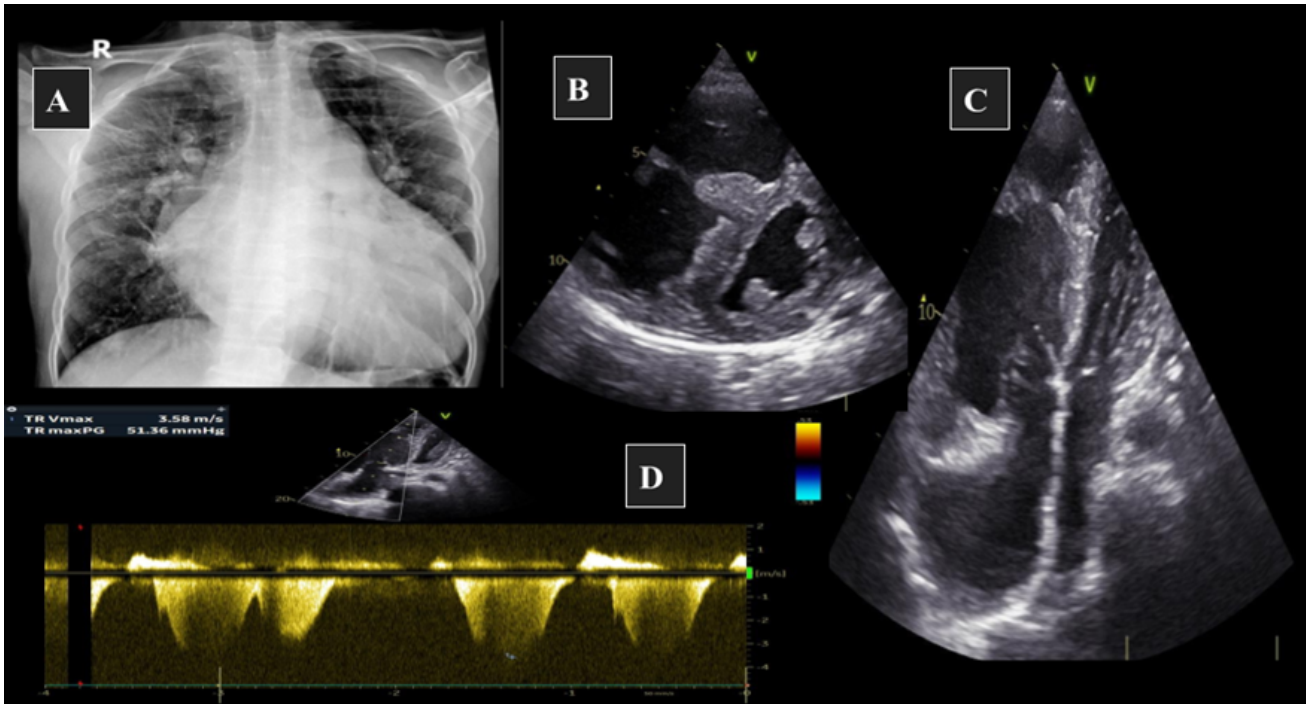

Figure 2. Case 1 X-ray and 2D echocardiography. A: Patient chest x-ray revealed increased cardiothoracic ratio with upward apex and increased pulmonary vascular marking in the hilus area; B: Parasternal short axis view 2D echocardiography showed D-shaped LV at systolic phase; C: apical 4-chamber view 2D echocardiography showed RA) and RV dilation (RV basal diameter 56 mm, RA area index 20.7 cm2/m2); D: severe TR with TR Vmax 3.79 m/s and high probability of PH with PASP 70 mmHg. LV: left ventricular; RA: right atrial; RV: right ventricular; TR: tricuspid regurgitation; PH: pulmonary hypertension; PASP: pulmonary artery systolic pressure.

after i.v diltiazem administration, which was followed by instantaneous symptom improvement. Repeat 12-lead ECG showed sinus rhythm with QRS rate 68x per minute with right axis deviation, right ventricular (RV) hypertrophy and strain and diffuse T wave inversion in precordial leads (Figure 3B). The patient was then transferred to the medical ward for further monitoring and management.

The TTE showed dilation of RA (RA area index 12.8 cm^2/m^2) and RV (Figure 4C) with severe TR, TR Vmax 4.5 m/s with high probability of PH with PASP of 93 mmHg (Figure 4D) and presence of PV notch of pulsed wave doppler (Figure 4B). Other valves were normal. Left ventricular (LV) appeared D-shaped at the systolic phase with normal LV and RV systolic functions (EF 60 %, TAPSE 2.3 cm, RV S' 14 cm/s). The interatrial septum is intact.

The patient was diagnosed with a high likelihood of pulmonary arterial hypertension (PAH). The group 2 and 3 PH were excluded for the same reason as case 1. The absence of RHC assessment and contrast CT pulmonary angiography for diagnostic workup also becomes a limitation of this case.

On admission, medical management was i.v furosemide 40 mg b.i.d, digoxin 0.25 mg o.d, bisoprolol 2.5 mg o.d, and sildenafil 20 mg t.i.d for pulmonary artery dilator purposes. On the second admission day, patient symptom improvement was observed with stable hemodynamics without other antiarrhythmic episodes during 24-hour monitoring. Patient was discharged on third admission uneventfully with bisoprolol 2.5 mg o.d and sildenafil 20 mg t.i.d. After 1 week post-discharge, the patient was followed up in an outpatient clinic without a tachyarrhythmia episode.

Case 3

A 36-year-old woman came to the emergency ward for respiratory distress. On admission: oxygen

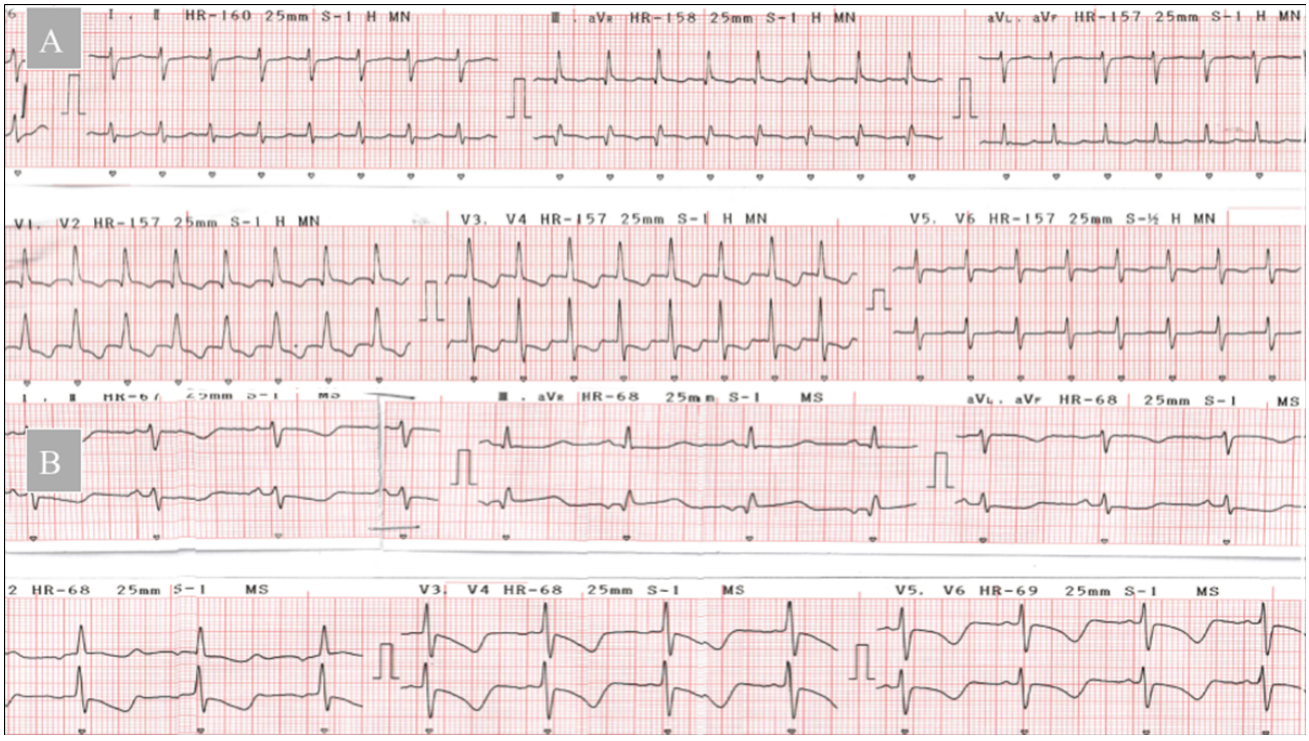

Figure 3. Electrocardiography in case 2. A: Initial ECG presentation at emergency ward showed narrow-complex tachycardia with QRS rate 155 x/minute with RBBB morphology with absence of P wave; B: Post pharmacological cardioversion ECG showed sinus rhythm with QRS rate 68x per minute with right axis deviation, RV hypertrophy and strain and diffuse T wave inversion in precordial leads. ECG: electrocardiography; RBBB: right bundle branch block; RV: right ventricular.

saturation was 70 %, blood pressure 110/71 mmHg, respiratory rate 40 per minute, and the heart rate was 160 bpm, regular. The electrocardiography (ECG) finding was wide complex tachycardia and right bundle branch block (RBBB) morphology. (Figure 5A) Serum lactate level was 2.40 mmol/l and PaO2 41 mmHg, TSH and FT4 were normal. Bedside echocardiography examination showed preserved ejection fraction (EF) with RA-RV dilation (Figure 6A and 6C). Pulmonary stenosis valvar type with peak pulmonary valve gradient 52 mmHg was also noted (Figure 6B and 6D). The patient was diagnosed with SVT with RBBB, type I acute respiratory failure with severe pulmonary stenosis.

The patient was administered intravenous diltiazem 20 mg and oxygen supplementation. ECG evaluation showed conversion to sinus rhythm with RBBB. (Figure 5B) The patient's symptom was relieved immediately after rhythm conversion and rate control with 98% oxygen saturation. Patient discharged on the 3rd day of admission.

Discussion

These case series highlight the importance of SVA as a precipitating factor for pulmonary hypertension exacerbation in RA enlargement.[3] Atrial tachyarrhythmia could precipitate RV failure in the setting of preexisting pulmonary hypertension, and the prevalence of SVA in PAH varied among studies from 9.9 % to 46.4 %. The most common SVA type in PAH is atrial fibrillation, followed by atrial flutter and AVNRT.[1] In paroxysmal SVA, female sex is predominant with 67.5% patients.[4] SVA causes profound hemodynamic insult in pre-existing RA dilation and pulmonary hypertension patients. Olsson et al reported that worsened right heart failure manifested in 30 % at the time of SVA detection in PH patients.[5]

These case series describe three cases with acute hemodynamic perturbation in pre-existing RA enlargement and PH, which were precipitated by supraventricular tachyarrhythmia. All cases were successfully managed with pharmacological management

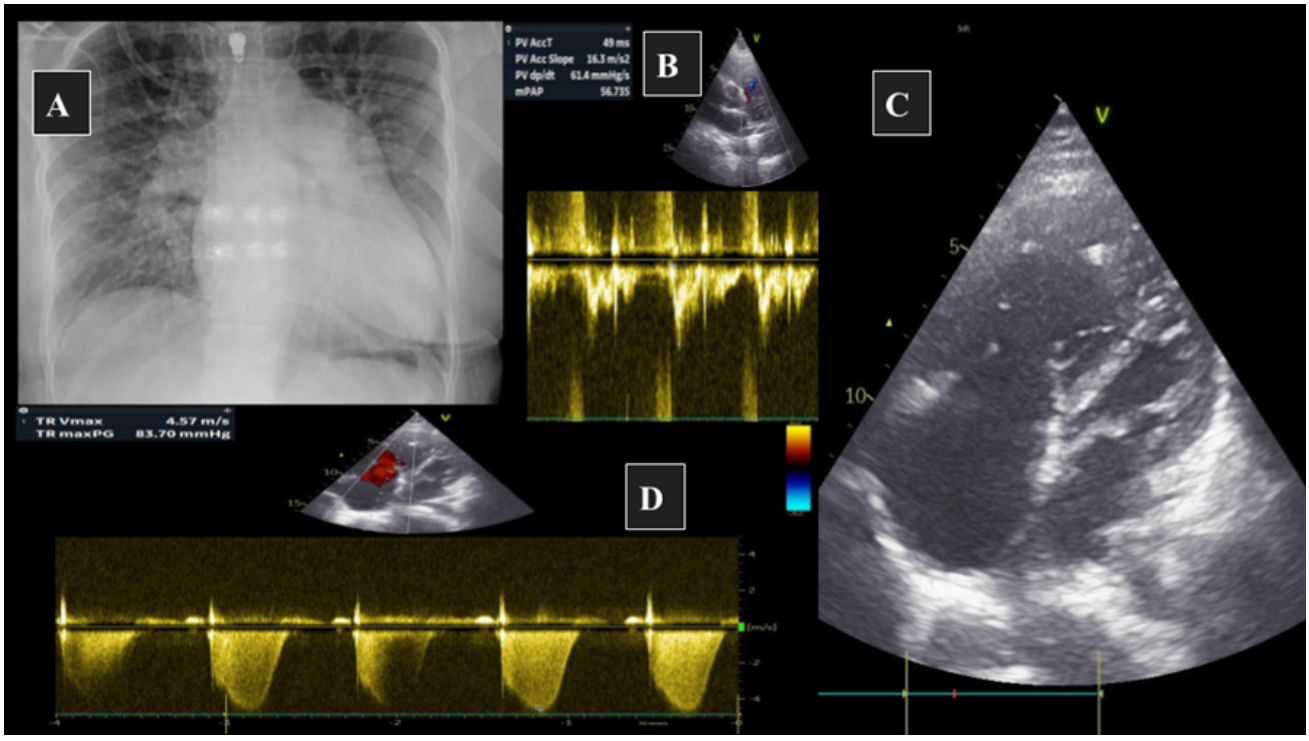

Figure 4. Case 2 X-ray and 2D echocardiography A: Patient chest x-ray revealed showed increased cardiothoracic ratio with upward apex, prominence of pulmonary arteries segment and pruning pulmonary vascular marking in the hilus area ; B: pulmonary artery notch by pulsed wave doppler; C: apical 4-chamber view 2D echocardiography showed dilation of RA (RA area index 12.8 cm2/m2) and RV; D: severe TR with TR Vmax 4.5 m/s with high probability of PH with PASP of 93 mmHg .RA: right atrial; RV: right ventricular; TR :tricuspid regurgitation; PH: pulmonary hypertension; PASP : pulmonary artery systolic pressure.

approaches, but with different antiarrhythmic (AAD) agent classes. The administration of AAD classes depends on the underlying arrhythmogenesis mechanism of each case according to the guidelines.[6,7]

In case 1, atrial flutter with 2:1 AV conduction and RBBB morphology is the type of SVA. The arrhythmogenesis origin of this SVA type arises from re-entry of an electrical circuit that is proposed to be caused by RA dilation via the cavotricuspid isthmus (CTI). Hence, amiodarone is chosen as an AAD in this circumstance because class III AAD will have prolonged action potential duration especially in phase III repolarization. This pharmacological mechanism will break the reentry circuit that formed and will terminate arrhythmia. Amiodarone is favorable and safe compared to other AAD in patients with structural heart disease.[5,8]

In the second and third cases, the patients develop AVNRT with unstable hemodynamics in case 2 but with respiratory distress in case 3. Unfortunately, given that both patients were still conscious in case 2 and 3, they were refused to electrically cardioverted due to personal reason; therefore pharmacological approach to manage the case undertaken. The class IV AAD, nondihydropyridine calcium channel blocker (CCB) is the drug of choice. Considering the arrhythmogenesis origin of AVNRT is a reentry process in the AV node via slow and fast pathway.[4,6] The conductive cells in AV nodes pose an action potential property of pacemaker cells. Hence, the influx of calcium is the crucial process of cell depolarization (phase 0). Administration intravenous nondihydropyridine CCB will terminate the micro-reentry electrical circuit in the AV node due to phase 0 rapid calcium influx suppression. A meta-analysis showed that conversion rate of non-dihydropyridine CCB does not differ significantly with adenosine administration in AVNRT patients. The limitation of nondihydropyridine CCB is its usage in patients with reduced EF is contraindicated.[4]

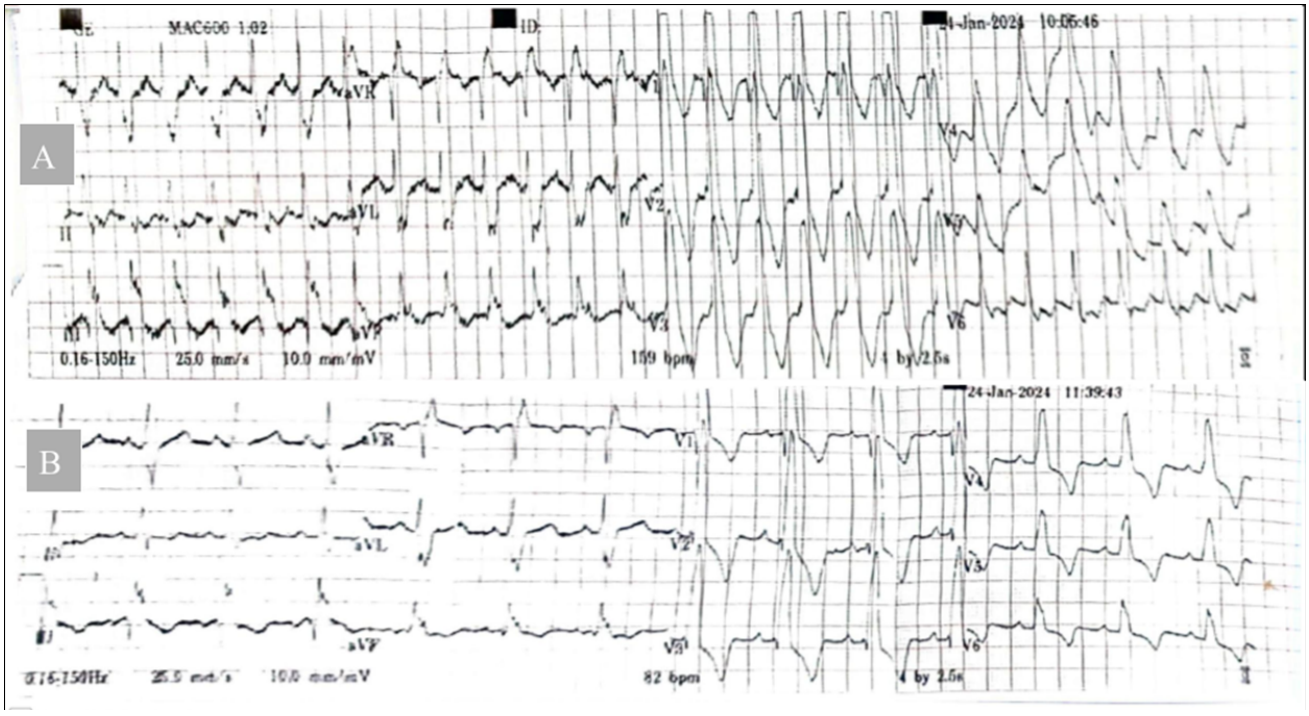

Figure 5. Electrocardiography in case 3. A: Initial ECG presentation at emergency ward showed wide complex tachycardia and RBBB morphology B: Post pharmacological cardioversion ECG showed sinus rhythm with RBBB. ECG: electrocardiography; RBBB: right bundle branch block.

All cases demonstrate successful rhythm conversion after AAD administration, which is followed by immediate symptom improvement. Supraventricular tachyarrhythmia in pre-existing RA enlargement poses a significant hemodynamic burden. Many reports which describe that SVA could precipitate acute RV dysfunction in PH, this situation prompted an early recognition and management to improve patient survival.[3,8,9,10]

There is a bidirectional relationship between PH and SVA, whereas RA dilation predisposes and triggers patients to develop SVA.[1,5,11] Waligóra et al found that in patients with PAH, the RA enlargement is associated with increased occurrence of SVA. In multivariate analysis, RA area index (RAai) is the only significant predictor of clinically significant SVA in PH (HR 1.23, $p < 0.001$). RAai value > 21.7 cm^2/m^2 becomes a discriminator for predicting significant SVA.1 On contrary our patiens RAai value were less than 21.7 cm^2/m^2. This finding highlighting that even in lower than referenced RAai value that mentioned, SVA is occurred in this patient population. Rajpold et al also reported that there is a significant association between RA dilation and SVA occurrence.[11] On the other hand, SVA becomes the precipitant factor for hemodynamic decompensation in previously enlarged RA or PH. These populations warrant for special attention, because correcting the precipitant cause of SVA will improve patient survival. The restoration and long-term sinus rhythm maintenance are of paramount importance in this special population, considering the lower survival in patients who developed atrial flutter or fibrillation compared to those who remained in sinus rhythm.[5,8,11.]

Conclusion

Supraventricular arrhythmia is a frequent arrhythmia that occurs in pulmonary hypertensive and RA dilation patients. The tachyarrhythmia in this patient population tolerated poorly and led to hemodynamic perturbation. Pharmacological cardioversion is one of the effective approaches to alleviate patient symptoms with significant clinical improvement.

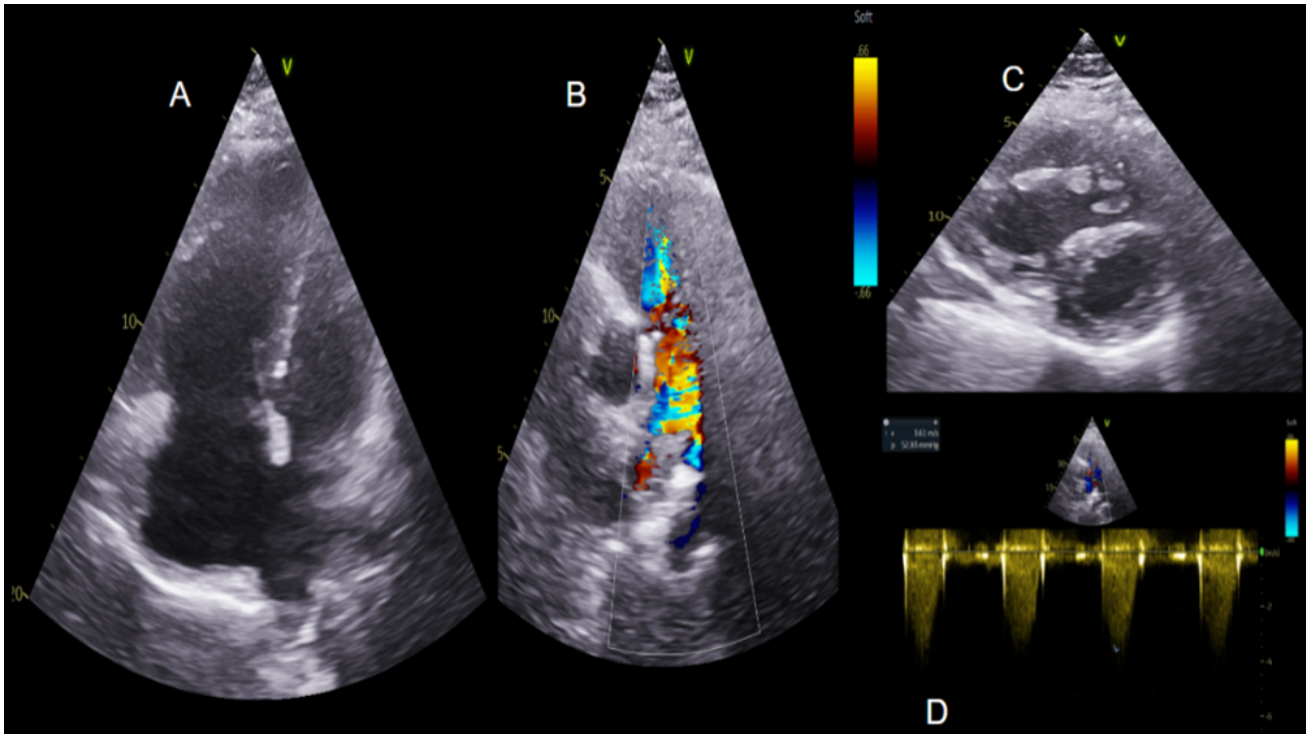

Figure 6. Case 3 2D echocardiography A and C: RA-RV dilation; B and D: Short axis view 2D echocardiography at great arteries level showed pulmonary stenosis valvar type with peak pulmonary valve gradient 52 mmHg. RA: right atrial; RV: right ventricular.

List of Abbreviations

AAD	Antiarrhythmic drugs
AVNRT	Atrioventricular-node reentrant tachycardia
AVRT	Atrioventricular reentrant tachycardia
AT	Atrial tachycardia
CCB	Calcium channel blocker
ECG	Electrocardiography
EF	Ejection fraction
LV	Left ventricular
ICU	Intensive care unit
PASP	Pulmonary artery systolic pressure
PAH	Pulmonary arterial hypertension
PH	Pulmonary hypertension
RA	Right atrial
RAai	RA area index
RBBB	Right bundle branch block
RHC	Right heart catheterization
RV	Right ventricular
SVA	Supraventricular arrhythmia
TR	Tricuspid regurgitation
TTE	Transthoracic echocardiography

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