Challenges in Clinical Electrocardiography

A Malignant Premature Atrial Contraction

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Case Presentation

A patient in their 60s presented to the emergency department with lightheadedness for few days that was not associated with exertional activities or orthostasis. The patient reported an episode in which they felt numb for 20 to 30 seconds all over their body. The patient felt presyncopal once. They did not report any chest pain, shortness of breath, palpitations, or leg edema. The patient did not have any recent acute illness or recent travel. They had no significant medical history except for impaired glucose tolerance and obesity. The patient was not taking any prescription medications and used no illicit drugs. On physical examination, the patient appeared in no acute distress. Their blood pressure was 139/80, with a regular pulse of 76 beats/min, and their lungs were clear. Heart auscultation revealed no extra cardiac sounds or murmurs. The rest of the physical examination was unremarkable. Blood cell counts and chemistry laboratory results were all normal except for a blood glucose level of 138 mg/dL (to convert to mmol/L, multiply by 0.0555). High-sensitivity troponin levels were normal. An electrocardiogram (ECG) was conducted (**Figure**).

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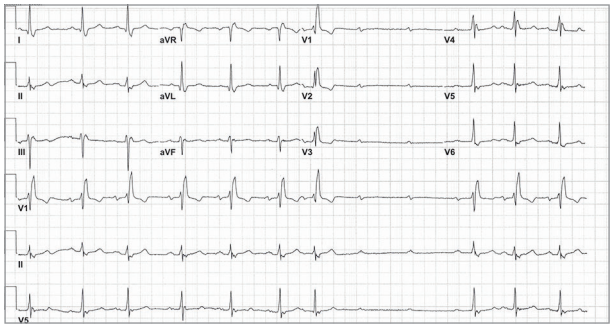
Questions: Is there a complete atrioventricular block? What is the trigger for complete atrioventricular block?

Figure. A 12-Lead Electrocardiogram (ECG)



Admission ECG showed normal sinus rhythm, right bundle branch block, and borderline first-degree atrioventricular block. A premature atrial contraction in the middle of the strip was followed by a high-grade atrioventricular block.

Interpretation

The ECG showed a normal sinus rhythm, borderline first-degree atrioventricular block, right bundle branch block, and a premature atrial contraction followed by 2 nonconducted P waves (high-grade atrioventricular block [ie, 2 or more nonconducted P waves]), resulting in a 2.8-second pause. Later, a 7.0-second pause due to sudden onset of complete heart block was detected on telemetry, again preceded by a premature atrial contraction. This condition is paroxysmal atrioventricular block (PAVB), a potentially lethal disease.

Discussion

Complete heart block is easily diagnosed and treated. Most conduction diseases are slowly progressive, recognizable, followed up over time, and treated appropriately with placement of a permanent pacemaker. Detailed guidelines have been published to address the various degrees of atrioventricular block based on severity, symptoms, and electrophysiology testing.[1] Rarely, complete heart block is intermittent (paroxysmal). PAVB is sometimes precipitated by an apparently innocuous event, such as a premature atrial contraction, or by other situations that cause bradycardia. PAVB may cause dizziness, lightheadedness, near syncope, syncope, and even death if unrecognized.

The most recognizable feature of PAVB is the sudden and unexpected occurrence of complete heart block, following normal 1:1 atrioventricular conduction, with an intervening event that produces a slowed heart rate. PAVB was first described in 1933.[2] PAVB occurs due to severe disease that involves the His-Purkinje system. The pathophysiology is an abnormal phase 4 depolarization. Phase 4 depolarization is a unique feature of the cardiac conduction system that is responsible for automaticity. During phase 4 of the action potential, a normal His-Purkinje fiber remains hyperpolarized. When diseased, a His-Purkinje fiber slowly depolarizes but remains excitable during normal heart rates. Any condition that slows the heart rate to a critical point leads to further depolarization of the His-Purkinje system, rendering it refractory to stimulation by the incoming sinus beat, resulting in atrioventricular block.[3] This is a phase 4 dependent block. A slowed heart rate precipitating PAVB could be due to a premature atrial, junctional, or ventricular beat that produces a pause, carotid sinus massage, Valsalva maneuver, or even sinus node slowing. This is in contrast to the physiological phase 3 atrioventricular block, which occurs at higher heart rates. Phase 3 and 4 blocks also lead to aberrant conduction at high and slow rates, respectively, with the former being a physiological while the latter a pathological process.

PAVB needs to be differentiated from a vagally induced heart block. The former is usually initiated by a pause due to a premature atrial or ventricular contraction. The latter is characterized by gradual slowing of the sinus rate (P-P lengthening) and atrioventricular conduction (progressive prolongation of the PR interval), then second-degree and/or third-degree atrioventricular block.[3,4] The prognosis of a vagally induced heart block is good, and attention needs to be directed at the underlying process. Pacing for a vagally induced heart block is not warranted.

The most common conduction abnormality on baseline ECG in patients with PAVB is right bundle branch block, followed by left bundle branch block and then nonspecific intraventricular conduction delay. However, 28% of patients with PAVB have a normal QRS duration.[3] This patient had a right bundle branch block and borderline first-degree atrioventricular block. The substantially wide QRS duration of 156 milliseconds, more than expected for right bundle branch block, indicated disease that involved the left bundle

branch. One of the ECGs (not shown) conducted during the patient's presentation showed transient left anterior fascicular block.

The natural history of PAVB is not well described in the literature. This patient had an ECG that showed a right bundle branch block with a QRS duration of 146 milliseconds 9 years earlier with a normal PR interval. They were asymptomatic for a very long time. PAVB is unpredictable. Event and implantable recorders could help detect PAVB. An electrophysiologic study is a specific but not a very sensitive test for diagnosing PAVB.[3]

Awareness of PAVB is essential for health care workers. High levels of suspicion and appropriate workup and monitoring are crucial to diagnosing and treating this potentially deadly disease.

This patient had transcutaneous pacing pads applied. Intravenous dopamine was initiated to minimize bradycardia. The patient was transferred to a tertiary care center, where they underwent placement of a permanent dual-chamber pacemaker the same day. Their echocardiogram showed normal left ventricular systolic function and mild aortic insufficiency. The patient was discharged home the following day in stable condition.

Take-Home Points

- PAVB is a potentially lethal disease. It is simply treated with placement of a permanent pacemaker.
- The presence of normal conduction, even at higher heart rates, does not rule out the diagnosis.
- PAVB is phase 4 dependent, and so it could be precipitated by any event that causes bradycardia.

ARTICLE INFORMATION

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