



The clinical significance of artery of Percheron in cerebral ischemia: a summary overview

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Abstract

The artery of Percheron (AOP) infarction is a rare vascular condition and a pattern of ischemia where a single arterial trunk supplies blood to the rostral midbrain and paramedian thalamic areas, causing neurological deficits. Due to its rarity and atypical presentation, AOP infarction is frequently overlooked in initial assessments, particularly on computed tomography (CT) scans. In addition, different types of AOP infarction have been identified, and it is shown that there can be four significant distinct types of this disease, which may manifest some different symptoms and have various vascular alterations. Knowledge of these variants in the AOP multisystem disorder is imperative to the diagnosis and treatment of infarctions in the condition, hence improving prognosis. To manage this uncommon but important condition, we need to increase awareness among healthcare providers and carry out more research on diagnosis and treatment.

Keywords: artery of Percheron, ischemia, stroke

Introduction

The artery of Percheron (AOP) infarction is a rare vascular condition and a pattern of ischemia where a single arterial trunk supplies blood to the rostral midbrain and paramedian thalamic areas (Fig. 1), causing neurological deficits. Occlusion of this artery leads to bilateral thalamic infarctions, often presenting nonspecific neurological symptoms such as altered mental status, impaired consciousness, and memory deficits. Due to its rarity and atypical presentation, AOP infarction is frequently overlooked in initial assessments, particularly on computed tomography (CT) scans^[1].

Epidemiological studies suggest that AOP infarctions account for approximately 0.1% to 0.3% of all ischemic strokes and 4% to 18% of thalamic strokes. Misdiagnosis is common, with the condition often being mistaken for tumors or other types of infarcts due to overlapping clinical presentations^[2,3]. The

HIGHLIGHTS

- The artery of Percheron (AOP) infarction is a rare vascular condition and a pattern of ischemia where a single arterial trunk supplies blood to the rostral midbrain and paramedian thalamic areas, causing neurological deficits.
- Knowledge of these variants in the AOP multisystem disorder is imperative to the diagnosis and treatment of infarctions in the condition, hence improving prognosis.
- To manage this uncommon but important condition, we need to increase awareness among healthcare providers and carry out more research on diagnosis and treatment.

condition is often mistaken for tumors or other types of infarcts due to relatively comparable clinical presentations. The presentation of AOP infarctions is characterized by drowsiness, decreased level of consciousness, memory disturbance, and cortical blindness with supranuclear vertical gaze palsy^[4]. Prompt recognition and management are crucial, as delayed treatment can result in significant morbidity or mortality. This review aims to consolidate current knowledge on AOP infarction, focusing on its anatomy, etiology, clinical presentation, diagnostic challenges, treatment strategies, and future research directions.

Epidemiology and etiology

AOP infarction is a relatively rare yet clinically significant condition, representing a small subset of ischemic strokes. Its incidence is estimated to range between 0.1% and 0.3% of all ischemic strokes and constitutes approximately 4% to 18% of all thalamic strokes^[5,6]. This variability in incidence underscores the unique vascular anatomy and the challenges associated with its diagnosis. The AOP is a singular arterial branch arising from the posterior cerebral artery (PCA) and is responsible for supplying the paramedian thalamic and, in some instances, the rostral

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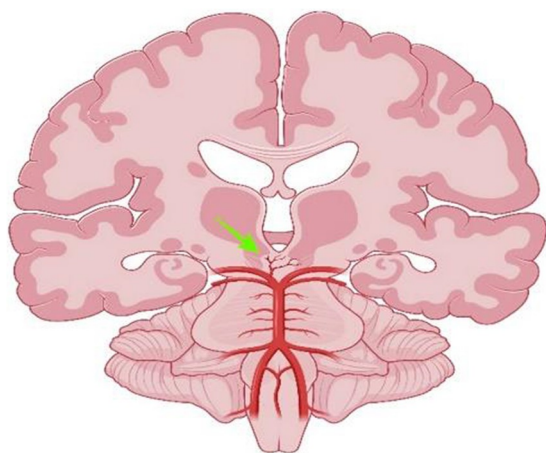


Figure 1. This illustration demonstrates an example of the AOP (green arrow). The image highlights a single arterial trunk branching from the right proximal PCA, which is responsible for supplying blood to the paramedian region of the thalami bilaterally and the rostral part of the midbrain.

midbrain^[6]. The existence of this vascular configuration is a variant, as the thalami are usually supplied by multiple perforating arteries. From a vascular perspective, the AOP is a critical structure because its occlusion can lead to bilateral thalamic infarctions, often with devastating neurological outcomes. Surgical ligation or thrombosis of the AOP results in ischemia in regions critical for consciousness, memory, and motor coordination due to the dual thalamic and midbrain involvement^[6]. Stroke is frequently encountered but can present a diagnostic challenge when presentation is non-specific or a lesion is not identified^[7]. AOP stenosis has a varied and often non-specific presentation, which can lead to delayed diagnosis and treatment of these patients^[7]. It is important that doctors recognize this entity for timely diagnosis and treatment, and for the best prognosis.

The pathophysiology of AOP infarction primarily involves mechanisms such as atherosclerosis, small vessel disease, and embolic events. Embolism formation is often the result of arterial occlusion and is particularly common in patients with a cardiogenic predisposition, including those with atrial fibrillation or other arrhythmogenic disorders. Furthermore, additional risk factors for AOP infarction include a history of cardiovascular disease (CVD), as well as modifiable risk factors such as smoking, diabetes mellitus, hypertension, and hyperlipidemia.

Key variants of AOP infarctions

Different types of AOP infarction have been identified, and it is shown that there can be four significant distinct types of this disease, which may manifest some different symptoms and have various vascular alterations (Fig. 2). Knowledge of these variants in the AOP multisystem disorder is imperative to the diagnosis and treatment of infarctions in the condition, hence improving prognosis^[5,6].

1. *Bilateral paramedian thalamic and rostral midbrain infarction.* The main cause of bilateral paramedian thalamic infarctions was small artery disease (60%), followed by cardioembolism (40%). A well-defined clinical picture is

shown in bilateral paramedian thalamic artery infarcts. These patients had disorders consisting of consciousness, memory dysfunctions, and various types of vertical gaze paresis and psychological changes.

2. *Bilateral paramedian thalamic infarction without midbrain involvement.* This variant occurs in about 38% of cases. The lesion directly impacts the paramedian thalami, yet the development of midbrain arteries remains unchanged^[6,8]. The variant may be observed in changes of mood, cognitive, neurological, and cognitive-shift forms. This is so because the important motor tracts are preserved in the remaining midbrain and as such the prognosis is slightly better than in cases where both thalami and the midbrain are impaired.
3. *Bilateral paramedian and anterior thalamic infarction with midbrain involvement.* Li et al^[8] reported that this variant is present in 14% of all patients. This happens to all the midbrain, paramedian, and anterior thalamic regions. Its features may include prolonged loss of consciousness, memory impairment, changes in behavior, and visual acuity.
4. *Bilateral paramedian and anterior thalamic infarction without midbrain involvement.* This variant is seen in roughly 5% of all cases, which is the least common form. It spreads across the anterior and paramedian subsections and does not involve the midbrain^[8]. It may present as a change in memory, sensory examination and cognitive function which is impaired universally except in areas governed by midbrain oculomotor and motor.

Clinical presentation

Four neurovascular anatomical variants have been described for the thalamus and midbrain^[1]. Type I is the most frequent, where two paramedian arteries arise from the proximal P1 segment of each posterior cerebral artery. In type IIa, both paramedian arteries arise from the P1 segment of either the left or the right posterior cerebral artery. Type IIb refers to the AOP, a single trunk originating unilaterally from one of the (P1 segment) posterior cerebral arteries and bifurcating to supply the paramedian thalamus and bilateral rostral midbrain. In contrast, type III refers to the existence of a communicating artery between the proximal aspect (P1 segments) of both the left and right posterior cerebral arteries, from which the paramedian arteries originate^[2,9]. AOP infarction can be characterized by memory impairment, vertical gaze palsy, and altered mental status^[10]. Pattern recognition is fundamental for early diagnosis and optimal care^[11]. Because of the high rareness of AOP infarctions and nonspecific symptomatology, the disease becomes difficult to diagnose in its early stages^[12]. The most reported variant is Percheron type IIb and supplies the paramedian thalami and the rostral midbrain^[13]. Type IIb occlusion usually causes a bilateral paramedian thalamic stroke, but rostral midbrain and anterior thalamus are involved in 57% and 19% cases. The rarer type III variant prevented the bilateral extension of infarction and involved the territory of tuberothalamic and paramedian perforating arteries^[13–15]. Bilateral infarction can cause a variety of upsets in the mental state varying from confusion to coma as the thalamus entirely controls consciousness and cognition^[9]. Other symptoms associated with the role of the thalamus in sensory tracts are neurologic decline, defects in higher mental functions, motor disability, amnesia, aphonia,

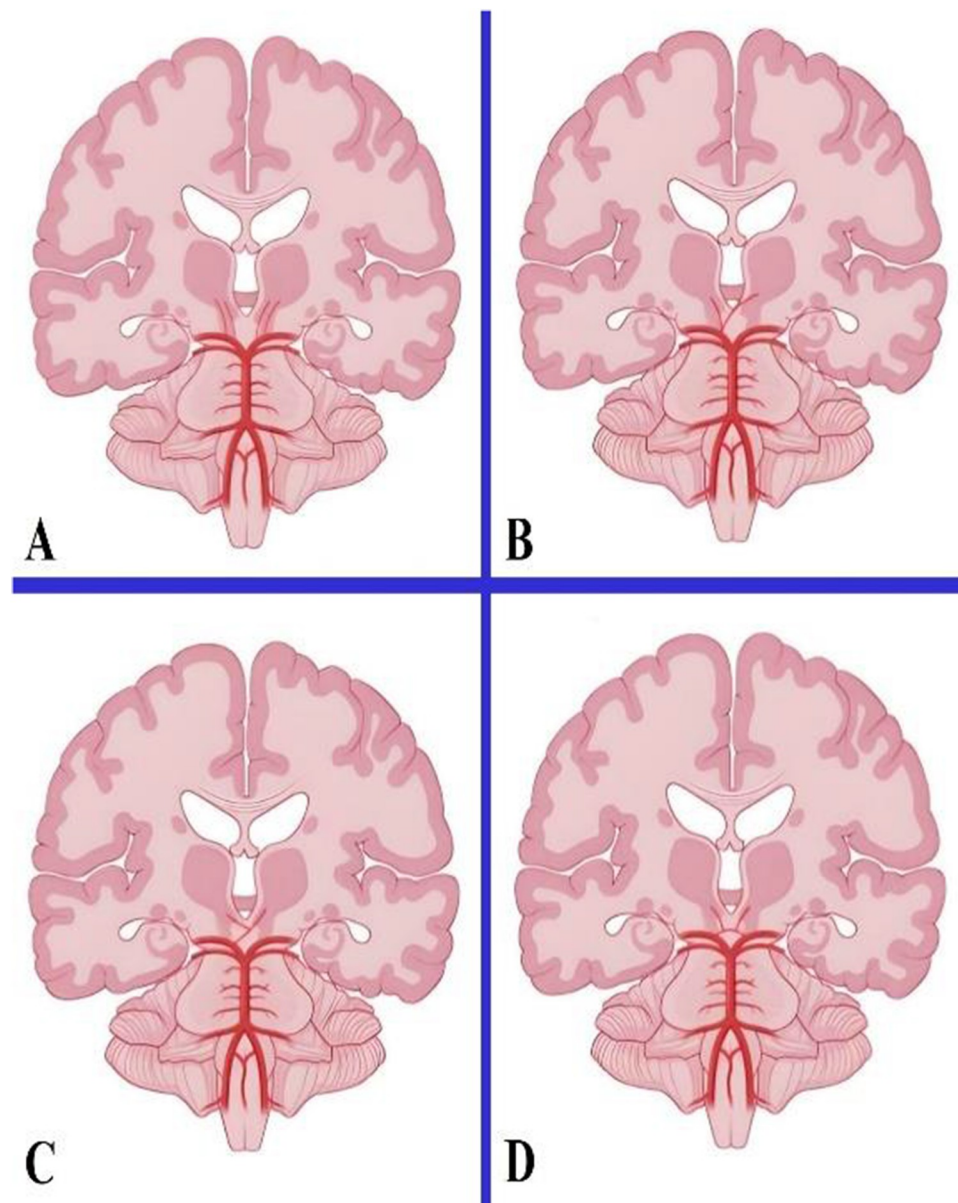


Figure 2. This illustration demonstrates four types of AOP infarction. (A) It shows paramedian arteries stemming from each PCA (type I). (B) It shows paramedian arteries arising from a communicating branch between the PCAs (type IIa). (C) It shows the AOP arises unilaterally from a PCA and gives rise to the paramedian arteries (type IIb). (D) It shows paramedian arteries arising from a communicating branch between the PCAs (type III).

and change in behavior manifested as apathy and failure to comprehend. Bilateral infarction manifested severe hypersomnolence and akinetic mutism with the variability of the extent of the ischemia in patients. Some of the effects of treatment involve patients who may regain their mental state and muscle coordination, while those with high ischemia levels are likely to suffer from permanent cognitive dysfunction^[16]. Most patients with an AOP infarction present with progressive drowsiness, amassing to a coma. However, clinical presentations may vary due to the disparate functions of the implicated structures^[17]. Further described manifestations include vertical gaze palsy and ataxia (commonly seen with midbrain involvement), behavior and memory deficits (often associated with disruptions to the hippocampus or its connections), motor dysfunction, and speech

difficulties^[17–19]. In addition, most of the signs attributed to AOP infarction have similarities to those observed in other neurological disorders, and thus at the early stages of presentation, it proves very difficult to distinguish it from other neurological disorders. Due to their shared risk factors, COVID-19 and stroke are a lethal combination for medical management to treat^[20]. Pantovic *et al*^[21] reported a young adult patient with infarction in the AOP territory as a presenting feature of COVID-19.

Diagnosis

Early diagnosis and proper management of patients are crucial for achieving the best outcomes in those with AOP infarctions. Although AOP infarction results in a characteristic pattern of

ischemia – namely bilateral paramedian thalamic infarct with or without midbrain involvement – it may cause diagnostic difficulties due to the variety of its clinical presentations and wide differentials, as well as its small diameter and the difficulty of obtaining visualization through diagnostic imaging^[22](Fig. 3). Early neuroimaging of AOP infarction and correct diagnosis are mandatory for early initiation of the appropriate treatment and better patient outcomes^[22]. Both aspects can be challenging due to the diverse manifestations in patients and the relatively low incidence of this type of infarction. Nalbantoglu *et al*^[9] and Rodriguez *et al*^[23] suggested using in emergencies a CT scan, commonly known as a CAT scan to evaluate the patient^[7,24,25]. Nevertheless, a CT scan is not particularly helpful in the early stages of a myocardial infarction. The second imaging technique, magnetic resonance imaging (MRI) with diffusion-weighted imaging (DWI), is considered the most effective for clearly identifying the presence or absence of ischemic lesions. If MRI reveals bilateral thalamic lesions, it is more likely that the patient will have an AOP infarction. Additionally, MR angiography can be conducted to examine the arterial supply, providing valuable information about blood flow and vascular structure^[11,7]. The thalamic arteries consist of a group of perforating arteries of the thalamus that originate from the posterior communicating and cerebral arteries, including the thalamogeniculate and medial and lateral posterior choroidal arteries^[26]. As there are several variants of thalamic blood supply, the identification of the potential presence and infarction of an AOP is important in diagnosis and treatment of ischemic strokes affecting the thalami and mesencephalon, especially because of the unusual and variable presentation of these forms of ischemic strokes^[27,28].

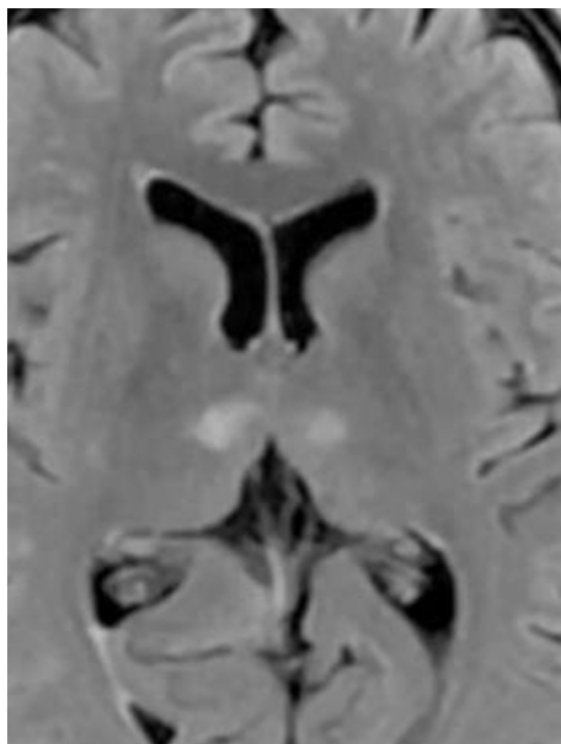


Figure 3. MRI image showing bilateral asymmetrical infarct in the artery of the Percheron area.

Jantre and Howlett^[29] presented a comprehensive visual spectrum of AOP infarction patterns, including bi-thalamic involvement (paramedian thalamic lesions) with or without midbrain involvement. Additionally, the “V” sign, observed on FLAIR and DWI sequences, is characteristic of AOP infarction. Other important vascular causes reported include top of basilar artery syndrome, internal cerebral vein thrombosis, and neoplastic lesions including diffuse midline glioma (DMG) H3 K27-altered.

Treatment and Prognosis

The preliminary management of AOP ischemia is comparable to that of acute ischemic stroke, which is thrombolysis is the primary management strategy for patients diagnosed early^[24]. When performed in the subacute phase of stroke using tissue plasminogen activator (tPA), intravenous thrombolysis increases a patient's outcomes^[30,31]. For patients who come in late, assisting the patient while concentrating on mitigating further complications and managing symptoms is the best approach^[8]. GCS score, size of the infarct, the degree of neurological deficit, and the timing of initiation of treatment are the major determinants of prognosis in a patient^[1]. Even though some patients may still suffer from certain permanent disabilities despite the administration of early thrombolytics or endovascular therapy that could enhance the patient's functional outcomes regarding the nervous system, such patients would be better off than otherwise^[32]. Survivors of patients with AOP infarction often have a prolonged and disabling form of cognitive impairment that remains insufficiently characterized^[33]. Çetin and colleagues^[33] examined the clinical and cognitive features of 10 patients with AOP infarction in the short and long term after stroke, showing that all patients (six with pure bilateral thalamic involvement and four with bilateral thalamic and rostral midbrain involvement) had cognitive impairment, consciousness, and behavioral disorders at stroke onset. In addition, most of the prefrontal cortex cognitive processes including executive functions such as planning and cognitive control, processing speed, and working memory were severely impaired at stroke onset^[33]. Similarly, Chang *et al*^[34] reported that 80% of their patients with AOP infarction had an unfavorable prognosis (mRS ≥ 3) at discharge, indicating that residual symptoms of AOP infarction often persist and affect daily life activity despite medical therapy and rehabilitation.

Future Studies

Research on AOP cannot be overstated as it improves diagnostic and treatment methods and gives us insight into stroke management overall^[34]. Since it is rare and can be mistaken for other neurological disorders, doctors need to be knowledgeable about AOP infarctions including and symptoms, especially in emergencies. For example, we need more research on advanced imaging techniques to detect AOP early so we can intervene timely and improve patient outcomes. Patients with bilateral thalamic infarctions need more specific treatment guidelines as existing ones are too general. The use of augmented reality for the study of intracranial vessels and their anatomical variants, as well as the use of 3D models, can improve knowledge and therapeutic strategies^[35–38]. To refine these guidelines, we need to develop rehabilitation strategies that target specific types of damage and neuroprotective therapies. We also need more research on the molecular and genetic factors that increase the risk of AOP infarction to support prevention.

Conclusion

AOP infarction is a rare but an important variant in cerebral ischemia as it can cause severe bilateral thalamic infarctions. Early recognition of this is key as treatment can make a big difference. Advanced imaging like MRI is crucial in detecting the specific lesions of AOP occlusion, so it is the key to diagnosis. To manage this uncommon but important condition, we need to increase awareness among healthcare providers and carry out more research on diagnosis and treatment. Knowledge of AOP variants is imperative to the diagnosis and treatment of infarctions in the condition, hence improving prognosis.

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Consent

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