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Tachycardia-Bradycardia Syndrome: Case Report and Literature Review

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Abstract **Case Report**

Sinus node dysfunction or sick sinus syndrome refers to a group of conditions characterized by abnormal cardiac pacing, leading to various cardiac bradyarrhythmia, tachyarrhythmia or bradycardia alternating with tachycardia. These arrhythmias can cause palpitations and reduced tissue perfusion, resulting in symptoms such as fatigue, lightheadedness, and sometimes syncope. Herein we report a case of sinus node dysfunction that initially manifested as supraventricular tachycardia but was later diagnosed as tachycardia-bradycardia syndrome.

Keywords: Sinus Node Dysfunction, Tachycardia-Bradycardia Syndrome, Syncope, Pacemaker.

INTRODUCTION

Sinus node dysfunction includes several conditions in the formation and/or conduction of impulses within the sinoatrial node. These abnormalities can manifest as sinus bradycardia, sinus pause, sinus arrest, sinoatrial nodal exit block, tachycardia-bradycardia syndrome or wandering atrial pacemaker [1].

Sinus node dysfunction is often associated with degenerative fibrosis of the sinus node tissue but may also result from medications, metabolic derangements, infiltrative diseases, ischemic diseases, etc. [2].

CASE REPORT

A 76-year-old male with past history of smoking presented to our facility with major palpitations. The history of palpitations began 9 months ago, accompanied by multiple episodes of presyncope.

Upon admission to the hospital, his blood pressure was 122/76 mmHg, heart rate of 140 bpm, normal respiratory rate, and oxygen saturation of 95%. His first ECG showed a regular supra-ventricular tachycardia (figure 1).

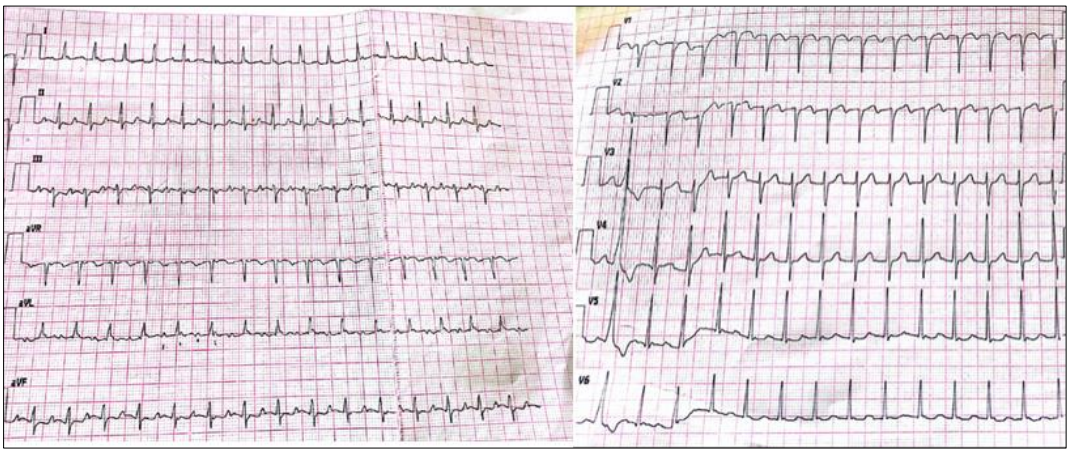

Figure 1: ECG showing regular supraventricular tachycardia

The patient was placed on continuous monitoring. Over the course of monitoring, the rhythm spontaneously converted to atrial tachycardia with variable conduction (Figure 2).

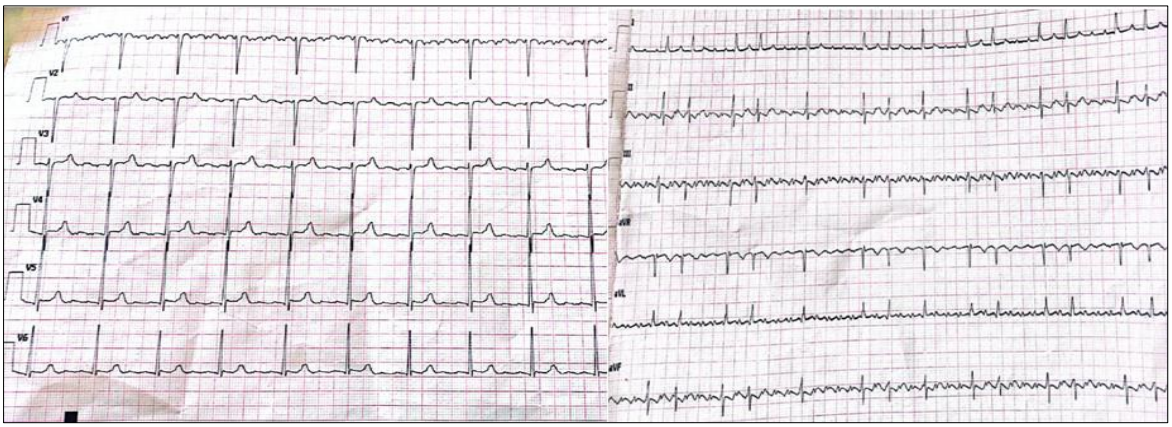

Figure 2: Atrial tachycardia with variable conduction

The next day the patient's rhythm changed to sinus bradycardia (figure 3)

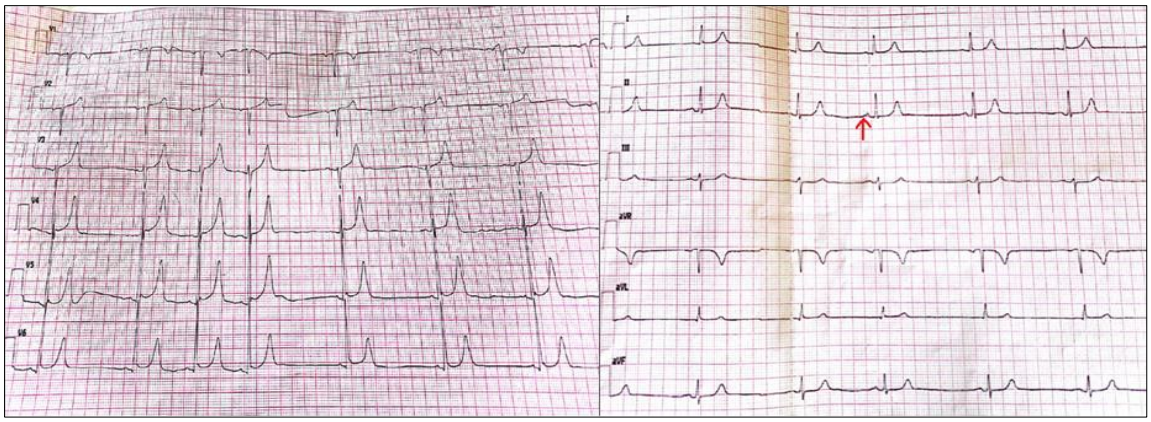

Figure 3: Sinus bradycardia; Interatrial block (red arrow)

The next day the patient changed again his rhythm to sinus tachycardia with multiple ectopic atrial contractions (Figure 4).

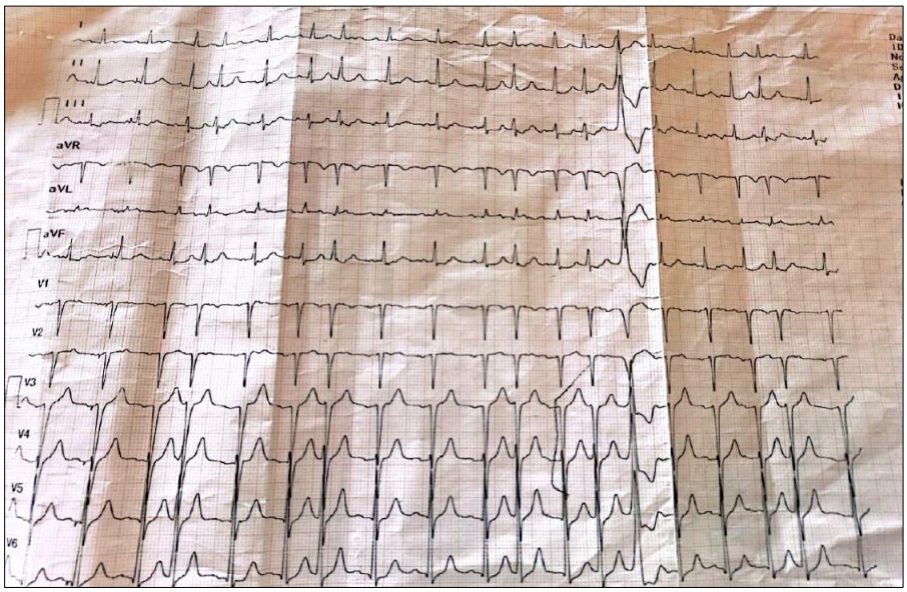

Figure 4: Sinus tachycardia and ectopic atrial contractions

No electrolyte imbalance had been detected during routine blood test.

Given the frequency of arrythmia and the risk of thromboembolism events, the patient was started on bisoprolol 2.5 mg and rivaroxaban 20 mg (CHA2DS2-VA: 2).

These frequently fluctuating arrhythmias observed on the resting ECG, along with the recurrence of presyncope episodes during monitoring, confirmed the diagnosis of sinus node dysfunction, more specifically identified as tachy-bradycardia syndrome.

The patient received a definitive treatment ➔ an implantable dual-chamber pacemaker.

→ EKG post implantation (Figure 5)

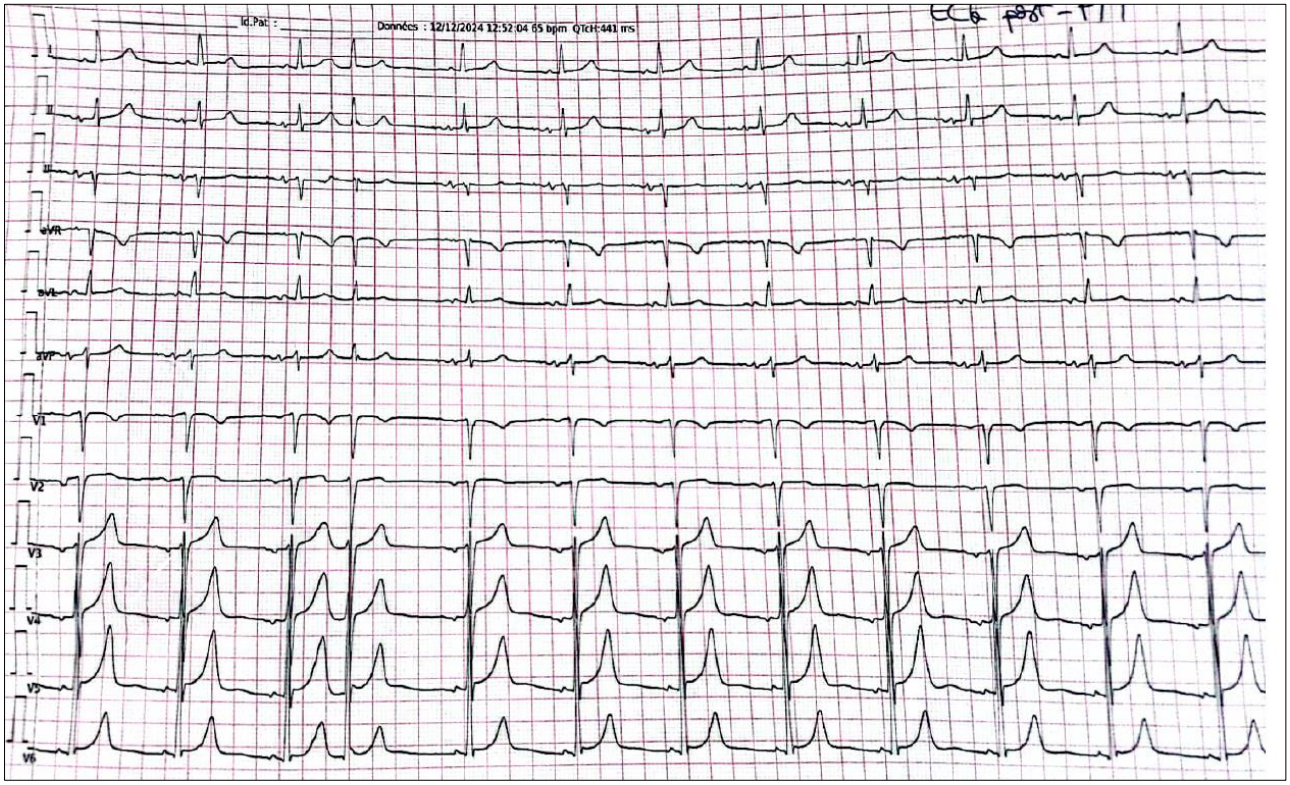

Figure 5: EKG post implantation of dual chamber pacemaker

DISCUSSION

Sinus node dysfunction affects approximately one in every 600 cardiac patients aged 65 or older and is one of the main causes of the pacemaker implantations [3]. The sinoatrial node is composed of two different functional types of cells: P cells and T cells. Dysfunction in one or both of these cell types can lead to sinus node dysfunction [3].

The etiologies are varied and can be intrinsic (e.g., degenerative fibrosis, infiltrative disease) or extrinsic (e.g., autonomic dysfunction, electrolyte imbalance, or induced by pharmacological agents such as anti-arrhythmics) [4].

Sinus dysfunction is a silent and progressive disease. The main symptoms are presyncope or syncope, which occur as a consequence of cerebral hypoperfusion [4].

SND can manifest in various arrhythmias such bradyarrhythmia (ectopic atrial bradycardia, pause >3 seconds after carotid massage, sino-atrial exit block, sinus bradycardia) or tachyarrhythmia (atrial flutter or fibrillation, ectopic atrial tachycardia) or alternating bradyarrhythmia and tachyarrhythmia [4].

Tachy-brady syndrome was first described in 1912 by Cohn and Lewis in a patient with Stockes-Adams attacks, and it's an association of paroxysmal atrial fibrillation, flutter, or atrial tachycardia followed by sinoatrial block, sinus arrest or sinus bradycardia [5].

Interatrial block translates the existence of a delayed conduction between the right and the left atrium due to atrial fibrosis and it serves as a potent clinical predictor of paroxysmal supraventricular tachyarrhythmias [6, 7].

Pharmacological agents such beta-blockers can help manage tachyarrhythmia but they suppress the sinus

node causing prolonged sinus pauses and bradycardia [8].

→ Implanted permanent dual-chambers pacemakers can prevent the occurrence of bradyarrhythmias, but they are ineffective at preventing the heart from developing tachyarrhythmias. Therefore, the proper treatment for tachycardia-bradycardia syndrome involves pacemaker implantation and the administration of medications such as beta-blockers [9].

Anticoagulation therapy should be considered in patient with paroxysmal atrial tachycardia to prevent to risk of systemic thromboembolism and stroke [4-10].

CONCLUSION

Tachycardia-bradycardia syndrome results from an impaired sinus node and manifests with alternating arrhythmias. It is a condition observed primarily in the elderly and is mainly caused by degenerative fibrosis.

The current treatment is pacemaker implantation and betablocker or non-dihydropyridine calcium-channel blockers to treat tachyarrhythmias.

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