

The Clock Struck Three and the Head-ached Up: A Case Report of Hypnic Headache in Psychiatric OPD: Diagnostic and Therapeutic Insights of a Rare Nocturnal Headache Syndrome

Rajamani VM¹, Mallika Biswas²

Received on: 03 August 2025; Accepted on: 26 December 2025; Published on: XX XXXX XX

ABSTRACT

Headache is a common complaint encountered by psychiatrists. While migraines and tension-type headaches are relatively well-known, uncommon presentations, especially in the elderly, pose diagnostic challenges. Hypnic headache (HH), a primary headache disorder infrequently encountered in psychiatry settings, is underdiagnosed, leading to a delay in the right treatment. We present a 54-year-old male with a complaint of headaches over the last 9 months, characteristically occurring at the same time of the night and awakening him from sleep, referred for psychiatric evaluation after poor response to multiple medications. Clinical examination, neuroimaging, polysomnography, and blood investigations were unremarkable. A diagnosis of HH was considered, and the patient improved on a combination of caffeine and paracetamol with a short course of melatonin alongside relaxation exercises and maintained symptom-free at 1-month follow-up. We discuss the approach to evaluation and management of this condition to improve awareness and competency.

Keywords: Caffeine, Case report, Elderly, Hypnic headache, Nocturnal headache.

Indian Journal of Private Psychiatry (2025): 10.5005/jp-journals-10067-0208

INTRODUCTION

Hypnic headache (HH) is a rare primary headache, first described by Raskin in 1988.¹ It was also called a clockwise or alarm clock headache because it occurred exclusively during sleep, awakening the patient almost always at the same time.² It is more frequent after the age of 50 years and is more common in women but may also occur in young adults and children.³ This entity, however, remains underdiagnosed, and pathophysiological mechanisms are poorly understood. Patients are therefore subjected to many investigations and multiple drug trials before the correct diagnosis is made. Lack of awareness of these disorders can lead to delays in diagnosis, even when patients have the classic presentation. We present a patient with HH, who did not respond to standard migraine and tension-type headache medications, but dramatically improved and had a sustained response on caffeine, underscoring the importance of accurate diagnosis and tailored therapy in HH.

CASE DESCRIPTION

A 54-year-old male, with a history of hypertension and hypothyroidism well-controlled on Tab Telmisartan 40 mg and levothyroxine 50 µg, was referred to the psychiatry outpatient department (OPD) of a multidisciplinary tertiary care hospital by his local physician, with complaints of nocturnal headache over the last 9 months. The headache was diffuse, dull aching, moderate in intensity without radiation but reported to occur only at night, at around 3:00 a.m., waking the patient and lasting 30–45 minutes. He did not report any daytime headaches. It occurred twice every week initially but has increased over the last two months as he reports waking up daily, as if an alarm was set in his body for that time. There were no specific aggravating or relieving factors reported. There

¹Department of Psychiatry, Centre for Addiction Medicine, National Institute of Mental Health and Neurosciences, Bengaluru, Karnataka, India

²Department of Psychiatry, Rajendra Institute of Medical Sciences, Ranchi, Jharkhand, India

Corresponding Author: Rajamani VM, Department of Psychiatry, Centre for Addiction Medicine, National Institute of Mental Health and Neurosciences, Bengaluru, Karnataka, India, Phone: +91 7680030033, e-mail: rajumbbs2017@gmail.com

How to cite this article: Rajamani VM, Biswas M. The Clock Struck Three and the Head-ached Up: A Case Report of Hypnic Headache in Psychiatric OPD: Diagnostic and Therapeutic Insights of a Rare Nocturnal Headache Syndrome. *Ind J Priv Psychiatry* 2025;xx(x):xx–xx.

Source of support: Nil

Conflict of interest: None

Patient consent statement: A written informed consent was obtained from the patient for the publication of details, which can include photographs and/or videos and/or case history to be published in any printed/online journals.

was no change in the quality and intensity of these headaches. His wife checked his blood sugar during the headache on two occasions and found it to be normal. There was no associated nausea, vomiting, photophobia or phonophobia, tearing, nasal stuffiness, or conjunctival injection alongside these headaches. There was no history of trauma prior to the onset of these headaches, no history of sleep-related problems, including insomnia before the episodes, snoring or parasomnias, or any leg discomfort or apnea. There was no past history of migraines or recurrent headaches,

strokes, seizures, neuralgias, or psychiatric disorders. Family history was unremarkable. He had three prior medical consultations from private physicians, with no improvement on ibuprofen (2 weeks), amitriptyline (6 weeks), flunarizine with propranolol (8 weeks), and sodium valproate (5 weeks), and was suggested a psychiatry consultation by a relative. He is currently off medications.

General physical examination and systemic exam revealed normal findings. Neurological examination was unremarkable. He had a blood pressure of 124/70 mm Hg and a heart rate of 80 beats/min. Fundoscopy was normal, and temporal arteries were nontender. Recent hematological and biochemical investigations were normal. The magnetic resonance imaging (MRI) brain was unremarkable. Polysomnography done prior to presentation had ruled out sleep apnea.

Mental status examination (MSE) revealed a tidy, cooperative, euthymic individual with dysphoric affect and occasional worry regarding lack of improvement, and no other psychiatric symptoms of depression, anxiety disorder, or somatization. The case and investigations were reviewed, and a diagnosis of primary HH was considered in view of the characteristic presentation. He was started on caffeine 50 mg plus paracetamol 650 mg combination tablet and melatonin 3 mg for 10 days alongside Telmisartan and Thyroxine. Relaxation exercises were also advised. At the follow-up visit after 10 days, the patient reported significant improvement with complete resolution of headaches from the 3rd day itself. Melatonin was stopped. At 1-month follow-up, the patient remained headache-free on caffeine 50 mg plus paracetamol 650 mg alongside regular relaxation exercises without any reported adverse effects.

DISCUSSION

Hypnic headache, often referred to as “alarm clock headache,” is a rare primary headache disorder characterized by exclusively nocturnal attacks that awaken patients from sleep, typically at a consistent time each night. Our patient’s age of presentation and headache characteristics—a strictly nocturnal, dull, diffuse pain occurring exclusively during sleep at a fixed circadian time—are most suggestive of HH but raise key diagnostic challenges.

The approach to new-onset headache in individuals over 50, however, required systematic and evidence-based exclusion of secondary causes.

A detailed history was taken, and a physical examination evaluating red flag signs of headache was done. The absence of temporal tenderness, jaw claudication, visual disturbances, fever, and neurological deficits argued against temporal arteritis, intracranial mass, or infections. The patient had well-controlled hypertension, and fundoscopy did not reveal any features of retinopathy.

Investigative workup—including complete blood counts, liver and renal function tests, electrolytes, inflammatory markers, and thyroid function—was unremarkable, further lowering the possibility of temporal arteritis and metabolic causes. Magnetic resonance imaging brain excluded intracranial mass lesions, subdural hematoma, and other space-occupying lesions, which are important etiologies in elderly patients presenting with new-onset headaches. Polysomnography ruled out obstructive sleep apnea—a common cause of nocturnal headache in this age-group—while blood glucose monitoring during symptoms ruled out hypoglycemia.

Treatment review neither suggested features of medication overuse headache nor were there features of chronic pain

syndromes or sleep disorders (e.g., restless leg syndrome, parasomnias). The lack of autonomic features, photophobia, phonophobia, or nausea and vomiting made cluster headache and migraine unlikely. Furthermore, the patient’s lack of response to standard migraine and tension-type headache prophylactics (amitriptyline, flunarizine, propranolol, sodium valproate) further supported a diagnosis outside these primary headache disorders.

After excluding these differentials, the patient’s syndrome aligned closely with International Classification of Headache Disorders (ICHD-3) criteria for HH.

Lithium, indomethacin, and caffeine have been reported to be effective in the management of HH.^{3,4} The adverse effects of lithium in an elderly patient with hypothyroidism, the need for serum lithium estimations for titration and the risk of subsequent discontinuation discouraged its use. Similarly, tolerability to indomethacin in the elderly, the phenomenon of “paradox of indomethacin,” where it could lead to daytime headaches, was a clinical concern.⁵ Hence, our first-line principal management strategy centered on low-dose caffeine (50 mg) plus paracetamol (650 mg) at bedtime, initially combined with a brief course of melatonin (3 mg).

The administration of caffeine in our patient immediately aborted the nocturnal attacks (evident by cessation of headaches from day three) and also provided continued prophylactic benefit when used nightly. Caffeine was safe and effective as both an abortive and prophylactic agent, aligning with previous studies, showing it was effective in approximately 70% of all patients.^{4,6} Antagonism of adenosine A1, A2A, and A2B receptors seems to explain its analgesic effects.⁷ The cyclic nature of some headache disorders indicates the role of melatonin and the rhythm-regulating centers of the brain.⁸ The patient wakes up consistently at the same time every night. We considered a short course of melatonin 3 mg as a rational chronobiotic adjunct and also to mitigate caffeine-induced insomnia, if any. Melatonin in doses 3–5 mg has been shown to have response rates around 50% in elderly patients with HH.⁹

The use of combination analgesics in HH is not widely discussed in the literature. While moderate doses of caffeine (10–35 mg/kg) augment the analgesic effect of medications such as acetaminophen, a higher risk of medication overuse headache compared with single-substance formulations has been reported alongside nervousness, dyspepsia, dizziness.^{10–12} The combination was well-tolerated and effective in our patient, supporting its pragmatic benefit as an adjunct in elderly patients. However, adverse events should be assessed at every follow-up.

The use of relaxation exercises in this case was advised as part of multimodal management, given the minor dysphonic effect observed on MSE. It might have synergistically contributed to improved sleep hygiene, psychological well-being, and subsequent headache reduction. While the literature on their use in HH is sparse, similar pathways of its efficacy in migraines and tension-type headaches may apply and require further studies.

Our approach led to rapid and sustained remission of HHs and was well tolerated, with no adverse effects reported at 1 month. The short follow-up period limits definitive conclusions; hence, further studies with longer follow-up are required for determining an effective dose range and optimal treatment duration with caffeine in HH.

CONCLUSION

The case highlights the need to consider HHs as an important differential diagnosis of sleep-related headaches, especially in

the elderly. To our knowledge, only a few cases of HH have been reported from India, and fewer still demonstrating successful management with a caffeine-paracetamol combination with melatonin. Caffeine, in this case, demonstrated both abortive and prophylactic efficacy, expanding upon published evidence. It is important to raise awareness of HH among psychiatrists, as it is conceptualized as depression, insomnia, or other headache syndromes. This could minimize unnecessary investigations and optimize patient outcomes.

ORCID

Rajamani VM  <https://orcid.org/0000-0002-0072-2498>

Mallika Biswas  <https://orcid.org/0009-0006-6918-8832>

REFERENCES

1. Raskin NH. The hypnic headache syndrome. *Headache* 1988;28(8):534–536. DOI: 10.1111/j.1526-4610.1988.hed2808534.x.
2. Al Khalili Y, Tan CW, Lui F. Hypnic Headache. [Updated 2025 Sep 24]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK557598/>.
3. Silva-Néto RP, Santos PE, Peres MF. Hypnic headache: A review of 348 cases published from 1988 to 2018. *J Neurol Sci* 2019;401:103–109. DOI: 10.1016/j.jns.2019.04.028.
4. Tariq N, Estemalik E, Vij B, et al. Long-term outcomes and clinical characteristics of hypnic headache syndrome: 40 patients series from a tertiary referral center. *Headache* 2016;56(4):717–724. DOI: 10.1111/head.12796.
5. Dodick DW, Jones JM, Capobianco DJ. Hypnic headache: Another indomethacin-responsive headache syndrome? *Headache* 2000;40(10):830–835. DOI: 10.1046/j.1526-4610.2000.00150.x.
6. Holle D, Naegel S, Krebs S, et al. Clinical characteristics and therapeutic options in hypnic headache. *Cephalalgia* 2010;30(12):1435–1442. DOI: 10.1177/0333102410375727.
7. Sawynok J, Yaksh TL. Caffeine as an analgesic adjuvant: A review of pharmacology and mechanisms of action. *Pharmacol Rev* 1993;45(1):43–85. PMID: 8475169.
8. Peres MF. Melatonin, the pineal gland and their implications for headache disorders. *Cephalalgia* 2005;25(6):403–411. DOI: 10.1111/j.1468-2982.2005.00889.x.
9. Gelfand AA, Goadsby PJ. The role of melatonin in the treatment of primary headache disorders. *Headache* 2016;56(8):1257–1266. DOI: 10.1111/head.12862.
10. Granados-Soto V, Castañeda-Hernández G. A review of the pharmacokinetic and pharmacodynamic factors in the potentiation of the antinociceptive effect of nonsteroidal anti-inflammatory drugs by caffeine. *J Pharmacol Toxicol Methods* 1999;42(2):67–72. DOI: 10.1016/s1056-8719(00)00044-7.
11. Holle D, Obermann M. Hypnic headache and caffeine. *Expert Rev Neurother* 2012;12(9):1125–1132. DOI: 10.1586/ern.12.100.
12. Migliardi JR, Armellino JJ, Friedman M, et al. Caffeine as an analgesic adjuvant in tension headache. *Clin Pharmacol Ther* 1994;56(5):576–586. DOI: 10.1038/clpt.1994.179.