CASE REPORT

Symptomatic First- and Second-Degree Atrioventricular Block in a Young Soldier: A Diagnostic Dilemma in a Resource-Limited Setting

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ABSTRACT

Background: First-degree AV block and Mobitz I second-degree AV block are largely asymptomatic. However, severe prolongation of the PR interval in patients with first-degree AV block could result in simultaneous atrial and ventricular contractions (loss of AV synchrony) and Talle MA symptoms akin to pacemaker syndrome. **Case Summary:** We present a 25-year-old male soldier who presented with a three-year history of exertional shortness of breath, fatigue, palpitations, and pre-syncope. Examination revealed bradycardia (42 bpm), other cardiovascular findings were normal. ECG showed first-degree AV block (PR interval: 347ms) with a broad QRS complex, later progressing to Mobitz I second-degree AV block (45 bpm). Exercise testing revealed an inadequate PR shortening despite an increase in heart rate. Other investigations, including echocardiography and blood tests, were normal. Limited access to Holter ECG and genetic testing hindered definitive diagnosis. Given the risk of progression to complete heart block and sudden cardiac death, early electrophysiology evaluation and pacemaker implantation are recommended.

Keywords: Symptomatic bradycardia, First-degree AV block, Mobitz I second-degree AV block

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Introduction

Symptomatic Atrioventricular (AV) conduction block commonly results from degenerative changes of the AV node occurring in the elderly or as a complication of acute myocardial infarction. The occurrence of Atrioventricular (AV) conduction block is roughly 30% in certain populations and tends to rise with advancing age. However, A-V block without other evidence of organic heart disease is seen at younger ages.[1, 2]

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Other aetiologies for AV block in the younger population include calcific changes associated with rheumatic heart disease, infective endocarditis, myocarditis and acute myocardial infarction. In this condition, electrical impulses from the atria are either delayed or completely blocked from reaching the ventricles when the AV conduction pathway is not in a state of physiological refractoriness.[3]

First-degree AV block and Mobitz I second-degree AV block are largely asymptomatic. However, severe prolongation of the PR interval in patients with first-degree AV block could result in simultaneous atrial and ventricular contractions (loss of AV synchrony) and symptoms akin to pacemaker syndrome.1 Similarly, failure of the sympathetically mediated shortening of the PR interval with exertion could result in profound symptoms in patients with first-degree heart block.[4] We present a case of symptomatic first-degree and Mobitz type I AV block in a 25-year-old soldier.

Case Report:

A 25-year-old male soldier was referred to our clinic with a recurrent history of exertional shortness of breath for the past three years, associated with easy fatigability, palpitations, and episodes of pre-syncope. He denied any history of chest pain, cough,

orthopnoea, paroxysmal nocturnal dyspnoea (PND), or childhood palpitations.

There was no ascertainable history of childhood sore throat or anterior neck swelling. The patient reported no symptoms suggestive of heat or cold intolerance. There was no history of hypertension, diabetes mellitus, smoking, ingestion of alcohol, or use of recreational drugs. There is no family history of sudden cardiac death, similar presentations or cardiovascular diseases.

When examined, he was found to be a lean young man (body mass index of 20.3 kg/m^2), otherwise well-nourished. There was no pallor, jaundice, digital clubbing, pedal oedema or anterior neck swelling. He had a regular resting pulse rate of 42 bpm with a large volume but not collapsing. All peripheral pulses were present and synchronous. The blood pressure was 120/80mmHg and the JVP was not raised. The precordium, apical impulse and heart sounds were normal. Respiratory, abdominal and other examinations were essentially within normal limits.

The first ECG (Figure 1) revealed a sinus rate at 72 bpm with a PR interval of 347ms consistent with a first-degree AV block. The first sinus impulse was conducted with a broad QRS complex (consistent with an LBBB or a fusion beat). The second ECG obtained while presenting at the UMTH (Figure 2) revealed a second-degree Mobitz I atrioventricular block with a bradycardic rate of 45 beats per minute. When chronotropic competence was assessed using Master's two-step exercise test, he reverted to a sinus rhythm at the rate of 81 beats per minute (80% increase from baseline) while maintaining a first-degree atrioventricular block with a PR interval of 352ms (figure 3). The exercise was stopped after 5 minutes due to fatigue. The QRS duration remained normal on all ECGs except for the isolated broad QRS noted in Figure 1. Holter ECG was requested but not obtained due to non-availability.

A transthoracic Echo revealed normal cardiac structure and function. Chest X-ray and other ancillary investigations, including complete blood count, blood urea Nitrogen (BUN), creatinine, and TSH were within normal range. All medications were suspended, and he was referred for EP study and possibly pacemaker (PM) implantation.

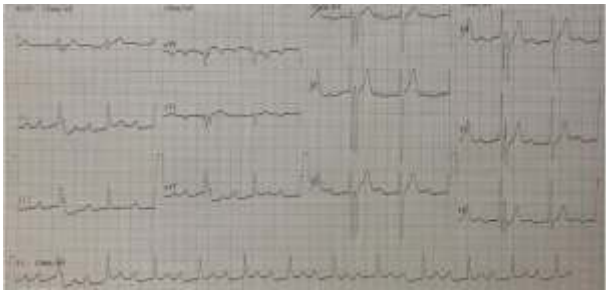

Figure 1. Electrocardiogram showing a first-degree heart block, sinus rate at 72 bpm with a PR interval of 347ms with the first sinus impulse was conducted with a broad QRS complex (consistent with an LBBB or a fusion beat).

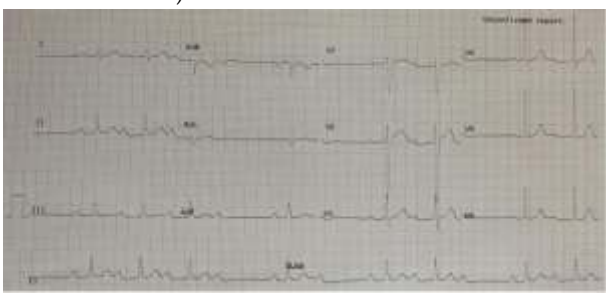

Figure 2. Electrocardiography showing second degree second-degree Mobitz I atrioventricular block with a bradycardic rate of 45 beats per minute.

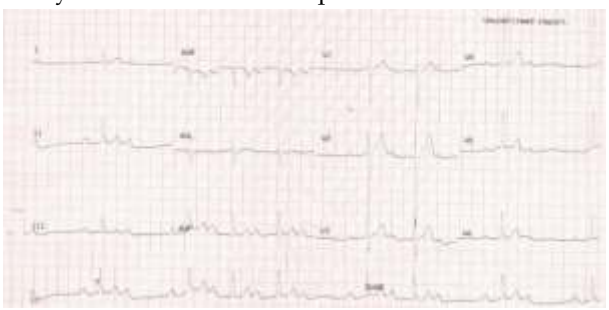

Figure 3. Electrocardiography following Master's two-step exercise test. Showing second-degree Mobitz I atrioventricular block AV block with PR interval 352ms with a ventricular rate of 81beats/minute

Discussion

The case illustrates a probable symptomatic first-degree or second-degree Mobitz I atrioventricular block in a young soldier presenting with paroxysms of effort intolerance, palpitation and occasional dizziness. Although most cases of first-degree AV block and Mobitz I second-degree AV block are asymptomatic, simultaneous atrial and ventricular contraction (loss of AV synchrony) in patients with markedly increased PR interval could result in symptoms like pacemaker syndrome. This may be the case in our patient, with a PR interval of 347 while in sinus rhythm. Similarly, failure of the sympathetically

mediated shortening of the PR interval with exertion despite almost doubling of the heart rate could be responsible for the symptoms experienced. Despite the purported associations between the symptoms and ECG findings, a conclusive causal link will require a concordance of the rhythm with symptoms, since the symptoms experienced may as well be related to a more advanced form of AV conduction disease not captured by the routine resting ECG. This will require other forms ambulatory ECG including Holter ECG, event recorders, etc.

Our patient's initial ECG showed bradycardia, a prolonged PR interval, and a wide QRS complex (LBBB), indicating a first-degree atrioventricular (AV) block (figure 1). This may occur in the AV node or the His-Purkinje system. While first-degree AV block due to increased vagal tone is typically seen in physically fit young individuals with asymptomatic bradycardia, [5] the fact that our patient was experiencing symptoms suggests that there may be additional complexities related to the first-degree heart block.

Second-degree AV block is considered when intermittent failure of AV conduction occurs. Second-degree atrioventricular (AV) block can be divided into two main types: type I, also known as Mobitz I or Wenckebach, and type II, known as Mobitz ll. The classic Mobitz type I second-degree AV block is defined by a progressive PR interval prolongation before the non-conducted P wave (Wenckebach phenomena). In a stable sinus rhythm, the first conducted P wave after a non-conducted P wave will have the shortest PR interval in that cycle. As a result, the pause between the QRS complexes on either side of the non-conducted P wave will be shorter than twice the P-P interval. Classic Type I or Mobitz l block cycle, there is a consistent P: R ratio (such as 3:2, 4:3, or 5:4).[6]

Mobitz II second-degree AV block is defined by a single non-conducted P wave with constant PR intervals before and after the blocked impulse (with constant PP and RR intervals). The pause that includes the blocked P wave is equivalent to 2 P-P cycles. Mobitz II second-degree AV block commonly occurs alongside intraventricular block.[7]

A 2:1 AV block, also known as an advanced AV block, cannot be definitively identified as either type I or type II second-degree AV block based solely on a single short recording of the surface ECG that shows only one PR interval preceding the blocked P wave. The block may be either in the AV node or the His-Purkinje system. It is important to note that both type I and type II second-degree AV blocks have the potential to either progress to or regress from a 2:1 block. Our patient's repeat ECG showed a 2:1 AV block coupled with the exertional symptoms suggesting the block was progressive and beyond Mobitz I.[8]

Advanced AV block and the Mobitz II are disorders that encompass abnormalities in the electrical activity of the heart that can lead to bradyarrhythmias, pre-syncope/syncope, and in severe cases, sudden cardiac death. These conditions are rare in young adults without structural heart disease, idiopathic degeneration, infiltrative diseases, or genetic abnormalities.[6]

Challenges in diagnosis

This case highlights the importance of early recognition and management of conduction disorders in young adults. The patient's presentation, in the context of normal cardiac structure, progressive AV conduction disorder, negative family history and normal laboratory findings, strongly suggests idiopathic progressive cardiac conduction disease.

Lenegre's disease is an idiopathic sclerodegenerative change affecting the cardiac conduction system and affects mainly young and middle-aged adults. It is progressive and frequently results in advanced or complete A-V block.[2,8] The diagnostic criteria proposed by Dhingra and associates include: (1) the emergence of progressive delays in intraventricular conduction that ultimately lead to complete heart block, (2) identification of the block occurring at a location beyond the His bundle, and (3) the lack of any identifiable organic heart disease.[9]

Inherited conditions involving ion channels, structural proteins, or other cardiac components can manifest as advanced AV block in young individuals. This includes mutations in SCN5A, which are associated with Brugada syndrome, progressive conduction disease, and idiopathic AV block. These mutations can lead to progressive AV block, wide QRS complexes, and arrhythmias, with sudden cardiac death occurring in some cases.[10] The presence of conduction system disease without arrhythmias in this patient may be consistent with SCN5A-related conduction abnormalities, and genetic testing could confirm this diagnosis. Genetic testing is not available in low resource settings like ours.

Another hereditary condition, LMNA (Lamin A/C) mutations, is linked to dilated cardiomyopathy and

conduction system disorders. It typically presents with progressive AV block, eventual ventricular dysfunction, and arrhythmias.[11] Although there is no evidence of ventricular dysfunction or arrhythmias in this patient, early-stage disease cannot be excluded.

Congenital heart block, often associated with maternal autoantibodies (anti-Ro/SSA), can also cause fetal AV node damage.[12] However, this condition is less likely given the patient's history and late presentation.

Infiltrative diseases cause conduction system dysfunction by direct involvement of myocardial and conduction tissues, presenting with AV block. The absence of systemic symptoms, normal echocardiography, and absence of restrictive physiology make sarcoidosis less likely, and normal QRS voltage and the lack of restrictive physiology make amyloidosis unlikely.[13] A cardiac MRI could help exclude these possibilities,[14] but it is unavailable in our low-resource setting.

This case underscores the need for heightened clinical awareness and the timely referral for specialized electrophysiology services, as permanent pacemaker implantation remains the definitive treatment for advanced conduction system disease.

Conclusion

This case highlights the significance of early recognition and management of progressive atrioventricular (AV) conduction abnormalities in young adults, particularly when occurring without structural heart disease or systemic symptoms. The patient's presentation, characterized by symptomatic bradycardia, a progressive AV block, and normal echocardiographic and laboratory findings, is consistent with idiopathic progressive cardiac conduction disease, such as Lenegre's disease.

The absence of systemic or infiltrative disease features, coupled with the patient's normal cardiac structure, narrows the differential to an idiopathic degenerative cause, which is genetic, with SCN5A-related conduction abnormalities being the most plausible diagnosis.

Unfortunately, the unavailability of advanced diagnostic tools such as genetic testing or cardiac MRI in resource-limited settings poses challenges in confirming the underlying aetiology.

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