Case Report

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A Rare Case of Artery of Percheron Infarction: Diagnostic Challenges and Management

Internal Medicine Section

MADHULIKA MAHASHABDE[1], RISHIKESH SUNILSINGH CHAUHAN[2], SAIKRISHNA REDDY[3], JUGAL SRIRAM[4]

ABSTRACT

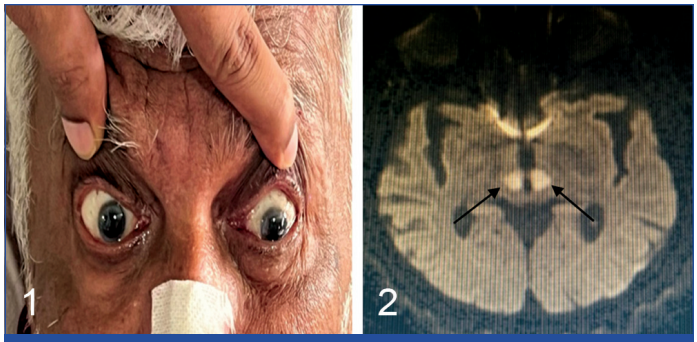
The Artery of Percheron (AOP) is a rare anatomical variant in the posterior circulation, where a single arterial trunk supplies both the paramedian thalami and the rostral midbrain bilaterally. Infarction of the AOP can result in altered mental status, memory deficits and vertical gaze palsy. However, early diagnosis remains challenging, as initial Computed Tomography (CT) scans often fail to detect subtle ischaemic changes. This underscores the critical need for the early use of advanced imaging techniques, particularly Magnetic Resonance Imaging (MRI), to improve diagnostic accuracy and expedite management. In this case, a 66-year-old male presented with sudden-onset severe drowsiness and unresponsiveness. Initial CT imaging did not reveal abnormalities, delaying diagnosis and ruling out thrombolytic therapy, which is most effective within 4.5 hours of symptom onset. Subsequent MRI identified bilateral thalamic and left midbrain infarction consistent with AOP occlusion. Management focused on supportive care, secondary stroke prevention with antiplatelet therapy, and rehabilitation. Despite the delayed intervention, the patient demonstrated gradual neurological improvement, highlighting the importance of tailored care in rare ischaemic stroke subtypes. Early identification and treatment remain essential for optimising outcomes and minimising long-term deficits. This case emphasises the importance of advanced imaging for diagnosing AOP infarction, a condition often missed on early CT scans, and highlights the challenge of timely diagnosis in ischaemic stroke management.

Keywords: Neuroanatomy, Neuroimaging, Pathophysiology, Perfusion, Prognosis, Thrombosis

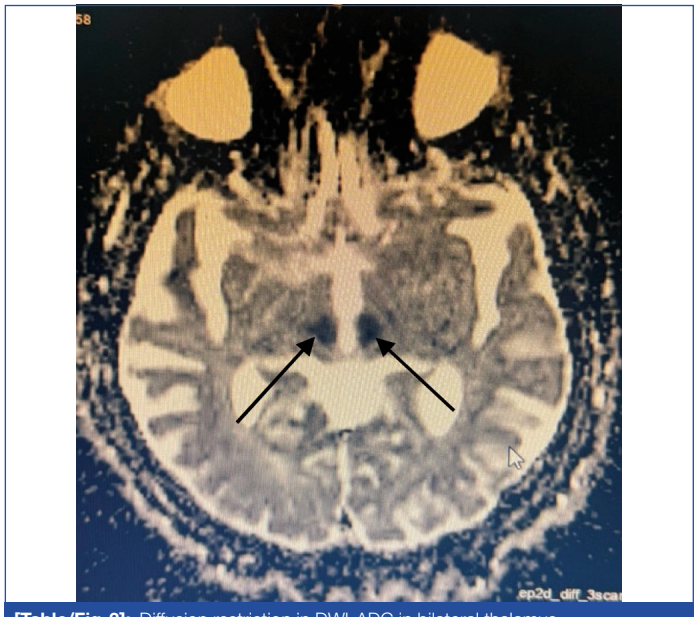
CASE REPORT

A 66-year-old male presented to the emergency department with a sudden onset of severe drowsiness that rapidly progressed to unresponsiveness. Earlier that day, he had been asymptomatic. Upon arrival, his Glasgow Coma Scale (GCS) score was 3, and examination revealed left pupil dilation and leftward eye deviation [Table/Fig-1], necessitating intubation for airway protection. General and systemic examinations were unremarkable. The patient was vitally stable, with normal blood pressure, pulse rate, and respiratory rate. Cardiovascular examination revealed normal heart sounds (S1 and S2) with no murmurs, while respiratory examination showed normal vesicular breath sounds with equal air entry bilaterally. Routine blood tests, including a complete blood count, serum electrolytes, and renal function tests, were all within normal limits. Additionally, an Electrocardiogram (ECG) showed normal sinus rhythm, ruling out arrhythmias as a contributing factor. The patient had no significant medical history, including hypertension, diabetes, atrial fibrillation, or hypercholesterolemia, which are common risk factors for ischaemic stroke. There was also no prior history of Transient Ischaemic Attacks (TIAs), thrombotic events, head trauma, or other potential causes such as infections or metabolic derangements. These findings ruled out alternative diagnoses such as traumatic brain injury, seizures, sepsis, or metabolic dysfunction.

An urgent non contrast CT scan of the brain was performed as the initial imaging modality. Despite a high clinical suspicion of ischaemic stroke, the CT scan revealed no acute abnormalities. The choice of CT as the first imaging technique was due to its accessibility and rapid acquisition time, a standard approach in acute stroke protocols to exclude haemorrhage. However, the lack of significant findings on CT delayed a definitive diagnosis, highlighting its limitations in identifying subtle posterior circulation strokes, such as those caused by AOP occlusion. On the second day, an MRI of the brain revealed acute infarction in the bilateral thalami and the left midbrain, characterised by hyperintensity on T2-weighted and FLAIR sequences [Table/Fig-2], restricted diffusion on Diffusion-Weighted Imaging (DWI), and a corresponding low Apparent Diffusion Coefficient (ADC) [Table/Fig-3], confirming the diagnosis of AOP infarction.



[Table/Fig-1]: Pupil dilatation and left eye deviation.
[Table/Fig-2]: Flair hyperintense signals in bilateral thalamus.



[Table/Fig-3]: Diffusion restriction in DWI-ADC in bilateral thalamus.

The management of the patient focused on supportive care and the prevention of further ischaemic events. As the patient arrived outside the therapeutic window for thrombolysis (4.5 hours from symptom onset), thrombolytic therapy was not initiated. Instead, antiplatelet therapy with aspirin was started to minimise the risk of subsequent thrombotic events. The patient was closely monitored in the intensive care unit for haemodynamic stability and potential complications, such as cerebral oedema or secondary haemorrhage. Hydration and nutritional needs were addressed with intravenous fluids and enteral feeding as necessary. Rehabilitation began once the patient's condition stabilised, focusing on physical therapy to address motor deficits and occupational therapy to enhance functional independence. Regular follow-ups were scheduled to evaluate neurological recovery and assess for the recurrence of ischaemic events. Over time, the patient demonstrated gradual improvement in neurological status, with partial recovery of motor function. Medications for secondary prevention, including antiplatelets and statins, were continued to reduce the likelihood of future strokes. This case highlights the importance of individualised care and a multidisciplinary approach in managing rare ischaemic stroke syndromes like AOP infarction.

DISCUSSION

The AOP infarction is a rare subtype of ischaemic stroke resulting from the occlusion of a single arterial trunk that supplies the bilateral paramedian thalami and, occasionally, the rostral midbrain. Early recognition is crucial for initiating timely therapeutic interventions, such as thrombolysis, within the narrow therapeutic window, thereby potentially preventing persistent neurological deficits, such as altered mental status, memory impairment and vertical gaze palsy [1-3].

Anatomical variations in the blood supply to the thalamus and midbrain have been classified into four types [4]. Type I, the most common, involves the bilateral paramedian arteries arising from each PCA. Type IIa refers to a single paramedian artery arising from the P1 segment of one PCA. Type IIb, which includes the AOP, describes a solitary trunk originating from one PCA that bifurcates to supply the bilateral thalami and midbrain. Type III features a communicating artery between the two PCA's P1 segments, giving rise to the paramedian arteries [5].

The clinical manifestations of AOP infarction are typically linked to the area of the brain affected by the stroke. The classic triad includes altered mental status (ranging from confusion to coma), vertical gaze palsy and memory impairment. Other symptoms may include severe cognitive dysfunction, amnesia, apathy, inappropriate social behaviour, and emotional changes such as depression. When the anterior thalamus is involved, patients often experience significant memory deficits. Midbrain infarctions can result in a wide range of motor and sensory deficits, including oculomotor disturbances, dysarthria, aphasia, hemiplegia, cerebellar ataxia and movement disorders [5].

A 2022 review examined the neuropsychological outcomes of patients with AOP infarction over a one-year period, emphasising the importance of early recognition and management to mitigate cognitive impairments [6]. Differentiating AOP infarction from other ischaemic strokes is essential due to its unique presentation. Unlike lacunar or cortical strokes, which typically present with focal deficits, AOP infarctions often manifest as bilateral thalamic involvement, leading to symptoms such as decreased levels of consciousness, memory disturbances and vertical gaze palsy. This bilateral and often symmetrical presentation contrasts with the typically unilateral findings seen in other stroke subtypes [7].

Pathophysiologically, AOP infarction results from the occlusion of a solitary arterial trunk arising from the posterior cerebral artery, which supplies both paramedian thalami and sometimes the rostral midbrain. This unique vascular anatomy means that an occlusion can lead to bilateral infarction, presenting with a constellation of symptoms, including altered consciousness, cognitive deficits and ocular movement disturbances [7,8].

Regarding management, the mainstay of treatment for AOP infarction is thrombolytic therapy, ideally administered within the therapeutic window of 4.5 hours. This underscores the importance of rapid diagnosis in order to maximise the efficacy of thrombolytic therapy. Thrombolytic treatment has been shown to result in good outcomes when administered promptly. Current imaging modalities, such as MRI with DWI, are the gold standard for detecting small vessel infarctions like AOP. However, limitations persist, particularly in the acute setting, where early changes may be subtle or overlooked. A 2022 study highlighted that head CT was often non diagnostic in the early stages, whereas MRI demonstrated bilateral thalamic infarcts in all cases, underscoring the need for high clinical suspicion and appropriate imaging techniques [9,10].

AOP infarctions can be difficult to diagnose due to subtle or absent findings on initial CT imaging, potentially delaying critical interventions like thrombolysis. Clinicians should maintain a high index of suspicion in patients presenting with sudden neurological deterioration, altered mental status, or eye movement abnormalities. Non contrast CT should be the first step to exclude haemorrhage, but if findings are inconclusive and clinical suspicion remains high, MRI with DWI should be performed promptly to confirm the diagnosis and guide management. Comparative analyses with larger case series have provided deeper insights into AOP infarction. A comprehensive review of 37 patients detailed the imaging patterns and clinical spectrum of AOP infarction, aiding in better recognition and differentiation from other stroke types [11].

CONCLUSION(S)

The AOP infarction, though rare, is a significant clinical condition that warrants early detection and management to improve patient outcomes. Healthcare providers should be vigilant in evaluating patients who present with symptoms such as sudden confusion, impaired consciousness, vertical gaze palsy, or bilateral neurological deficits. These features, particularly when subtle, necessitate advanced imaging, such as MRI with DWI, for accurate diagnosis, as conventional CT scans are often inadequate in identifying early ischaemic changes in the deep brain regions.

Management of AOP infarction depends on early intervention, including thrombolytic therapy within the recommended timeframe. This approach has been associated with favourable outcomes in cases where timely diagnosis and treatment are achieved. Beyond acute management, tailored rehabilitation programs focusing on cognitive and motor recovery are essential, as memory deficits and motor dysfunction often persist even after initial treatment.

Current literature highlights variability in outcomes, with some patients recovering significantly while others face long-term impairments. This variability underscores the need for further studies to understand prognostic factors and optimise management strategies. Future research should explore the pathophysiological mechanisms underlying AOP infarction, refine imaging protocols to enhance early detection and investigate novel therapeutic approaches for cases presenting beyond the thrombolytic window. By fostering clinical awareness and addressing existing knowledge gaps, the potential for improved diagnostic accuracy and patient outcomes in AOP infarction can be realised.

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PARTICULARS OF CONTRIBUTORS:
1. Professor and Head, Department of General Medicine, Dr. D. Y. Patil Medical College, Hospital and Research Centre, Pimpri, Pune, Maharashtra, India.
2. Resident, Department of General Medicine, Dr. D. Y. Patil Medical College, Hospital and Research Centre, Pimpri, Pune, Maharashtra, India.
3. Resident, Department of General Medicine, Dr. D. Y. Patil Medical College, Hospital and Research Centre, Pimpri, Pune, Maharashtra, India.
4. Resident, Department of General Medicine, Dr. D. Y. Patil Medical College, Hospital and Research Centre, Pimpri, Pune, Maharashtra, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Rishikesh Sunilsingh Chauhan,
PG Boys Hostel, Dr. D. Y. Patil Medical College, Pune, Maharashtra, India.
E-mail: rishikesh.s.19@gmail.com

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