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Spontaneous conversion to sinus rhythm in atrial fibrillation after dual antiplatelet and anticoagulant therapy in patients with unstable angina



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ABSTRACT

Introduction: Atrial fibrillation (AF) is a common arrhythmia and often becomes persistent with a high risk of thromboembolism event. Spontaneous conversion to sinus rhythm can occur in 50% of cases with new-onset AF. In this case report we report the spontaneous conversion of AF to sinus rhythm in patients with Unstable Angina without any thromboembolic complications.

Case description: A 65-year-old man with unstable angina pectoris (UA) with new-onset atrial fibrillation normal ventricular rate (AF-NVR) came to the Emergency Department Bali Mandara General Hospital. Patients have a history of uncontrolled hypertension and active smokers. Standard management of UA using dual antiplatelet, nitrates, and anticoagulants was given to

this patient. There were no anti-arrhythmia drugs given to seek for AF cardioversion. Within 6 hours after initial therapy, spontaneous conversion of AF to sinus rhythm occurs. The patient was treated for five days in a stable condition without thromboembolic complications. Long-term anticoagulants were not given to patients because the CHA₂DS₂-Vasc score is less than two indicating a low risk of thromboembolism.

Conclusion: New-onset AF has the chance of spontaneous conversion to sinus rhythm within 48 hours, proper management of the trigger factors of AF and optimal rate control are determinants of prognosis. Provision of long-term anticoagulants must be based on the CHA₂DS₂-Vasc score.

Keywords: atrial fibrillation, spontaneous, conversion, acute coronary syndrome

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INTRODUCTION

Atrial fibrillation (AF) is the most common arrhythmia and requires long-term therapy to prevent complications related to possible thromboembolism that can occur. In the United States, the prevalence reached 33 million population in 2010. AF is a very important focus by this condition increasing cardiovascular deaths due to heart failure, increasing 20-30% risk of stroke due to thromboembolism, increasing hospitalization by 10-40%, and reduce quality of life.^{1,2}

The strategy for newly diagnosed AF in less than 48 hours is to return it to sinus rhythm, either through direct-current cardioversion or pharmacological cardioversion techniques. However, there is a risk of thromboembolism that is often associated with cardioversion procedures. The first episode AF that occurs has a 51-70% chance to return to sinus rhythm spontaneously.^{3,4}

A study by Alasady et al.⁵ found among 149 patients with acute coronary syndrome (ACS), 4.9% developed having AF within the first week. The presence of AF in ACS is associated with a higher

outcome of major cardiovascular events (MACCE) compared to ACS patients who have sinus rhythm (SR).⁶ The presence of AF in the event of an acute coronary syndrome may have a causal relationship, although the mechanisms underlying it are not fully understood in detail. Is it ischemia that triggers the occurrence of AF or worsens existing AF or vice versa the AF that triggers ACS through a prothrombotic mechanism.⁸ Current case report will be presented a new-onset AF that undergoes spontaneous conversion in patients with Unstable Angina (UA).

CASE DESCRIPTION

A 65-year-old man comes to the emergency department Bali Mandara General Hospital with acute chest pain that is felt 3 hours before admission to hospital, the chest pain were felt blunt in the central part of the chest with penetrating sensation to the back with radiating pain to the neck and accompanied with cold sweating. Patients have many risk factors for coronary heart disease (CAD) such as uncontrolled hypertension for

approximately 10 years, which has recently been treated with Valsartan 80 mg for the last two years. Another risk factor is smoking one pack for three days. History of systemic conditions such as diabetes, previous stroke was denied. Examination of vital signs obtained blood pressure of 100/60 mmHg, irregular heart rate of 86 bpm. Irregular heart sound and no murmurs or thrills were found. Electrocardiogram (ECG) records found an irregular atrial rhythm, rate of 90 bpm with a duration of QRS 80 ms without the presence of concomitant ST-T segment changes (Figure 1). The blood chemistry panel showed insufficient increase in cardiac markers (Quantitative Troponin I <50 ng / mL and CK-MB = 12 U/L), other blood parameters were also within normal limits (Table 1). Chest X-ray results showed a 58% cardiothoracic ratio

with concomitant grounded heart apex suggestive for left ventricular hypertrophy (Figure 2). Based on these findings patients were diagnosed with UA with new-onset AF normal ventricular rate (AF-NVR).

Oral loading dose of aspirin (325 mg) and clopidogrel (300 mg) followed by daily doses of 80 mg and 75 mg respectively, isosorbide dinitrate 5 mg, atorvastatin 20 mg o.d, trimetazidine 35 mg b.i.d, pantoprazole 40 mg intravenous, and subcutaneous dose of sodium enoxaparin twice daily is given as part of the management of UA (non-ST segmentation acute coronary syndrome). After initial treatment, there is an improvement in chest pain symptoms and patient was admitted to intensive coronary care unit for further intensive monitoring.

Approximately 6 hours after initial therapy, there is a spontaneous conversion of cardiac rhythm from AF NVR to SR with a pulse of 60 bpm with a duration of QRS 90 ms (Figure 3). The patient is hemodynamically stable with no shock or bradycardia. On the following day the patient's blood pressure was 150/100 mmHg, and was given an additional drug laxative lubricant, nebivolol 5 mg o.d, valsartan 80 mg o.d, and no symptoms of repeated chest pain were reported nor any changes in ECG rhythm. Patients were discharged on the fifth day with home medication aspirin 80 mg and clopidogrel 75 mg o.d, nebivolol 5 mg o.d, atorvastatin 20 mg o.d, valsartan 80 mg once daily, long-term anti-coagulant was not given to this patient. On 7th day after discharge from hospital the patient controls the cardiology outpatient clinic without any atrial fibrillation findings on ECG examination.

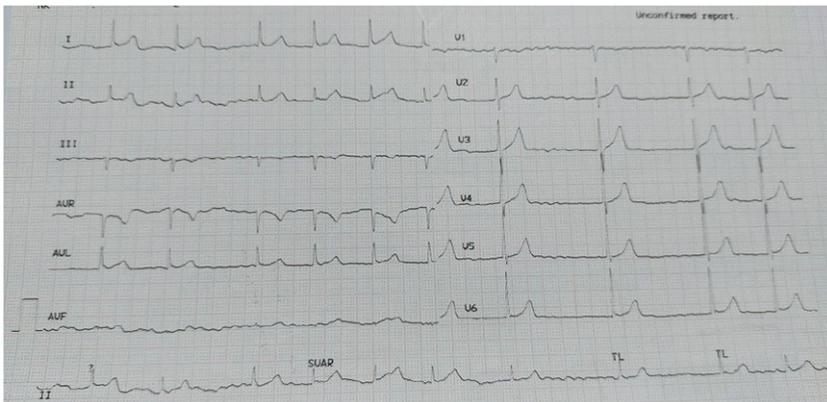


Figure 1. First ECG findings in emergency department with AF rate of 90 bpm, QRS complex duration of 80 ms without changes in ST-T segments



Figure 2. Chest x-ray showing an increase in cardiothoracic ratio (CTR) with sign of left ventricular hypertrophy (grounded apex)

DISCUSSION

An acute coronary syndrome is a clinical spectrum caused by impaired coronary circulation blood flow which is broadly divided into ST-Elevation myocardial infarction and Non-ST-ACS (NSTEMI and UA).⁹ The occurrence of ACS is associated with the presence of atherosclerotic plaque rupture which results in the formation of a total or partial occlusion thrombosis. While AF is an episode of acute arrhythmia that is rapid, irregular, and chaotic atrial activity that is new.¹ The primary keys in controlling AF are prevention of rate or rhythm control, management of the underlying disease (in this case is UA), assessment of stroke risk and thromboembolism prophylaxis, and symptoms severity assessment.¹⁰

Based on recommendations from the European Society of Cardiology (ESC guidelines for management of atrial fibrillation) in acute atrial

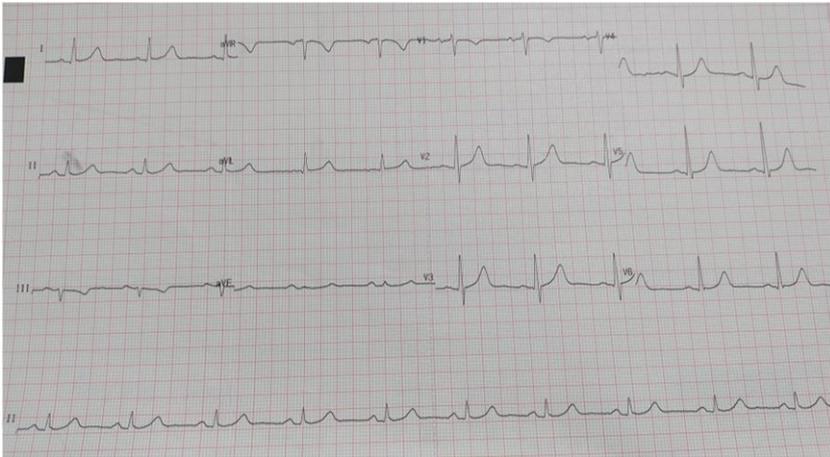


Figure 3. ECG findings of spontaneous conversion to sinus rhythm from AF-NVR approximately six-hour after initial therapy

Table 1. CBC and blood chemistry panel at first admission

Laboratory Parameters	Values
Haematology complete blood count	
WBC	9.83x10 ³ /μl
#Neu	6.35x10 ³ /μl
#Lym	4.32x10 ³ /μl
#Mono	0.644x10 ³ /μl
#Baso	0.122x10 ³ /μl
#Eos	0.072x10 ³ /μl
MPV	4.66 fL
Platelet	297x10 ³ /μl
Hb	11.8 g/dL
HCT	38.6%
MCV	87 fL
MCH	28.6 pg
MCHC	33.7 g/dL
RDW	13.2%
Kidney function (mg/dL)	
Ureum	29
Creatinine	1.4
Electrolyte (mmol/L)	
Na ⁺	138
K ⁺	3.8
Cl ⁻	110
Liver function test (U/L)	
SGOT	26
SGPT	18

Abbreviations: CBC, complete blood count; MPV, mean platelet volume, Hb haemoglobin; WBC, white blood cell; HCT, hematocrit; MCH, mean corpuscular haemoglobin; MCHC, mean corpuscular haemoglobin concentration; RBC, red blood cell; RDW, red cell distribution width; SGOT, serum glutamic oxaloacetic transaminase; SGPT, serum glutamic pyruvic transaminase.

fibrillation with hemodynamic instability requires immediate electrical cardioversion.² Meanwhile, in AF conditions without hemodynamic instability, optimizing rate control is treatment of choice using medical therapy with a target heart rate of less than 110 bpm (verapamil/diltiazem, beta-blockers, digoxin).^{2,10,11} Cardioversion for AF patients has a risk for a thromboembolic event, because of the transient atrial stunning that makes it easy for thrombus formation and then when the atrial kick returns to normal, the thrombus that has formed will become a thromboembolism.^{7,12} In a case reported by Yan et al.¹³ thromboembolic events occurs in AF patients undergoing spontaneous cardioversion caused by severe hyperkalemia treated with triple-drug therapy (calcium gluconate, oral kayexylate, rapid-acting insulin with 10% dextrose) and emergency hemodialysis, spontaneous conversion occurs in less than 48 hours after normalizing serum potassium and patient experiencing ischemic stroke.

Acute coronary syndrome has become a risk factor for AF with an incidence ranging from 6%-21% in ACS patients.^{8,12} The high incidence of AF in ACS may be secondary to neurohormonal factors that occur and affect the atrial substrate due to ischemia. Structural remodeling of the atria structure is key in the onset of AF. Activation of fibroblasts, atrial fatty infiltrate, myocyte hypertrophy, and, inflammatory infiltrate are the initial hallmark process of atrial remodeling.² The results of structural remodeling cause electrical dissociation in the muscle bundle, which causes re-entry and repetition of arrhythmias combined with shorter atrial refractory periods causing arrhythmias in AF to be persistent.¹²

Other hypotheses underlying such phenomenon may be due to the presence of thrombosis that occurs in the branches of the coronary arteries that supply blood to the sinus nodes and atria.⁵ This is supported by the findings of the case report by Sharma et al.¹⁴ which shows the spontaneous conversion of AF-NVR to sinus rhythm after percutaneous coronary intervention in the significant occlusion of right coronary artery.

The possibility of spontaneous conversion can occur in 50% of new-onset AF within 48 hours.^{1,12} The study conducted by Daniaz et al.¹⁶ in 365 patients with atrial fibrillation (40% of patients with prior history of AF and 60% of patients with new-onset AF), found spontaneous conversion to sinus rhythm in 242 patients (64%) in a duration of less than 24 hours. The study conducted by Choundhary et al.¹⁶ showed a higher likelihood of AF with fpm <350 to experience spontaneous conversion to sinus rhythm within 18 hours (adjusted OR: 3.7; 95% CI 1.3-10.5; p = 0.016).

In this case the patient did not experience a thromboembolic event after spontaneous conversion to sinus rhythm, long-term oral anticoagulants were not given because the CHA₂DS₂-Vasc score was less than two, thus indicating low-risk systemic thromboembolism.

CONCLUSION

Spontaneous conversion may occur in patients with new-onset AF, appropriate treatment of underlying disease and optimal rate control provide a good prognosis. The choice of cardioversion or rate control depends greatly on the clinical condition of the patient. Anticoagulant recommendations are highly recommended based on the results of the CHA₂DS₂-Vasc score.

CONFLICT OF INTEREST

The author declares there is no conflict of interest regarding publication of current case report.

ETHICAL CONSIDERATION

Patient had received information and signed informed consent regarding data publication prior to any data collection.

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