Letter to the Editor

Magnetic resonance angiography validation of bilateral thalamic infarction induced by artery of Percheron occlusion: a case description

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Introduction

The artery of Percheron (AOP) is a rare anatomic variant originating from the P1 segment of the posterior cerebral artery (PCA), which nourishes the thalami and may also supply the midbrain through vascular variations (1). Occlusion of the AOP may lead to bilateral thalamic infarction (AOP infarction) and be accompanied by midbrain dysfunction (2). There are scarce reports of this particular clinical condition (3). Considering the complex structure and function of the thalamus, AOP infarction is accompanied by complex clinical manifestations, with its diagnosis relying on imaging examinations. However, the size of the AOP is extremely small and difficult to identify using conventional angiography (4). Over the past 20 years, only a few reports have been published on the confirmation of the AOP with digital subtraction angiography (4-11), and cases of AOP detection by computed tomography angiography (CTA) (12,13) or magnetic resonance angiography (MRA) (8,14,15) are even rarer (Table S1).

Here, were report a case of AOP infarction validated by MRA. Our aim is to expand the knowledge of clinical neurologists and radiologists of AOP, as well as to reduce the rate of misdiagnosis. We also conducted a literature review focusing on the anatomical and imaging characteristics of AOP.

This study was approved by the Research Ethics Committee of the Second Hospital of Hebei Medical University. All procedures in this study were performed in accordance with the ethical standards of the institutional and/or national research committee(s) and the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patient for the publication of this case report and accompanying images.

Case presentation

A 51-year-old man was admitted to a medical institute, having been unconscious for 5.5 hours prior to admission for no obvious reason. The patient did not exhibit sudden limb twitching, foaming at the mouth, or incontinence although there were involuntary movements of all extremities. Approximately 2 hours after admission, the patient gradually recovered consciousness and was able to communicate with his family and walk with assistance. The patient was subsequently transferred to the emergency room of the Second Hospital of Hebei Medical University.

Head CT scans performed 3 hours after the onset of unconsciousness showed suspicious low-density foci in the left thalamus (*Figure 1A*). Approximately 5 hours after admission, the patient fell into a persistent coma again with the same clinical manifestations as described above and was then admitted to the Neurology Department for diagnosis and treatment, as described below.

The patient had no medical history of diabetes,

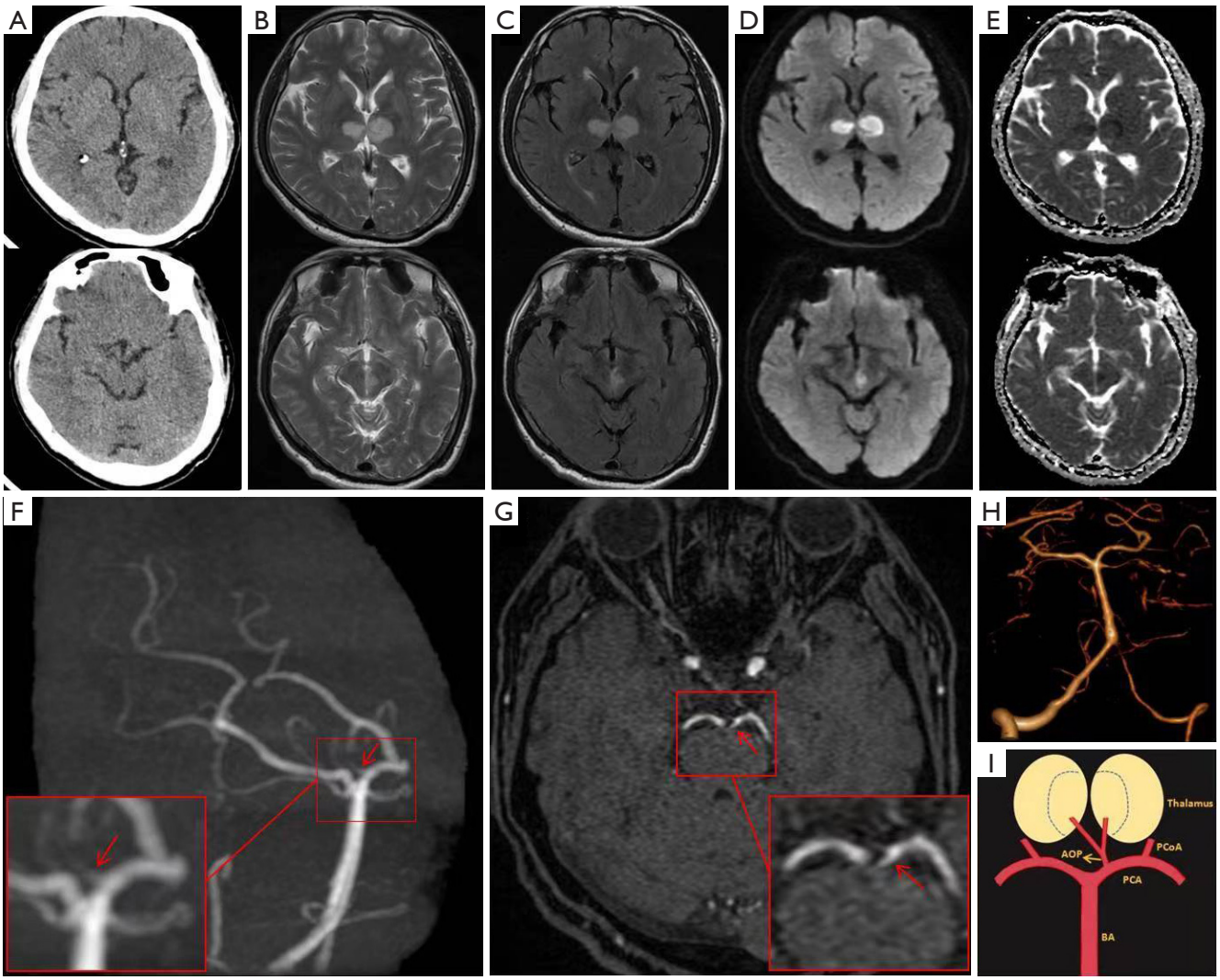

Figure 1 Brain imaging of the patient and schematic diagram of the AOP. (A) Head computed tomography showing cross-sectional images of the thalamus and midbrain. (B-E) Head MRI cross-sectional images of the thalamus and midbrain: T2-weighted images (B), fluid attenuated inversion recovery (C), DWI (D), and ADC (E) sequences. There is a high abnormal signal in the bilateral thalamus and left midbrain on DWI, but the corresponding ADC image has a low signal, indicating limited diffusion. (F,G) Magnetic resonance angiography image and raw data. Red arrows indicate the AOP. (H) Head computed tomography angiography. (I) Schematic diagram of the AOP. DWI, diffusion-weighted imaging; ADC, apparent diffusion coefficient; AOP, artery of Percheron; MRI, magnetic resonance imaging; BA, basilar artery; PCA, posterior cerebral artery; PCoA, posterior communicating artery.

hypertension, or coronary heart disease and denied a history of substance abuse or exposure to specific toxicants. Physical examination showed no abnormalities in the chest or abdomen. Neurological physical examinations showed that the patient was in a coma. The left and right pupil diameters were 3.5 and 1.5 mm, respectively; a bilateral pupil light reflex was present; the nasolabial fold and head wrinkle were symmetrical; and the eyes had been forced into an abduction position. Muscle tension was normal, but there was a lack of voluntary muscle control. The tendon reflex was active, the Babinski sign was positive, and the patient had a Glasgow Coma Scale (GSC) score of 6.

Magnetic resonance imaging (MRI) performed 2 days after the onset of unconsciousness showed an abnormal signal on the left side of the midbrain. There was a high signal in the bilateral paramedian thalami on diffusion-weighted

imaging (DWI), with a low signal in the corresponding apparent diffusion coefficient (ADC) image, indicating acute or subacute infarction (*Figure 1B-1E*). MRA showed a small artery branching from segment P1 of the left PCA with severe narrowing of the proximal lumen, which was considered to indicate severe stenosis or occlusion of the AOP (*Figure 1F,1G*). MRA also showed a slight decrease in the signal in the intracranial segment of the left vertebral artery. CTA completed 10 hours after the onset of unconsciousness showed basilar artery fenestration and severe luminal stenosis or occlusion of the left vertebral artery (*Figure 1H*). Transthoracic echocardiography showed no intracardiac thrombosis, and the left ventricle ejection fraction was 62.03%. Lower extremity venous ultrasonography showed no deep vein thrombosis, and a chest CT scan showed no space-occupying lesion. The results of all routine laboratory tests were within the normal range. In addition, the results of 24-hour Holter monitoring were normal.

A transcranial Doppler test with bubble study (TCD-b) was performed, with the results indicating a right-to-left shunt, which suggested the possible presence of patent foramen ovale (PFO), possibly leading to cardioembolism.

In terms of treatment, the patient was administered anticoagulants (low molecular weight heparin, subcutaneous injection), antiplatelets (aspirin), and statins (atorvastatin) and received other supportive treatments such as eureklin to improve vascular circulation and edaravone to scavenge free radicals. The patient was gradually awakened 24 hours after admission, and14 days later, he was discharged from hospital. At the time of discharge, the patient was conscious and was experiencing diplopia. His left eye was still in the forced abduction position, his right eye was in a centered position, and pupil diameter was symmetrical. There was no limb weakness or paresthesia, and the patient had a GSC score of 15.

At the 6-month follow-up, PFO was diagnosed by transesophageal echocardiography (TEE) performed at another hospital. The results did not suggest high-risk PFO, so no surgical treatment was performed. The patient continued to experience intermittent diplopia.

Discussion

Anatomy of AOP infarction

Typically, at least 1 of the 4 major vascular regions is involved in thalamic infarctions: posterolateral, anterior, paramedial, and/or dorsal (16). Of these, the paramedial part of the thalamus is primarily supplied by the thalamic paramedian artery, which bifurcates from segment P1 of the PCA and has 4 anatomic variations (1). Of these variations, Type IIb is a bilateral thalamic paramedian artery that originates from a unilateral P1 segment of the PCA, namely the AOP (*Figure 1I*). It is a very rare anatomic variant, with an incidence of 4–12% (17). In addition to supplying the paramedial part of the thalamus, the AOP may also (rarely) supply other parts of the brain. Several researchers have reported that the thalamic paramedian artery and the midbrain upper paramedial artery could originate from the same artery and that AOP infarction could lead to a distinctive pattern of ischemia, namely bilateral paramedian thalamic infarction with or without midbrain involvement (5). When the thalamotuberal artery is congenitally absent, the AOP is responsible for supplying blood to the anterior thalamus. Therefore, AOP occlusion will affect the paramedial part of the thalamus, the anterior part of the thalamus, and a part of the midbrain (5,18).

Clinical manifestations of AOP infarction

The complex structure and function of the AOP, combined with the highly varied artery anatomy, may lead to considerable variations in the clinical manifestations of AOP infarction, depending on the type of AOP blood supply (4). Monet *et al.* (19) summarized 4 main clinical manifestations of AOP infarction: vertical gaze palsy (65%), memory impairment (58%), confusion (53%), and coma (42%). An AOP-related coma manifests with extreme volatility, with patients experiencing disorders of consciousness that typically last for several hours or days. As the edema of the ischemic brain tissue decreases and the collateral circulation is gradually established, the disorders of consciousness are correspondingly alleviated (18).

Imaging characteristics of AOP infarction

For the early diagnosis of AOP infarction, DWI and fluid-attenuated inversion-recovery (FLAIR) sequences should be obtained on MRI. There could be hyperintensities within bilateral paramedial parts of the thalami (with or without midbrain involvement). In cases of impairments in the bilateral midbrain, 67% of patients show a "v" sign, which could be considered a characteristic sign (4,20). Although initial head CT or MRI results could be negative, a diagnosis of AOP infarction should not be excluded (21).

There have been only 3 case reports (4-6) since Roitberg

et al. (7) first diagnosed AOP by digital subtraction angiography. Godani *et al.* (22) reported a patient who had an obvious filling defect on CTA in the anterior part of segment P1 (which may be the origin of the AOP). CTA subsequently showed complete recanalization of the anterior part of segment P1 after the patient's manifestations had resolved, indirectly demonstrating the existence of AOP (22).

A new MRI sequence with relatively short scan times, self-calibrating echo-planar imaging perfusion-weighted imaging, has recently been proposed for the detection of infarct cores and ischemic penumbra (23). In the future, this technique may provide more immediate and accurate information for the diagnosis of AOP infarction.

Etiology of AOP infarction

To date, the etiology of AOP occlusion remains unknown, but most researchers believe that AOP infarction is caused by embolism or thrombosis; indeed, embolism, caused by cardiogenic, arthritogenic, and unknown etiologies, accounts for most cases of AOP infarction (16). Aaron *et al.* (24) and Perren *et al.* (18) found PFO to be involved in 1 of 4 and in 5 of 9 patients with cardioembolism, respectively. Therefore, in our patient, in whom AOP infarction with cardioembolism was highly suspected, TEE was critical to an early diagnosis (24). In addition, recent studies have reported that quantitative susceptibility mapping of thrombi based on susceptibility-weighted imaging sequences can help discriminate between cardioembolism and other stroke subtypes (25), which may prove useful in determining the etiology of AOP infarction.

In the present case, the patient drifted in and out of unconsciousness, and except for eye signs, there were no obvious focal neurologic deficits. The patient had no common risk factors for stroke, and laboratory examinations showed no obvious abnormalities. Bilateral paramedial thalamus and midbrain hyperintensities were apparent on MRI, and thus acute brain infarction was considered. MRA revealed the AOP branching from the P1 segment of the left PCA, and TCD-b, and TEE demonstrated PFO. Thus, the patient was diagnosed with AOP infarction.

Conclusions

AOP is a rare anatomic variant. AOP infarction is clinically rare, and the manifestations of the disease are complex and variable, lending it to misdiagnosis. Brain imaging is the main diagnostic method, but there is a low positive rate with head CT in the early stages. Thus, practitioners should use FLAIR and DWI sequences on MRI to facilitate early diagnosis. Although it is difficult to identify AOP, when AOP infarction is highly suspected, further angiography is necessary.

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Footnote

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://qims.amegroups.com/article/view/10.21037/qims-22-389/coif). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. The study was approved by the Research Ethics Committee of the Second Hospital of Hebei Medical University. All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patient for the publication of this case report and accompanying images.

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Supplementary

Table S1 Case reports of AOP discovered by different vascular imaging techniques in the past 20 years

Year of publication	Author	Journal	Vascular imaging	Descriptions
2021	Naldi A, *et al.*(8)	Neurol Sci	MRA	a single perforator originates from the proximal P1 segment of the left PCA.
			DSA	confirm the MRI evidence of a single dominant perforating artery compatible with AOP originating from the proximal P1 segment of the left PCA
2021	Kheiralla OAM, *et al.*(14)	Radiol Case Rep	MRA	an abnormal tiny vessel arising from the P1 segment of the left PCA, consistent with AOP
2019	Ranasinghe T, *et al.*(12)	Case Rep Emerg Med	CTA	demonstrated an AOP arising from the right PCA
2018	Lin PC, *et al.*(9)	Radiol Case Rep	DSA	contrast accumulation in a small branch of the right PCA, AOP.
2016	Lee HY, *et al.*(13)	Ann Rehabil Med	CTA	an unpaired thalamic perforating artery arising from the proximal P1 segment of the left PCA supplying the bilateral thalami.
2015	Jiménez Gómez E, *et al.*(10)	Neurologia	DSA	Recanalización del segmento P1 izquierdo con repleción de una arteria de Percheron
2013	Rodriguez EG, *et al.*(15)	Radiol Case Rep	MRA	a single branching vessel originating from the P1 segment of the right PCA which appears to supply both thalami
2012	Cao W, *et al.*(11)	Acta Radiol Short Rep	DSA	a stenotic AOP originating from the left P1 segment
2010	Lazzaro NA, *et al.*(4)	AJNR Am J Neuroradiol	DSA	a large unpaired thalamic perforating artery arising from the proximal P1 segment supplying the bilateral thalami (ie, an AOP).
2007	Kostanian V, *et al.*(6)	AJNR Am J Neuroradiol	DSA	normal visualization of the full length of the artery of Percheron
2004	Stefan Weidauer , *et al.*(5)	Eur Radiol	DSA	unpaired origin of the PTPA with a diencephalo-mesencephalic common trunc (Percheron type II) arising from the right superior cerebellar artery
2002	B. Z. Roitberg, *et al.*(7)	Acta Neurochir	DSA	a single unpaired central thalamic perforating artery

MRA, magnetic resonance angiography; MRI, magnetic resonance imaging; DSA, digital subtraction angiography; PCA, posterior cerebral artery; AOP, Artery of Percheron; CTA, computed tomographic angiography; PTPA, posterior thalamoperforating artery.

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