

## Isolated Hypersomnia Due to Bilateral Thalamic Infarcts: A Case Report

Mohamed Amine MNAILI<sup>1,2\*</sup> and Ahmed BOURAZZA<sup>3</sup>

<sup>1</sup>Neurology Department, Agadir Military Hospital, Agadir, Morocco.

<sup>2</sup>Neurology Department, University of Hassan II, Casablanca, Morocco.

<sup>3</sup>Neurology department, Mohammed V Military Hospital, Morocco.

### \*Correspondence:

MNAILI Mohamed Amine MD, Neurology Department, Agadir Military Hospital, Agadir, Morocco, 80000, Tel: +212660558267.

Received: 28 Feb 2024; Accepted: 29 Mar 2024; Published: 04 Apr 2024

**Citation:** Mohamed Amine MNAILI, Ahmed BOURAZZA. Isolated Hypersomnia Due to Bilateral Thalamic Infarcts: A Case Report. Int J Psychiatr Res 2024; 7(2): 1-2.

### ABSTRACT

*Thalamic pathology should be considered in patients with vague nonlateralizing neurological symptoms. Diagnosis of Percheron artery infarction is challenging and often made later in presentation due to lack of clinical awareness and the nonclassic stroke signs/symptoms on presentation. In this article, we report a case of bilateral thalamic stroke whose clinical presentation was dominated by a sudden onset of hypersomnia.*

### Keywords

Hypersomnia, Stroke, Bithalamic, Case report.

### Introduction

Hypersomnia is a well-known manifestation of bilateral thalamic ischemic stroke [1]. This uncommon ischemic stroke syndrome, with its broad spectrum of clinical presentations, is often misdiagnosed and therefore represents a diagnostic challenge for physicians. Acute hypersomnia is an unusual complication of this stroke [2]. Here, we report a case of bithalamic stroke whose clinical presentation was dominated by hypersomnia.

### Case Report

An 72-year-old man with a past medical history of hypertension, diabetes mellitus, and myocardial infarction presented to the emergency department with altered mental status for 1 day. On examination, she was frequently yawning and sleeping and difficult to arouse. There were no new neurological deficits. No ocular paresis or nystagmus was found. There was no recent history of drug abuse, head injury, trauma, or seizure. During the hospital course, the patient presented many periods of irrepressible need for sleep, and he was unable to stay awake and alerted during the major daytime waking episodes. Brain scan showed bilateral paramedian thalamic infarcts.



**Figure 1:** Brain scan showed bilateral paramedian thalamic infarcts.

All blood tests (e.g., electrolytes test, complete blood count [CBC], and liver function tests) were normal. A 9-lead electrocardiogram showed a right bundle-branch block. A transthoracic and transesophageal echocardiogram, carotid, and vertebral Doppler ultrasound revealed no abnormalities. The patient was diagnosed with ischemic stroke accompanied by excessive daytime sleepiness or hypersomnia. And given dual antiplatelet therapy, atorvastatin, and modanafil. During the post-stroke consultation at 3 months, the patient reported sleeping 09 hours per day (pre-stroke requirement, 5 to 6 hours).

## Discussion

The wide range sensorium impairment ranging from hypersomnia to coma secondary to bilateral thalamic infarction usually leads to diagnostic confusion. In this case, the anatomy of the Ascending reticular activating system (ARAS) will be discussed. Hypersomnolence (i.e., excessive daytime sleepiness and/or prolonged sleep) in bithalamic stroke is explained by the crucial role of the thalamus in sleep regulation and in maintaining arousal. In this case, the anatomy of the Ascending reticular activating system (ARAS) will be discussed. ARAS is a central nervous system that functions as a promoter of the sleep-wake process. This section is located in the reticular formation in the brain stem, which consists of several groups of cells and nuclei and a large number of interneurons and ascending and descending tracts that are interconnected with one another [3]. Bilateral lesions of the posterior region of the hypothalamus, left hemisphere, bilateral cerebral hemisphere (if the lesion is broad), tegmental mesencephalon (midbrain), thalamus, and anterior pectoral tegmentum underlie the occurrence of hypersomnolence due to involvement of the tract that forms ARAS even though only one lesion [3].

Our patient had a unique presentation, with isolated hypersomnia without the associated cranial nerve, motor, sensory and visual deficits commonly seen in paramedian thalamic stroke (PTS) [4]. Hypersomnia in PTS has been attributed to interruption of arousal pathways that are relayed from ascending reticular activating system through the thalamus and diffusely to the cortex [5]. There are 2 routes of projection of the arousal systems (anatomically and chemically distinct neuronal networks) in the brainstem and posterior hypothalamus to the cortex: thalamic (dorsomedial and intralaminar nuclei), and extrathalamic (basal forebrain) [6,7]. Recovery in bilateral PTS is attributable to the existence of an extrathalamic route of cerebral activation [8].

## Conclusion

This case highlighted the difficulties of recognizing AOP stroke in the acute phase. Bilateral thalamic infarction (BTI) can present as sleep-like coma without focal neurological signs which can lead to delay in the diagnosis. Due to the diagnostic challenge, treatment is often delayed.

## Research Quality and Ethics Statement

This case report did not require approval by the Institutional Review Board / Ethics Committee. The authors followed applicable EQUATOR Network (<http://www.equator-network.org/>) guidelines, specifically the CARE guideline, during the conduct of this research project.

## Declaration of Patient Consent

The authors certify that written informed consent was obtained from the patient to allow publication of de-identified findings, which may include images and other clinical information. The individual providing consent understood that although due efforts will be made to conceal the subject's identity, absolute anonymity cannot be guaranteed.

## References

1. Bassetti CL, Valko P. Poststroke hypersomnia. *Sleep Med Clin.* 2006; 1: 139-155.
2. Bollu PC, Pandey A, Pesala SP, et al. Sleepiness after stroke: Case report and review of literature on hypersomnia as a result of stroke. *Madridge Journal of Neuroscience.* 2017; 1: 4-6.
3. Mathias B, Frotscher M. Diagnosis of duus neurology topics: Anatomy, physiology, signs, symptoms. Jakarta EGC Medical Book Publisher. 2010.
4. Hermann DM, Siccoli M, Brugger P, et al. Evolution of neurological, neuropsychological and sleep-wake disturbances after paramedian thalamic stroke. *Stroke.* 2008; 39: 62-68.
5. Jouvet M. Waking mechanisms: Multiple networks in the mesencephalic reticular formation. *Arch Physiol Biochem.* 1996; 104: 762-769.
6. Bassetti C, Mathis J, Gugger M, et al. Hypersomnia following paramedian thalamic stroke: A report of 12 patients. *Ann Neurol.* 1996; 39: 471-480.
7. Castaigne P, Lhermitte F, Buge A, et al. Paramedian thalamic and midbrain infarct: Clinical and neuropathological study. *Ann Neurol.* 1981; 10: 127-148.
8. Jones BE. Basic mechanisms of sleep-wake states. In: Kryger MH, Roth T, Dement WC, eds. *Principles and Practice of Sleep Medicine.* 3rd ed. Philadelphia: Saunders. 2000; 134-115.