EURASIAN JOURNAL OF EMERGENCY MEDICINE
Case Report
DOI: 10.4274/eajem.galenos.2026.02328
Eurasian J Emerg Med. 2026;25: 294-7

Hypersomnolence as the Presenting Feature of Thalamic Stroke

Navami Sanjeev Rao, Viswanathan Pandurangan, Bhargavi Madhaviah Vasudev, Rajkumar Mani

Sri Ramachandra Hospital, Clinic of General Medicine, Chennai, India

Abstract

Posterior circulation stroke affecting thalamo-cortical arousal networks may present with increased sleepiness, with a lack of motor symptoms. Here, we present the case of a 41-year-old man with acute-onset hypersomnolence, without focal neurological deficits. The initial computed tomography (CT) brain was normal. Metabolic and infectious workup were negative. Magnetic resonance imaging (MRI) of brain was performed and revealed bilateral paramedian thalamic infarcts with hemorrhage and a left occipital infarct. MRI angiography demonstrated reduced flow in the posterior cerebral artery segments. This case highlights the prominent clinical feature of profound hypersomnolence in thalamic stroke and emphasizes how such patients are easily misdiagnosed when early CT imaging is unrevealing. Recognition of this pattern is critical to avoid delays in the diagnosis of thalamic infarction.

Keywords: Thalamus, hypersomnolence, posterior circulation, arousal network, stroke

Introduction

A drowsy patient with no focal deficits and a normal computed tomography (CT) brain is almost always worked up for metabolic encephalopathy, toxic or infectious causes. When focal neurological signs are absent, stroke is not often considered a differential diagnosis. In the upper pons and midbrain is located the ascending reticular activating system, which functions as a critical regulator of cortical arousal and consciousness (1). Wakefulness is maintained by interconnected brainstem, hypothalamic, and basal forebrain nuclei through pathways that connect to the cortex. Sleep is promoted by inhibitory GABAergic neurons in the ventrolateral preoptic area of the hypothalamus. These neurons suppress wake-promoting circuits through reciprocal inhibition. The thalamus is not merely a sensory relay. It is the gateway through which the brainstem maintains cortical wakefulness. Bilateral injury to this relay does not cause coma. It causes a patient who has increased sleepiness and difficulty staying awake (2). We describe a patient whose only manifestation of thalamic stroke was increased sleepiness lasting for around 20 hours.

Case Report

A 41-year-old man with no known comorbidities was brought to the emergency room with complaints of difficulty staying awake. He was functioning normally the previous day, had gone to sleep at 4 pm, and was reportedly normal before sleeping. At 11:00 am the next day, attenders tried to wake him up. He opened his eyes to calls and drifted back to sleep. There was no history of fever, seizures, vomiting, headache, trauma, or limb weakness. Attenders denied any history suggesting sedative overdose. They had gone to another hospital initially, CT brain done, showed a normal study. Capillary blood glucose recorded in the emergency room was 123 mg/dL.

On examination, he was drowsy, arousable to calls, able to obey simple commands like opening and closing of eyes, finger movements, limb movements briefly, and then goes back to sleep quickly. Pupils were bilaterally equal and reactive, and there was no restriction of eye movements. He moved all four limbs spontaneously. Deep tendon reflexes were normal bilaterally, and plantar responses were mute bilaterally. As the CT brain was normal, and the patient had no focal neurological deficits, a provisional diagnosis of metabolic encephalopathy vs. septic

Corresponding Author: Navami Sanjeev Rao MD, Sri Ramachandra Hospital, Clinic of General Medicine, Chennai, India
E-mail: navamisrao1097@gmail.com **ORCID ID:** orcid.org/0009-0008-4836-8409
Cite this article as: Rao NS, Pandurangan V, Vasudev BM, Mani R. Hypersomnolence as the presenting feature of thalamic stroke. Eurasian J Emerg Med. 2026;25: 294-7.

Received: 04.03.2026
Accepted: 09.04.2026
Published: 22.04.2026

encephalopathy was kept, and the patient was worked up for the same. Comprehensive laboratory evaluation was done. Hemoglobin was 12.4 g/dL, white blood cell counts were 9860 cells/mm^3, platelet count was 3.24 lakhs/mm^3. As there was no neutrophilic leukocytosis, bacterial infection was less likely. Renal profile showed serum creatinine of 0.8 mg/dl and BUN of 9 mg/dL. Serum electrolytes were also within the reference range-sodium 136 mmol/L, potassium 4 mmol/L, chloride 102 mmol/L, bicarbonate 21 mmol/L, calcium 8.8 mg/dL, magnesium 2.1 mg/dL, phosphate 2.8 mg/dL. Glycemic status assessed-HbA1c was 6.3%. Liver function test and thyroid function tests were unremarkable. Electrocardiogram demonstrated normal sinus rhythm with transthoracic echocardiogram revealing good left ventricular function and no regional wall motion abnormality. Because the clinical picture was unexplained, an magnetic resonance imaging (MRI) brain was obtained. MRI brain including diffusion weighted imaging and apparent diffusion coefficient revealed acute hemorrhagic infarct of bilateral paramedian thalami, with non-hemorrhagic infarct in the left occipital and posterior temporal lobes (Figures 1, 2). Magnetic resonance angiogram demonstrated decreased signal intensities in P3 and P4 segments of left posterior cerebral artery (PCA). Magnetic resonance venogram showed altered signal intensities in the straight sinus and left transverse sinus with corresponding T1 hyperintensities. A CT angiogram confirmed low contrast opacification of P3 and P4 segments of the left PCA. A CT venogram was done which showed no evidence of cerebral venous thrombosis. A repeat CT brain was done 2 days later, which showed a new hemorrhage in the left occipital lobe infarct. This pattern indicated posterior circulation ischemia involving thalamic perforators and cortical PCA branches rather than an isolated perforator infarct.

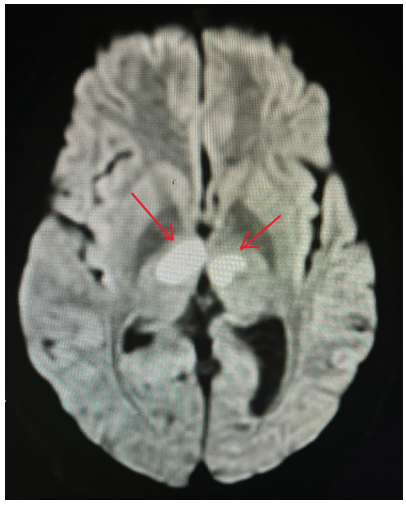

Figure 1. Axial DWI images showing restricted diffusion at the bilateral paramedian thalami (arrows)

DWI: Diffusion-weighted imaging

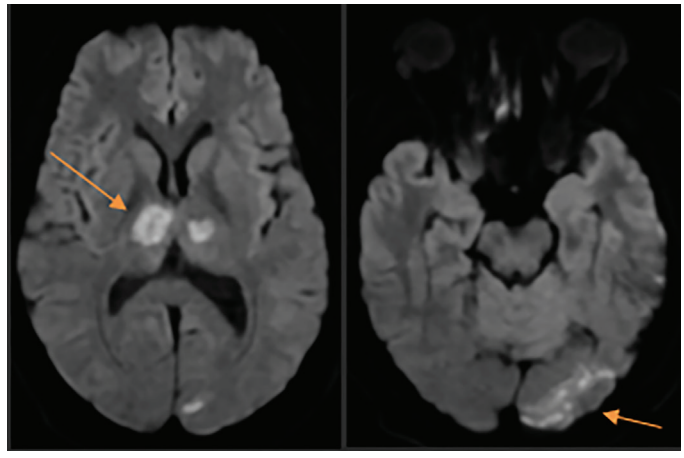

Figure 2. Axial DWI images showing restricted diffusion in bilateral thalami and the left occipital lobe reflecting the cytotoxic edema secondary to an acute infarction (arrows)

DWI: Diffusion-weighted imaging

Hospital Course

The patient was treated as an acute ischemic stroke with secondary hemorrhagic transformation, with aspirin 75 mg OD, atorvastatin 40 mg HS, levetiracetam 500 mg BD, armodafinil 100 mg OD, and antiedema measures. Blood and urine cultures were sterile. Over the next 6 days, alertness gradually improved. A detailed medication history was obtained, and the patient reported no use of sleeping pills or other centrally acting agents. No major motor deficits developed. Stroke workup, including cardiac evaluation with 24 hr holter monitoring, antinuclear antibody by immunofluorescence, antiphospholipid antibodies were unremarkable except for dyslipidemia with elevated cholesterol of 229, triglycerides 265, low-density lipoprotein 130, and high-density lipoprotein of 32. A repeat CT brain done showed the same findings and no worsening. The patient was discharged after 5 days for outpatient follow-up. The most striking feature throughout hospitalization remained excessive sleepiness out of proportion to other neurological findings.

Timeline of Clinical Events

Day 0: The patient went to sleep at 4 pm and was reportedly normal before sleeping

Day 1: Family noted difficulty waking the patient at 11 am. Visit to hospital, CT brain done, normal. Patient brought to our emergency department. After initial workup, MRI brain done showed bilateral thalamic infarct with secondary hemorrhagic transformation.

Days 1-5: Patient treated as acute ischemic stroke with secondary hemorrhagic transformation. Gradual improvement in alertness observed.

Day 5: Patient discharged with outpatient follow-up.

Follow-up and Outcomes

At outpatient follow-up, the patient reported significant improvement in daytime alertness and was able to resume routine daily activities without neurological deficits.

Discussion

Acute hypersomnia from a is not normal and should alert towards a potential stroke, making timely intervention important to patient's outcome (3). The thalamus is a deep grey matter structure on either side of the lateral wall of the third ventricle. It plays an important role in neurological functioning by acting as a transmission station for signals from nearly all pathways in the body. The thalamus participates in sensory processing, motor integration, cognition, and regulation of arousal (4). Damage to the thalamus causes impaired consciousness (42%), vertical gaze palsy (65%), memory issues (58%), and confusion (53%). If the corticospinal tract is involved, the patient may also develop weakness. Owing to its arterial connections with the brainstem, bilateral thalamic infarcts may cause cranial nerve palsies (5). The paramedian regions of the thalamus receive blood supply from small perforating branches of the first segment of the posterior cerebral arteries PCA (P1) bilaterally. In most individuals, each side is supplied independently. However, anatomical variations exist in which a single arterial trunk supplies both paramedian thalami, called the artery of percheron. The exact prevalence of this anatomical variant is not known; however, bilateral paramedian thalamic infarcts account for only 0.1% to 2% of ischemic strokes (6).

The intralaminar and dorsomedial nuclei of the thalamus serve as relays between the ascending reticular activating system in the brainstem and the cerebral cortex. Bilateral paramedian thalamic infarction disconnects the cortex from arousal input, and the patient gets a "sleeping stroke." A major diagnostic challenge arises from the radiology perspective that early CT imaging is frequently normal in paramedian thalamic infarction. Such patients closely resemble those with metabolic or toxic encephalopathy. Several reports have highlighted delays in diagnosis, leading to evaluation for other causes before a definitive MRI was obtained (7,8). While bilateral thalamic infarcts are often attributed to artery of Percheron occlusion, the occipital infarct and PCA flow abnormalities in this patient indicate a broader posterior circulation ischemic process involving both perforator and cortical branches (9,10).

Despite extensive bilateral thalamic infarction, the National Institutes of Health Stroke Scale score (NIHSS) at presentation was retrospectively calculated to be 1. This underscores a well-recognized limitation of the NIHSS in posterior circulation strokes, where deficits related to arousal, cognition, and brainstem function may be underrepresented.

Conclusion

Bilateral thalamic strokes are rare because of dual arterial blood supply. Early CT often fails to detect these infarcts. In emergency settings, it can lead clinicians towards alternative diagnoses, delaying definitive management. Persistent hypersomnolence without focal neurological deficits should prompt consideration of thalamic infarction and early MRI evaluation. This can directly influence early correct management and eligibility for reperfusion therapy.

Patient Perspective

The patient reported progressive improvement in alertness during hospitalization and expressed relief after the diagnosis was established and appropriate treatment was initiated.

Ethics

Informed Consent: Written informed consent was obtained from the patient for publication of this case report and accompanying images. The patient's identity has been protected in accordance with ethical standards.

Footnotes

Authorship Contributions

Surgical and Medical Practices: N.S.R., V.P., B.M.V., R.M., Concept: N.S.R., V.P., B.M.V., R.M., Design: N.S.R., V.P., B.M.V., R.M., Data Collection or Processing: N.S.R., V.P., B.M.V., R.M., Analysis or Interpretation: N.S.R., V.P., B.M.V., R.M., Literature Search: N.S.R., V.P., B.M.V., R.M., Writing: N.S.R., V.P., B.M.V., R.M.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

References

1. Edlow JA, Rabinstein AA, Traub SJ, Wijdicks EF. Diagnosis of reversible causes of coma. Lancet. 2014;384:2064-76.
2. Schmahmann JD. Vascular syndromes of the thalamus. Stroke. 2003;34:2264-78.
3. Alattar M, Nguyen T. 0977 bilateral thalamic strokes presenting as acute hypersomnia. SLEEP. 2023;46(Suppl 1):A431.
4. Bordes S, Werner C, Mathkour M, McCormack E, Iwanaga J, Loukas M, et al. Arterial supply of the thalamus: a comprehensive review. World Neurosurg. 2020;137:310-8.
5. Sheikh Hassan M, Osman Sidow N, Adam BA, Adani AA. Superior alternating hemiplegia (Weber's syndrome): case report. Ann Med Surg (Lond). 2022;77:103674.

6. Hawkes MA, Arena JE, Rollán C, Pujol-Lereis VA, Romero C, Ameriso SF. Bilateral paramedian thalamic infarction. Neurologist. 2015;20:89-92.
7. Phate N, Pawar T, Andhale A, Singh R, Talwar D, Acharya S, et al. Artery of percheron stroke: a case report with a diagnostic challenge. Cureus. 2022;14:e21939.
8. Rajapakshe I, Senanayake B. Paramedian thalamic syndrome due to artery of percheron infarction effectively treated with early anticoagulation: a series of 3 cases. J Neurol Sci. 2019;405:62-3.
9. Naum R, Alattar M. A case of sudden onset of hypersomnolence. Annals of Internal Medicine: Clinical Cases. 2025;4.
10. Mnaili M, Bourazza A. Isolated hypersomnia due to bilateral thalamic infarcts: a case report. Int J Psychiatr Res. 2024;7:1-2.