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

Abstrak

Blok atrioventricular (AV) derajat tiga atau Blok AV komplit terjadi saat tidak ada jaringan konduksi yang menghubungkan atrium dan ventrikel. Pada orang dewasa penyebab paling sering adalah infark miokard akut (IMA) dan gangguan konduksi yang berhubungan dengan usia degeneratif. Insidensi gangguan konduksi pada infark miokard akut terjadi sebanyak 25-30 %, dan 2-3 kali lebih sering berhubungan dengan infark miokard akut lokasi inferior dibandingkan anterior. Diagnosis yang tepat adalah dengan pendekatan yang tidak invasif seperti EKG (12sandapan elektrokardiogram), holter ECG, EKG dengan beban/aktivitas.

Kata kunci: Blok AV derajat tiga,Blok AV komplit, infark miokard akut

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Introduction

Third-degree AV block (complete block) occurs when none of the impulse of atrial is conducted to the ventricles hence the atria and ventricle are controlled by independent pacemakers. Such failure is one type of complete AV dissociation that can be congenital or acquired. Whether the block develop in the level of AV node (usually congenital), within His bundle, or bundle branches (usually acquired).[1]

Arrhythmias and conduction disturbances usually occurs in the early hours after myocardial infarction (MI). Sinus bradycardia or av block is one of the examples of conduction failure in the first hours of STEMI, especially in inferior infarction.[2] According to recordings from cardiac monitors implanted within 11 ± 5 days of an acute MI, the incidence for third-degree AV block is 10% and 7% for sinus bradycardia.[2,3].

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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 the 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. She had no other cardiac risk factors. 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).

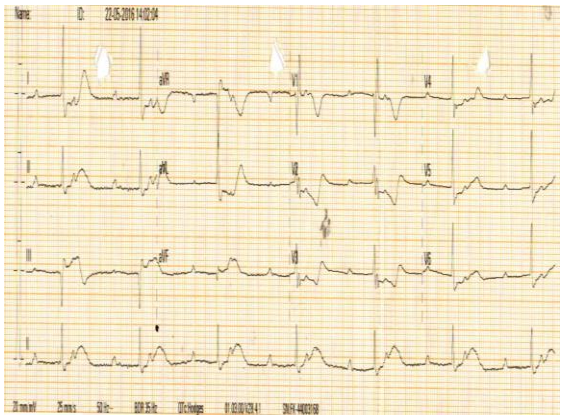

Figure 1. Third-degree AV block

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.

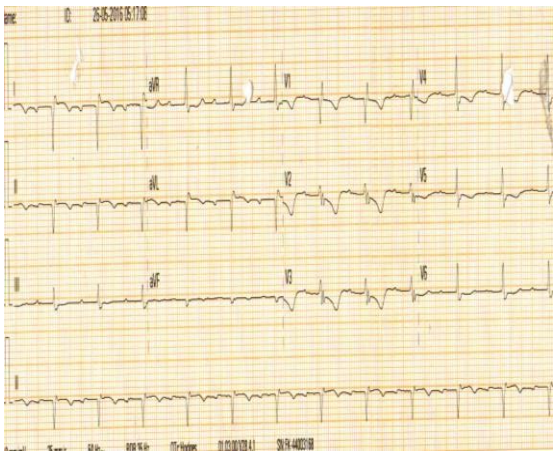

Figure 3. First-degree AV block

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).[4] 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).[5,6]

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.[7] 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.[7,8] 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.[8-10]

AV block is usually due to proximal occlusion of the RCA or a dominant circumflex artery.[11] 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).[5,12]

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.[1] When persistent symptoms occurs 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.[4]

AV block complicating myocardial infarction usually resolves spontaneously or with revascularization within days or weeks,[11] with only under 10 % requiring permanent pacing.[4,11] Reversible AV block is not recommended for permanent cardiac pacing because it does not influence the outcome.[4] 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.[4,13] Even so in third-degree AV block complicating inferior MI that does not resolve with revascularization, permanent pacing is indicated.[4] 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.[14] Possible mechanisms include different pain threshold, high endogenous endorphins, and sensory denervation due to autonomic neuropathy.[15] Several factors such as age, history of MI or CABG, mental stress, or certain drugs play a role in pathogenesis of silent ischemia.[14,15]

Conclusion

In this case, ECG was proven as a simple and valuable dagnostic 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

1. Braunwald E, Mann DL. Braunwald's heart disease : a textbook of cardiovascular medicine. Philadelphia, Pa.: Elsevier Saunders; 2015.

2. Steg PG, James SK, Atar D, Badano LP, Lundqvist CB, Borger MA, et al. ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. The Task Force on the management of ST-segment elevation acute myocardial infarction of the European Society of Cardiology (ESC). 2012 2012-10-01 00:00:00;33(20):2569-619.

3. Bloch Thomsen PE, Jons C, Raatikainen MJ, Moerch Joergensen R, Hartikainen J, Virtanen V, et al. Long-term recording of cardiac arrhythmias with an implantable cardiac monitor in patients with reduced ejection fraction after acute myocardial infarction: the Cardiac Arrhythmias and Risk Stratification After Acute Myocardial Infarction (CARISMA) study. Circulation. 2010 Sep 28;122(13):1258-64. PubMed PMID: 20837897. Epub 2010/09/15. eng.

4. Brignole M, Auricchio A, Baron-Esquivias G, Bordachar P, Boriani G, Breithardt OA, et al. 2013 ESC guidelines on cardiac pacing and cardiac resynchronization therapy: the task force on cardiac pacing and resynchronization therapy of the European Society of Cardiology (ESC). Developed in collaboration with the European Heart Rhythm Association (EHRA). Europace : European pacing, arrhythmias, and cardiac electrophysiology : journal of the working groups on cardiac pacing, arrhythmias, and cardiac cellular electrophysiology of the European Society of Cardiology. 2013 Aug;15(8):1070-118. PubMed PMID: 23801827. Epub 2013/06/27. eng.

5. Brady WJ, Jr., Harrigan RA. Diagnosis and management of bradycardia and atrioventricular block associated with acute coronary ischemia. Emergency medicine clinics of North America. 2001 May;19(2):371-84, xi-xii. PubMed PMID: 11373984. Epub 2001/05/26. eng.

6. Vogler J, Breithardt G, Eckardt L. Bradyarrhythmias and conduction blocks. Revista espanola de cardiologia (English ed). 2012 Jul;65(7):656-67. PubMed PMID: 22627074. Epub 2012/05/26. eng

7. Klabunde RE, Klabunde RE. Cardiovascular physiology concepts. Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins; 2012.

8. JAMES TN. Anatomy of the Coronary Arteries in Health and Disease. Circulation. 1965;32(6):1020-33.

9. Richard W, James F, Valentin F, Robert OR. Hurst's the Heart Manual of Cardiology, Thirteenth Edition: McGraw-Hill Professional; 2012. -1 p.

10. Anderson KR, Murphy JG. The atrio-ventricular node artery in the human heart. Angiology. 1983 Nov;34(11):711-6. PubMed PMID: 6638606. Epub 1983/11/01. eng.

11. Priori SG, Blomström-Lundqvist C, Mazzanti A, Blom N, Borggrefe M, Camm J, et al. 2015 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death. The Task Force for the Management of Patients with Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death of the European Society of Cardiology (ESC)Endorsed by: Association for European Paediatric and Congenital Cardiology (AEPC). 2015 2015-08-29 00:00:00.

12. Cardoso R, Alfonso CE, Coffey JO. Reversibility of High-Grade Atrioventricular Block with Revascularization in Coronary Artery Disease without Infarction: A Literature Review. Case reports in cardiology. 2016;2016:1971803. PubMed PMID: 26925272. Pubmed Central PMCID: PMC4746340. Epub 2016/03/01. eng.

13. Epstein AE, DiMarco JP, Ellenbogen KA, Mark Estes INA, Freedman RA, Gettes LS, et al. ACC/AHA/HRS 2008 Guidelines for Device-Based Therapy of Cardiac Rhythm Abnormalities: Executive SummaryA Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the ACC/AHA/NASPE 2002 Guideline Update for Implantation of Cardiac Pacemakers and Antiarrhythmia Devices) Developed in Collaboration With the American Association for Thoracic Surgery and Society of Thoracic Surgeons. Journal of the American College of Cardiology. 2008;51(21):2085-105.

14. Tabibiazar R, Edelman SV. Silent Ischemia in People With Diabetes: A Condition That Must Be Heard. Clinical Diabetes. 2003;21(1):5-9.

15. Chiariello M, Indolfi C. Silent Myocardial Ischemia in Patients With Diabetes Mellitus. Circulation. 1996;93(12):2089-91.