CASE SERIES

Artery of Percheron Infarct: A Case Series

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Abstract

The artery of Percheron (AOP), which is a rare anatomical variant, is characterized by a solitary common arterial trunk arising from the posterior cerebral artery (PCA). Occlusion of this artery is rare and may lead to a bilateral paramedian thalamic infarction with or without midbrain involvement. The AOP is a rare anatomical variant that is present in 4–12% of the population. The AOP stroke represents 0.1–2.0% of ischemic strokes and 4–18% of thalamic infarcts. This case series describes the varied clinical manifestations and the imaging findings of AOP infarct. The symptoms noted in the series are altered mental state, hypersomnolence, vertical diplopia, memory disturbances, hemiparesis, and ataxia. The magnetic resonance imaging (MRI) findings showed bilateral paramedian thalamic infarct with or without the involvement of the midbrain. The complexity and polymorphism of AOP stroke semiology explain why bilateral thalamic infarction is often misdiagnosed, lately detected, or even not detected. It is a real diagnostic challenge for clinicians to detect this condition in a timely fashion. Its diagnosis and treatment may be delayed because of the wide spectrum of its clinical features.

Keywords: Diplopia, Hypersomnolence, Paramedian thalamus, Posterior cerebral artery, Skew deviation, Thalamogeniculate arteries, Thalamopeduncular syndrome, The artery of Percheron, Top of basilar syndrome.

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Introduction

The AOP is an anatomical variant where a single common artery arises from the proximal PCA. If it is occluded, it will lead to infarction in the bilateral thalamus–paramedian region, with or without the involvement of the midbrain. It is very rare and present in 4–12% of people. The prevalence of bilateral thalamic infarction caused by AOP occlusion is unknown since it is often misdiagnosed. It constitutes 0.1–2.0% of the total ischemic strokes and approximately 4–18% of thalamic strokes.[1] In this case series, we elucidate the varied clinical manifestations and imaging findings in AOP stroke.

Case Description

Case 1

A 46-year-old male, who was with no comorbidity, came with a history of sudden onset giddiness followed by unresponsiveness. On admission, he was found to be stuporous. Two days later, his sensorium gradually improved but continued to be in a state of hypersomnolence. On examination, he had right hemiparesis, restriction of vertical gaze—both upgaze and downgaze—left eye ptosis, and dilated pupil on the left side. He also had impairment of recent memory. The MRI brain showed well-defined minimally enhancing T2/fluid-attenuated inversion recovery (FLAIR) hyperintensities with diffusion restriction in the bilateral paramedian thalamus (Fig. 1A), subthalamic region, left rostral midbrain (Fig. 1B) suggestive of an AOP infarct. Hypersomnolence improved after a week, whereas memory impairment and vertical gaze palsy persisted for 3 weeks.

Case 2

A 46-year-old male, who was diabetic for 5 years, presented with acute onset giddiness and diplopia which was binocular and vertical. He did not have an altered mental state, limb weakness, sensory disturbances, or memory impairment. His examination revealed partial ptosis of the right eye, vertical gaze restriction, and skew deviation. The MRI brain showed an acute infarct in the bilateral medial thalami (Fig. 2A) and the right side of the upper midbrain (Fig. 2B) suggestive of an AOP infarct. He was treated with antiplatelets and oral hypoglycemic drugs and his ocular disturbances resolved after a period of 2 weeks.

Case 3

A 45-year-old male, who was a hypertensive for 10 years, came with history of excessive sleepiness in the last 4 days. It was sudden in onset; 1 day later, he also noticed binocular vertical diplopia. However, there was no sensory disturbances or weakness. On admission, he was drowsy and hypersomnolent. He had skew deviation with hypertropia in right eye and hypotropia in left eye. He also had left eye ptosis and anisocoria – left pupil 6 mm and right pupil 3 mm. There were no motor or sensory deficits. The MRI brain showed T2/FLAIR hyperintensity with diffusion restriction in the bilateral paramedian posteromedial thalamus and left anterior part of the midbrain (Fig. 3) suggestive of an AOP infarct. His vertical gaze palsy and skew deviation improved completely in 3 weeks, but he had persistent left eye ptosis.

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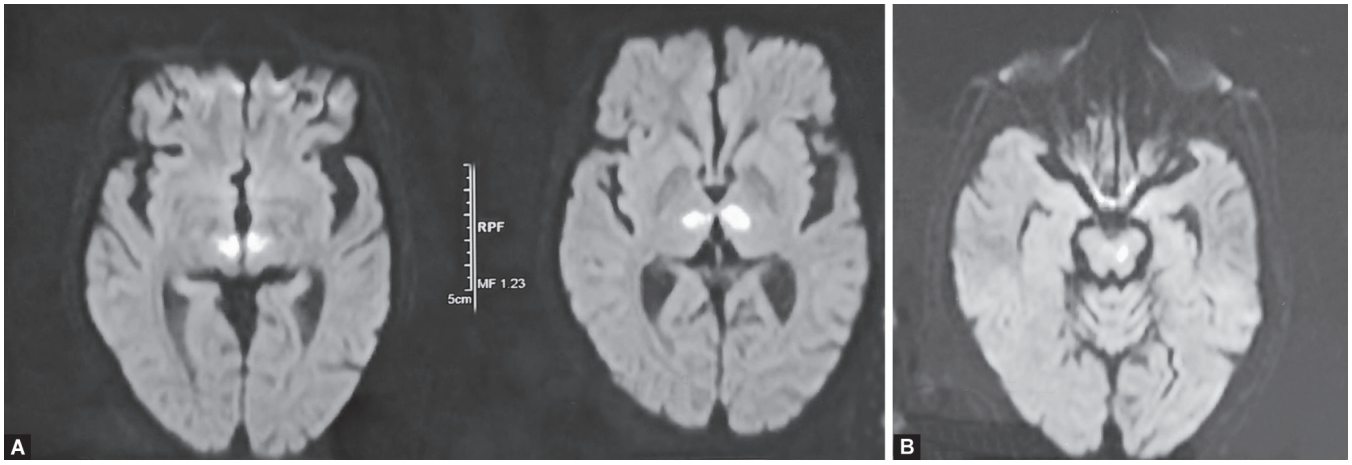
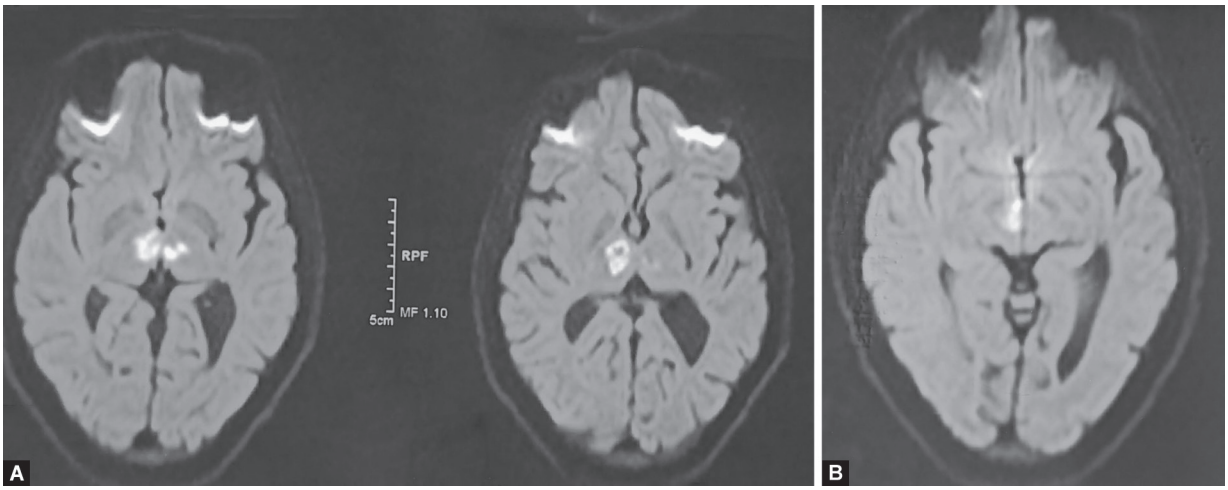
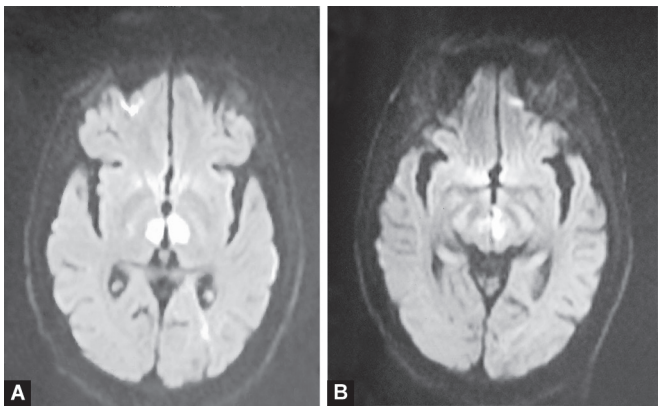


Figs 1A and B: (A) Acute infarct in bilateral paramedian thalamus; (B) Acute infarct in left rostral midbrain



Figs 2A and B: (A) Acute infarct in bilateral medial thalami; (B) Acute infarct in right upper midbrain



Figs 3A and B: Acute infarct in (A) bilateral paramedian posteromedial thalamus and (B) left anterior part of midbrain

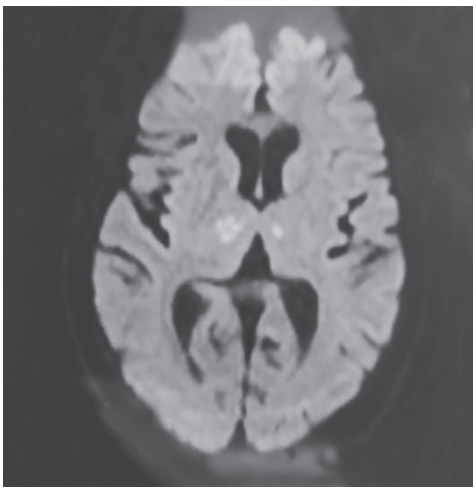


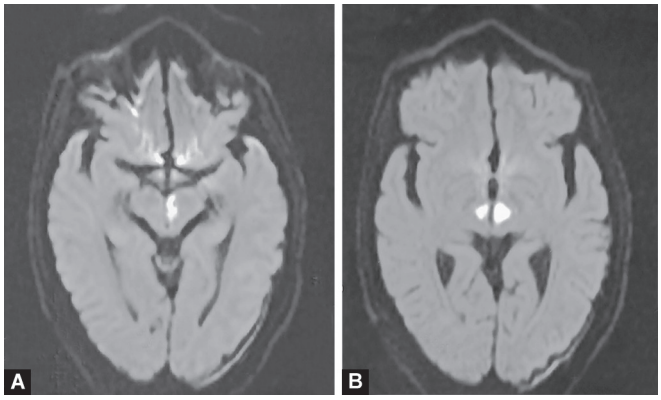
Fig. 4: Acute infarct in the paramedian bilateral thalamus

Case 4

A 60-year-old female with no comorbidity, presented with sudden giddiness followed by memory disturbances in the form of difficulty recollecting recent conversations, food consumed that day. There was no diplopia, limb weakness, sensory disturbances or altered mental state. Her neurological examination was normal except for impaired recent memory. She also had confabulations. The MRI brain showed subtle foci of diffusion restriction in the paramedian bilateral thalamus—possibly AOP infarct (Fig. 4). It took almost a month for the recent memory to improve completely.

Case 5

A 55-year-old male, who was a hypertensive for 8 years, came with sudden onset giddiness followed by transient loss of consciousness which lasted for around 4 hours. Once he regained consciousness, he complained of weakness in the right upper and lower limbs. He also had vertical diplopia. His neurological examination revealed hemiparesis and hemiataxia on right side, vertical gaze palsy, skew deviation, and impairment of recent memory. The MRI brain revealed an acute infarct in the medial aspect of the bilateral thalamus and left hemi-midbrain-AOP infarct (Fig. 5). At discharge, hemiataxia and vertical gaze improved partially and the patient lost follow-up after that.



Figs 5A and B: Acute infarct in the medial aspect of bilateral thalamus and left hemi-midbrain

Case 6

A 35-year-old male with no comorbidity was admitted with sudden onset loss of consciousness. On admission, he was stuporous and no paucity of limb movements noted. There was no papillary asymmetry. Two days after admission, he became conscious, but was hypersomnolent most of the times. Examination at this point revealed no neurological abnormality. The MRI brain showed acute infarct in bilateral paramedian thalami (Fig. 6). He was discharged 5 days later with no neurological deficit and without any altered mental state.

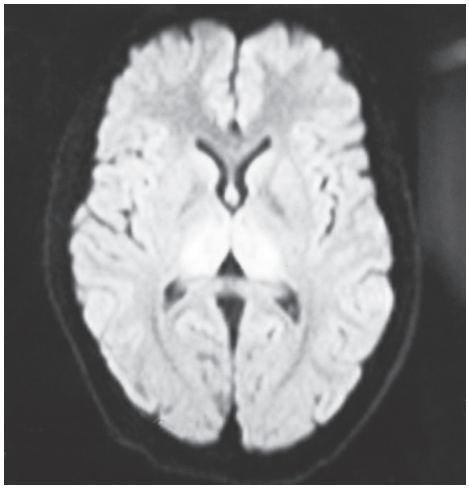


Fig. 6: Acute infarct in bilateral paramedian thalami

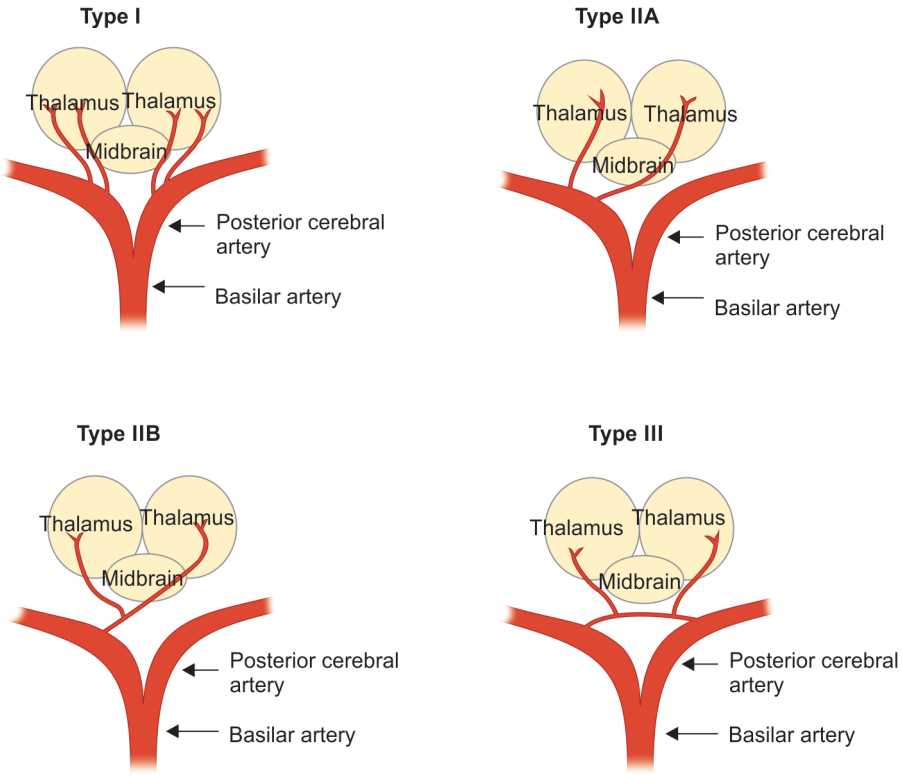


Fig. 7: Variants of blood supply to thalamus and midbrain

Discussion

The perforating branches from the posterior cerebral and posterior communicating arteries supply blood to the thalamus and the midbrain. There are four vascular territories: Anterior, paramedian, posterior, and inferolateral. The anterior vascular region is nourished by the polar artery also known as the thalamotuberal artery. It originates from the posterior communicating artery. The thalamogeniculate artery, which comes from the PCA (P2 segment), supplies the inferolateral region Blood supply to the posterior territory comes from the posterior choroidal arteries, which also come from the P2 segment of the PCA. Finally, the paramedian parts of the thalamus are supplied by perforating arteries, also known as paramedian arteries. It has four normal variants (Fig. 7). In variant I, which is the most common type, the perforating branches originate from the left and right-sided PCA separately. In variant IIA, the left P1 segment gives rise to both paramedian arteries. The perforating artery comes from the AOP in variant IIB, where the AOP originates from the P1 segment of PCA. Variant III is also called the arcade variant, where smaller perforating branches come from an arterial arc, which bridges the PCA and P1 segments.[2]

Four characteristic variants of AOP infarct are identified in 43%, there will be involvement of bilateral paramedian thalamus along with rostral midbrain. Bilateral paramedian thalamus without the involvement of midbrain is noted in 38%. Anterior and bilateral paramedian thalamus with midbrain involvement, and Anterior and bilateral paramedian thalamus without midbrain is seen in 14 and 5% cases, respectively.

The most frequent clinical presentations of AOP stroke include vertical gaze palsies memory disturbances and coma. Other presentations that are reported include excess sleepiness, akinetic mutism, and behavioral disturbances such as apathy and agitation. The altered sensorium in AOP stroke can vary from hypersomnolence to coma. Thalamus has an essential role in regulating sleep and arousal maintenance. Hypersomnolence is caused due to the interruption of dopaminergic and noradrenergic discharges from the reticular activating system (RAS) to the thalamus.[3]

Vertical gaze restriction is caused due to midbrain involvement. However, it is also seen in patients without midbrain lesions. Here it is due to a disconnection in the fibers which goes through the thalamus to the rostral interstitial part of the medial longitudinal fasciculus (riMLF). Severe memory impairment is seen in strokes involving polar or anterior territory. Various thalamic structures play a crucial role in memory: the mammillothalamic tract, dorsomedial and anterior nucleus. The mammillothalamic tract and dorsomedial nucleus come under the paramedian territory; the anterior nucleus comes under the polar territory.[3]

Additional involvement of the midbrain will cause "mesencephalothalamic" or "thalamopeduncular" syndrome. Apart from vertical gaze restriction, altered sensorium, and memory disturbance, the syndrome has oculomotor disturbances, hemiparesis, cerebellar ataxia, and movement disorders. They are due to the involvement of the structures like the interpeduncular nucleus, superior cerebellar peduncle, red nucleus, oculomotor nucleus, and periaqueductal gray matter.[2]

Apart from the AOP infarct, the bilateral thalamic infarct is also seen in deep venous sinuses thrombosis and top of the basilar syndrome. In thrombosis of deep veins, the clinical features are usually different from an arterial infarct, and it also involves other structures. In ''top of the basilar syndrome,'' infarcts will also involve the pons, cerebellum, and occipital lobes, in addition to bilateral thalami.[4] Other causes of bilateral thalamic lesions include toxins like carbon monoxide, cyanide, and methanol, metabolic disorders such as Osmotic demyelination syndrome (ODS), Wernicke's encephalopathy, Wilson's disease, hypoxic–ischemic encephalopathy, neurodegenerative diseases such as Fahr disease and Creutzfeldt–Jakob disease), infections [e.g., human immunodeficiency virus (HIV), flavivirus, and toxoplasmosis].

Conclusion

There is often misdiagnosis and underdiagnosis in AOP infarction due to its complexity and polymorphic clinical presentation. Hence, there is a real diagnostic challenge for physicians in detecting this condition. The treatment may also be delayed because of this. The initial computed tomography (CT) scan will be normal in the majority of cases. Hence, MRI is the diagnostic modality of choice, when an AOP stroke is suspected. When elderly persons present with altered sensorium, the differential diagnosis should also include an AOP infarct.[4] An AOP infarct also poses a neurodiagnostic challenge since the small vessels are difficult to be picked up on digital angiography.

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