SINUS NODE DISEASE IN A YOUNG FEMALE WITH CARDIOGENIC SYNCOPE

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ABSTRACT

Sinus node dysfunction (SND) is characterized by disease of the sinus node and/or contiguous atrial tissue resulting in the generation of heart rate that is not commensurate with the physiologic need of the body due to various causes. SND is an ailment commonly found in the advanced age group, with a mean age of 68 years, and is rarely found in the young. Making a diagnosis requires the presence of symptoms to coincide with the abnormal electrocardiographic (ECG) findings, and the symptoms of SND are non-specific. There is a limited report of this condition in the young, and available reports show that most cases of SND occur in the young and children with underlying heart defects and mostly in males. The report describes an unusual presentation of ‘sinus node disease’ in a young Caucasian female with no underlying cardiac abnormalities that presented with syncope and sinus bradycardia. A 36-year-old Caucasian female presented to our facility with complaints of recurrent episodes of a brief period of loss of consciousness associated with intermittent palpitations and no background cardiovascular disease. Rest 12-lead ECG showed bradycardia, while a 24-hour Holter ECG showed periods of bradycardia, ‘supraventricular tachycardia’, and tachycardia-bradycardia occurring with symptoms. A diagnosis of SND was made and a permanent pacemaker was offered, but she and her spouse opted to have the procedure done in her home country. ‘Sinus node disease’ through an ailment of the aged can be found in the young without underlying cardiac defects and can be suspected in people with unexplained syncope.

Keywords: Electrocardiography (ECG), Holter ECG, Palpitation, Pacemaker, Sinus node, Syncope

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INTRODUCTION

The ‘sinoatrial node’ dominates the pacing activity of the heart and has been described as a defined area that lies at the intersection of the ‘superior vena cava’ and the ‘right atrium’ (Jabbour and Kanmanthareddy, 2022). ‘Sinus node dysfunction (SND) was first reported as a cardiovascular disease in 1968 although Wenckebach narrated the electrocardiographic presentation of ‘sinus node dysfunction’ in 1923 (Zipes and Jalife, 2004). It was formerly known as ‘sick sinus syndrome’, and describes a disorder related to abnormal conduction and propagation of electrical impulses at the ‘sinoatrial node’. During periods of increased physical activity or exertion the abnormal rhythms do not adapt adequately to meet the physiologic demands of the body, hence producing symptoms in the patient (Hawk et al., 2021).

‘Sinus node dysfunction’ is a disease of older adults, although it can present at any age. The average age of a patient with ‘sinus node dysfunction’ is 68 years (Dakkak and Doukky, 2022), it was also found to be one of the most commonest indications for pacemaker insertion in the elderly population (Ewy, 2014).

Reported cases of ‘SND’ are very few in Nigeria, Falase et al. (2013) reported five cases of ‘sick sinus syndrome’ with bradycardia among their patients that had ‘permanent pacemaker implantation’. Common risk factors are advancing age, hypertension, high ‘body mass index’ (BMI), and prior cardiovascular event. However, sinoatrial disease is rare in the young, and even amongst the young, males are more affected than females (Mackintosh, 1981).

The intrinsic causes of ‘SND’ include degeneration and fibrosis of the sinoatrial node and/or tissues around the node from myocardial infarction, infiltrative cardiomyopathies (amyloidosis, sarcoidosis), and cardiac surgery, while extrinsic factors include drugs, thyroid diseases, hypoxia, hypothermia, electrolyte derangement and infections(diphtheria) (Semelka, Gera, Usman, 2013). The clinical presentation of ‘SND’ may include syncope, pre-syncope, palpitations, or dizziness; however, it can be asymptomatic or present with subtle or nonspecific symptoms. ‘Sick sinus syndrome’ has multiple electrocardiogram manifestations, including ‘sinus bradycardia’, ‘sinus arrest’, ‘sinoatrial block’, and alternating patterns of bradycardia and tachycardia (bradycardia-tachycardia syndrome) (Adan & Crown, 2003). These abnormal rhythms are responsible for the end organ hypoperfusion which is the underlying pathogenesis for the common symptoms experienced by the patient, and the Holter ECG is pivotal in diagnosing ‘SND’.

This is a case report of ‘sinus node dysfunction’ in a 35-year-old Caucasian female who presented with syncopal attacks, intermittent palpitations, ‘sinus bradycardia’, and no underlying cardiac disease, to highlights the fact that although SND is a disease commonly seen in the aged population, it can also be seen in the young without any background cardiac disease, even in the female sex too.

CASE REPORT

A 36-year-old female Caucasian came to the cardiology clinic with complaints of recurrent transient loss of consciousness and intermittent palpitations. The last episode of the brief period of loss of consciousness was said to have occurred around 2:30 pm the day before her presentation, which started with some abdominal discomfort in the
office. She decided to use the restroom, and after standing up from her desk she started feeling light-headed but decided to continue to proceed to the restroom. Just before she could reach the entrance of the restroom, she suddenly found herself on the floor just before the said entrance. The period of loss of consciousness was estimated at 15 seconds because she was a couple of steps away from the entrance to the restroom before she found herself on the floor. No injuries were sustained during the fall. Her husband also reported that he had witnessed similar attacks that occurred in their home, which first started about 3 months before the presentation.

She also complained of intermittent palpitations which occurred at random times of the day and were usually associated with dizziness, but no associated chest pains, diaphoresis, or shortness of breath. No identifiable triggering factors and they usually lasted for between 3-5 minutes. She was not on any routine medications, and she did not report any underlying cardiovascular disease or significant family history.

On examination in the clinic, she was not in any distress, not pale, anicteric, afebrile, and had no pedal edema. A cardiovascular examination revealed a pulse of 59 beats/minute, which was regular with occasional missed beats. Her blood pressure reading was 100/66 mmHg with no orthostasis after 1 and 5 minutes of standing. First and second heart sounds auscultated with some ectopic beats, and examination of other systems was essentially normal. An initial assessment of ‘syncope’ caused by bradyarrhythmia was made, and some investigations were ordered which had the following results. (Table 1)

### Table 1: Investigation results.

<table>
<thead>
<tr>
<th>PARAMETERS</th>
<th>RESULT</th>
<th>REFERENCE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calcium</td>
<td>2.9 mmol/L</td>
<td>2.3 - 2.7 mmol/L</td>
</tr>
<tr>
<td>Magnesium</td>
<td>1.0 mmol/L</td>
<td>0.8 - 1.1 mmol/L</td>
</tr>
<tr>
<td>Cardiac Troponin T</td>
<td>&lt; 50 ng/L</td>
<td>&lt; 50 ng/L</td>
</tr>
<tr>
<td>Free Thyroxine test (fT4)</td>
<td>0.73 ng/ml</td>
<td>0.8 - 2.24 ng/ml</td>
</tr>
<tr>
<td>Free Triiodothyronine test (fT3)</td>
<td>2.33 pg/ml</td>
<td>1.4 - 4.2 pg/ml</td>
</tr>
<tr>
<td>Thyroid-Stimulating Hormone (TSH)</td>
<td>1.93 mIU/L</td>
<td>0.39 - 6.16 mIU/L</td>
</tr>
</tbody>
</table>

An urgent rest 12-lead ECG was done which showed ‘sinus bradycardia’ with poor R wave progression in the precordial leads (Figure 1), prompting a ‘24-hour Holter ECG’ to be ordered.
The ‘24-hour Holter ECG’ was done with a CONTEC ECG (3-lead) Holter machine, model TLC: 9803. The patient’s chest was cleaned with methylated spirit and dried, and then 3M Red Dot adhesive electrodes were attached to five spots on her chest, representing V1, V3, V5, V- and N. The positions were the 4th intercostal space to the right of the sternum, 5th rib mid-clavicular line, 5th intercostal space left anterior axillary line, below the angle of Louis and the right lower chest respectively. After lead attachment, the device function was assessed with a pretest, and after a satisfactory assessment, rhythm detection and recording were commenced with the device. She was then given a ‘Holter-diary’ to record the times she experienced any symptoms during the Holter ECG monitoring period. Twenty-four hours later, she returned with the device, and the information from the portable device was downloaded into a 3-channel ECG Holter System analyzer software. Then the 3 ECG strips were analyzed by two Consultant Cardiologists.

RESULTS
The patient’s Holter-diary presented two periods of palpitations both lasting about 36 minutes and 24 minutes, which coincided respectively with the timeline of ‘atrial fibrillation’ and ‘supraventricular tachycardia’ on the Holter strips. The Holter ECG report also showed varying periods of different rhythms, including the following; regular sinus rhythm (Figure 2), sinus bradycardia (Figure 3), sinus bradycardia with unifocal premature ventricular complexes (Figure 4), atrial fibrillation (figure 5), supraventricular tachycardia (figure 6) and alternating tachycardia-bradycardia (Figure 7).
**Figure 2:** The patient’s holter ECG strip showing regular sinus rhythm

**Figure 3:** The patient’s holter ECG strip showing sinus bradycardia
**Figure 4:** The patient’s holter ECG strip showing ‘sinus bradycardia’ with unifocal premature ventricular complexes

**Figure 5:** The patient’s holter ECG strip showing atrial fibrillation
She was not on any medications that could be implicated as a possible cause of the rhythm abnormalities that caused her symptoms, so she was advised to do coronary angiography to ensure there was no ‘coronary artery disease’, and was also offered a ‘permanent pacemaker’. However, she decided to travel back to the United State for further treatment.
**DISCUSSION**

‘Sinus node dysfunction’ occurs when there is ‘sinus node’ failure. This makes the conducting system of the heart produce a rhythm that is unable to meet the physiologic need of the body due to various causes (Dobrzynski, Boyett, Anderson, 2007). Intrinsic causes of ‘SND’ are due to either disease affecting the ‘sinus node’ or the ‘sinoatrial tissue’ (Dobrzynski et al., 2007; Adan & Crown, 2013). As noted in this case, the abnormal pacing function of the ‘sinus node’ resulted in reduced cardiac output and the various symptoms she experienced, that is, the ‘syncope’ and ‘presyncope’ (Lamas et al, 2000).

SND is commonly a medical disorder of the age group with an average age of 74 years and affects males and females equally (Falase et al., 2013), but some studies report the occurrence of ‘sinus node dysfunction in the young. A study was done by Albin et al. (1985), where 49 patients who had undergone ‘permanent pacing’ for ‘sinus node dysfunction’ were studied. The average age was 23 years, the most common initial symptom was syncope and the dominant rhythm abnormality was tachycardia-bradydysrythmia syndrome. Results showed that 64% of the participants had underlying cardiovascular disease, and 51% of them had previously undergone cardiac surgery. This case report described a 35-year-old woman who was diagnosed with SND after presenting with syncope without any underlying cardiac disease or previous cardiac surgery. Possible causes in the young include some childhood and familial disease e.g., ‘congenital heart disease’, post ‘corrective cardiac surgery’, a mutation in SCN5A ‘cardiac sodium channel gene’ etc (Benson et al., 2003). She was not able to give a detailed family history in this case.

The patient in this case presented with ‘syncope’ and ‘presyncope’, which were found to have coincided with ECG abnormalities leading to the diagnosis of SND. These clinical features are found in about 50% of cases and are due to decreased perfusion of the brain (Rodriguez and Schocken, 1990). Other symptoms like fatigue, dyspnea, and chest discomfort or pressure were not present during the clinical assessment of this patient. A possible explanation for this may be due to the highly nonspecific, transient, and infrequent nature of the symptom which makes it very difficult for patients to identify.

ECG findings that occur together with symptoms are a crucial key factor in diagnosing SND. The ECG may also be the only easily accessible tool to diagnose this condition in asymptomatic patients. These abnormalities include ‘bradycardia’, ‘sinus pauses’, ‘sinus arrest’, ‘SA nodal exit block’, ‘supraventricular tachycardias’ (‘atrial fibrillation’, ‘atrial flutter’, ‘atrial tachycardia’) and alternating bradycardia and atrial tachyarrhythmia (Fuster et al., 2001). The presenting ECG in this case showed bradycardia while the 24-hour Holter ECG showed varying periods of ‘sinus rhythm’, bradycardia, ‘atrial fibrillation’, and ‘supraventricular tachycardia’. This patient likely experienced episodes of ‘syncope’ and ‘presyncope’ during a period of ‘sinus arrest’, as it was reported in a study by Arthur et al. (2000), that ‘cardiogenic syncope’ due to arrhythmias using occur during ‘sinus arrest’, or during the long pause that follows the spontaneous conversion of a supraventricular tachyarrhythmia to regular sinus rhythm.

Using an ‘event loop recorder’ or ‘implantable event monitors’ have been found to have more yield than 24-hour Holter ECG in SND due to the fleeting and unpredictable nature of the symptoms (Furukawa et al., 2012; Lipski et al., 1976). However, in this case, the 24-hour study was enough to identify the underlying rhythm abnormalities. Stress
ECG (Kusumoto et al., 2019), a pharmacologic challenge with atropine or adenosine (Viskin, Justo, Halkin, 2007), and electrophysiologic testing are other investigative modalities that are pivotal in clinching the diagnosis of ‘SND’ (Kusumoto et al, 2019).

Treatment of ‘SND’ involves inserting a ‘permanent pacemaker’ to relieve symptoms after attending to extrinsic causes (Epstein et al., 2008, 2009). The use of anticoagulants to prevent thrombus formation in patients with ‘atrial fibrillation’ and tachycardia-bradycardia syndrome is essentially the only indication for drug administration in SND as there is currently no pharmacologic therapy recommended in patients with symptoms. The patient was offered a permanent pacemaker which she declined. Instead, she opted to travel back to her country for further management.

CONCLUSION
Although SND is more common with increasing age, it can very well be seen amongst the young. The diagnosis depends on matching the patient’s abnormal rhythm to his/her symptoms, and this can be conveniently done with a 24-hour Holter ECG together with a Holter diary, as done in this case. A high index of suspicion in the young can lead to early diagnosis and management with a ‘permanent pacemaker’.

CONFLICT OF INTEREST
There is no conflict of interest to disclose

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REFERENCES


