

# Sick Sinus Syndrome and Cerebral Venous Thrombosis: A Connection or Coincidence? A Case Report and Literature Review

Alawi A. Al-Attas\*, Njood A. Alhrkan, Muteb A. Alwatid, Saifullah M. Zaman, Mohammed F. Salawaty and Nouf A. Mansour

Department of Neurology, King Saud Medical City, Riyadh, Saudi Arabia

Received: 18 July 2023

Accepted: 17 August 2023

\*Corresponding author: alawiattas@yahoo.com

DOI 10.5001/omj.2025.21

## Abstract

It has been hypothesized that increased intracranial pressure (ICP) may cause sick sinus syndrome (SSS) because of the effect of high ICP on vagal discharge to the heart, which results in disruption to the sinus cycle, even though age-related degeneration of the SA node is the most common intrinsic factor leading to sinus node dysfunction. In this case report, a 47-year-old man who is medically free arrived at the emergency room at King Saud Medical City complaining of headache, confusion, and chest pain that had been bothering him for 4 days. He also reported feeling dizzy and lightheaded. Extensive cerebral venous thrombosis (CVT) and SSS were discovered in the patient. We looked at the origin of the SSS and how it related to CVT. Our conclusion is that SSS caused by elevated ICP secondary to CVT.

**Keywords:** Sick Sinus Syndrome; Intracranial Pressure; Cerebral Venous Sinus Thrombosis; Sinoatrial Node; King Saud Medical City.

## Introduction

A group of clinical diseases known as sick sinus syndrome (SSS) are caused by an irregularity in the sinoatrial nodal system (SA); in fact, this sickness typically affects elderly people due to the degeneration of the SA node. Though generally idiopathic, it can be both hereditary and acquired.<sup>1</sup>

Clinically, it appears as arrhythmias, which may include sinus bradycardia, sinus pauses or arrests, sinoatrial exit block, or alternating bradyarrhythmias and tachyarrhythmias.<sup>2</sup> Indeed, it has been hypothesized that increased intracranial pressure (ICP) may cause SSS because of the effect of high ICP on the vagal discharge to the heart, which results in disruption to the sinus cycle, even though age-related degeneration of the SA node is the most common intrinsic factor leading to sinus node dysfunction.<sup>2,3</sup>

Herein, we describe a case of SSS in a relatively young man with a rare underlying pathology and the presentation that resulted from it.

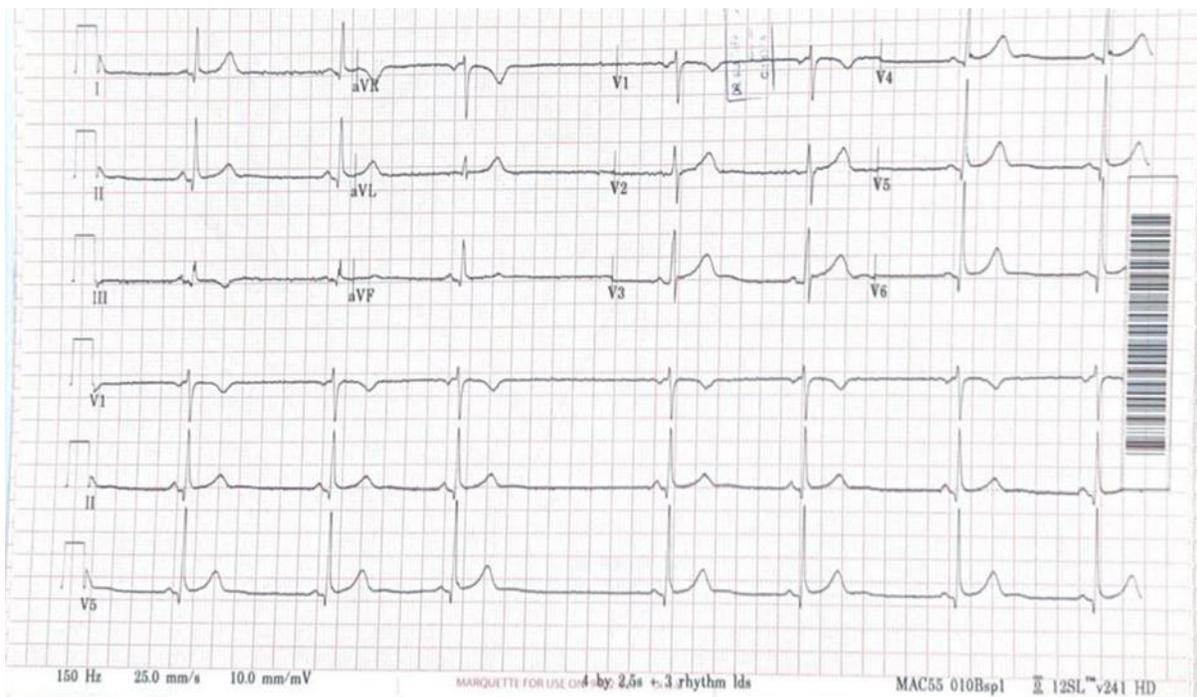
## Case Report

Herein, we report a 47-year-old man, who is medically free, who presented to the emergency department with a 4-day history of episodes of unprovoked syncope with nausea, somnolence, and worsening headache and confusion associated with chest discomfort and dizziness. The headache was generalized, persistent, progressive, severe, and increased with recumbent position. It was not relieved by analgesics and did not vary in frequency or intensity

throughout the day. The chest pain was mild, localized to the left side of the chest and did not radiate elsewhere. In addition, neither aggravating nor alleviating factors were present. The condition did not worsen with deep respiration, and there were no associated symptoms such as cough, hemoptysis, dyspnea, or orthopnea. He denied similar previous symptoms, and he denied fever, chills, vomiting, photophobia, phonophobia, tinnitus, transient visual blurring on standing, or sensorimotor symptoms. His family history was noncontributory. He had no previous medical history; he was not on any medications, and his socioeconomic situation was good.

Clinical examination in the emergency department revealed blood pressure was 128/75 mmHg, heart rate was 44 beats per minute, respiratory rate was 19 breaths per minute, he was afebrile, and he had an oxygen saturation of 95% in room air. The patient was lying down in bed; he was awake, oriented, and following commands. Fundoscopic examination revealed bilateral papilledema, mild left facial weakness, and left upper limb drift. The rest of the neurological exam was unremarkable.

An EKG showed sinus bradycardia with multiple intermittent sinus pauses of duration 4-5 seconds [Figure 1].



**Figure 1:** The EKG showed sinus bradycardia with a HR of about 40 beats per minute, sinus arrhythmia, and sinoatrial exit block. The sinoatrial exit block is due to the failed propagation of pacemaker impulses beyond the SA node. The combination of these EKG abnormalities (sinus bradycardia, sinus arrhythmia, and associated dropped P waves) consistent with sinus node dysfunction and sick sinus syndrome.

All blood tests, including the basic tests and those for vasculitis and thrombophilia, and spiral CT of the chest came back normal. A computed tomography (CT) scan of the brain with and without contrast and CT cerebral venography (CTV) revealed a picture consistent with extensive cerebral venous thrombosis (CVT) [Figure 2].

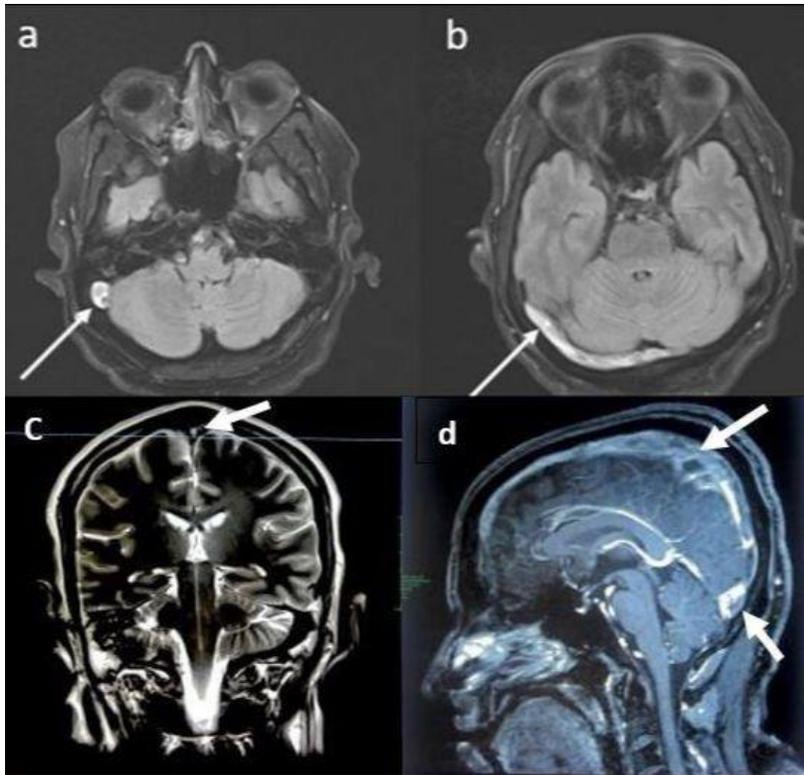


**Figure 2:** (a) Non-enhanced axial CT of the brain revealed hyperdense transverse sinuses on the right side as well as edema in the right occipitoparietal regions. (b) An enhanced axial CT of the brain showed an empty delta sign due to thrombosis in the superior sagittal sinus. The sign consists of a triangular area of enhancement with a low attenuating center, which is the thrombosed sinus. (c) An axial CT venogram shows a lack of flow in the right transverse and the right sigmoid sinus, a filling defect due to a thrombosed sinus.

Magnetic resonance imaging (MRI) of the brain demonstrates increased signal intensity in the right sigmoid sinus and the right transverse sinus [Figure 2].

The echocardiogram was normal. Furthermore, Holter monitoring demonstrated sinus bradycardia. Therefore, therapeutic anticoagulation was initiated, and the patient recovered clinically despite continuing to experience mild headaches and dizziness. Based on the Holter report, the cardiologist diagnosed persistent symptomatic bradycardia with sinus bradycardia ranging from 30 to 45 beats per minute with pauses lasting 4 to 5 seconds. After diagnosis of SSS, the patient was transferred to the cardiac care unit (CCU) for pacemaker implantation. After 24 hours of observation, the post-procedure period was uneventful, and telemetry revealed sinus rhythm without a pacing beat. The heart rate recorded was within the normal range. A chest X-ray performed after pacemaker implantation revealed a cardiac pacemaker implanted in the right atrium and right ventricle [Figure 3C]

Finally, he was transferred to the acute stroke unit, where he remained for five days under the supervision of the neurology team following pacemaker implantation. We resumed anticoagulation and monitored it to ensure that he was neurologically and cardiologically stable enough to be discharged. The absence of migraines and the disappearance of other symptoms indicated his improvement. The patient was then instructed to return to both the pacemaker and neurology clinics for further evaluation.



**Figure 3:** (a) An axial T2-weighted MR image demonstrates an area of abnormality with increased signal intensity in the right sigmoid sinus consistent with CVT. (b) Fluid attenuated inversion recovery (FLAIR) weighted MR image demonstrates. (c) A coronal T2-weighted MR image shows hypersignal intensity in the superior sagittal sinus. (d) A sagittal contrast-enhanced T1-weighted image shows filling defects along the superior sagittal sinus and involving the other cerebral sinuses, indicating extensive CVT.

## Discussion

According to estimates, cerebral venous thrombosis (CVT) causes less than 1% of all strokes. CVT tends to affect youthful patients with female predominance and has a wide range of clinical manifestations. These and other characteristics make CVT a difficult disease to diagnose without knowledge of its evolving epidemiology, clinical characteristics, associated conditions, and the neuroimaging findings typically required to confirm the diagnosis.<sup>4</sup>

On the other hand, SSS, also known as sinus node dysfunction (SND), is a disorder of the SA node characterized by a constellation of aberrant rhythms resulting from impaired pacemaker function and impulse transmission. These include atrial bradyarrhythmias, atrial tachyarrhythmias, and occasionally bradycardia alternating with tachycardia, which is commonly known as "tachy-brady syndrome." These arrhythmias may cause palpitations and tissue hypoperfusion, resulting in fatigue, dizziness, presyncope, and syncope.<sup>5</sup>

Due to age-related degenerative fibrosis of the SA node, SSS has been linked to aging (mean age of 68 years).<sup>6</sup> Given the patient's relatively young age, this is a less likely cause of SSS in him.<sup>7</sup>

It is essential to note that while there may be a link between CVT and SSS, this association is uncommon. Massive ICP elevation is associated with the sympathoadrenal mechanism known as the Cushing response (CR), which causes the hypertension, bradycardia, and respiratory irregularities.<sup>8</sup>

There are a variety of neurology and neurosurgery diseases such as stroke, subarachnoid hemorrhage, CVT, autoimmune diseases, tumors, infectious diseases, and even some medications and vaccines that have been implicated

to elevate ICP and subsequently lead to SSS. For instance, an elevated ICP because of extensive CVT was the root cause of our patient's SSS. Makhaly et al. reported a case of glioblastoma in the right frontal nodule, corpus callosum, and right pontine area. They thought that SSS was most likely caused by an imbalance of autonomic tone at the SA node. They said that this could be further broken down into either hyperactivity of parasympathetic nerve outflow or hypoactivity of sympathetic nerve outflow.<sup>1</sup> Arguija also reported a case of cerebral venous sinus thrombosis after an epidural injection in the cervical spine. The patient had a slow heart rate, which was thought to be caused by an increase in ICP due to CVT. The bradycardia resolved after she received acetazolamide.<sup>9</sup> Furthermore, bradycardia was reported in patients with isolated intracranial hypertension syndrome (IIHS) with subsequent CVT.<sup>10</sup>

Additionally, N-methyl-D-aspartate receptor (NMDAR) encephalitis caused a case of profound sinus node dysfunction reported by Wong et al.<sup>11</sup> Similar case of a 33-year-old Japanese woman with no significant medical history who presented with a picture of encephalitis and movement disorder and was found to be positive for NMDAR limbic encephalitis. The telemetry monitoring strip showed a long period of sinus arrest of 9.28 seconds.<sup>12</sup> Interestingly, ischemic stroke of the thalamus was also the culprit where Asavaaree et al. reported a rare case of the artery of percheron causing thalamic infarction that resulted in severe bradycardia.<sup>13</sup>

It has also been documented that increased ICP brought on by a subarachnoid hemorrhage can impact the autonomic nervous system. According to Kawahara et al., the aberrant sinus cycle caused by the high ICP, which enhanced vagal discharge to the heart.<sup>14</sup> Following intracranial hypertension, the classic definition of CR is the occurrence of hypertension, bradycardia, and apnea. This was evident in the case report of spontaneous subarachnoid hemorrhage and CR, who self-aborted and had a good outcome.<sup>15</sup>

Surprisingly, it was determined that sinus arrest during rapid eye movement sleep occurred due to a similar physiological process.<sup>16</sup> Moreover, Blitz et al. reported intracranial complications of hypercoagulability and superinfection in the setting of COVID-19 in three cases in which one of them developed bradycardia.<sup>17</sup> Khan et al. reported a case of post-COVID-19 vaccine patients who developed CVS and ultimately bradycardia.<sup>18</sup>

Autonomic and cardiovascular dysfunction are established effects of brainstem lesions. According to several case studies, lesions that are located inside or close to the medulla oblongata are the main cause of compression of the vasomotor region.<sup>1</sup> Brainstem compression is the same mechanism by which both increased ICP and brainstem injuries result in autonomic dysfunction.<sup>19,20</sup>

Dadlani et al. highlighted the possibilities of varied causes of coexisting bradycardia in a neurosurgical patient and made the neurosurgical community aware of them. They reported a case of posterior fossa surgery in which persistent bradycardia developed in the postoperative period. They recommended that a very high degree of suspicion is essential for the diagnosis of SSS since the patient would benefit from early medical management and possible external cardiac pacing in selected cases.<sup>21</sup>

Hamaguchi et al. discovered that the patient who had neuromyelitis optica spectrum disorder (NMOSD) with dorsal medulla and cervical cord lesions initially displayed area postrema syndrome (refractory nausea and vomiting) and potentially deadly bradycardia and later required pacemaker insertion due to a diagnosis of SSS.<sup>20</sup> In addition, SSS happened shortly after taking the anesthetics fentanyl and vecuronium, which were known to trigger SSS.<sup>22</sup> The patient we had, however, was special because SSS could not have been caused by any other confounding factors.

## Conclusions

SSS is a rare and serious cardiac rhythm disorder that can be caused by increased ICP. The link between SSS and CVT needs to be investigated. Early recognition and prompt treatment with pacemaker implantation are warranted. Despite these potential connections, SSS and CVT are distinct medical conditions that affect different parts of the body and have different underlying causes. More research is needed to fully understand any potential links between these conditions and identify any shared risk factors or treatment strategies.

## Disclosure

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors. Written consent was obtained from the patient.

## Reference

1. Makhaly S, Fallah P, Giannetti N. Elevated Intracranial Pressure as a Cause of Sick Sinus Syndrome. *Case Reports Cardiol* 2018; 2018: 1–3.
2. Semelka M, Gera J, Usman S. Sick sinus syndrome: A review. *Am Fam Physician* 2013; 87: 691–696.
3. Sick Sinus Syndrome - PubMed, <https://pubmed.ncbi.nlm.nih.gov/29261930/> (accessed August 8, 2023).
4. Liberman AL. Diagnosis and Treatment of Cerebral Venous Thrombosis. *Continuum (Minneapolis)* 2023; 29: 519–539.
5. De Ponti R, Marazzato J, Bagliani G, et al. Sick Sinus Syndrome. *Card Electrophysiol Clin* 2018; 10: 183–195.
6. Dobrzynski H, Boyett MR, Anderson RH. New Insights Into Pacemaker Activity. *Circulation* 2007; 115: 1921–1932.
7. Devinsky O, Pacia S, Tatabhota G. Bradycardia and asystole induced by partial seizures: a case report and literature review. *Neurology* 1997; 48: 1712–1714.
8. Schmidt EA, Despas F, Traon AP Le, et al. Intracranial pressure is a determinant of sympathetic activity. *Front Physiol* 2018; 9: 308284.
9. Arguija A, Arguija AF. Cerebral Venous Sinus Thrombosis After Cervical Spine Epidural Injection. *J Med Cases* 2012; 4: 170–172.
10. Sikka G, Sharma D, Allen AV, et al. Critical Care | Rare Case of Isolated Intracranial Hypertension Syndrome (IHS) With Subsequent Cerebral Venous Sinus Thrombosis (CVST) Abstract SESSION TITLE: Critical Care Student/Resident Case Report Posters II SESSION TYPE: Student/Resident Case Report Poster PRESENTED. *No 4 Meeting Abstracts Chest*; 148. Epub ahead of print 2015. DOI: 10.1378/chest.2277458.
11. Wong SW, Pang RY. Bradycardia in a Woman Presenting with Altered Behaviour. DOI: 10.6515/ACS.202301\_39(1).20220820A.
12. Chia PL, Tan K, Foo D. Profound sinus node dysfunction in anti-N-methyl-D-aspartate receptor limbic encephalitis. *Pacing Clin Electrophysiol*; 36. Epub ahead of print March 2013. DOI: 10.1111/J.1540-8159.2011.03154.X.
13. Asavaaree C, Doyle C, Smithason S. Artery of Percheron infarction results in severe bradycardia: A case report. *Surg Neurol Int*; 9. Epub ahead of print 2018. DOI: 10.4103/SNI.SNI\_254\_18.
14. Kawahara E, Ikeda S, Miyahara Y, et al. Role of autonomic nervous dysfunction in electrocardiographic abnormalities and cardiac injury in patients with acute subarachnoid hemorrhage. *Circ J* 2003; 67: 753–756.
15. Wan WH, Ang BT, Wang E. The Cushing Response: A case for a review of its role as a physiological reflex. *J Clin Neurosci* 2008; 15: 223–228.
16. Coccagna G, Capucci A, Pierpaoli S. A case of sinus arrest and vagal overactivity during REM sleep. *Clin Auton Res* 1999; 9: 135–138.
17. Blitz SE, McMahon JT, Chalif JI, et al. Intracranial complications of hypercoagulability and superinfection in the setting of COVID-19: illustrative cases. *J neurosurgery Case lessons*; 3. Epub ahead of print May 1, 2022. DOI: 10.3171/CASE22127.
18. Khan S, Khalil M, Waqar Z. Post Covid-19 Vaccine (Sinovac) Cerebral Venous Sinus Thrombosis. *Pakistan J Neurol Sci*; 17.
19. Nadkarni T, Kansal R, Goel A. Right cerebellar peduncle neurocysticercosis presenting with bradycardia. *Acta Neurochir (Wien)* 2010; 152: 731–732.
20. Hamaguchi M, Fujita H, Suzuki T, et al. Sick sinus syndrome as the initial manifestation of neuromyelitis optica spectrum disorder: a case report. *BMC Neurol* 2022; 22: 1–5.
21. Dadlani R, Challam K, Garg A, et al. Can bradycardia pose as a “red herring” in neurosurgery? Surgical stress exposes an asymptomatic sick sinus syndrome: Diagnostic and management dilemmas. *Indian J Crit Care Med* 2010; 14: 212–216.
22. Ishida R, Shido A, Kishimoto T, et al. Prolonged cardiac arrest unveiled silent sick sinus syndrome during general and epidural anesthesia. *J Anesth* 2007; 21: 62–65.