



Hypersomnia Related to Artery of Percheron Infarction

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Infarction of the artery of Percheron (AOP) is rare. Its diagnosis is challenging because of its varied symptoms depending on the area affected, and it is often difficult to detect radiologically. Herein, we describe a case of AOP infarction that presented with a rare symptom, delaying its diagnosis. A 65-year-old woman presented with a 1-week history of excessive daytime sleepiness despite adequate quality and duration of nighttime sleep. Physical and laboratory examinations were inconclusive. Noncontrast-enhanced computed tomography revealed hypodense lesion in the right thalamus. T2-weighted and fluid-attenuated inversion recovery magnetic resonance images displayed high-signal-intensity lesions with restricted diffusion in the bilateral paramedian thalamus. A diagnosis of AOP infarction was established. Patients with AOP infarction benefit from the immediate initiation of thrombolytic therapy, particularly within 4.5 hours, necessitating a prompt and accurate diagnosis. This case report emphasizes that AOP infarction should be considered a differential diagnosis in patients presenting with hypersomnia.

Key words: artery of Percheron, hypersomnia, magnetic resonance imaging

Introduction

Infarction of the artery of Percheron (AOP) is rare. Its diagnosis is challenging because of its varied symptoms depending on the area affected, and it is often difficult to detect radiologically. Herein, we describe a case of AOP infarction that presented with a rare symptom, which delaying its diagnosis.

Case Report

A 65-year-old woman without any under-

lying medical conditions visited our emergency department with excessive sleepiness for 1 week, even during the day and at mealtimes. She was unable to stay awake during activities. A thorough examination did not reveal fever, headache, tremors, limb weakness, dyspnea, or focal neurologic deficits. The laboratory test results were unremarkable. Noncontrast-enhanced computed tomography (CT) revealed hypodense lesion in the right thalamus (Fig. 1A), ischemic infarction was suspected. Carotid and transcranial duplex sonography and electroencephalography were normal. Brain magnetic resonance (MR) imaging revealed high-signal

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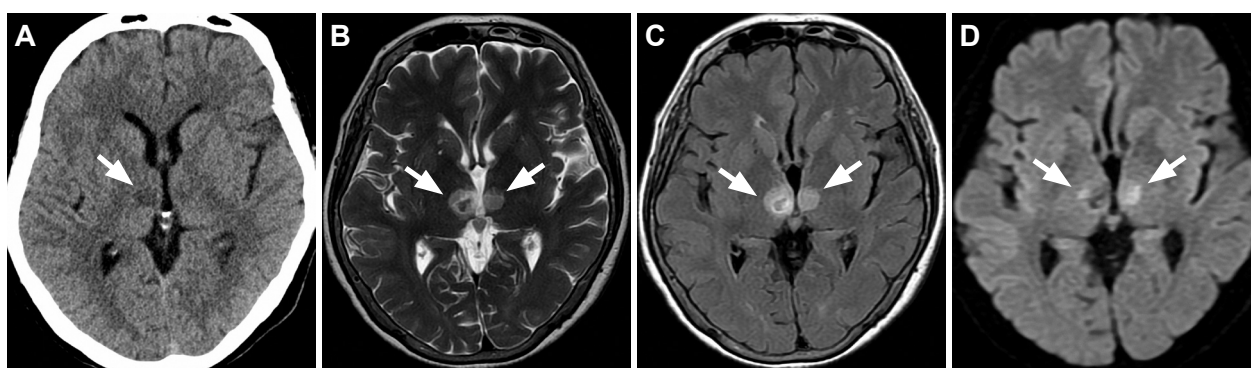


Fig. 1 Computed tomography (CT) and magnetic resonance (MR) images showing acute ischemic infarction consistent with artery of Percheron occlusion in the paramedian thalamus. (A) Noncontrast-enhanced brain CT showing a right thalamus hypodensity, which indicates an ischemic infarct (arrow). Axial (B) T2-weighted and (C) fluid-attenuated inversion recovery (FLAIR)-weighted MR images showing hyperintensities in bilateral paramedian thalamus (arrows). (D) Diffusion-weighted MR images showing restricted diffusion in the bilateral paramedian thalamus (arrows).

intensity lesions on T2-weighted (Fig. 1B) and fluid-attenuated inversion recovery images (Fig. 1C), with restricted diffusion (Fig. 1D) in bilateral paramedian thalamus. Therefore, AOP infarction was diagnosed. Empirical and central stimulants were administered, and she was discharged based on improvement.

Discussion

The blood supply to the thalami and midbrain arises from the posterior cerebral artery (PCA) and communicating arteries and can be divided into four territories: anterior, paramedian, posterior, and inferolateral (Fig. 2).¹ The branches to paramedian territory greatly vary and are divided into four types: Type I, both paramedian arteries arise from proximal (P1) segment of ipsilateral PCA; Type IIa, both paramedian arteries arise from left or right PCA; Type IIb, bilateral thalamic branches arise from a single trunk originating from PCA; and Type III, communicating artery exists between the P1 segments of both PCAs. Type IIb was first described in 1973 by Gérard Percheron, and the trunk was named “artery of Percheron”.^{2,3} Its incidence is 4 – 12%.¹ It creates a unique radiographic pattern, which can be classified into four types.²

Thalamus plays an essential role in various

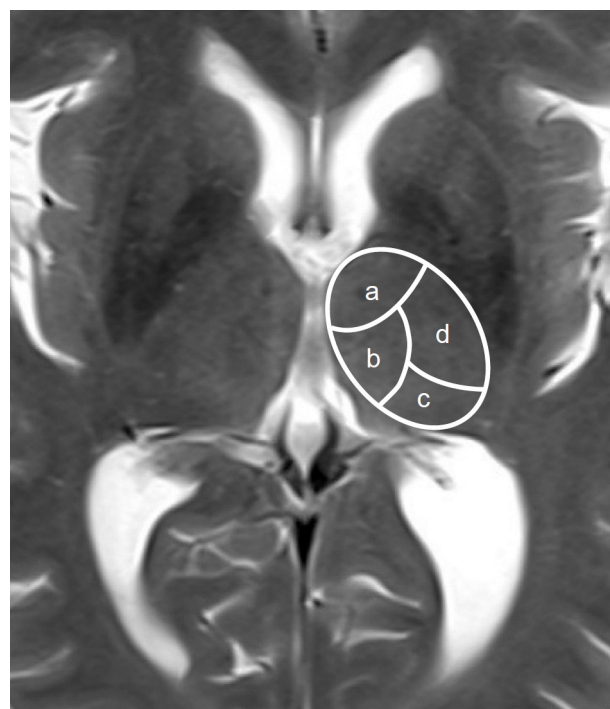


Fig. 2 Anatomy territory of the thalamus. (a) Anterior territory. Blood supply from tuberothalamic artery of posterior communicating artery. (b) Paramedian territory. Blood supply from paramedian artery of posterior cerebral artery (PCA). (c) Posterior territory. Blood supply from posterior choroidal artery of PCA. (d) Inferolateral territory. Blood supply from thalamogeniculate artery of PCA.

brain functions, including memory, emotion processing and regulation, sleep-wake cycle, executive functions, general cortical alerting response mediation, and sensory information processing.⁴ AOP infarction has three major

manifestations: altered mental status, vertical gaze palsy, and memory impairment. However, diagnosing AOP infarction remains challenging because of the wide array of symptoms. Moreover, visualization of any AOP occlusion using CT or MR angiography is difficult due to its small caliber,^{1,5} which further delays diagnosis. The radiological differential diagnoses for AOP infarction include top of basilar artery syndrome, internal cerebral vein thrombosis, and Wernicke's encephalopathy. Top of the basilar artery syndrome can be excluded if there is no evidence of basilar artery occlusion. Deep vein thrombosis can be excluded if cerebral venous system is patent. Wernicke's encephalopathy may be excluded if there is a lack of corresponding history and vitamin B1 (Thiamine) deficiency.

Hypersomnolence is characterized by excessive daytime sleepiness despite adequate quality and duration of nocturnal sleep. The reticular activating system (RAS) is a network of neurons originating in the pons, midbrain, and bilateral thalamus and entering bilateral cerebral cortices that mediate behavior and wakefulness. RAS impairment may engender altered mental status, such as hypersomnia. Although hypersomnia in AOP infarction is rare, it has been reported.⁶ Bollu *et al.* asserted that bilateral lesions of the posterior hypothalamus, tegmental midbrain, upper pons, stratum, pontine tegmentum, medulla, and cerebral hemisphere may cause hypersomnolence.⁷ Although several mechanisms have been proposed, the relationship between hypersomnolence and AOP infarction remains unclear.

The effective management of AOP infarction involves immediate initiation of thrombolytic therapy within 4.5 hours. A prompt and accurate diagnosis of AOP infarction is paramount to maximizing treatment efficacy. Several AOP infarction cases, diagnosed by clinical and radiological observations following rehabilitation, have been managed conservatively.⁸ Although no consensus has been

reached on the treatment of AOP infarction-related hypersomnia, prompt diagnosis can lead to earlier treatment, lower morbidity, and improved patient outcomes.

Conclusions

This case report highlights the difficulty of timely recognition of AOP infarction. Furthermore, it emphasizes the importance of considering AOP infarction as a differential diagnosis for hypersomnia.

Author Contributions

Study Design, ICL and CSC; Data Collection, PLL and CLL; Data Interpretation, CSC, ICL and CLL; Manuscript Preparation, ICL and CHO; Literature Search, CHO and PLL. All authors have read and agreed to the published version of the manuscript.

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Institutional Review Board Statement

Not applicable for studies not involving humans or animals.

Informed Consent Statement

According to our hospital's ethical policy for case reports, this study had received the patient consent form agreement.

Data Availability Statement

All relevant data is provided in the manuscript.

Conflicts of Interest

The authors declare no conflict of interest.

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