CLINICAL VIGNETTE

Sinus Arrest in a Young Adult

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Introduction

Sinoatrial (SA) node dysfunction is most common in elderly patients, accounting for more than 50% of pacemaker implantations in the United States.[1] However, it can also affect younger, otherwise healthy adults. The underlying etiology in these cases includes congenital heart diseases, familial genetic mutations, and endocrinopathies. Early recognition and management of sinus node dysfunction in these individuals is important to prevent complications including congestive heart failure, sudden cardiac arrest, and death. We present a young adult with severe sinus node dysfunction to raise awareness of evaluation and management in younger patients.

Case Presentation

A 27-year-old male with history of prior syncope presented to the emergency room after a witnessed syncopal episode two hours prior to arrival. His only other syncopal episode had occurred about seven years prior during a dental procedure with no specific intervention performed. The patient had been seated when he developed blurry vision and diaphoresis during a conversation about a coworker's recent surgery. He lost consciousness and fell off his seat, and regained consciousness within 5-10 minutes. Upon awakening, he reported feeling confused but denied other symptoms. He had no bladder incontinence, tongue biting, or myoclonic jerking. He endorsed good nutritional intake and denied consumption of alcohol prior to the event.

Past medical and surgical histories were otherwise unremarkable, and the patient did not take any medications. His family history was negative for cardiovascular or neurological conditions. He reported occasional alcohol consumption but no tobacco or other drug use.

On exam, the patient had a temperature of 36.8C, blood pressure 129/83, heart rate 63, respiratory rate 18, and oxygen saturation of 100% on room air. Orthostatic vitals were unremarkable. His cardiac exam revealed a regular rate and rhythm without abnormal heart sounds. His exam was otherwise unremarkable without neurologic deficits. Labs were only notable for a mild leukocytosis of 11.0 K/cu mm. Troponin was negative and basic metabolic panel was unremarkable. Initial electrocardiogram (EKG) had sinus arrythmia with an incomplete right bundle branch block (RBBB) (Figure 1). CT head showed no acute intracranial process.

The patient's syncope was initially thought either to be vasovagal or from a conduction abnormality in the setting of incomplete RBBB, and he was admitted for monitoring on telemetry. He reported feeling dizzy when a nurse was placing a peripheral intravenous catheter. He subsequently became unresponsive with loss of pulses and telemetry showing asystole. Code blue was called, and chest compressions were started. Return of spontaneous circulation was achieved and he regained consciousness after approximately 30 seconds. Telemetry review showed progressively worsening bradycardia followed by 37 seconds of sinus arrest without ventricular escape rhythm and subsequent return of normal sinus rhythm (Figure 2). The patient was admitted to the intensive care unit for transvenous pacemaker placement.

The patient's cardiac arrest was thought to be from excessive vagal response to noxious stimuli with underlying sinus node dysfunction. The etiology of the sinus node dysfunction was unclear. One consideration was Brugada syndrome given presence of incomplete RBBB, though the patient did not meet full criteria. Lyme disease may involve the sinus node. However, this is uncommon and Lyme serologies resulted negative. Echocardiogram showed normal left ventricular ejection fraction without any structural abnormalities. Given the severity of his symptoms with prolonged asystole on telemetry, he underwent successful placement of dual chamber permanent pacemaker. There were no further episodes of bradycardia. He was seen in cardiology a week later where he remained asymptomatic with a well-functioning pacemaker. There was one episode of sinus bradycardia that had required pacing. He remained well on multiple follow up visits over three years with no further adverse events.

Discussion

The sinus node is the dominant pacemaker of the heart, producing a cyclical, organized electrical rhythm to maintain good cardiac output. Sinus node dysfunction refers to the inability of the sinus node to produce an adequate heart rate to meet physiological needs.[1] It can occur either at rest or on exertion, with the latter representing an inadequate chronotropic response. In an otherwise healthy heart, additional pacemaker cells such as the atrioventricular (AV) node, the Bundle of His, and the Purkinje fibers can initiate pacing signals if the sinus node fails. These cells fire at slower rates compared

to the SA node, which is why SA node dysfunction can lead to bradycardia or cardiac arrest.[2]

Multiple causes can lead to sinus node dysfunction in young adults. Congenital heart diseases may be the most common cause, with transposition of the great arteries reported as most common.[3] In these cases, sinus node dysfunction results from the absence, displacement, or malformation of the sinus node. Iatrogenic injury from corrective cardiac surgery is another common etiology.[4] Familial gene mutations have also been implicated in sinus node dysfunction. One example is Brugada syndrome, which involves mutations in the cardiac sodium channel α-subunit SCN5A.[5] Neuromuscular disorders are another possible cause. Muscular dystrophies such as myofibrillar myopathy can lead to dilated, restrictive, or hypertrophic cardiomyopathies which can lead to sinus node dysfunction.[6] Other less frequent causes include extrinsic insults to the SA node including electrolyte disturbances, hypothermia, and hypothyroidism. Sinus node dysfunction can be purely idiopathic, as presumed in this case.

Diagnosing sinus node dysfunction requires the presence of both relevant clinical and pertinent EKG findings. Symptoms vary depending on the severity of dysfunction and on the individual, but can include lightheadedness, dizziness, fatigue, and syncope. Those with underlying cardiac comorbidities may experience anginal symptoms including chest pain or shortness of breath.[1] Characteristic EKG findings include inappropriate or severe sinus bradycardia, sinus pauses, sinus arrests (>3 seconds), sinus exit blocks with or without escape rhythms, and alternating tachycardia and bradycardia.[7] Sinus node dysfunction is diagnosed when there is a correlation between symptoms and these EKG findings. If there is concern that EKG did not capture the patient's symptoms, exercise stress testing or ambulatory EKG monitoring may be obtained.[7]

The first step in treatment of sinus node dysfunction is to ensure hemodynamic stability. Unstable patients should be treated using the advanced cardiac life support protocol for symptomatic bradycardia. They may require chronotropic agent infusion or transvenous pacing. Treatment for stable patients depends on their symptoms. Any reversible causes should be corrected to assess if the sinus node dysfunction resolves. If this is unsuccessful, symptomatic patients require placement of a permanent pacemaker. Observation is recommended in asymptomatic patients.[1]

The clinical course following treatment for sinus node dysfunction in young adults depends on the presence of underlying cardiac abnormalities. One retrospective review found that all symptomatic patients experienced resolution of their symptoms after pacemaker placement. Prognosis appeared excellent for patients without underlying heart disease.[3]

Conclusion

Sinus node dysfunction is uncommon in young adults, but can lead to serious complications including cardiac arrest. Early recognition and treatment of this condition is critical. Management involves addressing any reversible causes and considering pacemaker placement based on symptoms. The prognosis in young adults is typically excellent in the absence of underlying cardiac disease.

Figures

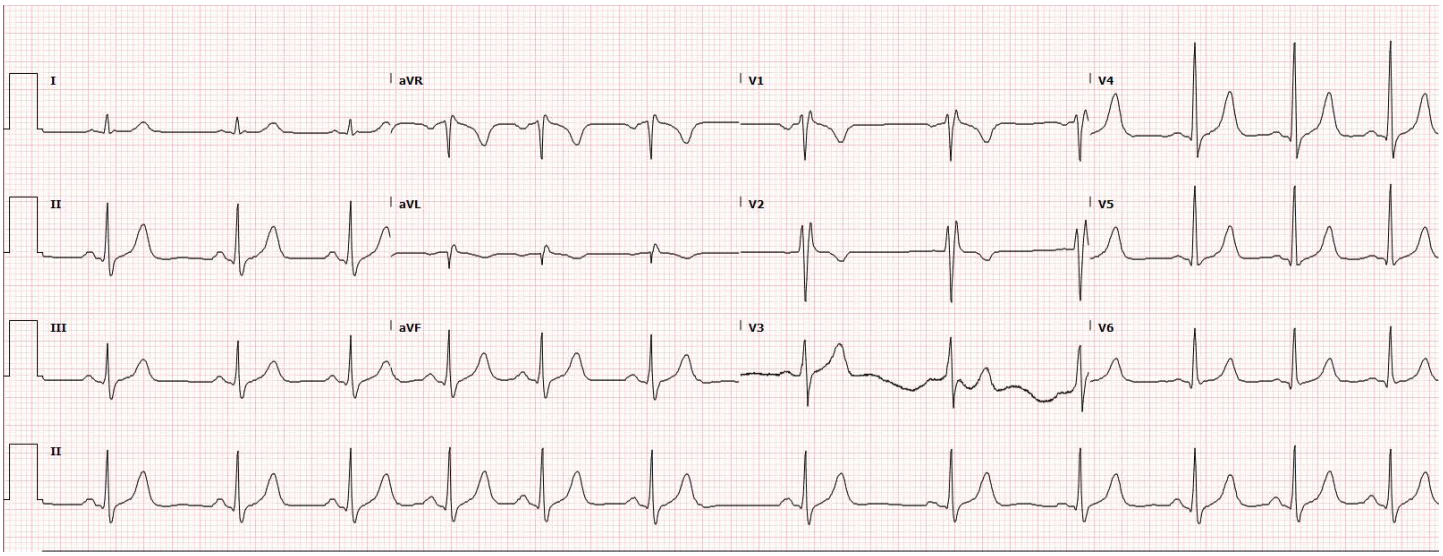

Figure 1: EKG showing incomplete RBBB and sinus arrythmia.

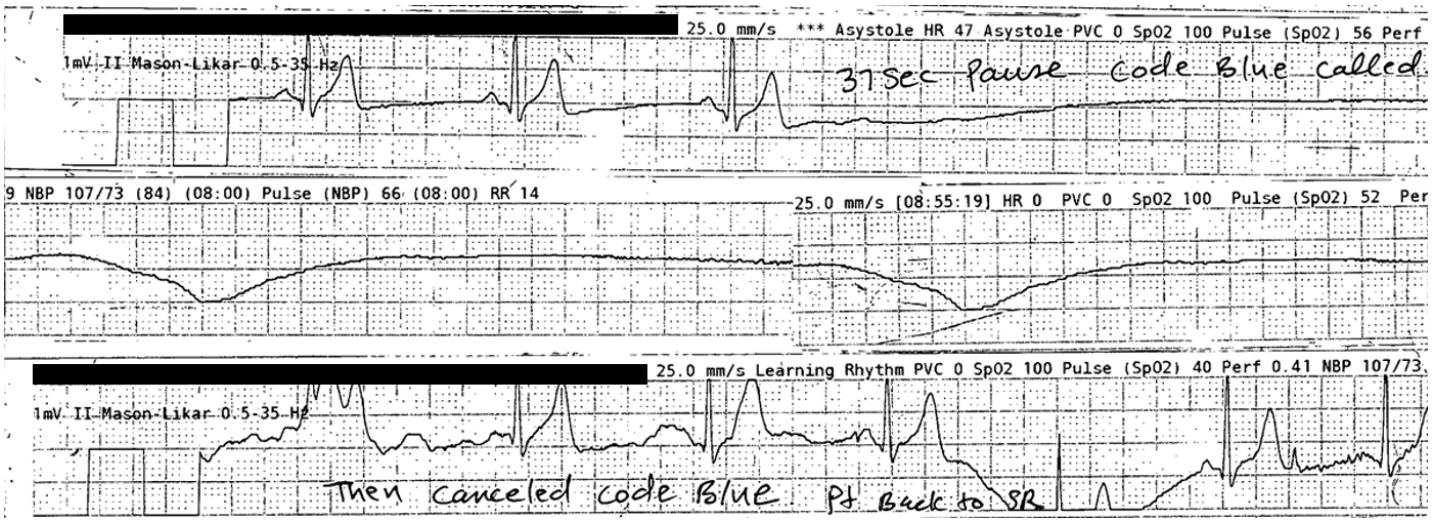

Figure 2: Telemetry showing 37 second sinus pause with subsequent normal sinus rhythm.

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