Case Report

Varying degrees of advanced heart blocks in association with acute inferior myocardial infarction

Kashif Bin Naeem, Abbas Jarrahi, Najiba Abdulrazzaq

From Department of Internal Medicine and Cardiology, Al Baraha Hospital, Dubai, Ministry of Health and Prevention, UAE

Correspondence to: Kashif Bin Naeem, MRCP (United Kingdom), Al Baraha Hospital, Dubai, Ministry of Health and Prevention, UAE. Tel.: 00971 569443145. E-mail: kbncardiology@yahoo.co.uk

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ABSTRACT

Acute inferior myocardial infarction (MI) is often associated with heart blocks as acute occlusion of the right coronary artery (RCA) compromises flow to sinoatrial nodal and atrioventricular nodal branches that arise from the proximal and distal parts of RCA, respectively. This phenomenon can happen in up to 20% of the cases, is usually transient and rarely requires pacemaker implantation. Still, when present, it raises alarms and poses significant monitoring and management challenges to the physicians. We report a similar case where the patient presented with inferior MI complicated by complete heart block. The patient went through varying degrees of advanced heart blocks before finally reverting to normal sinus rhythm. Subsequent coronary angiogram showed total thrombotic occlusion of proximal RCA. To the best of our knowledge, such a case has not been described before in literature where a complete array of heart blocks is reported in the context of acute MI. This case illustrates the importance of recognizing advanced heart blocks in acute MI. Although usually transient, these arrhythmias need careful monitoring during the initial days after MI.

Key words: *Acute myocardial infarction, Electrocardiogram, High-degree atrioventricular block*

Acute myocardial infarction (AMI) is a common cardiac presentation with significant morbidity and mortality. Data from the national registry of MI - 4 estimates that there are 500,000 cases of infarction with ST-segment elevation in the USA per year [1]. Heart block is a complication of AMI, especially the inferior wall infarction. The incidence rate reaches up to 20% in patients who suffer from inferior AMI [2]. Although this event is transient, it poses a significant challenge to physicians and influences management decisions. We report a case where the patient presented with inferior MI and an array of advanced heart blocks that regressed gradually as active ischemia resolved.

CASE REPORT

A 63-year-old, diabetic and hypertensive, male, presented to the emergency department of our center with acute onset chest pain of 3 h duration accompanied with profuse sweating. He did not complain of any shortness of breath, orthopnea, dizziness, or loss of consciousness. He had a cerebrovascular accident 4 years ago without any residual neurological deficit. He had no family history of the premature coronary disease and denied smoking and consuming alcohol.

On physical examination, he was awake, conscious and oriented but still in chest pain with a blood pressure of 95/60 mmHg, heart rate of 32 beats/min, respiratory rate of 18/min, afebrile, and peripheral oxygen saturation of 97% on room air. He had normal heart sounds with no added sounds or murmurs. In addition, his chest was clear, and his venous pressures were normal with no evidence of leg edema. His electrocardiogram (ECG) showed ST-segment elevation in leads II, III, and aVF (inferior leads), accompanied by a third-degree atrioventricular (AV) heart block with ventricular escape rhythm (ventricular rate 32 beats/min) (Fig. 1a). He was immediately administered an intravenous (IV) fibrinolytic agent (tenecteplase). He was also treated with IV atropine for bradycardia that showed a temporary response. He was then commenced on IV dopamine infusion.

His laboratory tests revealed high Troponin-T (2.2 ng/mL). Chest X-ray was normal. Right-sided ECG excluded right ventricular infarction. He was then transferred to the coronary care unit (CCU) for further monitoring and treatment. His chest pain resolved within 30 min. The case was discussed with the cardiologist at the tertiary center to consider for transfer for angioplasty as the facility of primary coronary angioplasty was not available in our center. As per his advice, the patient was managed conservatively at our center as the patient became pain-free and remained hemodynamically stable. Transthoracic echocardiography showed left ventricular inferior hypokinesia, ejection fraction of 50%, Grade 1 diastolic dysfunction, no valve disease, and no effusion or masses. His was optimized on aspirin, clopidogrel, statin, and subcutaneous enoxaparin; he remained in complete heart block (CHB). However, on day 3, his ECG showed CHB with junctional escape rhythm (Fig. 1b). He was kept in CCU, although, he

remained asymptomatic and maintained his blood pressure. On day 6, his ECG showed second-degree Mobitz Type 1 heart block (Wenckebach phenomenon) (Fig. 1c). He remained stable and was transferred to the medical ward. On day 7 his ECG revealed sinus rhythm with first-degree AV heart block (Fig. 1d). He was then discharged from the hospital on day 9 while on aspirin, clopidogrel, atorvastatin, lisinopril, gliclazide, and insulin glargine.

He underwent coronary angiography immediately after discharge that showed a dominant right coronary artery (RCA) that had a long, thrombotic, total occlusion of its proximal to mid segment with no antegrade flow (Fig. 2). Distal branches were faintly opacified through collaterals from the left coronary artery. There was significant proximal calcification. The interventional cardiologist recommended aggressive medical therapy. His ECG post catheterization showed normal sinus rhythm with Q waves and T inversions in the inferior leads (Fig. 1e).

DISCUSSION

Despite noteworthy improvements in the prognosis of AMI over the past decade, it still remains one of the leading causes of morbidity and mortality worldwide [3]. Disturbances in heart rhythm are a common association of AMI. It may occur due to obstruction of any coronary artery, but most commonly associated with RCA lesions. Our patient had RCA occlusion that led to the varying degrees of heart blocks observed.

The sinoatrial (SA) and AV nodes, which function as the pacemaker and pacesetter, respectively, receive their blood supply mainly from the RCA (Fig. 3a and b) and the left circumflex artery. Pejković *et al*. performed anatomical dissection of 150 hearts from adults aged 18 to 80 years [4]. They found that the SA node artery was most frequently a large atrial branch of the RCA (63%). In 37% of cases, it was a branch of the left coronary artery or one of its branches. The AV node artery was the first and longest inferior septal perforating branch of the right (90%) or left (10%) coronary artery.

The arrhythmias associated with AMI include sinus bradycardia and AV blocks. Sinus bradycardia is the most common arrhythmia associated with the inferior AMI, occurring in up to 40% of the cases in the first 2 h and decreasing to 20% at the end of the 1st day. Initially, it may be attributed to the increase of vagal tone in the first 24 h of AMI. After this period, it may be the consequence of sinus node ischemia or atrial ischemia [5]. The association between heart block and AMI has been noticed as far back in history as 1956 by Penton *et al*. [6] who noted that out of 251 cases of CHB, 49 (19.5%) had AMI. Similar incidence was noted by Zoob and Smith [7] who reported 15.6% of cases of CHB due to AMI. The incidence of heart blocks reaches up to 20% in patients who suffer from inferior AMI, depending on the adopted criteria in the studies [2]. However, the incidence rates of CHB associated with AMI have decreased in the past decades due to advances in management techniques.

During the pre-thrombolytic era, the incidence of high-degree AV block (HAVB) in AMI was reported to be 8.6% [8], and the incidence of CHB in AMI was reported by another

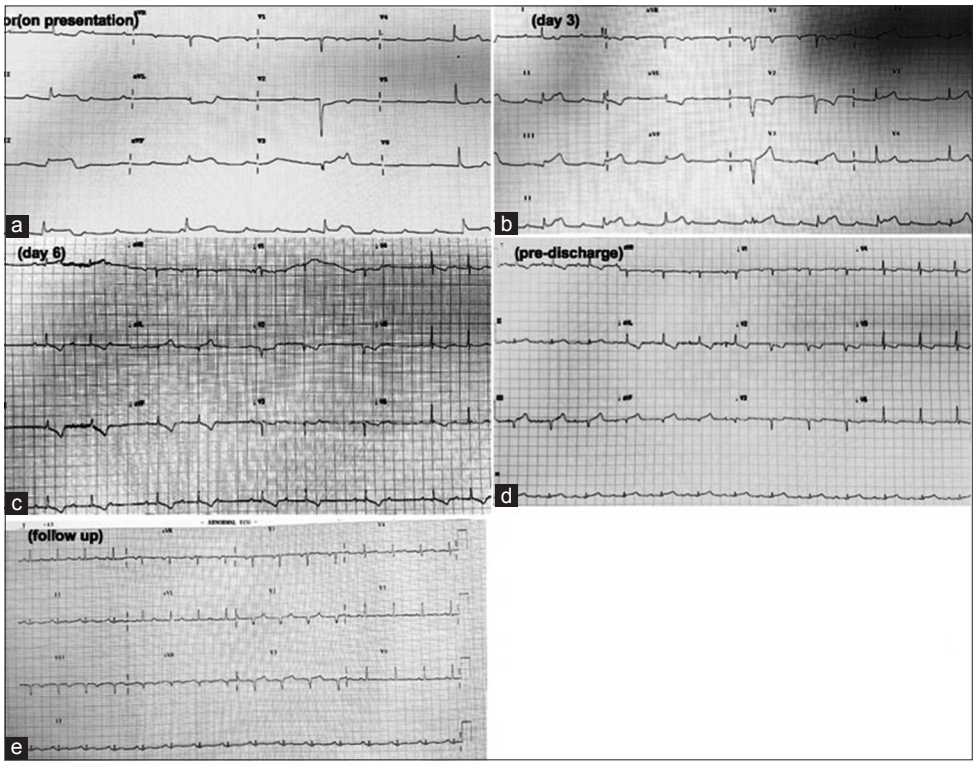

Figure 1: Serial electrocardiograms (ECGs) showing varying degrees of heart blocks. (a) Shows ECG on presentation: Inferior ST-elevation, complete heart block (CHB) with ventricular escape rhythm, (b) shows ECG on day 3: CHB with junctional escape rhythm, (c) shows ECG on day 6: Second-degree Mobitz Type 1 atrioventricular (AV) block (Wenckebach phenomenon), (d) shows pre-discharge ECG: Sinus rhythm with first-degree AV block, (e) shows normal ECG on follow-up

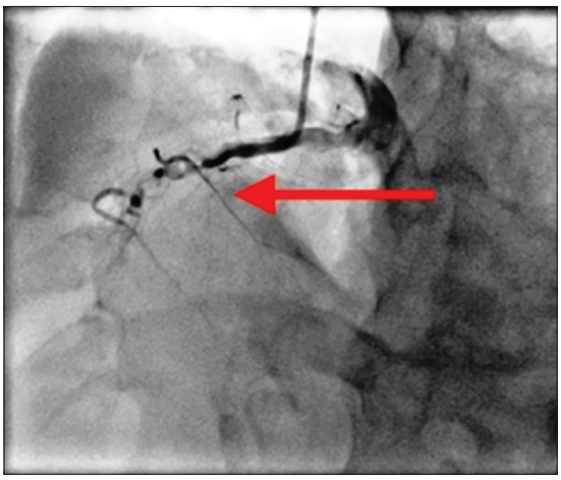

Figure 2: Coronary angiogram showing completely occluded right coronary artery

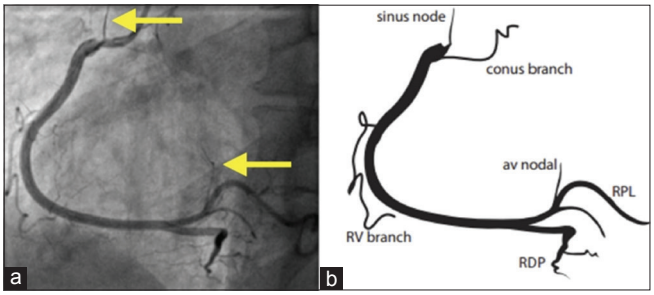

Figure 3: (a) Coronary angiographic image of a normal right coronary artery (RCA), (b) a diagram with major branches of RCA indicating the origin of sinus nodal and atrioventricular nodal branches

study to be 5.8% [9]. After the availability of thrombolysis, the incidence of HAVB in AMI was recorded as 6.9% according to one study [10]. The utilization of primary percutaneous coronary intervention (PCI) had an impact on the incidence rates as well, where one study reported the incidence of HAVB in AMI as 3.2% of which 2.7% was CHB and 0.5% was second-degree AV block [11]. AV nodal conduction tissues are resistant to permanent ischemic damage because of their complex arterial blood supply, high intracellular glycogen content and ability to absorb nutrients and oxygen from surrounding venous sinusoids. However, significant ischemia can result in HAVB. Furthermore, an exaggerated parasympathetic activity or local release of potassium or adenosine or a combination of them has also been suspected to be the underlying mechanism for HAVB. Interestingly, revascularization post-thrombolysis has been postulated to generate a surge of afferent vagal activity leading to the development of the transient HAVB [11].

Arrhythmias in acute MI are usually transient. However, high-grade (second- or third-degree) AV block and persistent bundle branch block are independently associated with worse short- and long-term prognosis in both inferior/posterior and anterior/lateral MI but are more ominous in anterior/lateral MI because of a relatively greater extent of myocardial injury [12]. Independent risk factors for the occurrence of heart blocks are inferior infarction, advanced age, cardiac insufficiency symptoms, female gender, smoking, hypertension, and diabetes mellitus [2]. First-degree AV block does not require treatment. HAVB with inferior/posterior ST-elevation MI usually is transient and associated with a narrow complex/junctional escape rhythm that can be managed conservatively. However, transcutaneous pacing shall remain standby in case hypotension or heart failure ensues. Prophylactic placement of a temporary transvenous pacing system is recommended for high-grade AV block and/or new bundle branch (especially left bundle branch block) or fascicular block in patients with anterior/lateral MI.

Our patient presented with inferior MI (that was treated by thrombolysis) complicated by CHB. As our center was not equipped with PCI facility, the case was discussed with the cardiologist at the tertiary center for transfer for urgent PCI as it is the preferred therapy. However, he suggested that the patient can be managed conservatively at present at our center as the patient became pain-free and was hemodynamically stable. He maintained his blood pressure, and therefore, was managed with IV atropine. Transvenous pacing wire was not inserted, and transcutaneous pacemaker was kept standby. He was managed conservatively and improved spontaneously over the course of next few days. As such patients have an unusual presentation; these can pose significant management challenges for the physicians.

CONCLUSION

Acute MI is common and can be associated with varying degrees of advanced heart blocks. Although the arrhythmias are usually transient, such patients need careful monitoring and management.

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