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**Case Report**

# Innocent Sinus Node: Tachycardia-Bradycardia Syndrome Revealing a Compressing Esophageal Tumor

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## Abstract

Sinus node disease (SND) is a leading indication for permanent pacemaker implantation and is classically attributed to age-related degenerative changes. Nevertheless, functional conditions may result in similar clinical and electrocardiographic presentations, particularly in atypical cases. Extra-cardiac thoracic masses causing cardiac compression are rare and often underrecognized. We report the case of a patient presenting with a tachycardia-bradycardia syndrome initially suggestive of SND, in whom a comprehensive etiological evaluation revealed an esophageal tumor causing significant left atrial compression. This unusual presentation highlights the importance of considering extracardiac structural abnormalities in the differential diagnosis of apparent sinus node dysfunction.

**Keywords:** sinus node disease, tachycardia–bradycardia syndrome, extra-cardiac compression.



## INTRODUCTION

Sinus node disease (SND), also known as sick sinus syndrome, encompasses a spectrum of rhythm disorders caused by impaired impulse generation or conduction within the sinoatrial node, resulting in an inadequate heart rate response to physiological demands. It is most commonly related to age-associated degenerative fibrosis of the sinus node and represents one of the leading indications for permanent pacemaker implantation [1-3]. However, potentially reversible mechanisms may mimic intrinsic sinus node dysfunction, including medications, metabolic disturbances, autonomic imbalance, or structural abnormalities affecting the atria [4,5]. Rarely, extra-cardiac thoracic masses may cause cardiac compression by adjacent mediastinal structures, leading to atypical arrhythmic manifestations that can resemble sinus node dysfunction [6-8].

Recognizing these uncommon etiologies is essential to avoid misdiagnosis and potentially unnecessary pacemaker implantation.

## CASE PRESENTATION

### 1/Patient information:

A 65-year-old man with multiple cardiovascular risk factors, including advanced age, male sex, and a three-year history of hypertension well controlled with a fixed-dose combination of perindopril 10 mg and amlodipine 5 mg daily, and a significant smoking history estimated at 51 pack-years, with smoking cessation two years before admission, was referred to the emergency department after experiencing a syncopal episode. The event occurred suddenly at rest and prompted urgent medical evaluation because of its recurrent nature and the progressive deterioration of the patient's general condition.

### 2/Clinical findings:

Further questioning revealed that the patient had been experiencing recurrent episodes of syncope over the preceding five months. These episodes occurred spontaneously, without obvious triggering factors, and had initially been underestimated by the patient because of their transient character. Over the same period, he also reported progressive dysphagia predominantly affecting solid foods, associated with an unintentional weight loss estimated at approximately 15 kilograms during the month prior to admission. No chest pain, fever, or hemoptysis was reported.

On admission, the patient was hemodynamically stable. Physical examination showed normal blood pressure and adequate peripheral perfusion. Cardiac auscultation revealed regular heart sounds without murmurs, gallops, or pericardial rub. Pulmonary examination did not demonstrate crackles or

signs of pulmonary congestion, and there were no clinical findings suggestive of right- or left-sided heart failure. The remainder of the physical examination was unremarkable.

**3/Diagnostic assessment:**

Initial electrocardiography performed in the emergency department demonstrated sinus rhythm with marked bradycardia (heart rate 46 beats per minute), complete right bundle branch block, and preserved atrioventricular conduction **(Figure 1).**

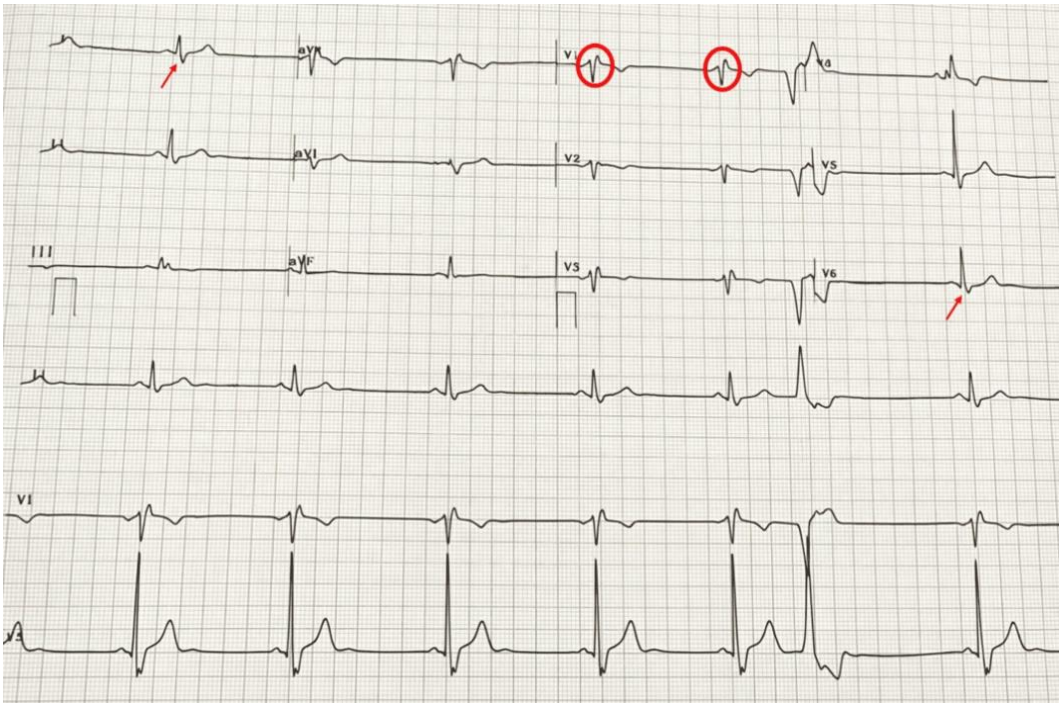


**Figure 1: Shows sinus rhythm with marked bradycardia (heart rate 46 beats per minute) and complete right bundle branch block (The circles in lead V1 indicate the rsR′ pattern, while the arrows in lead I and lead V6 indicate broad terminal S waves, findings consistent with complete right bundle branch block (QRS duration: 120 ms)**

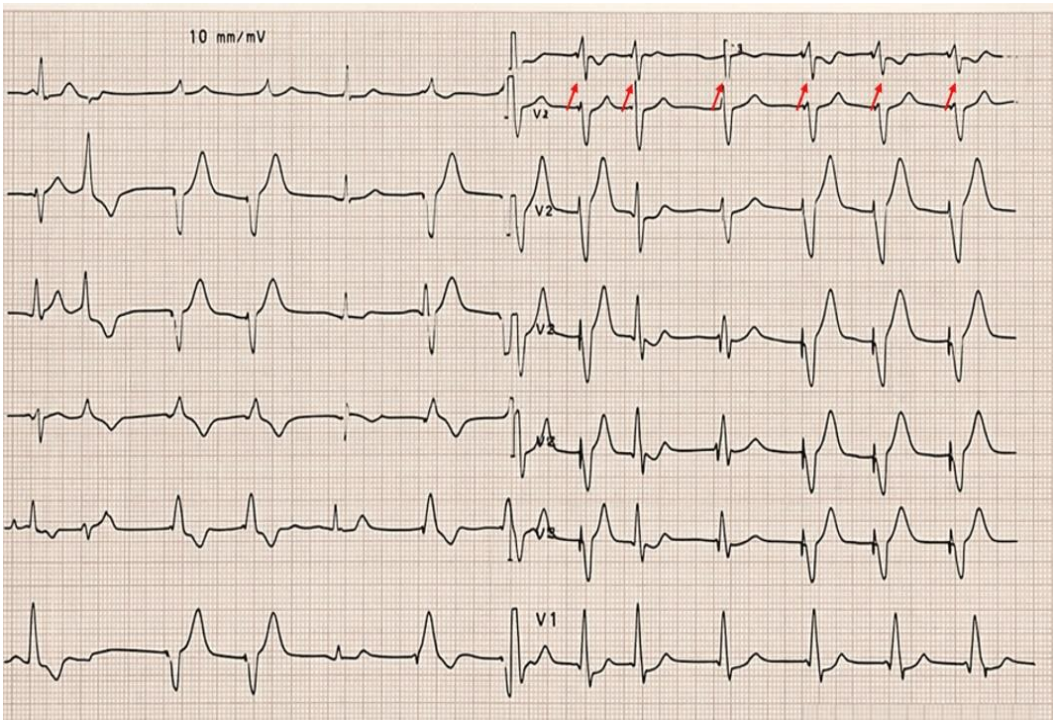


**Figure 2: Electrocardiogram documenting the first episode of paroxysmal atrial fibrillation (Arrows indicate the irregular ventricular rhythm and absence of organized atrial activity consistent with atrial fibrillation.)**

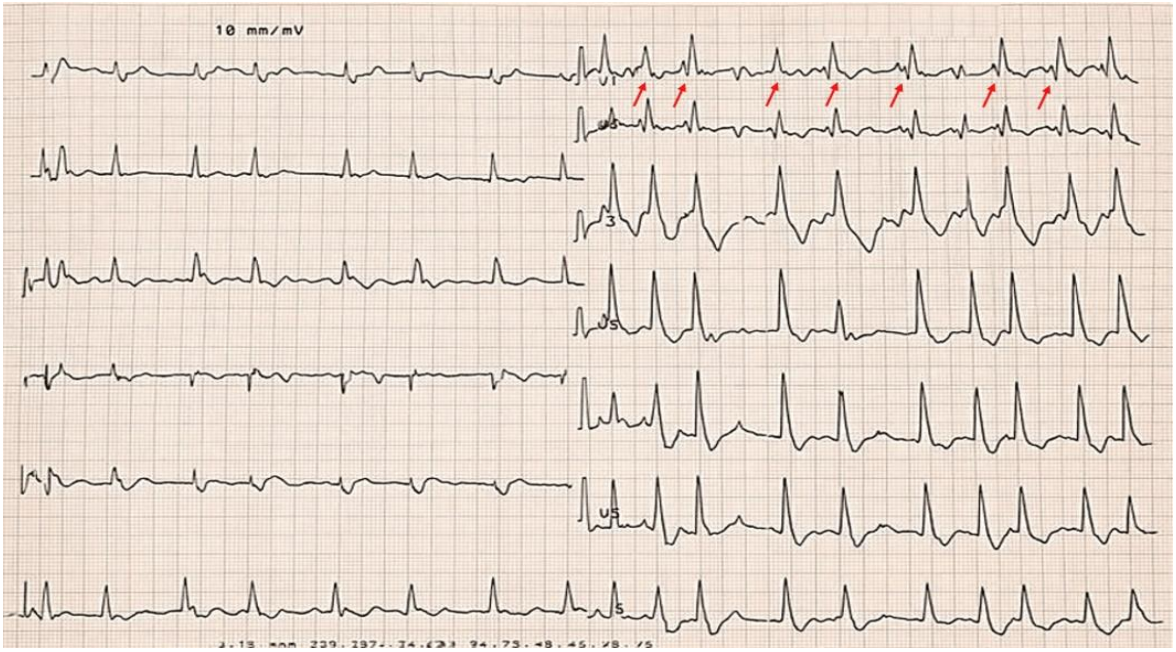


**Figure 3: Electrocardiogram documenting the second episode of paroxysmal atrial fibrillation (Arrows indicate the irregular ventricular rhythm and absence of organized atrial activity consistent with atrial fibrillation.)**

A subsequent electrocardiogram showed two documented episodes of paroxysmal atrial fibrillation **(Figures 2, 3)**.

Laboratory investigations, including routine blood tests and tumor markers, were within normal limits. Chest radiography revealed a suspicious opacity in the posterior mediastinum.

Twenty-four-hour Holter monitoring showed a predominantly sinus rhythm with episodes of atrial flutter with variable atrioventricular conduction and a significant sinus pause lasting up to 7.4 seconds, without evidence of an atrioventricular block **(Figure 4).**

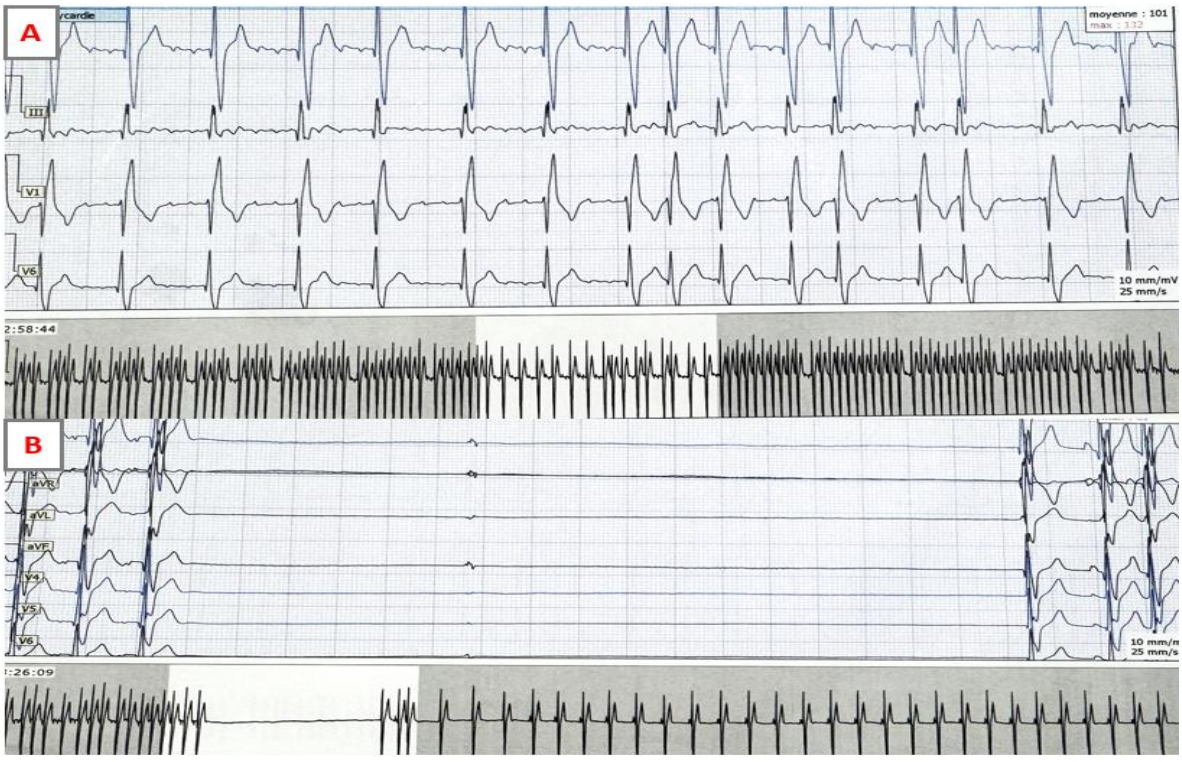


**Figure 4: Twenty-four-hour Holter ECG showing, in panel A, an episode of atrial flutter with variable atrioventricular conduction and, in panel B, a significant sinus pause lasting up to 7.4 seconds**

Transthoracic echocardiography demonstrated preserved cardiac chamber dimensions and systolic function but revealed extrinsic compression of the left atria by a heterogeneous extra-cardiac mass (**Figure 5**).

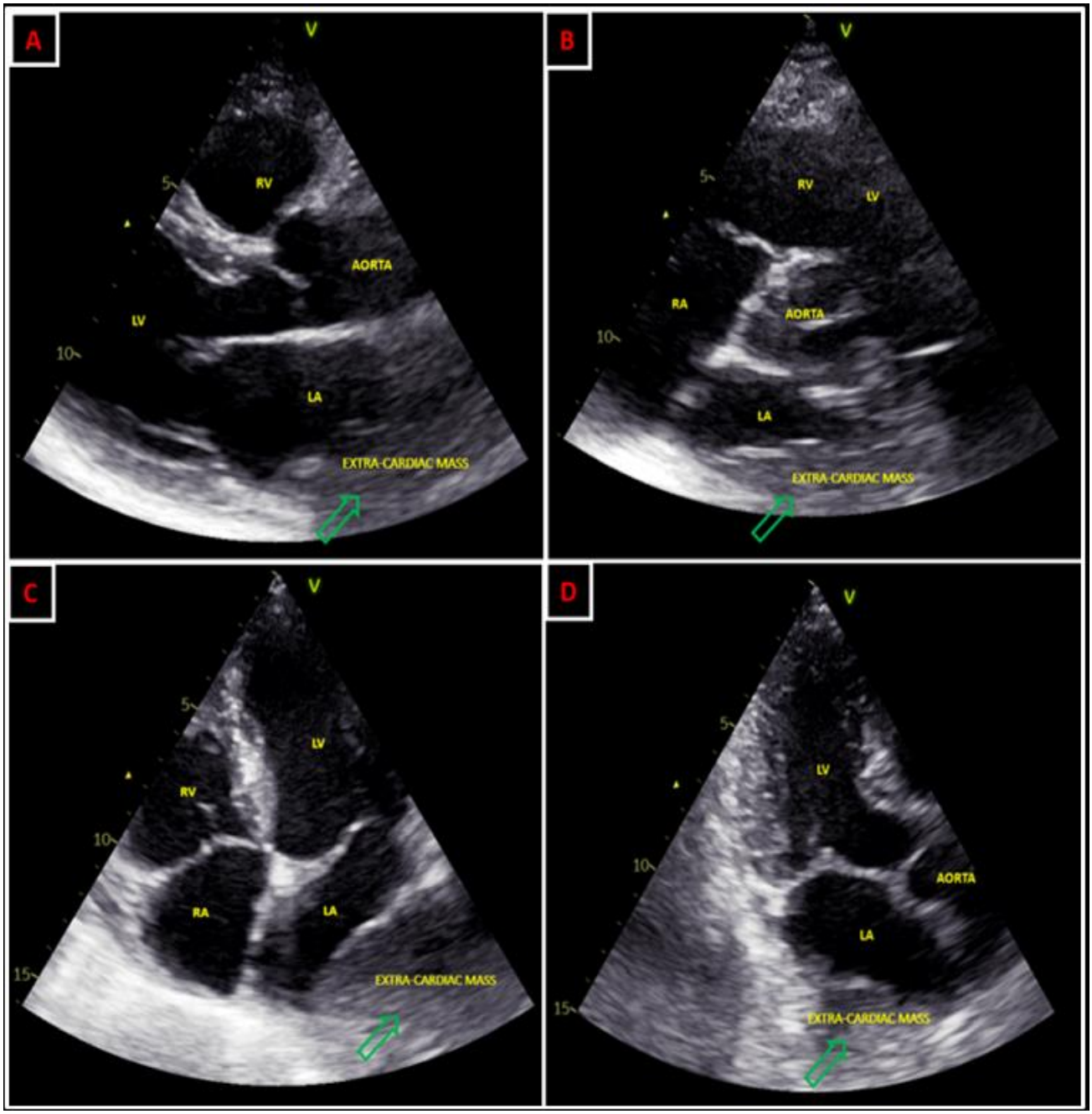


**Figure 5: Transthoracic echocardiography images demonstrating extrinsic compression of the left atrium by a heterogeneous extra-cardiac mass assessed in the parasternal long-axis (A), parasternal short-axis (B), apical four-chamber (C), and apical three-chamber views (D)**

Thoracic computed tomography subsequently revealed a large posterior mediastinal mass located in the subcarinal region, measuring approximately 77 × 50 × 95 mm, with spiculated margins, central necrosis, and mild contrast enhancement. The lesion encased the middle third of the esophagus, infiltrating the surrounding fat and causing significant luminal stenosis over a length of approximately 95 mm, with distal re-permeabilization. Anteriorly, it exerted a mass effect on and infiltrated the pulmonary arterial branches, which remained patent. Posteriorly, the mass was in contact with the anterior aspect of the thoracic vertebral bodies from T6 to T9, without evidence of bone destruction. Inferiorly and laterally, it contacted the descending aorta, with focal loss of the intervening fat plane **(Figure *6*).**

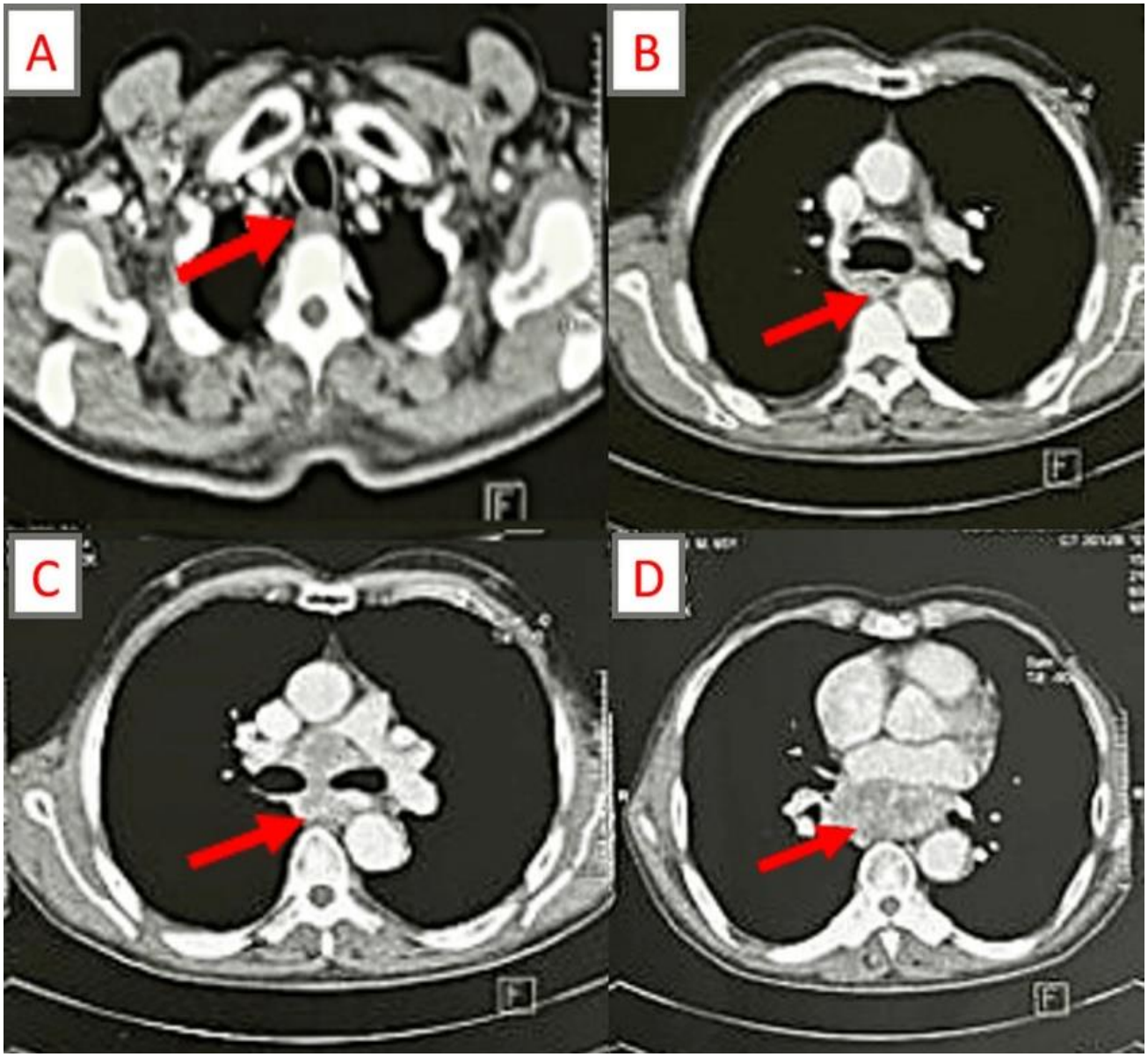


**FIGURE 6: Axial contrast-enhanced thoracic computed tomography : Demonstrating a large posterior subcarinal mediastinal mass (red arrows); (A) Axial section at the thoracic inlet level showing the posterior mediastinal component of the lesion; (B) Axial section at the upper thoracic level demonstrating esophageal invasion associated with tracheal compression; (C) Axial section at the mid-thoracic level showing invasion of both pulmonary arterial branches and extension to the esophagus; (D) Axial section demonstrating close contact between the lesion and the left atrium, with significant extrinsic compression of the atrial cavity**

Histopathological examination of a computed tomography (CT)-guided biopsy of the mediastinal mass revealed a poorly differentiated adenocarcinoma. Immunohistochemical analysis demonstrated positivity for cytokeratin 7 (CK7) and cytokeratin 19 (CK19), while thyroid transcription factor-1 (TTF-1), napsin A, cluster of differentiation 3 (CD3), CD20, caudal-type homeobox 2 (CDX2), synaptophysin, and chromogranin A were negative.

After multidisciplinary discussion, the findings were considered most consistent with a bronchopulmonary adenocarcinoma presenting as a posterior mediastinal mass with secondary cardiac compression.

For oncological staging, fluorine-18 fluorodeoxyglucose (18F-FDG) positron emission tomography/ computed tomography (PET/CT) revealed a large hypermetabolic mediastinal mass with central necrosis, along with a hypermetabolic lesion in the right lung, and no evidence of distant hypermetabolic involvement **(Figure 7).**

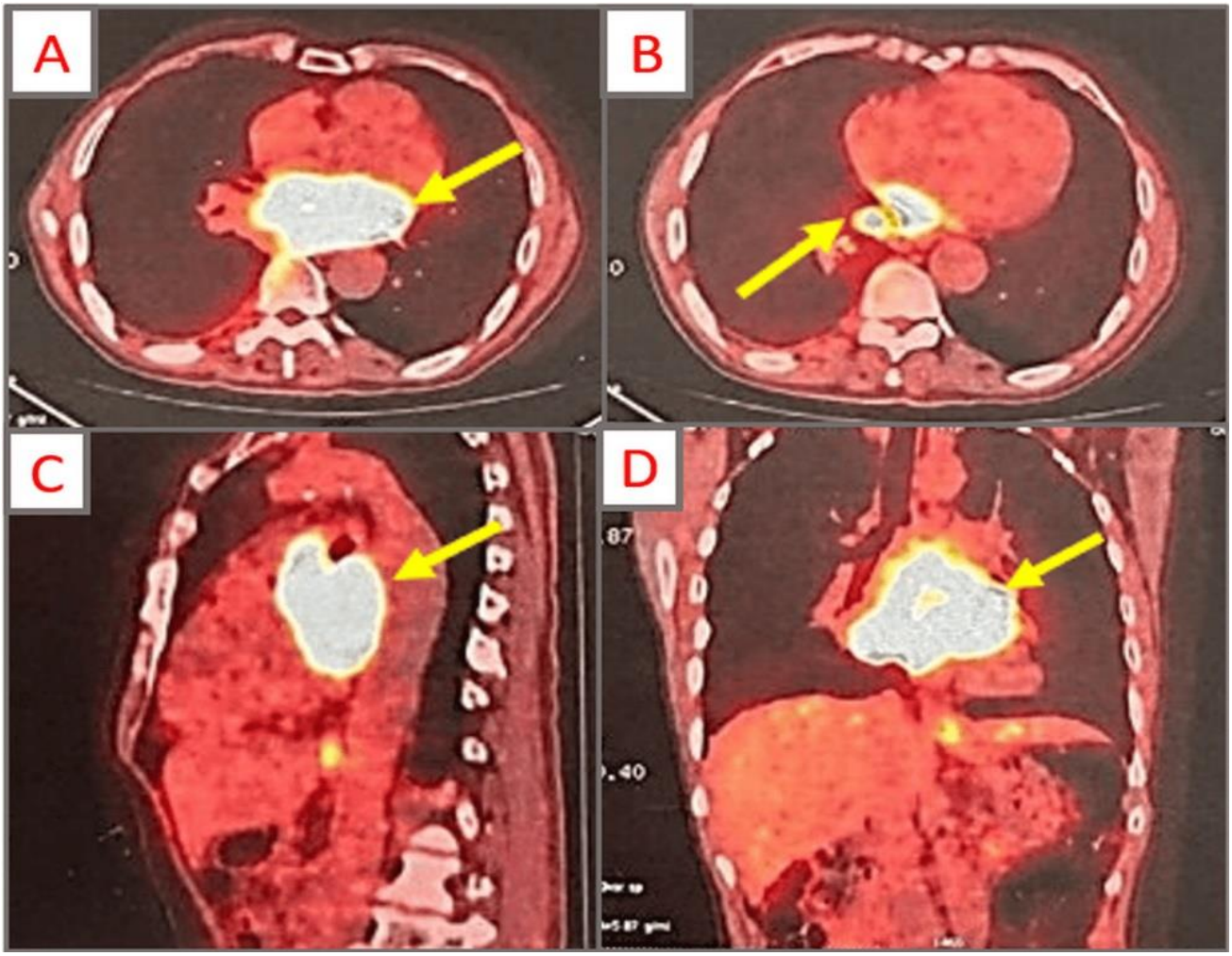


**Figure 7: Fluorine-18 Fluorodeoxyglucose Positron Emission Tomography-Computed Tomography (18F-FDG PET/CT) imaging:Demonstrates a large hypermetabolic posterior mediastinal mass with central necrosis (yellow arrows), without evidence of distant hypermetabolic involvement; (A,B) Axial fused PET/CT images showing intense peripheral FDG uptake surrounding the necrotic center of the mediastinal mass; (C) Sagittal fused PET/CT reconstruction illustrating the longitudinal extension of the hypermetabolic lesion; (D) Coronal fused PET/CT reconstruction demonstrating the relationship of the mediastinal mass with adjacent thoracic structures**

**4/ Diagnosis:**

Functional sinus node dysfunction secondary to extrinsic left atrial compression by a non-metastatic mid-esophageal adenocarcinoma

**5/Therapeutic intervention:**

In light of the extra-cardiac etiology of the rhythm disturbances, implantation of a permanent pacemaker was considered unwarranted at that stage. In accordance with the 2024 European Society of Cardiology (ESC) guidelines [9] for atrial fibrillation, oral anticoagulation therapy with rivaroxaban 20 mg once daily was initiated due to documented atrial fibrillation/flutter and a Congestive heart failure (or left ventricular dysfunction), hypertension (high blood pressure); age ≥ 75 years; diabetes mellitus; prior stroke, transient ischemic attack (TIA), or thromboembolism; vascular disease (such as prior heart attack, peripheral artery disease, or aortic plaque); Age 65-74 years ($CHA_2DS_2$-VA) score of two (one point for hypertension and one point for age between 65 and 74 years). Antiarrhythmic drugs were deliberately avoided because of their potential to exacerbate bradyarrhythmias in the context of sinus node dysfunction. Following multidisciplinary discussion, the patient was referred to the oncology department for further evaluation and management.

**6/Follow-up and outcomes:**

Cardiac rhythm monitoring was planned during follow-up to assess the evolution of sinus node dysfunction after management of the compressive tumor and to reassess the indication for permanent pacemaker implantation if symptoms persisted. Unfortunately, the patient died during the course of combined chemoradiotherapy, before completion of the planned oncological treatment. Consequently, long-term rhythm evolution and the potential reversibility of sinus node dysfunction could not be fully evaluated.

**7/Informed consent:**

Oral informed consent for publication of this case report and the accompanying images was obtained from the patient's next of kin in accordance with institutional ethical standards.

## DISCUSSION

SND encompasses a spectrum of rhythm disturbances related to sinoatrial node dysfunction, including sinus bradycardia, sinoatrial block, sinus pauses or arrest, and the characteristic alternation between bradyarrhythmias and atrial tachyarrhythmias known as the tachycardia-bradycardia syndrome [1-3]. Its prevalence in the general population is estimated at 0.03-0.05%, rising to 0.5-1% in individuals over 65 years of age, and it accounts for approximately 30-50% of permanent pacemaker implantations worldwide [2,10]. Although SND is classically attributed to age-related degenerative fibrosis of the sinus node and adjacent atrial myocardium, extrinsic and potentially reversible causes are increasingly recognized as mimickers of intrinsic sinus node dysfunction [3,5].

Extra-cardiac etiologies include mediastinal masses, pericardial disease, pulmonary tumors, bulky lymphadenopathy, and more rarely. esophageal tumors causing external atrial compression [6-8]. In the present case, the compressive lesion involved the left atrium, reflecting its close anatomical relationship with the esophagus. Left atrial compression may alter atrial compliance, induce mechanical stress, and promote atrial tachyarrhythmias through mechano-electrical feedback and conduction heterogeneity [7,11,12].

Left atrial compression may result in a tachycardia-bradycardia syndrome through an indirect and functional mechanism. Atrial stretch and pressure overload can facilitate atrial fibrillation or flutter, and recurrent atrial tachyarrhythmias may subsequently induce post-tachycardia suppression of sinus node activity, prolonged sinus pauses, and functional bradycardia mediated by transient autonomic imbalance rather than direct structural damage to the sinus node [3,13].

From a mechanistic standpoint, extrinsic left atrial compression may ultimately lead to atrial stretch, altered compliance, mechano-electrical feedback, and autonomic dysregulation. Although it does not directly affect the sinus node, left atrial compression may produce the same clinical phenotype through tachyarrhythmia-mediated functional suppression of sinus node automaticity. The final expression is therefore a tachycardia-bradycardia syndrome driven by functional rather than degenerative mechanisms [3,11,13,14].

Clinically, patients may present with intermittent and nonspecific symptoms such as palpitations, syncope or presyncope, fatigue, and dyspnea, often accompanied by manifestations related to the compressive lesion itself, including dysphagia in cases of mediastinal or esophageal tumors [6,8]. Electrocardiography is essential for documenting sinus bradycardia, pauses, and atrial tachyarrhythmias, but does not identify the underlying cause, emphasizing the need for systematic etiological investigation in patients without typical risk factors for degenerative SND [1,2].

In cases of tachycardia-bradycardia syndrome, current guidelines recommend oral anticoagulation only when atrial fibrillation or flutter is objectively documented, with treatment guided by thromboembolic risk assessment using validated scores such as $CHA_2DS_2$-VA. Antiarrhythmic drugs should be used cautiously or avoided due to their potential to worsen bradyarrhythmias in the absence of pacing support [1,3,15].

Importantly, international guidelines stress that reversible causes must be excluded before diagnosing intrinsic SND or proceeding with permanent pacemaker implantation [1,15]. Management should therefore be etiology-driven, prioritizing treatment of the underlying extra-cardiac pathology. Permanent pacing should be reserved for patients with persistent symptomatic bradycardia or pauses that do not resolve after correction of the causal condition, underscoring the functional and potentially reversible nature of sinus node dysfunction in this setting.

## CONCLUSIONS

SND should not be considered exclusively degenerative, as functional and potentially reversible mechanisms may also be involved. This case illustrates a rare presentation of tachycardia-bradycardia syndrome secondary to extrinsic left atrial compression by a mid-esophageal tumor. In patients with atypical clinical features or associated compressive symptoms, a thorough etiological investigation and multimodal imaging are essential to identify reversible extra-cardiac causes of arrhythmia. Recognition of these uncommon mechanisms has important therapeutic implications, as appropriate treatment of the underlying condition may improve rhythm disturbances and help avoid unnecessary permanent pacemaker implantation. This report further highlights the importance of a multidisciplinary approach in the diagnosis and management of secondary sinus node dysfunction.

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## REFERENCES

- Brignole M, Auricchio A, Baron-Esquivias G, *et al.,* 2013 ESC guidelines on cardiac pacing and cardiac resynchronization therapy: the task force on cardiac pacing and resynchronization therapy of the European Society of Cardiology (ESC). Developed

in collaboration with the European Heart Rhythm Association (EHRA). Eur Heart J. 2013, 34:2281-329. 10.1093/eurheartj/eht150
- Jensen PN, Gronroos NN, Chen LY, Folsom AR, deFilippi C, Heckbert SR, Alonso A: Incidence of and risk factors for sick sinus syndrome in the general population. J Am Coll Cardiol. 2014, 64:531-8. 10.1016/j.jacc.2014.03.056
- Monfredi O, Boyett MR: Sick sinus syndrome and atrial fibrillation in older persons - a view from the sinoatrial nodal myocyte. J Mol Cell Cardiol. 2015, 83:88-100. 10.1016/j.yjmcc.2015.02.003
- Mangrum JM, DiMarco JP: The evaluation and management of bradycardia . N Engl J Med. 2000, 342:703-9. 10.1056/NEJM200003093421006
- Alboni P, Holz A, Brignole M: Vagally mediated atrioventricular block: pathophysiology and diagnosis . Heart. 2013, 99:904-8. 10.1136/heartjnl-2012-303220
- Tang KS, Zargarian E, Patel PM: Case report: a crushing sensation-a rare case of mediastinal germ cell tumor causing cardiac tamponade. Front Cardiovasc Med. 2024, 11:1539900. 10.3389/fcvm.2024.1539900
- Scagliola R, Rosa GM, Porto I: Left atrial compression by extracardiac structures: a comprehensive multimodality approach. Anatol J Cardiol. 2021, 25:524. 10.5152/AnatolJCardiol.2021.162
- Breall JA, Goldberger AL, Warren SE, Diver DJ, Sellke FW: Posterior mediastinal masses: rare causes of cardiac compression. Am Heart J. 1992, 124:523-6. 10.1016/0002-8703(92)90626-7
- Van Gelder IC, Rienstra M, Bunting KV, *et al.,* 2024 ESC guidelines for the management of atrial fibrillation developed in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS). Eur Heart
- J. 2024, 45:3314-414. 10.1093/eurheartj/ehae176
- Mond HG, Proclemer A: The 11th world survey of cardiac pacing and implantable cardioverter-defibrillators: calendar year 2009-a World Society of Arrhythmia's project. Pacing Clin Electrophysiol. 2011, 34:1013-27. 10.1111/j.1540-8159.2011.03150.x
- Ho SY, Sánchez-Quintana D: The importance of atrial structure and fibers . Clin Anat. 2009, 22:52-63. 10.1002/ca.20634
- Argyris TS: The growth-promoting effects of wounds on hair follicles already stimulated by plucking . Anat Rec. 1962, 143:183-8. 10.1002/ar.1091430303
- Shen MJ, Zipes DP: Role of the autonomic nervous system in modulating cardiac arrhythmias . Circ Res. 2014, 114:1004-21. 10.1161/CIRCRESAHA.113.302549
- Wijffels MC, Kirchhof CJ, Dorland R, Allessie MA: Atrial fibrillation begets atrial fibrillation: a study in awake chronically instrumented goats. Circulation. 1995, 92:1954-68. 10.1161/01.cir.92.7.1954
- Kusumoto FM, Schoenfeld MH, Barrett C, *et al.,* 2018 ACC/AHA/HRS guideline on the evaluation and management of patients with bradycardia and cardiac conduction delay: a report of the American College of Cardiology/American Heart Association task force on clinical practice guidelines and the Heart Rhythm Society. Circulation. 2019, 140:e382-482. 10.1161/CIR.0000000000000628

## ABBREVIATIONS (ALPHABETICAL ORDER)

- **18F-FDG PET/CT**: Fluorine-18 fluorodeoxyglucose positron emission tomography/computed tomography
- **CD3**: Cluster of differentiation 3
- **CD20**: Cluster of differentiation 20
- **CDX2**: Caudal-type homeobox 2
- **$CHA_2DS_2$-VA**: Congestive heart failure, Hypertension, Age ≥75 years, Diabetes mellitus, Stroke/TIA/systemic embolism, Vascular disease, Age 65–74 years
- **CK7**: Cytokeratin 7
- **CK19**: Cytokeratin 19
- **CT**: Computed tomography
- **ECG:** Electrocardiogram
- **SND**: Sinus node disease
- **TTF-1**: Thyroid transcription factor-1