

Reversible total atrioventricular block in a very high-risk non-ST-elevation myocardial infarction (NSTEMI) during conservative treatment in a limited resource setting: a case report

Susanti Mareta Anggraeni*, Ruth Grace Aurora

Jailolo General Hospital, North Maluku, Indonesia

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ABSTRACT

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Total atrioventricular (AV) block is the most common type of conduction disorder found in acute coronary syndrome (ACS), which requires timely recognition and treatment. This case report aimed to present conservative medical treatment for managing total AV block (TAVB) in a very high-risk non-ST-elevation myocardial infarction (NSTEMI) in a rural area. We reported a patient with TAVB in a very high-risk NSTEMI. The patient was hemodynamically unstable and needed immediate percutaneous coronary intervention (PCI). Due to limited facilities and difficult access to immediate PCI, the patient was treated conservatively with the NSTEMI protocol. Epinephrine, as a β -adrenergic agonist, was administered to improve hemodynamic status. During conservative treatment, TAVB was converted into the first-degree AV block on the third day of intensive care. In a setting where revascularization strategies and pacemaker implantation are not feasible, administration of antithrombotic agents and β -adrenergic agonists can be considered to manage TAVB with NSTEMI with close monitoring.

ABSTRAK

Blok atrioventricular (AV) total merupakan bentuk gangguan konduksi jantung yang paling sering ditemukan pada kasus sindrom koroner akut (SKA). Laporan kasus ini bertujuan menjabarkan tatalaksana konservatif yang dapat dilakukan pada kasus blok AV total pada infark miokard akut non-segment ST elevasi (IMA-NEST) dengan risiko sangat tinggi di rumah sakit dengan fasilitas terbatas. Kami melaporkan pasien dengan blok AV total pada IMA-NEST dengan risiko sangat tinggi. Pasien datang dalam keadaan hemodinamik tidak stabil dan memerlukan akses untuk intervensi koroner perkutan (IKP) segera. Oleh karena keterbatasan fasilitas dan sulitnya akses untuk IKP segera, dilakukan tatalaksana konservatif dengan protokol IMA-NEST. Kami menggunakan epinefrin sebagai agonis β -adrenergik untuk memperbaiki status hemodinamik pasien. Selama perawatan konservatif tersebut blok AV total terkonversi menjadi blok AV derajat 1, yaitu pada perawatan intensif hari ketiga. Pada kondisi revaskularisasi dan pemasangan alat pacu jantung tidak dapat dilakukan, pemberian antitrombotik dan agonis β -adrenergik dapat dipertimbangkan sebagai terapi konservatif pada pasien blok AV total dengan IMA-NEST.

Keywords:

case report;
conservative treatment;
non-ST-elevation myocardial infarction;
total AV block;
 β -adrenergic agonists

INTRODUCTION

Acute coronary syndrome (ACS) was one etiology of atrioventricular (AV) conduction disorders or AV block. This blockage can manifest from first

degree to total AV block (TAVB). The previous study reported that 1.9% of all ACS patients presented with TAVB.¹⁻³ Total occlusion in ST-elevation myocardial infarction (STEMI) was the most common acute coronary

*corresponding author: susantimaretaanggraeni@gmail.com

event to be reported as the cause of AV block. Nevertheless, non-ST-elevation myocardial infarction (NSTEMI) and unstable angina (UA) also cause AV block in several cases. Hemodynamic profile was often deteriorated due to TAVB and thus required timely recognition and treatment.⁴

CASE

A 58 y.o. man came to the emergency department with the chief complaint of heartburn 2 hr before admission to the hospital. Other accompanying symptoms were dizziness, nausea, vomiting, and diaphoresis. Five days earlier, the patient came to the emergency department with a complaint of epigastric pain and nausea. There was no ECG recorded. He was diagnosed with dyspepsia and treated as an outpatient. The risk factors identified were type-2 diabetes mellitus (T2DM) without routine treatment and smoking for 30 yr.

There were indicators of hypoperfusion on the initial observation. The blood pressure (BP) was 60/40 mmHg, the heart rate was 24 beats per min and the distal extremities were cold. There was no previous medication identified to cause bradycardia. The heart auscultation revealed that S1 and S2 were normal and irregular without murmurs or gallops. Lung examination showed vesicular on both lungs, no rales or wheezing were found.

The electrocardiogram (ECG) showed a TAVB pattern with ST-segment depression in leads V4-V6 (FIGURE 1). Two times, 1 mg of atropine sulphate was administered, but there was no improvement in the rhythms. The fluid challenge of 250 mL crystalloid solution was then conducted after signs of congestion were excluded by lung auscultation. After the first fluid challenge, the BP was slightly increased (70/40 mmHg), and then the second fluid challenge was given.



FIGURE 1. Initial ECG at Emergency Department presented total AV block with junctional escape rhythm. Blue arrow: visible P wave; orange arrow: possible P wave buried in T wave; red arrow: possible P wave buried in QRS wave; black arrowhead: narrow QRS wave as junctional escape wave.

TABLE 1. Laboratory work up at the Emergency Department

Laboratory parameters	Flag	Results	Normal value
Haemoglobin (g/dL)	L	13.0	14.0-18.0
Haematocrit (%)	L	38.2	42-54
Erythrocyte (/mm ³)	L	4.33 x 10 ⁶	4.5-6.5 x 10 ⁶
Thrombocyte (/mm ³)	N	230 x 10 ³	150-450 x 10 ³
Leucocyte (/mm ³)	N	9.4 x 10 ³	4.5-11.0 x 10 ³
Neutrophile (%)	L	34	54-62
Lymphocyte (%_	H	56.8	25-33
Random plasma glucose (mg/dL)	H	345	<200
Troponin I (ICU) (ng/mL)	H	4.51	<1

Laboratory data (TABLE 1) revealed high random blood glucose (345 mg/dL) and elevated troponin I levels (4.51 ng/mL). The troponin could not be measured at the time of admission due to technical obstacles in our hospital. Due to the reagent's limitations, a serial troponin level measurement could not be achieved. The electrolyte levels of Na and K were within the normal limit. He was assessed for cardiogenic shock due to TAVB, TAVB due to a very high-risk NSTEMI, uncontrolled T2DM, and acute kidney injury (AKI) with a differential diagnosis of chronic kidney disease (CKD). Considering the fact of high-grade AV block with signs of hypoperfusion, it was decided to give epinephrine at 0.1 µg/kg/min, titrated to achieve hemodynamic stability.

The patient met the criteria for pacemaker implantation and immediate percutaneous coronary intervention (PCI), but since there was no catheterization laboratory in our province, only conservative treatment was given to the patient. Medications given during hospitalization were epinephrine, heparin, dual anti-platelet therapy (DAPT), statin, and other symptomatic treatments. Unfractionated heparin (UFH) was used with loading dose 60 unit/kg followed by maintenance dose 12 unit/kg/hr for 5 d. Anti-platelet

therapy monitoring was not available in our setting, so we monitor the adverse effect of UFH use, such as bleeding (melena, epistaxis), purpura due to heparin-induced thrombocytopenia (HIT), and anaphylactic reaction of UFH. The use of DAPT consists of a loading dose of 320 mg aspirin and 300 mg clopidogrel at admission, followed by the DAPT daily dose of 80 mg aspirin and 75 mg clopidogrel. We used high dose atorvastatin (40 mg) at admission, followed by a daily dose of 20 mg.

The patient was transferred to the intensive care unit (ICU) for further treatment. Echocardiography revealed normal left ventricle (LV) function with an ejection fraction (EF) of 53%, grade I diastolic dysfunction, LV dilatation with eccentric LV hypertrophy (RWT 0.33), regional wall motion abnormality (RWMA) with infero-septal hypokinetic, normal RV contractility (TAPSE 2.1 cm), and mild mitral regurgitation (E/A < 1). The electrocardiogram was converted to first-degree AV block on day 3 of hospitalization (FIGURE 2A). Epinephrine was subsequently down-titrated until it was finally stopped on day 5 of hospitalization, then he was transferred to the regular ward. On day 8 patient was discharged, and ECG showed sinus rhythm with first-degree AV block (FIGURE 2B).

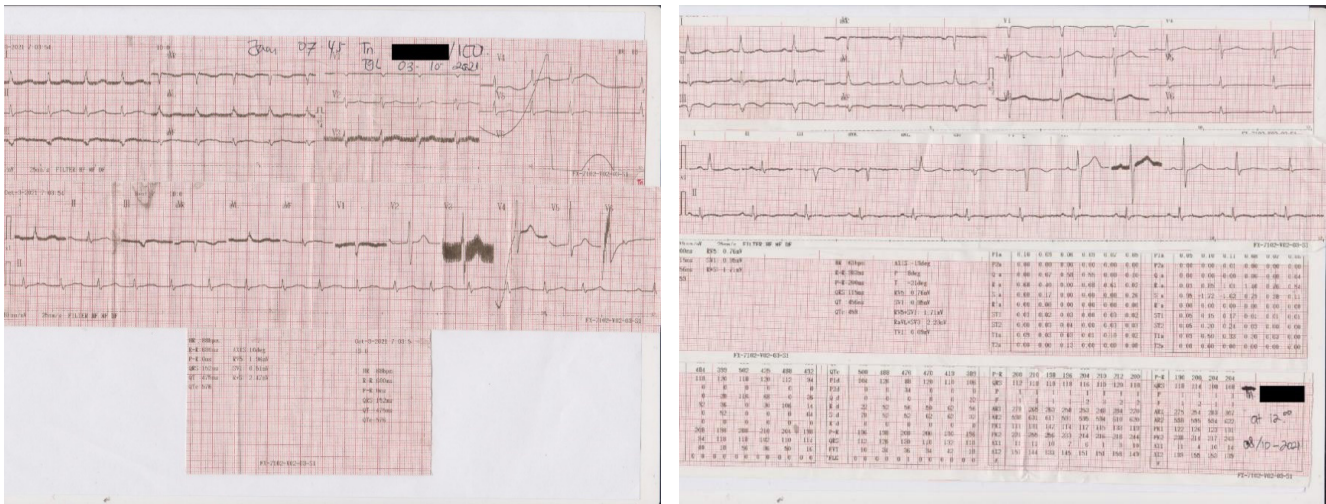


FIGURE 2. A) ECG on day 3 of admission; B) ECG on day 8 of admission

DISCUSSION

The case of TAVB in NSTEMI is not commonly found.^{4,5} The AV node was supplied by the right coronary artery (RCA) in 90% of patients (AV nodal branch); and by the left circumflex artery (LCx) in 10% of patients. Therefore, inadequate RCA blood flow would disrupt the conduction system. Two mechanisms that are hypothesized to induce AV block in NSTEMI are vagal and ischemic theories. Vagal theory is established from Bezold-Jarisch reflex. Unmyelinated C-fiber afferent receptors originating from the inferior and posterior walls are sensitive to mechanical and chemical stimuli. Chemical substances such as prostaglandin, serotonin, and free radicals are released from the infarct area, causing parasympathetic activity (hypotension and bradyarrhythmia).^{6,7} The second hypothesis is the ischemia theory. The AV node is commonly vascularized by RCA or LCx in the predominance of the left coronary artery. Meanwhile, the bundle of His and conducting fibers is then supplied by the left anterior descending artery (LAD) septal perforators and either RCA or LCx. Inadequate vascularization due to partial or total occlusion leads to disruption of the conduction system. Conduction

disorders were commonly found in inferior MI due to RCA occlusion.^{8,9}

Based on the 2020 ESC Guideline for the management of ACS in patients presenting without persistent ST-segment elevation, our patient was classified as a very high-risk NSTEMI due to the presence of cardiogenic shock and life-threatening arrhythmia. Therefore, an immediate invasive strategy should be conducted to manage our patient. Due to inadequate resources and no catheterization laboratory facilities in our area, this revascularization strategy was unfeasible. In TAVB with reversible causes, treating the etiology would resolve the conduction abnormality, as in thyroid disorders, Lyme disease, cardiac sarcoidosis, viral myocarditis, and intoxication of some heart medications.¹⁰ Apart from those etiologies, Acute coronary syndrome has been reported as a reversible cause of TAVB.¹¹⁻¹³

Conservative management was then conducted by our team. Initial DAPT and anticoagulants were administered to our patient based on standard medical therapy of ACS. Meanwhile, the management of TAVB was based on the 2018 ACC/AHA/HRS guideline. Bradycardia due to AV block with hemodynamic compromise is managed by removal of potential causative factors

concomitant with medical therapy. Second or third grade AV block should be managed by atropine administration to increase ventricular rate, improve AV conduction, and relieve symptoms. Aminophylline can be considered to be added if the etiology of AV block is acute inferior MI. Meanwhile AV block due to other causes can be managed by β -adrenergic agonists such as isoproterenol, dopamine, dobutamine, or epinephrine. Cardiac pacing is recommended if the AV block do not respond to medical therapy.

Epinephrine was chosen to improve the hemodynamic status in this case. Other β -adrenergic agonists may also be used, but in our setting, only epinephrine was available. Epinephrine acts on α - and β -adrenergic receptors. In high doses, epinephrine exerts action on α -1 receptors, increasing heart rate and myocardial contractility.¹⁴ Epinephrine is also preferred in a patient with a hypotensive state.¹⁵ Hemodynamic improvement was achieved by our patient after up-titrating the epinephrine dose with close monitoring. In this case, the ECG was converted into a first-degree AV block. It was suggested that restoration of perfusion in the setting of acute MI leads to improved conduction.

Symptomatic bradycardia in TAVB and NSTEMI should be managed altogether. In cases of myocardial ischemia, adequate reperfusion will improve electrical conduction. Implantation of cardiac pacing in acute MI is considered based on clinical presentation to avoid unnecessary pacemaker implantation. Implementation of the guideline as a standard of therapy should be accompanied by the availability of standard pharmacologic agents as well as the presence of cardiology specialists in every hospital in Indonesia. As an archipelago country, Indonesia has a lot of obstacles to equalizing health services, either in urban or rural areas. Therefore, government should arrange policies to

reduce the gap of inequalities in health services.

CONCLUSION

Standard medical treatment for TAVB with NSTEMI is reperfusion, followed by cardiac pacing as necessary. Due to lack of access to catheterization laboratory, conservative management based on both atrioventricular block and the NSTEMI guideline are conducted. In this case, antithrombotic agents and β -adrenergic agonists together improve cardiac conduction and hemodynamic status. In the setting of limited cardiovascular care, those antithrombotic agents and β -adrenergic agonists can be considered to manage TAVB with NSTEMI with close monitoring.

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