Third-Degree Atrioventricular Block in Clinically “Silent” Acute Myocardial Infarction

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Abstract
Third-degree atrioventricular (AV) block also termed complete heart block is present when there is complete absence of conduction between atria and ventricles. In adults the most common causes are acute myocardial infarction and age-related degeneration of conduction system. Incidence of conduction block is reported to be 25-30 % in the setting of acute myocardial infarction (AMI) and is 2 to 3 times as commonly associated with inferior than anterior infarction. Proper diagnosis involves noninvasive diagnostic tests (12 lead electrocardiogram (ECG), Holter ECG, or stress/exercise ECG).

Keywords: third-degree AV block, complete av block, acute myocardial infarction

In this article, we present a case of third-degree AV block associated with acute myocardial infarction

Case Illustration
The case we present here is about 46 years old female patient who admitted to the emergency department with a history of progressive general weakness since about five days before admission. She denied any similar symptoms before. There was no complaint of typical chest pain, palpitations, or syncope. She had a history of uncontrolled diabetes since 8 years ago. She denied taken any medication. At the time of admission her blood pressure was 100/60 mmHg, pulse rate approximately 35-40x/min, and respiratory rate approximately 20x/min. Physical examination within normal limits. Patient remained clinically and hemodynamically stable. The initial electrocardiogram (ECG) revealed a third-degree AV block and Q waves with slight T inversion in inferior lead (Figure 1) with high-sensitive troponin T elevation to 709.1 pg/mL (normal <14 pg/mL).
She was admitted to coronary intensive care unit. Temporary pacemaker was inserted, coronary angiography revealed 70% proximal stenosis of the right coronary artery (RCA), which was considered the culprit lesion. Left circumflex artery had 70% distal stenosis. Left main and left anterior descending artery had no significant lesion. Balloon angioplasty of the RCA lesion was performed with a much improved angiographic result and drug-eluting stent (DES) was successfully deployed to the RCA lesion with an excellent angiographic result and no residual stenosis. The patient was transferred to the intensive care unit. Upon revascularization, the patient immediately reverted to first-degree AV block (Figure 3) and then has remained normal sinus rhythm since then. Patient complaint entirely resolved and permanent pacemaker implantation was avoided.

Figure 1. Third-degree AV block

Figure 3. First-degree AV block

Discussion

The important finding in this case is the resolution of complete heart block upon revascularization of the RCA. The relative frequency of new onset AV block in patients with myocardial infarction has decreased with percutaneous coronary intervention (PCI). The mechanisms that caused most bradyarrhythmias in myocardial infarction are either reversible ischemia or irreversible necrosis of the pacemaker sites. Other factors include altered autonomic function, systemic hypoxia, electrolyte disturbances (hyperkalemia), local increases in adenosine, acid-base disorders (metabolic acidosis), and complications of medical therapies (beta- or calcium-channel-blocking agents). The most useful approach of the pathophysiology is related to the anatomic location of the infarction. Heart is a muscular pump that pushes blood through extensive network of blood vessels throughout myocardium. These vessels are the coronary arteries and veins. Failure of regulation mechanism of the coronary circulation can lead to insufficient oxygen delivery to myocardium which will impair cardiac function. The two main branches of coronary circulation are the left main and right main coronary arteries. The left main coronary artery divides into left anterior descending artery, which circulate through interventricular groove on anterior surface of the heart toward cardiac apex supplies the left ventricular free wall, medial one-third of anterior right ventricular wall, AV (his) bundle, and proximal left bundle branch; and the left circumflex artery, which travels posteriorly along the groove between left atrium and ventricle supplies the lateral left ventricular wall. The right main coronary artery circulates between right atrium and ventricle to posterior regions of the heart. This vessel and its branches serve the right ventricle and atrium. Over 70% individual the posterior descending artery arises from distal right coronary artery (right dominant coronary artery). It supply the basal and middle inferior wall, right bundle branch, AV node, AV (his) bundle, posterior portion of left bundle branch, and posteromedial mitral papillary muscle. AV block is usually due to proximal occlusion of the RCA or a dominant circumflex artery. The major association of RCA-related infarctions and third-degree AV block can be due to increased parasympathetic influence and reversible ischemia, that explains tendency of rhythm disturbance and relatively slow ventricular escape rates, because necrosis of the
AV node appears to be rare because of the presence of collateral circulation (RCA and LAD contributions). Diagnosis is usually difficult to assess knows that AV block can be entirely asymptomatic, but clinical signs of AV block typically include exercise intolerance, weakness, dizziness, angina, and/or syncope. When persistent symptoms occur, certain diagnosis usually made from a standard 12 lead ECG, Holter ECG or external loop recorder (ELR) or implantable loop recorder (ILR) when intermittent depends on the frequency, and electrophysiology study (EPS) when suspected.

AV block complicating myocardial infarction usually resolves spontaneously or with revascularization within days or weeks, with only under 10% requiring permanent pacing. Reversible AV block is not recommended for permanent cardiac pacing because it does not influence the outcome. AV block with anterior infarction with large necrosis or complicated by new-onset bundle branch block (BBB) shows no benefit of cardiac pacing, and more proper to evaluate the indications for CRT-D. Even so in third-degree AV block complicating inferior MI that does not resolve with revascularization, permanent pacing is indicated. In this case patient presented with no typical symptoms of myocardial infarction (silent ischemia). Clinically patients with diabetes more likely present without typical chest pain in the setting of AMI. Human perception of pain (angina) begins with mechanical and chemical stimulation in myocardium. Impaired perception caused failure of painful stimuli recognition. Possible mechanisms include different pain threshold, high endogenous endorphins, and sensory denervation due to autonomic neuropathy. Several factors such as age, history of MI or CABG, mental stress, or certain drugs play a role in pathogenesis of silent ischemia.

Conclusion
In this case, ECG was proven as a simple and valuable diagnostic tool to guide us for revascularization. Third-degree AV block complicating probably a recent myocardial infarction are resolves spontaneously upon revascularization of the RCA. Patient complaint entirely resolved, ECG remained sinus rhythm since then, and permanent pacemaker implantation was avoided.

Acknowledgement
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Conflict of interest
None

References


