A 38-year-old woman was admitted to the hospital for further evaluation of an unwitnessed syncopal episode at home. Her past medical history included known bradycardia and recurrent syncope since she was five years old. Of note, an electrophysiologist had recently implanted a loop recorder to further investigate these syncopal episodes. Prior evaluation, including a Holter monitor, transthoracic echocardiogram (TTE), and electroencephalogram to rule out seizures, was unremarkable.

On the day of admission, the patient reported lower abdominal cramping of moderate intensity associated with nausea that awakened her from sleep. She recalled having a bowel movement that relieved her cramping prior to losing consciousness. Her husband heard a loud thud and found her on the bathroom floor. She awoke shortly thereafter and could not recall the events described by her husband. She denied any preceding or current chest pain, palpitations, shortness of breath, light-headedness, diaphoresis, nausea, or headache prior to losing consciousness. There was no evidence of loss of bladder or bowel control, biting of her tongue, or witnessed seizure-like activity. Nonetheless, she presented to the emergency department (ED) for further evaluation.

The patient’s only medication was metronidazole. She rarely drank alcohol and did not use tobacco or illicit drugs. She exercised vigorously, including running marathons and skiing.

On arrival to the ED, her vital signs, including orthostatics, were within normal limits. A comprehensive physical exam was unremarkable. Her laboratory results were notable for a mildly decreased free T4 of 0.69 ng/dL (range 0.76-1.46 ng/dL). Her electrocardiogram (ECG) showed normal sinus rhythm without any atrioventricular block or ischemia. The patient was given a presumptive diagnosis of vasovagal syncope given that her syncopal episode was temporally associated with a bowel movement. The initial plan was for same-day discharge and outpatient follow-up with her electrophysiologist the following day. However, on further review of the history, the patient’s husband reported that his wife was snoring when he found her on the bathroom floor and that her face began turning blue once she stopped snoring. In light of this severe apneic episode, she was admitted for further evaluation including telemetry monitoring, TTE (unremarkable), and interrogation of her loop recorder, which revealed a 46-second asystolic pause as the etiology of her syncopal event. Figure 1 shows the beginning of the asystolic pause. Six pages of the interrogation were required to completely capture the event. Four pages showed the flat line associated with asystole, as seen in Figure 2. Given this alarming finding, a dual chamber pacemaker was placed the next day and the patient discharged home in stable condition.

**Discussion**

Syncope is a common condition frequently seen in the emergency department. Syncope requiring admission to the hospital is typically seen in elderly patients with vasovagal events or marked dehydration. This remarkable case presents a rare finding of prolonged asystolic pause leading to syncope in a healthy young woman that initially was attributed to vasovagal syncope. It highlights the pitfalls of anchoring bias and the importance of taking a detailed history, which ultimately led to the correct diagnosis and appropriate management of this patient.

Syncope is defined as the “abrupt and transient loss of consciousness caused by cerebral hypoperfusion.” The etiologies of syncope are divided into three broad categories: orthostatic hypotension, neurally-mediated, and cardiogenic. Patients considered to have low-risk syncope include those who are less than 50 years-old without cardiovascular disease, with a normal ECG and cardiovascular exam, and symptoms more consistent with orthostasis or neurally-mediated syncope.1 Cardiac etiologies account for approximately 20% of syncopal events and are potentially life-threatening with the majority caused by arrhythmias rather than structural disorders.1

A thorough history and physical exam help differentiate the different categories of syncope and other causes of transient loss of consciousness. Key features of a patient’s history suggestive of syncope due to an arrhythmic etiology include syncope during exertion, palpitations, or family history of sudden death.1 Features of cardiac nonarrhythmic etiologies include history of coronary heart disease, previous myocardial infarctions, and other signs and symptoms of structural heart disease.1 Essential components of the physical examination include orthostatic vital signs, a careful cardiovascular examination, and a thorough neurological examination.

This case underscores the importance of a meticulous history and obtaining witness accounts of syncope if available. The history provided by the patient and the benign examination initially categorized her into a low risk syncope. Specifically,
her account suggested a neurally-mediated etiology, such as a vasovagal event in the setting of defecation. The key history of severe apnea provided by her husband suggested an alternative, higher-risk, and possibly cardiac etiology of her syncope. Therefore, expedited investigation into potential life-threatening causes of her syncope was pursued as opposed to the initial plan of discharge home with close follow-up.

Further evaluation of syncope includes tests to assess for cardiac and neurological etiologies. A 12-lead ECG helps to identify cardiac causes of syncope, such as arrhythmias, high-grade atioventricular block, sinus bradycardia, QT prolongation, or ischemic changes. An echocardiogram assesses for structural heart disease. Suspicion of exertion syncope may warrant stress testing. Tilt-table testing evaluates for neurally mediated or reflex syncope. Imaging of the brain and its vasculature may be indicated if abnormalities are found on neurological examination. However, brain imaging is not recommended in the evaluation of a patient with simple syncope and a normal neurologic examination. Similarly, EEG is not recommended in patients without neurological evidence of seizures. Labs, including basic metabolic profile and serum or urine drug screens, are indicated to assess other etiologies of transient loss of consciousness.

Continuous ECG monitoring is useful for outpatient evaluation of patients with unexplained syncope. The type of monitor selected is based on the frequency and nature of the syncopal event. A Holter monitor is recommended if symptoms are frequent enough to be detected within 24-72 hours. External loop records (ELR), such as a ZioPatch, may be utilized in patients with a history of syncopal episodes occurring in a period of less than four weeks with ECG findings of arrhythmia. Internal loop records (ILR) are recommended for patients with infrequent syncopal episodes occurring within a period time of greater four weeks. Studies using ILR found that severe bradycardia or asystole are common findings for unexplained syncope. In this case, interrogation of the patient’s loop recorder revealed a 46-second asystolic pause as the etiology of syncope. Causes of asystole or marked bradycardia leading to syncope include dysfunction of the sinus node, high-grade atioventricular conduction block, carotid sinus syndrome, and pathologic vasodepressor reactions.

Management of patients with syncope is dependent on the underlying diagnosis. Pacemaker implantation is indicated for patients with symptomatic bradycardia or asystolic pauses, as in this patient. Education to help minimize injury, such as lying down when feeling presyncopal or performing physical counter-pressure techniques, such as hand grip, leg crossing, and squatting, is recommended when syncope is neurally mediated. Management of orthostatic syncope depends on the etiology of the patient’s hypovolemia and may include volume expansion or medication initiation.

Conclusion

The overall assessment and management of a patient with syncope is wholly dependent on a detailed history and physical exam. In the present case report, the patient had undiagnosed recurrent syncope without known risk factors for severe bradycardia or asystole. Her presentation appeared to be initially consistent with vasovagal syncope, but a key element of her history led to the correct and potentially life-saving diagnosis of prolonged asystolic pause and urgent placement of a pacemaker.

REFERENCES


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