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WHEN BOTH THALAMI FALL SILENT: ACUTE ARTERY OF PERCHERON INFARCTION PRESENTING WITH IMPAIRED CONSCIOUSNESS

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AUTHOR AND AFFILIATION

Samia Amar[1], Boutaina Essaher[1], Yahya Sqalli Houssaini[1], Asmaa Fariyou[1], Hassan Ennouali[2], Mohammed Ennmer[2]
[1]Department of radiology, Faculty of medicine and pharmacy, Mohammed V University, Rabat, Morocco
[2]Department of radiology, Mohammed V Military Instruction Hospital, Rabat, Morocco

Corresponding author: Samia Amar, samiaamar23@gmail.com

ABSTRACT

Artery of Percheron (AOP) infarction is an uncommon cause of bilateral paramedian thalamic ischemia and may present with nonspecific neurological manifestations that delay recognition of acute stroke. We report the case of a 76-year-old man with treated arterial hypertension and diabetes mellitus who presented with acute impairment of consciousness and was admitted approximately 3 hours after symptom onset. Brain magnetic resonance imaging (MRI) demonstrated bilateral paramedian thalamic diffusion restriction with corresponding low apparent diffusion coefficient (ADC) values and FLAIR hyperintensity, consistent with acute ischemic infarction. No mesencephalic involvement was observed. Three-dimensional time-of-flight (3D TOF) magnetic resonance angiography showed no proximal intracranial arterial occlusion; the AOP was not visualized. The characteristic bilateral paramedian thalamic distribution, together with the clinical presentation, supported the diagnosis of AOP territory infarction. The patient received intravenous thrombolysis within the therapeutic window. This case highlights the importance of recognizing the characteristic MRI pattern of AOP infarction in patients presenting with otherwise unexplained acute impairment of consciousness, even in the absence of midbrain involvement or direct visualization of the responsible perforating artery.

KEYWORDS

Artery of Percheron, Bilateral thalamic infarction, Ischemic stroke, Impaired consciousness

MAIN ARTICLE

INTRODUCTION

The thalamus has a complex vascular supply and plays a central role in consciousness, memory, behavior, and multiple sensory and motor functions. Its arterial supply is conventionally divided into anterior, paramedian, inferolateral, and posterior territories. The paramedian thalami are particularly involved in the regulation of consciousness and alertness, explaining the prominent disturbances of arousal that may accompany bilateral injury [1].

The artery of Percheron (AOP) is an anatomical variant of the paramedian thalamic circulation, corresponding to the type IIb configuration described by Gérard Percheron, in which a solitary perforating arterial trunk arises from the P1 segment of one posterior cerebral artery and supplies both paramedian thalami, with variable contribution to the rostral midbrain [1,2]. Occlusion of this single artery may therefore result in bilateral paramedian thalamic infarction, with or without associated mesencephalic involvement.

AOP infarction is rare, accounting for approximately 0.1–2% of all ischemic strokes [3]. Its clinical recognition can be challenging because manifestations are heterogeneous and may lack the typical lateralizing deficits associated with acute ischemic stroke. Altered consciousness, memory impairment, and vertical gaze palsy constitute the classically described clinical spectrum, although presentations may range from hypersomnolence and confusion to profound coma [1,3].

MRI, particularly diffusion-weighted imaging (DWI), plays a central role in early recognition by demonstrating the characteristic bilateral paramedian thalamic ischemic pattern. Direct visualization of the AOP itself is not required for diagnosis and is frequently not possible because of the vessel's small caliber [4].

We report a case of acute AOP territory infarction in a 76-year-old man presenting predominantly with impaired consciousness, in whom MRI performed approximately 3 hours after symptom onset demonstrated acute bilateral paramedian thalamic infarction without mesencephalic involvement, allowing intravenous thrombolysis within the therapeutic window.

CASE PRESENTATION

A 76-year-old man with a medical history of treated arterial hypertension and diabetes mellitus presented with acute-onset impairment of consciousness. He was admitted to our institution approximately 3 hours after symptom onset. On initial clinical assessment, the predominant neurological finding was an altered level of consciousness.

Given the sudden onset of symptoms, an acute cerebrovascular event was suspected, and brain MRI was performed in the acute setting. DWI demonstrated bilateral paramedian thalamic areas of marked diffusion restriction (Figure 1A), with corresponding low ADC values (Figure 1B), consistent with acute ischemic infarction. Corresponding bilateral paramedian thalamic hyperintensities were also identified on fluid-attenuated inversion recovery (FLAIR) imaging (Figure 1C). No mesencephalic involvement was observed (Figure 2). Three-dimensional time-of-flight magnetic resonance angiography showed no proximal intracranial arterial occlusion. The artery of Percheron was not visualized.

The bilateral paramedian distribution of the acute ischemic lesions, together with the clinical presentation dominated by impaired consciousness, was highly suggestive of an infarction in the AOP territory. The absence of mesencephalic involvement corresponded to the isolated bilateral paramedian thalamic pattern of AOP infarction.

As the patient presented approximately 3 hours after symptom onset and was within the therapeutic window, intravenous thrombolysis was administered according to the institutional acute ischemic stroke protocol.

Following intravenous thrombolysis, the patient's level of consciousness subsequently improved, although persistent somnolence was noted during the clinical course.

DISCUSSION

The AOP corresponds to the type IIb variant of the paramedian thalamic arterial supply described by Percheron. In this configuration, a single perforating arterial trunk arises from the P1 segment of one posterior cerebral artery and supplies both paramedian thalami, with variable contribution to the rostral midbrain [1,2]. This anatomical arrangement explains how occlusion of a single small perforating vessel can produce bilateral thalamic ischemia.

Although the precise prevalence of this anatomical variant remains uncertain, cadaveric studies have estimated its presence in approximately 7–11.7% of individuals [1]. AOP infarction itself represents only a small proportion of ischemic strokes. In a consecutive series of 4,705 patients with acute ischemic stroke, Macedo et al. identified eight cases of AOP infarction, corresponding to a frequency of 0.17% [5].

Four principal imaging patterns of AOP infarction have been described according to the distribution of ischemic injury. Bilateral paramedian thalamic infarction with rostral midbrain involvement is the most frequent pattern, accounting for approximately 43% of cases, followed by isolated bilateral paramedian thalamic infarction without midbrain involvement in approximately 38%. Bilateral paramedian and anterior thalamic involvement with midbrain infarction accounts for approximately 14%, whereas bilateral paramedian and anterior thalamic infarction without midbrain involvement represents approximately 5% [6]. The imaging findings in our patient therefore corresponded to the isolated bilateral paramedian thalamic pattern without mesencephalic involvement, the second most frequently reported configuration.

The clinical manifestations of AOP infarction reflect the functions of the paramedian thalami and, when involved, the rostral midbrain. Alterations in consciousness are particularly common and may range from somnolence and hypersomnolence to stupor or coma. Memory impairment, behavioral changes, speech abnormalities, and gaze disturbances may also occur [1,3,6,7]. Altered mental status, memory impairment, and vertical gaze palsy constitute the classically described clinical triad of AOP infarction [3]. Changes in the level of consciousness have been reported in 73–94.2% of patients with AOP stroke, emphasizing their central role in its clinical presentation [6].

In our patient, impairment of consciousness was the predominant presenting manifestation. This presentation is particularly relevant because an acute disturbance of consciousness without prominent lateralizing neurological findings may not immediately suggest an arterial ischemic event. Changes in the level of consciousness are nevertheless among the most frequently reported manifestations of AOP infarction [6]. Recognition of this association is therefore important in patients presenting with otherwise unexplained acute alteration of consciousness.

The absence of mesencephalic involvement in our patient is also relevant to the clinical and radiological presentation. Midbrain involvement may be associated with gaze abnormalities, oculomotor deficits, ataxia, and motor dysfunction [1,6]. Conversely, infarction confined to the bilateral paramedian thalami may predominantly manifest with disturbances of consciousness and cognition [6]. The absence of midbrain involvement therefore does not argue against AOP infarction; isolated bilateral paramedian thalamic infarction represents a well-established imaging pattern of this vascular syndrome.

Bilateral thalamic abnormalities have a broad differential diagnosis. In addition to AOP infarction, potential causes include deep cerebral venous thrombosis, metabolic disorders

such as Wernicke encephalopathy and osmotic demyelination and neoplastic lesions [8]. Clinical presentation and imaging characteristics are therefore essential for narrowing the differential diagnosis. In our patient, the abrupt onset of impaired consciousness together with marked DWI hyperintensity and corresponding ADC reduction in a bilateral paramedian distribution strongly supported acute arterial ischemia.

Neuroimaging is central to the diagnosis of AOP infarction. Non-contrast computed tomography may be unrevealing during the early phase, with CT reported to remain normal in up to 50% of cases. MRI is more sensitive for detecting acute AOP infarction, with DWI and FLAIR being particularly useful for identifying the characteristic bilateral paramedian thalamic abnormalities [6,7]. In our patient, bilateral paramedian thalamic diffusion restriction was clearly demonstrated approximately 3 hours after symptom onset, allowing the ischemic nature and characteristic distribution of the lesions to be recognized during the therapeutic window.

When the rostral midbrain is involved, a characteristic “V sign” may be observed as a V-shaped hyperintensity along the pial surface of the midbrain adjacent to the interpeduncular fossa on axial FLAIR and DWI. This finding has been reported in approximately 67% of AOP infarctions with midbrain involvement and may provide an additional diagnostic clue [6]. The absence of this sign in our patient was consistent with the lack of mesencephalic involvement.

The role of vascular imaging deserves particular consideration. The AOP is a small-caliber perforating vessel and may be difficult to demonstrate on magnetic resonance angiography, CT angiography, or even conventional catheter angiography [2,4,6]. Consequently, non-visualization of the AOP on vascular imaging should not, by itself, be interpreted as evidence of occlusion, as the vessel may not be visualized even under normal conditions. Direct visualization of its occlusion is therefore not required to establish the diagnosis [2,4,6]. In our patient, 3D TOF MRA showed no proximal intracranial arterial occlusion, while the AOP itself was not visualized. The diagnosis of AOP infarction was based on the characteristic bilateral paramedian thalamic distribution of acute ischemia in the appropriate clinical context. Recognition of this point is important because reliance on direct demonstration of the responsible perforating artery may result in unnecessary diagnostic uncertainty.

Timely recognition of AOP infarction has important therapeutic implications. Because the initial presentation may consist predominantly of altered consciousness, the condition can initially be mistaken for a metabolic, toxic, infectious, epileptic, or other nonvascular cause of encephalopathy, potentially delaying reperfusion therapy [4,6,7]. When identified within

the appropriate therapeutic window, eligible patients should be considered for intravenous thrombolysis according to established principles of acute ischemic stroke management [4,6,8].

Safan et al. reported an AOP infarction characterized by bilateral thalamic ischemia without midbrain involvement in a patient presenting with decreased consciousness who received intravenous thrombolysis within the therapeutic window [8]. The patient subsequently showed progressive neurological improvement, with successful extubation by day 6 and the ability to follow simple commands, followed by substantial recovery at clinical follow-up, although residual vertical gaze impairment and right-sided dysmetria persisted [8]. This report illustrates the importance of recognizing the characteristic imaging pattern sufficiently early to preserve the opportunity for reperfusion treatment.

In our patient, presentation approximately 3 hours after symptom onset and early identification of bilateral paramedian thalamic ischemia on MRI allowed the diagnosis of acute AOP territory infarction despite the absence of direct visualization of the responsible artery on 3D TOF MRA. Intravenous thrombolysis was subsequently administered within the therapeutic window. The patient's level of consciousness subsequently improved, although persistent somnolence was noted during the clinical course.

The prognosis of AOP infarction is heterogeneous and appears to depend partly on the extent and distribution of ischemic injury. Infarctions confined to the bilateral paramedian thalami appear to have a more favorable prognosis than patterns involving the rostral midbrain. Navickaitė et al. cite functional independence in 67% of patients with bilateral paramedian thalamic infarction compared with 25% when the rostral midbrain is involved [6]. Nevertheless, persistent hypersomnolence and cognitive impairment may occur. In the consecutive series reported by Macedo et al., persistent hypersomnia and vascular dementia were documented in some survivors at 6-month follow-up [5].

The present case reinforces an important diagnostic message: in a patient presenting with unexplained acute impairment of consciousness, bilateral paramedian thalamic diffusion restriction should immediately raise suspicion for AOP infarction. Neither the absence of mesencephalic involvement nor failure to visualize the AOP on routine angiographic imaging excludes the diagnosis. Recognition of this characteristic clinic-radiological pattern is particularly important in patients presenting early, in whom prompt diagnosis may preserve eligibility for reperfusion therapy.

CONCLUSION

Artery of Percheron infarction should be considered in patients presenting with acute unexplained impairment of consciousness when MRI shows bilateral paramedian thalamic infarction. The absence of mesencephalic involvement does not exclude the diagnosis, and direct visualization of the AOP on vascular imaging is not required. Early recognition of this characteristic clinic-radiological pattern is important to avoid diagnostic delay and preserve the opportunity for reperfusion therapy in eligible patients.

FIGURES:

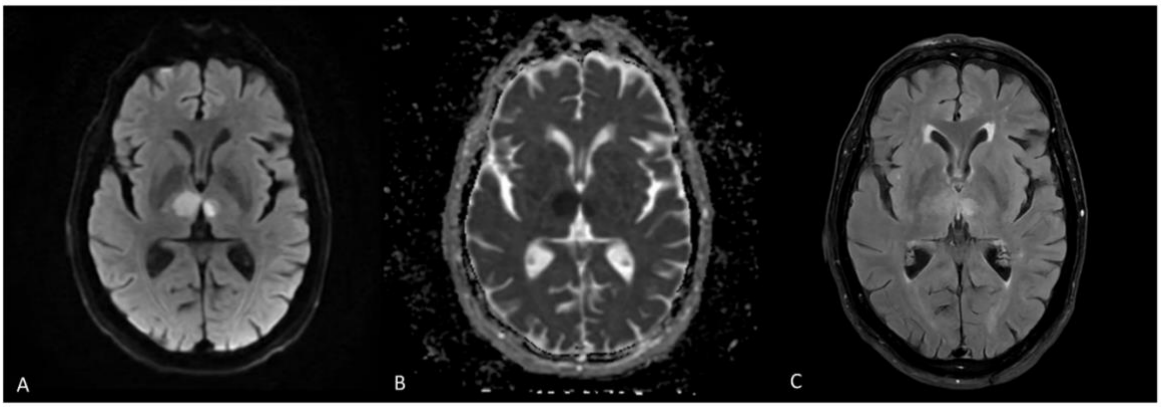

Figure 1. MRI findings at the level of the thalami
(A) Axial diffusion-weighted image (DWI) demonstrates bilateral paramedian thalamic hyperintensities. (B) Corresponding apparent diffusion coefficient (ADC) map demonstrates low signal intensity in the same regions, confirming restricted diffusion. (C) Axial FLAIR image demonstrates corresponding bilateral paramedian thalamic hyperintensities.

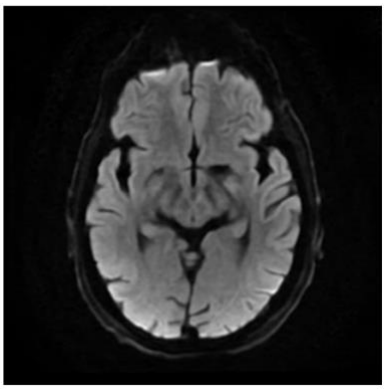

Figure 2. MRI findings at the level of the midbrain
Axial diffusion-weighted image (DWI) demonstrates no abnormal diffusion restriction within the midbrain, showing the absence of mesencephalic involvement.

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