


Nonreperfused Inferior Wall Myocardial Infarction with Total Atrioventricular Block

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ABSTRACT

Inferior wall myocadial infarction (IWMI) with total atrioventricular block (TAVB) is associated with poor clinical status, such as right ventricular infarction (RVI). In IWMI patients with RVI and TAVB, coronary reperfusion using primary percutaneous coronary intervention (PCI) or fibrinolysis can preserve ventricular function and reduce mortality and morbidity. Bradyarrhythmia with hypotension and TAVB is one of the main indications to temporary pacing. This is a case of infero-posterior wall myocardial infarction, RVI, with TAVB. Primary PCI or fibrinolysis and temporary pacing was suggested, but the patient refused. Treatments were atropine sulfate and dopamine for bradycardia and hypotension. Dual-antiplatelet with aspirin and clopidogrel along with fondaparinux was given for antithrombotic therapy. The ECG showed resolution of STEMI and improvement in heart rhythm after 48h.

Keywords: Inferior wall myocadial infarction, nonreperfused patients, right ventricular infarction, total atrioventricular blocks

ABSTRAK

Infark miokardial dinding inferior dengan blok atrioventrikular total dikaitkan dengan status klinis buruk, seperti infark ventrikel kanan. Pada pasien infark miokardial dinding inferior dengan infark ventrikel kanan dan blok atrioventrikular total, reperfusi koroner dengan intervensi koroner perkutan primer atau fibrinolisis dapat melindungi fungsi ventrikel dan mengurangi mortalitas serta morbiditas. Bradiaritmia dengan hipotensi dan blok atrioventrikular total adalah salah satu prioritas utama pemasangan pacu jantung sementara. Kasus ini membahas pasien pria dengan infark miokard infero-posterior, infark ventrikel kanan, dengan blok atrioventrikular total. Pasien menolak intervensi koroner perkutan primer atau fibrinolisis serta pacu jantung sementara. Terapi atropin sulfat dan dopamin untuk bradikardia dan hipotensi; terapi antitrombotik berupa *dual-antiplatelet* dengan aspirin dan *clopidogrel* bersama *fondaparinux*. EKG menunjukkan resolusi STEMI dan perbaikan irama jantung setelah 48 jam. Pada pasien non-reperfusi, diperlukan antitrombotik yang tepat. **Patricia Feliani Sitohang. Infark Miokard *Nonreperfused Inferior Wall* dengan Blok Atrioventrikular Total**

Kata kunci: Blok atrioventrikular total, infark miokardial dinding inferior, infark ventrikel kanan, pasien non-reperfusi

INTRODUCTION

Conduction disturbances and bradyarrhythmias are well documented complications of myocardial infarction (MI).[1] Complete atrio-ventricular (AV) blocks occur in more than 5% of MI patients.[1-3] Inferior wall infarcts account for half of all the ST-segment elevation myocardial incfarction (STEMI).[1,2] Complete AV blocks complicating inferior wall myocardial infarction (IWMI) appear in 11% to 15% of all cases.[3]

Patients with acute inferior or posterior MI are more likely to develop conduction system abnormalities compared to patients with an anterior or lateral acute MI.[1] Bundle branches blocks were more common in anterior infarction but AV node block occurred almost exclusively in inferior infarction.[4] Complete AV block in inferior or posterior MI is less fatal, often resolving within hours to a few days.[1,5-6] IWMI with complete AVB is associated with poor clinical status, such as right ventricular infarction (RVI), cardiogenic shock, and atrial fibrillation,[3] probably related to its association with larger infarct size.[1,3,7]

The frequency of RVI in IWMI was 43%, in accordance with other studies (50%).[8] Mortality rate in patients with IWMI and complete AV block varies from 12-23% and significantly high in patients with right wall myocardial infarction (RWMI).[7] One year mortality was significantly higher in patients with AV block compared to those without AV block (1.3 to 1.6), and when subdivided by location, death were 2.4 and 3.3 more likely in inferior and anterior MI.[9]

CASE REPORT

A 56 year-old male presented to the emergency department with substernal chest pain for 1 hour accompanied with diaphoresis and nausea. No history of chest pain. Prior history were diabetes, dyslipidemia, family history of MI, and 35 pack-year history of smoking. The initial vital signs were: blood pressure 80/48 mmHg, heart rate 42 beats/minute, respiratory rate 21 breaths/minute, oxygen saturation 94%, and a pain score of 8 out of 10. An electrocardiogram (ECG) was obtained immediately and showed an acute infero-posterior myocardial infarction, RWMI, and presence of complete AV block (**Figures**

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1). A blood sample was taken and abnormal results were as follows: leukocytes 15.700/µL; fasting blood glucose 145 mg/dL; 2-hours postprandial blood glucose 184 mg/dL; total cholesterol 222 mg/dL; LDL cholesterol 172 mg/dL; triglycerides 189 mg/dL; TnI > 10 µg/mL. Chest radiograph showed cardiomegaly with no pulmonary congestion. He was diagnosed as acute coronary syndrome – Infero-posterior wall myocardial infarction, RVI, with complete AV block.

The treatments were aspirin 160 mg oral, clopidogrel 300 mg oral, simvastatin 40 mg oral, and morphine 2,5 mg intravenous (iv). Atropine sulfate 1 mg iv was given twice but bradycardia reappear. Dopamine was initiated for hypotension after no response to second 200 mL Ringer Lactate fluid challenge test. Primary Percutaneous Coronary Intervention (PCI) or fibrinolysis and Temporary Pacemaker (TPM) was suggested but refused due to cost reasons. The patient was admitted to Cardiac Intensive Care Unit (CICU); 2.5 mg fondaparinux (low molecular weight heparin/LMWH) iv bolus was given, followed by subcutaneous injection, 2.5 mg once daily.

After 48h, his ECG showed STEMI resolution and improvement in heart rhythm with first-degree atrioventricular block (**Figure 2**) and stable hemodynamics. He was observed in hospital for the next 4 more days. At 3 months follow-up, he was asymptomatic, no evidence of bundle branch/AV block on ECG, Q-waves were present in the infero-posterior leads consistent with the recent infero-posterior MI.

DISCUSSION

The inferior wall of the heart is supplied by branches from the right coronary artery (RCA) and the circumflex branch of left coronary artery (LCA). The artery to AV node is derived from the RCA in 90% cases and LCA in 10% cases and it origin depends on topographic arterial dominance.[1,4,7,10] IWMI are caused by the occlusion of the dominant artery in more than 70% of cases. Because the AV nodal artery is derived from the dominant artery, it is believed that necrosis of the AV node leads to AV block.[7] An alternate hypothesis is increased of vagal tone activity.[10] Ischemic-mediated mechanical stretch and chemical substances stimulate receptors located in the inferior and posterior left ventricular walls. These receptors lead to activation of nonmyelinated afferent C-fibers from the vagus nerve, which in turn result in increased vagal tone and bradyarrhythmia. This mechanism is known as the Bezold-Jarisch reflex.[6] In a multicenter retrospective study, 95 patients (22 percent) went on to develop high grade AV block defined as Mobitz type II (7 patients) or complete AV block (88 patients) [2]. Twenty six of the 88 patients progressed from Mobitz type II to complete AV block, while the other 62 developed suddencomplete AV block.

Approximately 50% patients with IWMI have some involvement of the right ventricle, because both posterior wall of the right ventricle and the inferior wall of the left ventricle share a common blood supply from right coronary artery.[5] RVI is rare because of lower oxygen demands of the right ventricle, rich intercoronary collateral system, thin right ventricle driving some nutrition from the blood within right ventricular cavity itself, and systolic compressive forces of the left ventricle produces throttling effect on the left coronaries that drive blood from the left to right coronaries during systole.[8] A right ventricular infarct should be considered in all acute inferior wall MI patients with hypotension and cardiogenic shock.[7,11] Hypotension and shock occur when there is enough RV ischemia to decrease RV compliance, raising the diastolic filling pressure in the RV. Filling of the RV is reduced, and RV stroke volume decreased. As a result, filling of left ventricle is impaired, cardiac output is reduced, and systemic blood pressure decreases.[7]

Ninety percent of complete AV blocks cases in IWMI occur above the bundle of His, associated with a narrow QRS complex. It often results in

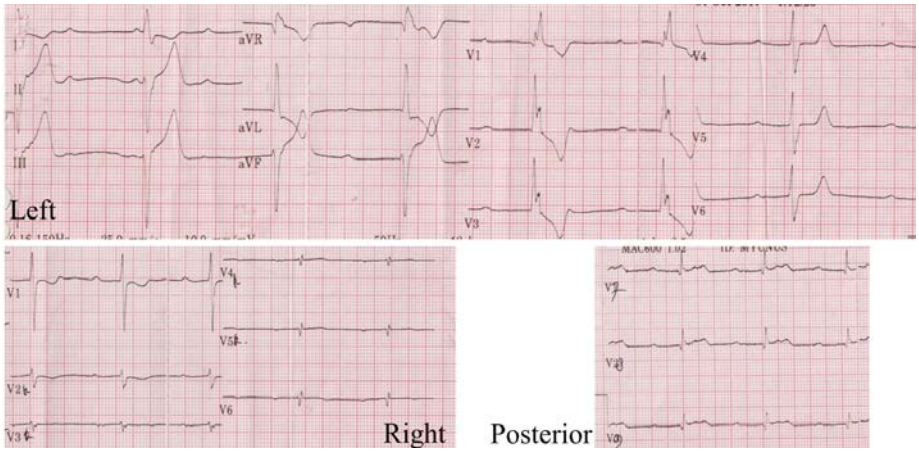

Figure 1. ECG on admission. Complete AV block at 42 bpm; Left Axis Deviation (LAD); narrow QRS complex; ST elevation in lead II, III, aVF, V7-V9 (early stages of STEMI) with reciprocal ST depression in lead I, aVL, V1-V3; ST elevation in lead III > lead II (RVI); No significant changes in right side ECG.

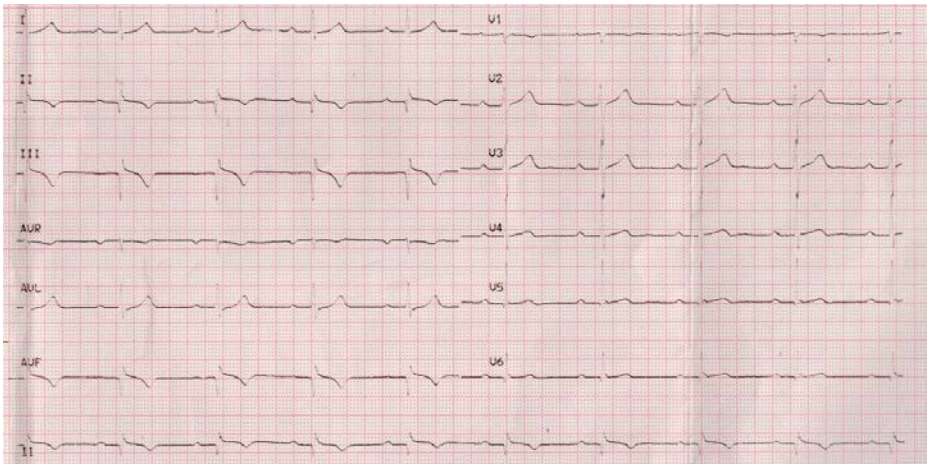

Figure 2. ECG on 48h after admission. Sinus bradycardia at 52 bpm; LAD; Pathologic Q waves and ST elevation with T wave inversion in lead II, III, aVF (STEMI evolution); First-degree atrioventricular block with 262 ms PR-interval.


an asymptomatic bradycardia (40 to 60 beats/min), usually transient, resolving within five to seven days.[1-2]

In IWMI with RVI and complete AV block, prompt and complete coronary reperfusion using fibrinolysis or primary percutaneous coronary intervention (PCI) can preserve both right and left ventricular function and reduce mortality and morbidity.[1-2,4,10-11] In study of 550 complete AV block patients,[3] reperfusion strategy was significantly associated with early resolution of AV block at different cut-off intervals and a significant reduction in in-hospital mortality (12.8% vs 45.2%) compared with conservative treatment. In 2017 ESC Guidelines for the management of acute MI in patients presenting with ST-segment elevation, reperfusion therapy is indicated in all patients with symptoms of ischaemia of ≤ 12 hours duration and persistent ST-segment elevation and primary PCI is superior to fibrinolysis in reducing mortality, reinfarction, or stroke.[12]

In the Treatment with Enoxaparin and Tirofiban in Acute Myocardial Infarction (TETAMI) registry, 30-day mortality was only 4.4% in patients who received reperfusion therapy, compared to 12% in control group. Similarly, the triple end point of death, myocardial reinfarction, or recurrent angina occurred is only 11% in patients receiving reperfusion compared to 19.1% in patients who did not.[13] Despite the benefits of reperfusion therapy, there is a wide variation of eligibility.[14] Four factors were found to be strongly related to failure to provide or receive reperfusion therapy in STEMI: age > 75 years, prior congestive heart failure, prior myocardial infarction (MI), or prior coronary artery bypass surgery. Other variables associated with not offering reperfusion were female sex, diabetes, and delayed presentation (>12 hours).[13-14] In the National Registry of Myocardial Infarction (NRMI), the proportion of patients with STEMI eligible for but not receiving any form of reperfusion therapy slowly decreased from 1992, but remained as high as 28.1% in 2006.[13]

This patient is eligible for reperfusion therapy, but refused due to cost reasons, so another management strategy is needed. Nonreperfused patients derive the most benefit from appropriate antithrombotic regiment. Early initiation of dual-antiplatelet therapy with aspirin and clopidogrel drastically reduces mortality and subsequent cardiovascular events in this population; available data suggest that for patients who do not receive any form of reperfusion, anticoagulation therapy with LMWH provides a clear additional mortality benefit versus placebo.[13] The findings from OASIS 5 and 6 indicate that the balance of benefit and risk of fondaparinux is better than enoxaparin for patients with non-ST elevation ACS and better than usual treatment for patients with STEMI who are not treated with primary PCI.[15]

Bradyarrhythmias that occur early in the setting of an IWMI (within the first 24 hours) may respond to atropine while those occurring later are often atropine-resistant.[2,10-11] Recommendations regarding temporary pacing in the setting of acute MI were included in the 2004 ACC/AHA Guidelines for the management of patients with STEMI, one of the main priorities is bradyarrhythmia with hypotension and complete AV block.[2] In 2012 ACCF/AHA/HRS Focused Update Incorporated Into the ACCF/AHA/HRS 2008 Guidelines for Device-Based Therapy of Cardiac Rhythm Abnormalities, permanent ventricular pacing is indicated only for persistent and symptomatic second- or third-degree AV block after the acute phase of MI (Level of Evidence: C).[16]

RVI is associated with hypotension, and right ventricular cardiac output optimize due to augmented right atrium contractility.[11] Refractory hypotension after correction of bradycardia with atropine usually indicates volume depletion; intravenous fluid should be given to patients with evidence of low cardiac output without pulmonary edema.[2]

In this case, temporary pacing is recommended due to complete AV block with hypotension, but is not done due to cost issues. Patient only respond briefly with atropine sulfate and hypotension does not respond with fluids. So based on the patient's condition, dopamine is chosen as an adjunctive therapy in this case.

In 2015 AHA Update,[16] intravenous infusion chronotropic agents (dopamine or epinephrine) is now recommended for symptomatic bradycardia or unstable bradycardia as equally effective alternative to external pacing when atropine is ineffective. When fluid resuscitation is insufficient, chronotropic agents are indicated for cardiogenic shock. Dopamine and dobutamine are the drugs of choice to improve cardiac contractility, dopamine is preferred in hypotension.[2]

CONCLUSION

In this nonreperfused patients, fondaparinux with dual-antiplatelet therapy (aspirin and clopidogrel) provide significant benefit; this regimen is supported by both current European and North American STEMI guidelines. Bradyarrhythmias due to complete AV block in IWMI (within the first 24 hours) may respond to atropine, and do not need permanent pacing.

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