

## Case Report

# Total atrioventricular block in patient with late onset acute inferior myocardial infarction

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### ABSTRACT

TAVB is common complication of acute inferior myocardial infarction (AMI). Total atrioventricular block (TAVB) occurs when none of the impulses are conducted from atrium to ventricles and move independently without coordination. In our case, A 61-year-old woman came to the emergency room (ER) with chief complaint of general weakness and nausea without any other complaints since 4 days before going to ER. She had TAVB which is a complication of undiagnosed AMI due to silent ischemia. She didn't get reperfusion therapy because of patient delay and limited resources. Pharmacological therapy had been given while awaiting implantation of temporary pacemaker (TPM). Two days after insertion, the heart rhythm returned to sinus rhythm, therefore permanent pacemaker implantation was not required. TAVB in AMI usually resolves spontaneously. Therefore, it is important to identify TAVB in AMI, so we can quickly diagnose and promptly treat the patient. Thus, it can reduce mortality and increase the probability of spontaneous resolution of TAVB, so physician practice management (PPM) insertion can be avoided.

**Keywords:** AMI, Total AV Block, TPM, PPM

### INTRODUCTION

Atrioventricular block (AV block) is a condition where there is a disturbance in normal electrical conduction of the heart that connects sinoatrial node (SA node) with ventricles through atrioventricular node (AV node). Complete heart block (total AV block) occurs when none of the impulses are conducted from the atrium to the ventricles. Atrium and ventricles move independently without going through the same pacemaker. The absence of normal impulses from the SA node passing through the AV node, causes the SA node can't control heart rate, and can lead to decreased cardiac output due to loss of coordination between the atrium and ventricles. This condition can be fatal if not treated quickly and appropriately.<sup>1</sup>

AV block is one of the common complications of acute coronary syndrome (ACS), especially in inferior

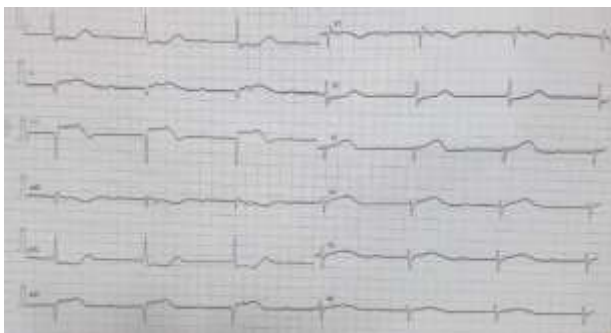
myocardial infarction, occurring in 9-33% of patients. The time of occurrence of atrioventricular block ranged from 30 minutes to 7 days from the onset of signs of acute coronary syndrome.<sup>2</sup> Total atrioventricular block (TAVB) is the most severe condition of AV conduction disorders. The presence of TAVB in patient with ACS is associated with a worse short-term prognosis, regardless of the location of myocardial infarction (MI).<sup>3</sup> TAVB is more common in inferior MI than anterior MI, but the mortality rate is higher in anterior MI with TAVB.<sup>4</sup>

In this article, we will discuss a case of a patient with TAVB associated with acute inferior MI.

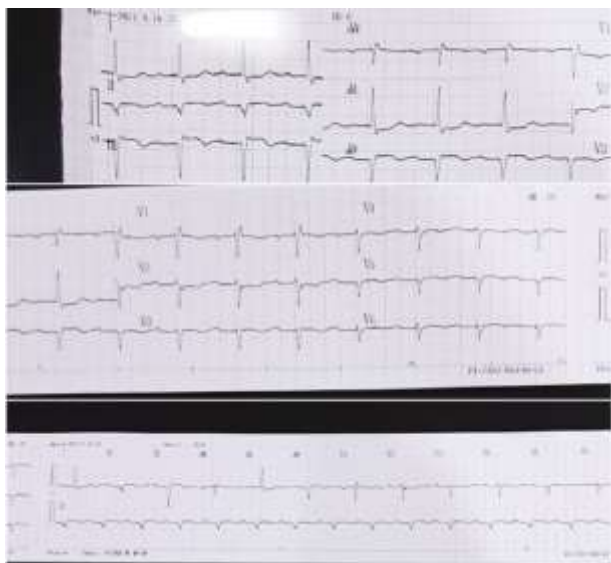
### CASE REPORT

A 61-year-old woman came to the emergency room (ER) with the chief complaint of general weakness since about 4 days before going to the ER. In addition, she

complained of nausea and vomiting. She thought that the complaint was due to acute gastritis. She had been taking antacids but the complaints did not improve at all. They were getting worse. She did not complaint of typical chest pain, tightness, palpitations or fainting. She had history of uncontrolled diabetes mellitus since 10 years ago and ischemic stroke 3 years ago. However, she had never taken any medication. She had no other cardiovascular risk factors and had never experienced other heart disease. On arrival, the patient's vital signs: BP 160/90 mmHg, HR 35-40 bpm, RR 20x/min, temperature 36.6°C and oxygen saturation 97% without oxygen supplementation. On examination of the heart and lungs, no other abnormalities were found. The patient was well perfused in all extremities. Other physical examination results were within normal limits. She was in clinically and hemodynamically stable. From the initial examination of the 12-lead ECG, there was a TAVB (complete heart block), elevation of the ST segment with pathological Q at II, III, aVF (Figure 1). The quantitative value of Troponin increased to 10.15 ng/ml (normal <0.03 ng/ml).



**Figure 1: Patient’s ECG in ER showed Total AV Block, late onset inferior myocardial infarction.**



**Figure 2: Patient’s ECG after insertion of TPM showed a rhythm conversion to Sinus Rhythm with first degree AV Block.**

The patient was sent to the intensive cardiovascular care unit room. Pharmacological therapy had been given while awaiting insertion of temporary pacemaker (TPM). Patient had received 320 mg aspirin, 300 mg clopidogrel, 8 mg candesartan, 6000 IU lovenox injection, 0.5 mg atropine sulfas injection, 3-10 mcg/kg/min Dopamine infusion. Sometime after that, temporary pacemaker was inserted. The patient's complaints had improved since the insertion of TPM. Two days after insertion, all of the patient’s complaints had disappeared. Her heart rhythm had returned to sinus rhythm (Figure 2). Because of this, it was not necessary to insert a permanent pacemaker (PPM).

**DISCUSSION**

Total atrioventricular block (TAVB) occurred in 10.3% of patients with acute coronary syndrome (ACS).<sup>4</sup> Acute coronary syndrome is a serious clinical condition with high mortality rate and it is the leading cause of death worldwide. This is due to the loss of blood supply in the myocardium which causes damage to heart tissue due to a lack of oxygen. Conduction disturbances associated with complications of acute coronary syndromes are often associated with increased morbidity and mortality in patients. This complication often occurs in delayed diagnosis and treatment of ACS.<sup>5</sup>

The mechanisms that cause bradyarrhythmias in patients with acute coronary syndromes are reversible ischaemia or irreversible necrosis of the pacemaker area. Several other factors include changes in autonomic function, systemic hypoxia, and electrolyte disturbances (hyperkalemia), local elevations of adenosine, acid-base disorders (metabolic acidosis), and complication of medical therapy (beta blockers, or calcium channel blockers).<sup>6</sup>

There are several mechanisms that most commonly cause TAVB in ACS patients. The presence of ischaemia that occurs due to blockages in the coronary arteries causes an increase in the release of acetylcholine by the infarcted area. It causes an increase in the sensitivity of the AV node to acetylcholine. In addition, activation of parasympathetic afferent nerves in the inferior-posterior wall of the left ventricle may result in increased vagal tone, reflex bradycardia and Atrioventricular Block. This mechanism is known as the Bezold–Jarisch reflex.<sup>7,8</sup> Furthermore, there is reversible ischaemia or necrosis in the pacemaker area which can result in disruption of the conduction system. Ischaemia can occur at the AV Node if blood flow to the AV Node area is impaired. The AV Node is very sensitive to the presence of oxygen deficiency conditions. Conduction disturbances may occur on second, third, fourth day after infarction, possibly due to expansion of inflammation or edema from the infarcted area.<sup>7,9</sup>

TAVB is more frequently a complication of inferior myocardial infarction than anterior myocardial infarction.

It results from a disruption of blood supply to the area of the atrioventricular node or the upper portion of His bundle. Blood flow to this area comes from the flow from right coronary artery and occlusion to these arteries responsible for 90% of inferior myocardial infarction. The remaining 10% is due to occlusion from left circumflex artery.<sup>10</sup> This is similar to what happened to our patient, who experienced TAVB due to complications from inferior MI, where the blockage probably in the RCA.

The clinical presentation of patients with acute inferior myocardial infarction with total atrioventricular block often depends on the extent of myocardial damage and also the complications resulting from myocardial damage, such as heart failure, cardiogenic shock, arrhythmias and mechanical complications.<sup>3,4</sup> To diagnose a total atrioventricular block sometimes can be difficult because atrioventricular block sometimes can be no complaints at all, but for clinical signs and symptoms that generally appear include the presence of intolerance to exercise, weakness, dizziness, chest pain (angina) and syncope. When these complaints persist and there is a suspicion of Total AV block, the diagnosis can be obtained by looking at a standard 12 lead ECG, Holter ECG or external loop recorder (ELR) or implantable loop recorder (ILR) and an electrophysiology study (EPS).<sup>11</sup> Our patient has total AV Block but her symptom is only general weakness and there are no other complaints.

TAVB that occurs as a complication of ACS usually resolves spontaneously or with revascularization within days or weeks. Only 10% of patients end up needing a permanent pacemaker. This temporary conduction disturbance may occur due to the resolution of the inflammatory condition in the atrioventricular node area and the anastomotic pathway has been able to work properly. Temporary anoxemia is another factor that causes the temporary block.<sup>9</sup> In addition, atrioventricular node has 2 protective mechanisms against ischaemia. The first atrioventricular node receives collateral supply from the septal perforators of the left anterior descending artery and adjacent venous sinusoids. Second, these structures have intracellular content that is rich in glycogen. The conduction system being relatively resistant to ischaemia and can recover function faster than the adjacent myocardium once the ischaemia is reversed. In reversible atrioventricular block, permanent use of a pacemaker is not recommended, because it does not improve the outcome of the patient.<sup>3,7,8</sup>

In this case, the patient did not show the clinical symptoms of a typical acute myocardial infarction (silent ischaemia). This is because the patient had a long history of uncontrolled diabetes. Usually, diabetic patients do not show symptoms of typical chest pain when experiencing acute myocardial infarction. Perception of pain in humans (angina) begins with mechanical and chemical stimulation of the myocardium. In diabetic patients there is impaired perception caused by impaired recognition of

pain stimuli. Other mechanisms that may be involved include individual pain thresholds, high endogenous endorphins, and sensory denervation due to autonomic neuropathy. Several other factors such as age, history of myocardial infarction or CABG, mental stress or certain medications can play a role in the pathogenesis of silent ischaemia.<sup>12,13</sup>

In our patient, spontaneous resolution occurred on the 3rd day after temporary pace maker (TPM) insertion, so there was no need for permanent pace maker (PPM) insertion. This is probably due to several reasons. Ischaemia have occurred in this patient, however, there is no extensive necrosis in the pacemaker area. Spontaneous recanalization or dual arterial blood supply explains the absence of necrosis.<sup>14-16</sup> The existence of a protective mechanism of AV node against ischaemia also increases the likelihood of spontaneous resolution, because the collateral blood vessels work properly and the AV node is rich in glycogen.<sup>7,8</sup> In addition, the AV node becomes relatively resistant to ischemia and can restore function more rapidly than the other area of myocardium, once the ischemia has subsided.<sup>15</sup> Another possibility is that the ongoing thrombotic process can be stopped by reperfusion, either invasively or pharmacologically. In our patient, due to limited resources and patient delay, reperfusion therapy was not performed either invasively or pharmacologically. Patients was given antithrombotics and anticoagulant which were expected to improve outcome and increase the likelihood of spontaneous resolution of TAVB.<sup>13,15,17</sup>

## CONCLUSION

It is important to identify the complications of acute inferior MI, such as total AV block. Besides that, precise and prompt treatment is needed in order to improve patient outcomes and increase the likelihood of spontaneous TAVB resolution.

In this case, the patient's complaints were completely resolve, her heart rhythm returned to the sinus rhythm after TPM insertion, so that PPM insertion was not required. Close monitoring and routine control is needed in this patient.

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