ECG IMAGE OF THE MONTH
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From Bradycardia to Tachycardia: Complete Heart Block

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PRESENTATION

Syncope in the setting of heart block is usually, but not invariably, due to bradycardia. This point was underscored when a patient's telemetry data proved surprising. The patient, a 74-year-old man with no history of cardiac disease, initially presented to an urgent care facility after noting an elevated reading on his home blood pressure monitor. He described a 4-week history of dyspnea on exertion and poor exercise tolerance but denied dizziness, syncope, chest pain, orthopnea, lower-extremity edema, or other symptoms. His medical history included obstructive sleep apnea, prostate cancer in remission, and gastroesophageal reflux disease, for which his only medication was ranitidine. He denied any use of tobacco or illicit drugs. His brother died of sudden cardiac death before the age of 50 years. The patient was a resident of the Pacific Northwest, and he had recently travelled to South America.

Upon arrival to the clinic, his blood pressure was 200/100 mmHg with a pulse of 37 beats per minute. His physical examination was otherwise unremarkable. A 12-lead electrocardiogram (ECG) showed complete heart block with a ventricular escape rhythm (**Figure 1**). Laboratory data revealed normal serum chemistry except for a creatinine level of 1.3 mg/dL. The complete blood count, iron studies, serum protein electrophoresis, and coagulation profile all returned normal results. A chest radiograph demonstrated an enlarged cardiac silhouette with evidence of mild pulmonary edema but no hilar lymphadenopathy.

Subsequently, the patient was admitted to the cardiac intensive care unit with continuous telemetry and external pacing pads in place. A few hours after admission, he had a brief episode of witnessed syncope corresponding to a wide complex rhythm on telemetry, and this was followed by several shorter occurrences of the arrhythmia (**Figures 2** and **3**). The subsequent rhythm abnormalities were associated with light-headedness but not syncope.

ASSESSMENT

A 12-lead ECG was in progress when the syncopal event occurred. This demonstrated pause-dependent polymorphic ventricular tachycardia (**Figure 4**). Because the rhythm self-terminated, the patient did not need external defibrillation. However, he underwent urgent transvenous pacer placement and coronary angiography, which demonstrated only non-obstructive coronary disease. Transthoracic echocardiography confirmed normal left ventricular systolic function without regional wall motion abnormalities and mild diastolic dysfunction. No further episodes of polymorphic ventricular tachycardia were observed after initiating ventricular pacing at 80 beats per minute.

DIAGNOSIS

Complete heart block is characterized by complete dissociation of atrial and ventricular electrical conduction, such that no signals generated above the atrioventricular node, including those emanating from the sinus node, are conducted to the ventricles. Typical junctional or ventricular escape rhythms result in pulse rates from 30-50 beats per minute. Patients often present with dizziness, syncope, or presyncope, as well as fatigue, poor exercise tolerance, and dyspnea on exertion. The differential diagnosis of acquired complete heart block includes myocardial ischemia, especially in the right coronary artery distribution, medications that impair atrioventricular nodal conduction, infiltrative disease (particularly sarcoidosis), infection

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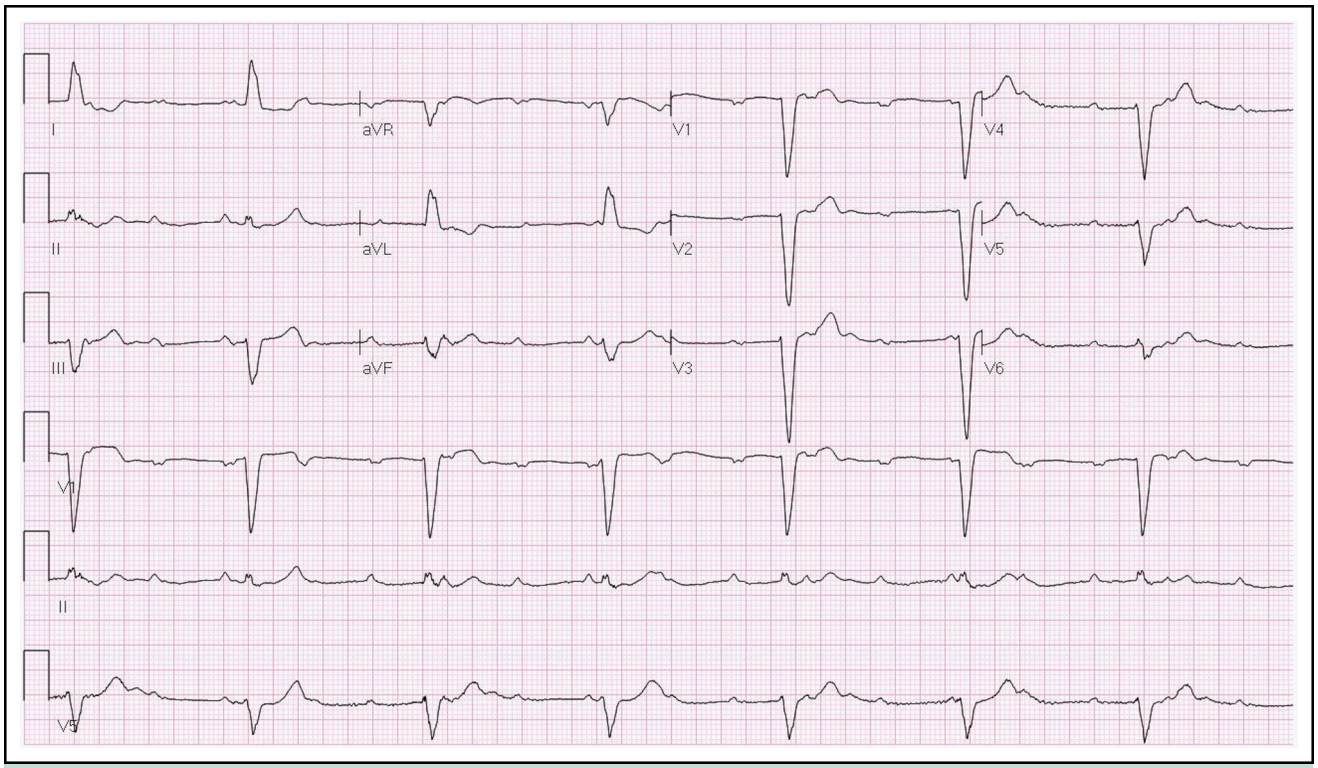

Figure 1 The baseline electrocardiogram (ECG) showed complete heart block with a sinus rate of 105 beats per minute, a wide complex regular ventricular escape rhythm of 38 beats per minute, and a QTc of approximately 440 ms.

(endocarditis with valvular abscess or Lyme carditis), inflammatory disorders, genetic conduction abnormalities, and idiopathic fibrosis and calcification of the conduction system.[1]

Our patient was presumed to have age-associated fibrosis of the conduction system, given the normal results on his coronary angiography and transthoracic echocardiogram. Ruling out structural heart disease was particularly important with his family history of sudden death. He took no culprit medications and had no history of cardiac surgery. His history indicated that he was at low risk for Lyme or Chagas disease, and the normal laboratory evaluation made cardiac amyloidosis and hemochromatosis unlikely. A chest radiograph showed that he did not have hilar lymphadenopathy or other findings to suggest sarcoidosis, a commonly overlooked cause of complete heart block in patients presenting prior to the sixth or seventh decade of life.[2]

When a patient presents with complete heart block and syncope, physicians should think beyond symptomatic bradycardia or prolonged ventricular asystole and also consider bradycardia-mediated polymorphic ventricular tachycardia or torsades de pointes as a possible cause.[3] Polymorphic ventricular tachycardia is identified by its frequently changing QRS morphology and axis. Torsades de pointes, translated as "twisting of the points," is a specific type of polymorphic ventricular tachycardia. First described in the setting of complete heart block, it is characterized by a rotating axis above and below the electric baseline and by a prolonged QT interval.[3]

QT interval prolongation in patients with heart block can be due to medications or acquired repolarization abnormalities.[4,5] In patients with concurrent torsades de points and heart block, the ECG usually shows exaggerated lengthening of the QT interval.[6] Importantly, the QT interval is dynamic and may only be extended just prior to the initiation of torsades de pointes. QT prolongations, and thus torsades de pointes, are often seen after a pause in electrical activity, such as occurs after a premature ventricular complex. Our patient's initial corrected QT interval, at 440 ms, indicated borderline prolongation (**Figure 1**), but it could be seen to lengthen before the initiation of polymorphic ventricular tachycardia (**Figures 2-4**). However, the variability of the wide complex escape morphology made it especially difficult to determine the true QTc interval.

Data suggest that there is a genetic predisposition to reduced repolarization reserve; patients who have torsades de pointes in the setting of atrioventricular block are more likely to have polymorphisms in sodium or potassium channel genes.[7] Interestingly, patients with a genetic variant are also more likely to have a family history of sudden death, as in our patient's case. The ECG captured during his