CASE REPORT

A CASE OF RECURRENT SYNCOPE: TO PACE OR NOT TO PACE IS THE QUESTION?

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Abstract: Syncope is a frequently encountered condition in the emergency room, with vagal syncope being the most prevalent form. However, overlapping symptoms can pose diagnostic challenges when distinguishing between vagal syncope and symptomatic sinus node dysfunction. A comprehensive history and careful evaluation of hemodynamics and electrocardiogram findings are crucial for accurate differentiation. Vagal syncope is typically associated with an upright posture, and supine syncope is considered atypical. In this case report, we describe an intriguing instance of supine vagal syncope, highlighting the importance of correct diagnosis to avoid unnecessary pacemaker implantation.

Keywords: Supine, Syncope, Vagal, Sinus Node Dysfunction

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INTRODUCTION

Syncope is a common and challenging presentation in the emergency room.[1] Vagal etiology is the most common cause of syncope, but it can overlap with symptomatic sinus node disease, making the diagnosis more challenging.[2] Distinguishing between the two entities is crucial, as one may require a pacemaker while the other can be managed with lifestyle modifications and counter-maneuvers.[3] A comprehensive patient history is crucial in differentiating between these conditions and guiding cost-effective diagnostics.[4] In this case report, we present the diagnostic quandary of a middle-aged man with an atypical presentation of syncope.

CASE PRESENTATION

A 63-year-old gentleman was referred to the cardiology department by internal medicine due to recurrent fainting episodes. He had been experiencing episodes of dizziness and near-fainting for the past year. Initially, the episodes were infrequent, occurring once every five months. However, the frequency had increased in the week leading up to his visit to the clinic. The patient felt well before and after the events, with no associated trauma or confusion. These episodes typically occurred at rest and were not triggered by exertion. The patient did not experience prodromal symptoms such as headache, palpitations, visual disturbances, nausea, sweating, or tinnitus. The syncope was not associated with seizure-like activity, and there were no characteristic signs such as up-rolling of the eyes, jerking of limbs, or urinary or bowel incontinence. On detailed questioning, he mentioned feeling lightheaded and dizzy before a typical attack. Each episode lasted a few minutes, followed by spontaneous regaining of consciousness without post-episode confusion, and he quickly returned to his baseline state. The patient had hypertension that was well-controlled with Losartan.

As these symptoms were affecting his quality of life, he sought consultation with an internist. Initial assessment in the clinic revealed normal vital signs, with a blood pressure of 135/75 mmHg and no postural drop. His general physical examination was unremarkable. A cardiovascular examination showed normal heart sounds without any additional sounds. Neurological examination showed no focal deficits. A resting 12-lead electrocardiogram (ECG) was within normal limits, showing normal sinus rhythm with a heart rate of 72 bpm, normal PR interval, normal axis, normal QRS duration, and normal ST and T waves. The corrected QT interval (QTc) was 393 msec (Figure 1).

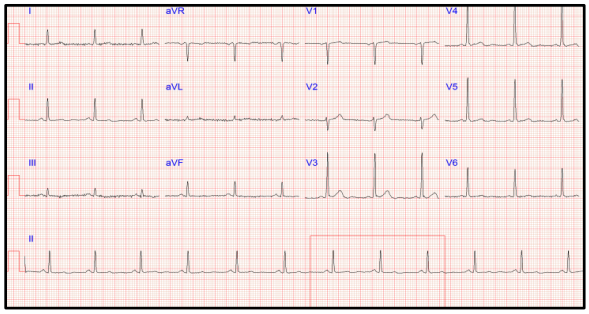

Figure 1: Normal sinus rhythm 72 bpm, normal PR interval, normal axis, normal QRS duration with normal ST, T waves, and a QTc of 393 msec

Further investigations, including complete blood count (CBC), creatinine, electrolytes, thyroid profile,

and cardiac biomarkers, all yielded normal results. An MRI of the brain revealed focal gliosis in the left occipital and temporal lobes, likely due to prior ischemic events and mild periventricular microvascular ischemic changes, with no acute infarction or intracerebral hemorrhage. Suspecting scar epilepsy, he was started on anti-epileptic medication and advised to undergo an advanced workup.

Holter monitoring and an electroencephalogram (EEG) were recommended. While undergoing EEG within the 24 hours of ECG surveillance, the patient experienced a witnessed syncopal episode while lying on the couch. This prompted a cardiology consultation as the syncope occurred in the supine position. The Holter monitoring revealed a sinus pause of 3.0 seconds (Figure 2).

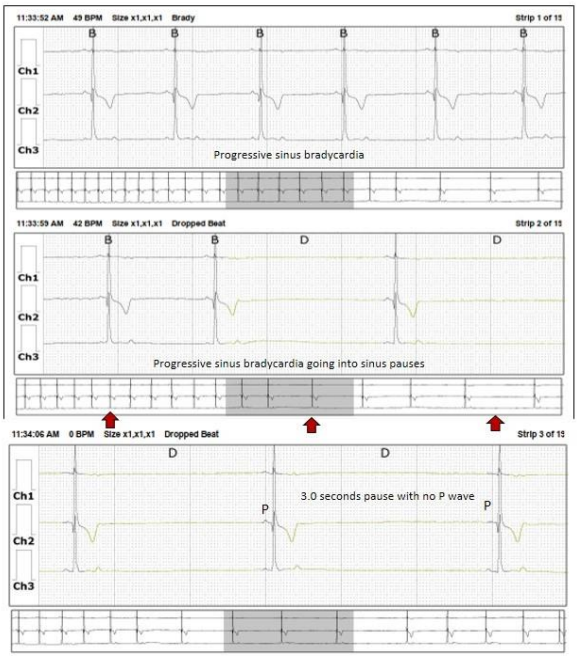

Figure 2: In a period of 14 seconds, the rhythm goes from sinus rhythm to sinus bradycardia and sinus pause. The longest was 3.0 seconds, noted at the time of syncope in the supine posture. This pattern appears to be typical of a vagally mediated sinus slowing and pause, and the immediate acceleration noted in the latter half of the lower panel

Following the syncopal episode, the patient spontaneously regained consciousness and was transferred to the critical observation area after a few minutes. The consulting question was whether a pacemaker was warranted, considering the syncope in the supine position and the associated 3.0 -second pause during wakefulness.

The Holter monitoring reported an average heart rate of 73 bpm with a normal diurnal variation. There were eight dropped beats, and the longest R-R interval observed was 3.8 seconds during the nocturnal period. The echocardiogram showed normal left ventricular systolic function (visually estimated LVEF = 55-60%), mildly dilated left atrium, grade I left ventricular diastolic dysfunction, moderate mitral regurgitation, and mild tricuspid regurgitation. Based on the typical sinus slowing with a pause followed by a warm-up period, the cardiology team requested a head-up tilt test (HUTT).

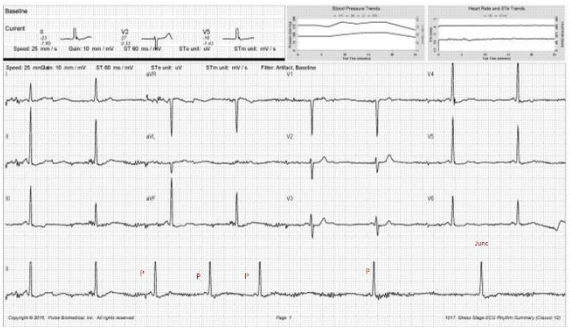

Figure 3A: At 16 minutes post provocation with nitroglycerine, the first syncope artifact is noted on the ECG as noise. There is sinus slowing with PR interval shortening. The PR interval is shorter than the normal 12 lead PR interval at baseline, and then there is a junctional beat, and the rate at that point is 34 bpm

The HUTT yielded positive results, showing bradycardia (heart rate decreased from 119 to 34 bpm, 16 minutes after provocation with 500 mcg of sublingual nitroglycerin) along with hypotension (blood pressure dropped from 130/85 mmHg to unrecordable levels), resulting in transient loss of consciousness. The ECG during the test demonstrated sinus bradycardia with intermittent junctional escape rhythm (Figure 3A). During the recovery period, transient atrial fibrillation with a controlled ventricular rate was recorded (Figure 3B).

Based on the fainting episodes accompanied by subtle prodromal symptoms that the patient recalled only after detailed questioning and considering the fact that a supine subject should tolerate a heart rate in the 30s without loss of consciousness, a diagnosis of vagal syncope of mixed type with a more predominant hypotensive response was made. All investigations confirmed a vagal etiology, and therefore, a

pacemaker was not considered necessary, as true symptomatic sinus node dysfunction would be an indication for one. The patient was advised to increase salt and fluid intake, taught counter maneuvers and preventive measures, and has been followed up without further recurrence of syncope.

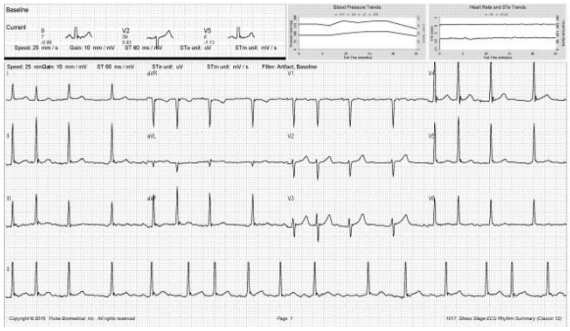

Figure 3B: Transient atrial fibrillation noted in the recovery phase of the head-up tilt test. The ventricular rate is controlled at 98 bpm. The rhythm settled spontaneously

DISCUSSION

Our case serves as an instructive example as it presents syncope occurring in the supine position, challenging the classic teaching that vagal syncope occurs exclusively in the upright posture. Although vagal syncope is typically associated with an upright position, there have been documented cases of supine syncope or sleep fainting with vagal etiology.[5-7] Comprehensive history-taking is crucial for diagnosing syncope, and while there is overlap in features, certain characteristics, such as a prolonged post-ictal state, may indicate syncope of neurogenic origin. It's important to note that without accompanying factors, pure bradycardia alone triggers a natural physiological response of vasoconstriction, which prevents syncope.[8] However, factors such as vasodilation or volume depletion can lead to concomitant hypotension and syncope, necessitating careful assessment.

In our case, the patient experienced syncope while lying down after being seated in the room awaiting an EEG. The Holter monitor recorded 14 seconds of sinus rhythm transitioning into sinus bradycardia. Since the patient was in a supine position with a heart rate in the 30s, cerebral perfusion could have been maintained. However, the profound vasodilatory response resulted in fainting. This response was later confirmed during the head-up tilt test (HUTT), and the patient acknowledged that the subtle symptoms he had previously downplayed were similar to those experienced during the syncope episode observed during the HUTT. Furthermore, the presence of a transient short-run of atrial fibrillation (AF) supports a vagal etiology. AF triggered by vagal activation is well-documented in the literature.[9]

Symptomatic sinus node dysfunction warrants pacemaker implantation. However, trials assessing the efficacy of pacemakers for all patients with vagal syncope have yielded mostly negative results, albeit with certain caveats. Pacemakers may have a role in a select group of patients with asystole as the predominant response. Based on available data, dual-chamber pacing with the closed-loop stimulation algorithm appears to be a viable alternative, if not the best, for preventing recurrent episodes.[10] Counter maneuvers are often effective for patients with vagal syncope and a mixed response. Implanting a pacemaker in cases of symptomatic bradycardia of vagal origin, which can be managed noninvasively, would be counterproductive. Pacemakers do not address the preceding vasodilation and may exacerbate the situation with inappropriate pacing or lead to complications.

CONCLUSION

Reflex vagal syncope is the leading cause of syncope and should be considered a primary differential diagnosis, especially in younger and middle-aged patients. Thorough and calm history-taking is paramount, resembling a detective's interrogation. In cases of ambiguous history, further investigations should be pursued to assess the need for a pacemaker. It is crucial to understand the vagal response system, as it causes paradoxical bradycardia and can initiate transient atrial fibrillation.

AUTHORS' CONTRIBUTION

FIH and AQ: Concept and design, data acquisition, interpretation, drafting, final approval, and agree to be accountable for all aspects of the work. IA, and AHK: Data acquisition, interpretation, drafting, final approval and agree to be accountable for all aspects of the work.

Conflict of interest: Authors declared no conflict of interest.

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