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### A Case Report of 80-Year-Old Male with Total Atrioventricular Block, Acute Decompensated Heart Failure, and Non-ST-Elevation Myocardial Infarction Very High Risk: A Complicated Case in Limited Resources Setting

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#### ABSTRACT

**Background:** Cardiovascular disease is still a major problem globally, with high morbidity and mortality rates. The progression of cardiovascular disease is so dynamic that it can leave the patient in a terminal condition. Given the high mortality and morbidity accompanied by rapid disease progression, understanding the appropriate management of each patient with comorbid heart disease is very important. **Case presentation:** We report an 80-year-old male with TAVB (Total Atrioventricular Block), ADHF (Acute Decompensated Heart Failure), and NSTEMI (Non-ST-Elevation Myocardial Infarction). The patient was treated and stabilized intensively in our center based on protocol. Due to the rapid progression of the disease, we referred the patient to the tertiary hospital in Bali for further advanced treatment. **Conclusion:** The mortality rate in cases of cardiovascular diseases is relatively high, requiring rapid and adequate treatment to provide better clinical outcomes.

#### 1. Introduction

Cardiovascular disease is still a major problem globally, with high morbidity and mortality rates. According to the World Health Organization (WHO), as many as 17.9 million deaths in 2019 were caused by cardiovascular disease, which accounted for 32% of the causes of death in the world. More than three-quarters of deaths from cardiovascular disease occur in low- and middle-income countries.<sup>1</sup> In Indonesia, in 1992-2000, the proportion of deaths from cardiovascular disease increased quite sharply. Based on data from the Household Health Survey (SKRT) in 1992, the death rate reached around 16.4% and continues to increase from year to year. From 1995 to

2001, the death rate increased from 24.5% to 26.4%.<sup>2</sup> According to Basic Health Research (Riskesmas) in 2013, the highest prevalence of cardiovascular disease in Indonesia was coronary heart disease at 1.5%, where the highest incidence was (3.6% in the age group 65-74 years, followed by the age group over 75 years as much as 3.2%.<sup>3</sup>

The progression of cardiovascular disease is so dynamic that it can leave the patient in a terminal condition. Given the high mortality and morbidity accompanied by rapid disease progression, understanding the appropriate management of each patient with comorbid heart disease is very important. In this case report, the author describes a patient with

complex heart disease in the form of total atrioventricular block (TAVB), acute decompensated heart failure (ADHF), and non-ST-elevation Myocardial Infarction (NSTEMI) very high risk, which was encountered in one peripheral hospital in Bali.

## 2. Case Presentation

An 80-year-old male came to the emergency room (ER) with chief complaints of shortness of breath for

more than six hours before the examination, and it was getting worse. He also had a cough, nausea, and vomiting. The patient felt weakness in his body. The patient had a history of chronic heart failure (CHF) et causa coronary artery disease, atrial fibrillation, with an ejection fraction (EF) of 39%, which was routinely controlled through the polyclinic. He used the cardiac medication, including Candesartan 8 mg, Bisoprolol 2.5 mg, Spironolactone 25 mg, and Warfarin 4-6 mg.

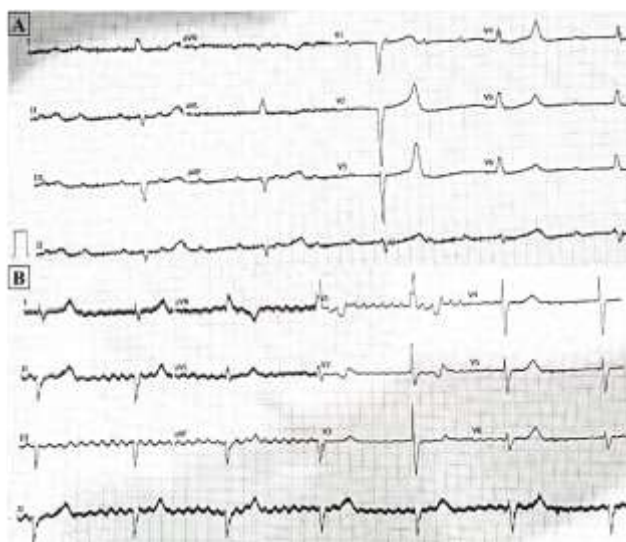


Figure 1. ECG results. (A) Examination in the ER. (B) Follow-up on the third day of treatment.

The patient was in a severely ill condition, fully alert (*compos mentis*), with normotension (110/70 mmHg), bradycardia (heart rate/HR was 30 beats per minute), dyspnea (respiratory rate was 26 times per minute), and desaturation (peripheral oxygen

saturation was 92% on room air). An electrocardiogram examination (Figure 1) revealed TAVB. Chest X-ray showed cardiomegaly with congestive pulmonum (Figure 2). Laboratory and electrolyte tests depicted normal results.



Figure 2. Chest X-ray result.

On initial assessment in the ER, he was diagnosed with symptomatic bradycardia et causa TAVB with ADHF Profile B. The patient was given Sulfas Atropine 0.5 mg bolus intravenously every five minutes up to a maximum dose of 3 mg with a target HR of >50 beats per minute. Then, he was observed in the intensive care unit with Oxygen 6 liters per minute (lpm) via a non-rebreathing mask, IVFD Futrolit 8 drips per minute, Furosemide 5 mg/hour, Candesartan 8 mg, Dopamine 3 mcg/BW/minutes, Spironolactone 25 mg, Lansoprazole 30 mg, and Amiodarone 400 mg.

On the second day of follow-up treatment, the patient planned to be referred to a tertiary hospital due to no significant increase in heart rate or clinical improvement. However, the patient's family refused, so the patient was still treated at our center.

On the third day of follow-up, the patient complained of chest pain accompanied by severe shortness of breath. Vital sign examination revealed hypotension (51/35 mmHg), bradycardia (38 beats per minute), and dyspnea (peripheral oxygen saturation was 93% on O<sub>2</sub> 6 lpm). Troponin T examination increased with a value of 111.31 pg/mL. The patient was diagnosed with NSTEMI, persistent TAVB, ADHF profile C. He treated with Norepinephrine drip with starting dose of 0.1 mcg/BW/minute, Acetylsalicylic Acid 80 mg, Clopidogrel 75 mg, Lovenox 0.4 cc subcutaneous, Spironolactone 25 mg, Lansoprazole 30 mg, Dopamine 5 mcg/BW/minute, stop Furosemide drip, and the patient referred to a tertiary hospital.

### 3. Discussion

TAVB is a total failure of conduction between the atria and ventricles, which caused no connection between the P wave and the QRS complex.<sup>4</sup> Many diseases and conditions can affect the AV conduction system, including congenital and acquired diseases caused by infection, inflammation, degenerative, ischemic, metabolic, and iatrogenic. The most common causes of TAVB are degenerative and ischemic causes. NSTEMI and unstable angina (UAP) also cause AV block in some cases. The hemodynamic

profile often worsens due to TAVB, requiring rapid recognition and treatment.<sup>5,6</sup> Determining the possible cause of TAVB is important because, in most cases, it can be resolved once the potentially reversible cause is corrected and placement of a temporary or permanent pacemaker can be avoided. In contrast, irreversible TAVB requires rate regulation, especially if the level of blockade is intra-His or infra-His, which does not respond to drug therapy.<sup>7,8</sup>

In general, patients with first- or second-degree Mobitz type 1 AV block do not require treatment. However, patients with higher degrees of AV block (Mobitz type 2 AV block, grade 3/TAVB) tend to have severe damage to the conduction system. They usually have a greater risk of developing asystole, ventricular tachycardia, or sudden cardiac death. Therefore, these patients require immediate care for cardiac monitoring, further evaluation, consideration of temporary pacing on a case-by-case basis, and ultimately, implantation of a permanent pacemaker.<sup>4,9</sup>

Management of TAVB, in this case, requires the implantation of a pacemaker that is unavailable at our center. Furthermore, the atropine injection could not change the rhythm or even improve the heart rate. The responsiveness of TAVB to therapy is highly dependent on the block's location. Based on the block's location, the AV block is classified into AV nodal, also called supra-His, intra-His, and infra-His. Supra-His or intra-His AV block mostly shows a narrow QRS complex, whereas infra-His shows a wide QRS complex.<sup>10</sup> Generally, supra-His block develops slowly, has a faster AV uncoupling mechanism, and has a greater response to autonomic manipulations such as atropine and epinephrine. Intra-His or infra-His AV block develops rapidly and unpredictably, is associated with a slower and unpredictable ventricular escape mechanism, and will not respond to atropine but sometimes improves with catecholamines.<sup>11,12</sup>

Our patient had a history of CHF, which faced an acute exacerbation, then was diagnosed with ADHF profile B. ADHF is a rapid change in symptoms and signs of heart failure. It can be a life-threatening condition that requires immediate medical attention

and usually results in hospitalization. Hospitalization for ADHF is recommended when the patient experiences dyspnea at rest, usually characterized by tachypnea at rest or oxygen saturation of less than 90%. Patients should also be hospitalized if they show signs or symptoms of low cardiac output, including hypotension, worsening kidney function, or changes in mental status. Any patient with hemodynamically significant arrhythmias (i.e., atrial fibrillation with rapid ventricular response) or acute coronary syndrome should be hospitalized.<sup>13,14</sup> Our patient was accompanied by bradyarrhythmia and desaturation, so treatment was carried out in the intensive care unit.

Most ADHF patients experience congestion due to volume overload, vascular redistribution, or both. The goal is to reduce filling pressure and relieve symptoms through diuresis, vasodilation, or both. Several recent trials have proven the detrimental effects of hypotension in ADHF. The diuresis rate should achieve the desired volume status without causing a rapid decrease in intravascular volume, which could lead to symptoms of hypotension or renal dysfunction.<sup>13,14</sup>

Current guidelines recommend intravenous administration of diuretics as first-line therapy for volume overload.<sup>15</sup> The loop diuretics, furosemide, bumetanide, and torsemide, are the initial diuretics of choice in ADHF.<sup>16,17</sup> Recently, preliminary results from the dopamine in acutely decompensated heart failure II (DAD II) Trial showed no differences between high-dose furosemide, low-dose furosemide, and low-dose furosemide plus dopamine, in the case of mortality or readmission to ADHF.<sup>18</sup> The patient in this case was treated with diuretics in the form of Furosemide 5 mg/hour and Spironolactone 25 mg. Apart from that, the patient was also given Dopamine 3 mcg/KgBB/minutes.

Patients with comorbid heart disease may experience worsening symptoms. One of them is experiencing Acute coronary syndrome (ACS). ACS is divided into ST-segment elevation myocardial infarction (STEMI), NSTEMI, and UAP subgroups. ACS carries significant morbidity and mortality, and

prompt diagnosis and appropriate treatment are essential. NSTEMI is diagnosed in patients with symptoms consistent with ACS and elevated troponin but without ECG changes consistent with STEMI. Unstable angina and NSTEMI differ primarily in the presence or absence of detectable troponin leaks.<sup>19,20</sup> Our patient experienced disease progression during treatment. On the third day, he experienced chest pain and an increase in Troponin T. In addition, the patient experienced hemodynamic instability accompanied by persistent bradyarrhythmia. Thus, it was categorized as a very high-risk NSTEMI.

In patients with a confirmed diagnosis of NSTEMI, anticoagulation should be initiated. Protocols vary by institution, so a cardiology consultation should be sought if available. Unfractionated heparin in bolus doses and continuous infusion is commonly used, and most institutions have protocols available. Other strategies may include enoxaparin, bivalirudin, fondaparinux, and dual antiplatelet therapy.<sup>19,20</sup> Our patient was initially treated with a Norepinephrine drip with a starting dose of 0.1 mcg/BW/minute, Acetylsalicylic Acid 80 mg, Clopidogrel 75 mg, Lovenox 0.4 cc subcutaneous. The patient was then referred for further treatment to a tertiary hospital.

#### **4. Conclusion**

Our patient finally received intensive care at a tertiary hospital after initial treatment and stabilization at our center. Due to the high mortality rates in cases of cardiac diseases, it is mandatory to carry out adequate treatment when this case is first encountered in the emergency department to provide better clinical outcomes.

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