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CASE SERIES

Artery of Percheron infarcts – study of five cases – case series

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An anatomical variation known as the artery of Percheron (AOP) has a single artery that emerges from the proximal posterior cerebral artery (PCA). It will result in an infarction in both the thalamus and the paramedian regions with or without the involvement of the midbrain. It is rare, occurring in roughly 4–12% of people. Due to its frequent misidentification, the frequency of bilateral thalamic infarction caused by AOP blockage is unknown. It accounts for approximately 4–18% of thalamic strokes and 0.1–2.0% of all ischemic strokes. We examine the various clinical signs and imaging findings associated with AOP stroke in this series. We report five cases of AOP stroke admitted in our hospital with varied symptoms like altered sensorium, hypersomnolence, reduced consciousness, and one patient with hallucinations. It poses a great diagnostic difficulty because of the varied clinical presentation.

Keywords: artery of Percheron, thalmaic infacrtion, thalamus, cerebrovascular accident, altered sensorium

Introduction

An anatomical variation known as the artery of Percheron (AOP) has a single artery that emerges from the proximal posterior cerebral artery (PCA). It will result in an infarction in both the thalamus and the paramedian regions with or without the involvement of the midbrain. It is rare, occurring in roughly 4–12% of people. Due to its frequent misidentification, the frequency of bilateral thalamic infarction caused by AOP blockage is unknown. It accounts for approximately 4–18% of thalamic strokes and 0.1–2.0% of all ischemic strokes. We examine the various clinical signs and imaging findings associated with AOP stroke in this series.

Case description

Case 1

A 58-year-old female with a known history of diabetes mellitus on oral medications was brought to the emergency department (ER) after being found unresponsive. On physical examination, the patient was found to be sleepy and only responded to painful stimuli by opening her eyes. Her pupils were pinpointed, and her Glasgow Coma Scale (GCS) was 9–10. She was moving all her limbs normally to pain. Magnetic resonance imaging (MRI) of the brain showed right thalamus hyperacute lacunar infarction and left thalamus acute/subacute infarction (**Figure 1**). She was not

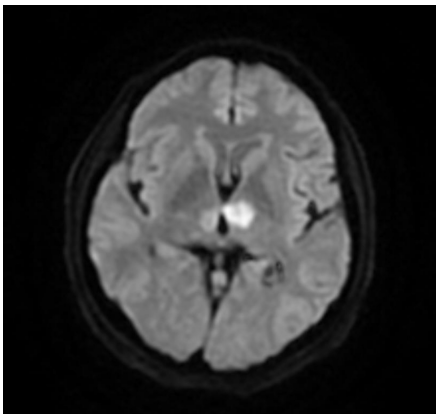

FIGURE 1 | DW image of patient 1 showing bilateral paramedian infarcts.

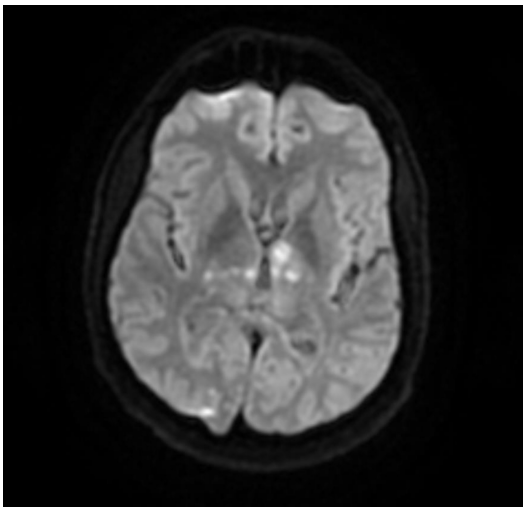

FIGURE 2 | DWI imaging of case 2 showing bilateral paramedian infarcts extending to anterior thalamus.

thrombolyzed as she was out of the window and was treated conservatively. She was discharged after 10 days; upon discharge, the patient had improved significantly without any focal neurological deficit.

Case 2

A 43-year-old male smoker with a history of hypertension who presented with fever and confusion for 2 days without any other neurological complaints. On examination, the patient was conscious but confused, with no other focal neurological deficit and no signs of meningeal irritation. Vitally, he was febrile with elevated blood pressure. His initial computed tomography (CT) brain was normal. His blood investigations and lumbar puncture to rule out encephalitis were normal. He tested positive for COVID-19. He was started on empirical encephalitis treatment. On the next day of admission, the patient's consciousness level deteriorated, and they had pupillary asymmetry with a dilated and nonreactive right pupil. He was intubated and ventilated. Repeated CT brain scans showed no newly developed vascular insult during the time interval. Brain MRI with Magnetic resonance angiography (MRA) was done and showed posterior circulation (AOP) stroke represented by bilateral medial thalamus and midbrain infarcts (shown in **Figures 2** and **3**) with no visualization of the PCA. The patient was started on antithrombotic. Acyclovir and steroids were stopped. He was on a mechanical ventilator for more than 30 days and was gradually weaned off. The patient was discharged 2 months later, and at the time of discharge he was conscious, oriented, pupils equal and reacting, extraocular movement full, and he was able to walk with support.

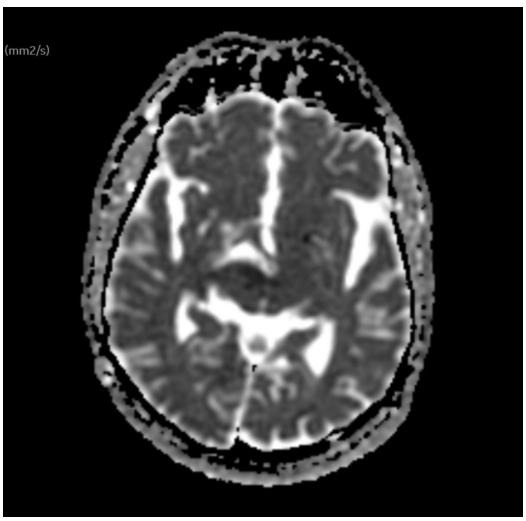

FIGURE 3 | ADC imaging of case 2 showing midbrain infract.

Case 3

A 71-year-old male was admitted through the ER with a history of unresponsiveness. On arrival, the patient's GCS was 7/15, and he had a right-sided facial droop and a pinpoint left pupil. He was intubated and transferred to the intensive care unit (ICU). MRI diffusion showed an acute non-hemorrhagic infarction, most compatible with an AOP territory infarct (**Figures 4** and **5**). He was not thrombolyzed as the time of onset was unknown and the MRI brain was not showing a diffusion-perfusion mismatch. He had a protracted course in the ICU as he developed two cardiac arrests and pneumonia. The patient underwent a tracheostomy on a later date, was extubated, and was transferred to the general ward. He was treated with antithrombotic and supportive care and was discharged. On discharge, the patient was conscious with a tracheostomy tube in situ and was able to be supported in a wheelchair.

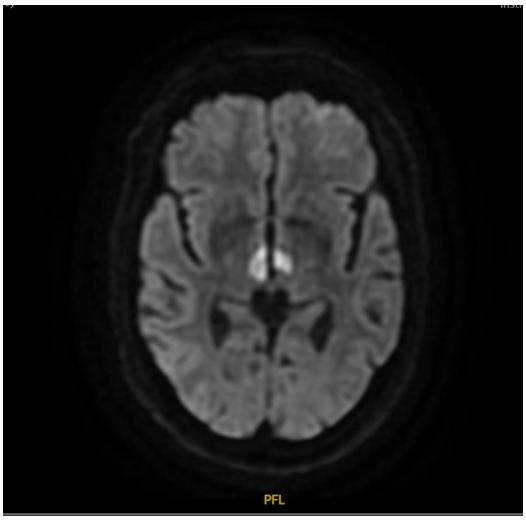

FIGURE 4 | DW images of case 3 showing bilateral paramedian infarcts.

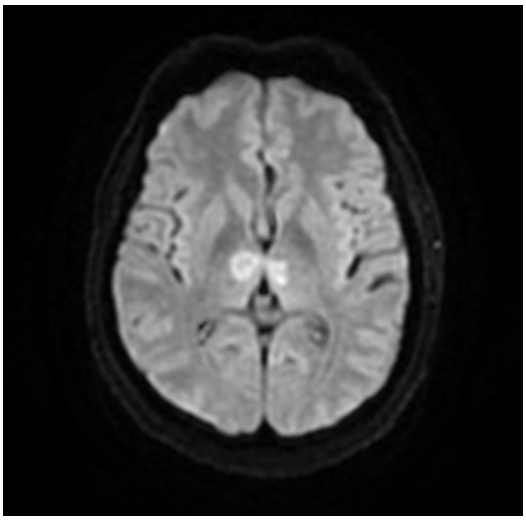

FIGURE 6 | DW images of case 4 showing bilateral paramedian infarct.

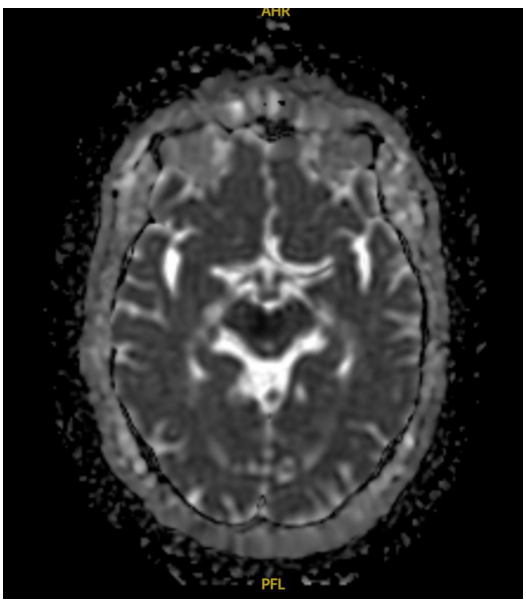

FIGURE 5 | ADC imaging of case 3 showing midbrain infracts.

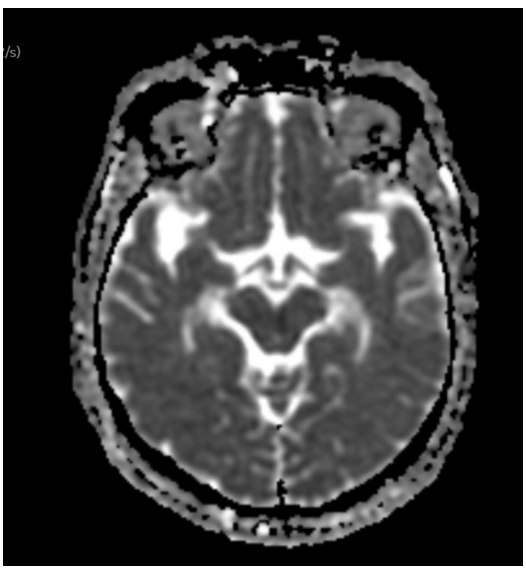

FIGURE 7 | ADC images of case 4 showing midbrain infarct.

Case 4

A 45-year-old male patient with a history of smoking and no chronic medical illness presented to the ER as he was found unresponsive. Upon arrival, his GCS was 6/15, and his pupils were unequal, with the left side dilated and non-reactive, and he was intubated on the same day. An electroencephalography (EEG) showed normal findings, while toxicology and autoimmune screens were negative. CT and MRI of the brain showed paramedial and midbrain infarction suggestive of AOP infarct (**Figures 6** and **7**).

The patient was not thrombolysed as the time of onset was not known and no mismatch was noted in the MRI brain. The patient developed a chest infection due to Staphylococcus aureus, which led to sepsis. Further examinations showed no acute changes in brain imaging. His echocardiography showed diastolic dysfunction. After a period of ventilatory and respiratory support, the patient was successfully extubated and discharged with recommendations for post-stroke rehabilitation. At the time of discharge after 2 months of hospital stay, he was conscious but had poor verbal communication, and he was able to move all limbs.

Case 5

A 36-year-old male smoker presented to the ER with 3-day history of intermittent dizziness, sleepiness, reduced concentration, hallucinations, slurred speech, short-term memory loss, and confusion. On examination, he was alert and oriented, well nourished, and not in acute distress. MRI

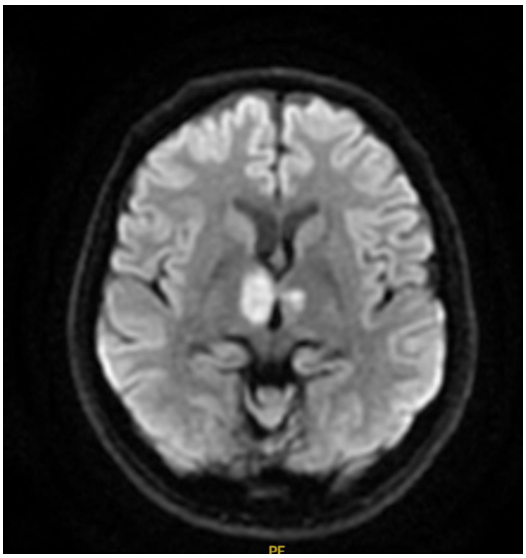

FIGURE 8 | DW images of case 5 showing bilateral paramedian and right anterior thalamic infarct.

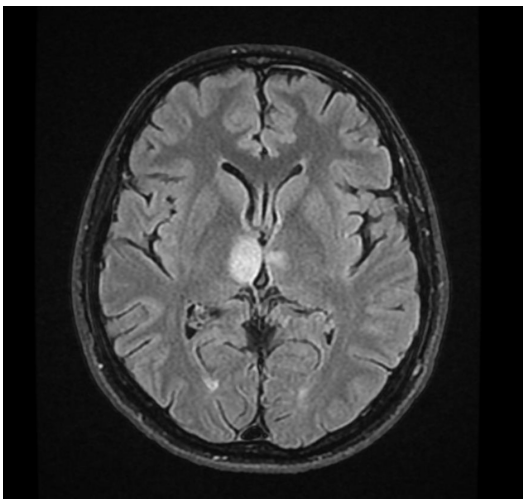

FIGURE 9 | Flair images of case 5 showing bilateral paramedian infarct and right anterior thalamic infarct.

brain confirmed bilateral thalamic hypodense areas, highly suggestive of AOP infarctions (**Figures 8** and **9**), more on the right side. He was treated with antiplatelets and statins. Upon discharge after 5 days of admission, the patient was awake, alert, and oriented with no focal deficits.

Discussion

The thalamus acts as a crucial relay center for managing sleep, alertness, and consciousness. The PCA and posterior communicating artery (PCOM) are the primary sources of blood supply to the thalamus. The vascular territories of the thalamus consist of anterior, posterior, inferolateral, and paramedian regions. The tuberothalamic artery, which branches from the PCOM, supplies the anterior area. The choroidal artery and the thalamogeniculate arteries, which emerge from the P2 segment of the PCA, supply the posterior and inferolateral regions, respectively (1). The paramedian arteries provide blood to the paramedian region. There are four known variants in this area. In type P1, the segment of each PCA gives the paramedian artery independently. Type 2 is divided into 2A and 2B, where in 2A either the right or left PCA gives both paramedian arteries, and in 2B, a single trunk originates from either of the PCA and divides into right and left paramedian artery. Type 3 also called as arcade variant, there is existence of a communicating artery between both P1 segments from which the paramedian artery arises (**Figure 10**).

The paramedian artery supplies primarily the dorsomedial nucleus, internal medullary lamina, and intralaminar nuclei, central lateral, centromedian, and parafascicular (2).

Paramedian infarctions caused by blockage of paramedian arteries are the second most common type of infarction, after inferolateral territory infarctions. Paramedian infarctions typically present with consistent and predictable symptoms, characterized by certain clinical features. Vigilance disorders with stupor and coma, memory loss, mood alterations, and vertical gaze palsy. Changes in mental status can vary from mild drowsiness or confusion to deep sleepiness or coma, with these symptoms appearing suddenly and potentially lasting a long time, although complete recovery is possible. Therefore, sudden increased drowsiness in patients should raise suspicion of bilateral thalamic infarction (3, 4).

The structures most involved with disorders of vigilance are the midline nucleus, where there is an extension of the mesencephalic reticular formation. Memory dysfunction and confabulation occur in some patients who do not lose consciousness, which typically improve over time. Memory disruptions become more intense when the polar territory is also affected (4), with the involvement of medial dorsal nuclei. The patient often does not report changes in mood and behavior, resulting in limited literature on the topic. However, they typically show signs of irritability and apathy. Vertical gaze palsy typically indicates midbrain involvement but can also happen without midbrain infarction. The involvement of the frontal fugal thalamic bundle traveling through the medial dorsal nucleus and internal medullary lamina is reportedly the cause (3, 4).

Radiological patterns of AOP involvement identified four distinct patterns of AOP infarction: bilateral paramedian thalamic with rostral midbrain, bilateral paramedian thalamic without midbrain, bilateral paramedian and anterior thalamic with midbrain, and bilateral paramedian and anterior thalamic without involvement of midbrain (4).

According to Bouglovasky et al., impairment in conscious level is commonly reported with a paramedian infarction. They also present with severe confusion, memory impairment, and confabulation. Headaches, gaze palsies, and dysphagia are a very less commonly reported (5).

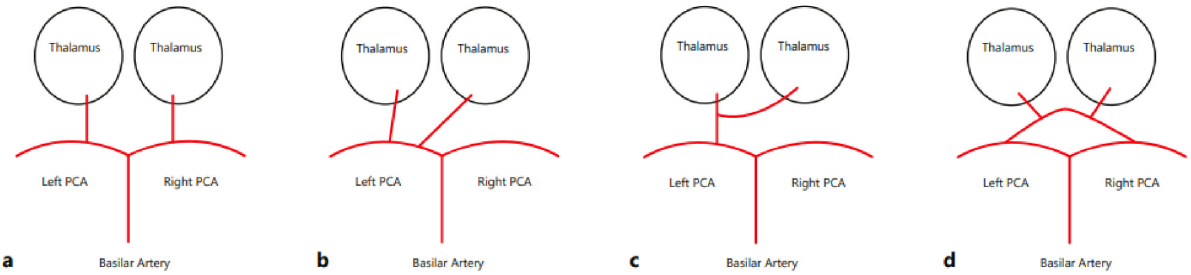

FIGURE 10 | Showing variants of AOP.

According to Witte et al., after bilateral thalamic lesions, patients usually display neurocognitive symptoms characterized by constructional apraxia, anosognosia, disorientation, global intellectual dysfunction, attention problems, memory disturbances, and behavioral and mood alterations (6). Both Joana et al. and Caruso et al. have reported that patients with AOP infarct exhibit complicated neuropsychological symptoms such as severe drowsiness and excessive sleepiness, and upon improvement, they show memory disturbances (7, 8). Krishnan et al. observed in their case series that the majority of patients exhibited an altered sensorium, with one patient displaying a vertical gaze palsy (1).

Conclusion

AOP infarcts present a diagnostic challenge for the physicians in the acute setting because of the diversity and inconsistency in the presentation. There are also poor localization signs, and acute imaging most of the time is negative. These factors can hinder the early diagnosis and early intervention, especially considering intravenous thrombolysis. MRI is the diagnostic choice for these patients, so we need a high index of suspicion when the patient presents with a diverse clinical presentation of acute onset and to plan for an early intervention.

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Abbreviations and acronyms

AOP: artery of Percheron
PCA: posterior cerebral artery
CVT: cerebral venous thrombosis
ER: emergency
GCS: glasgow coma scale
MRI: magnetic resonance imaging
CT: computed tomography
LP: lumbar puncture
ICU: intensive care unit
EEG: electroencephalography

Disclosures

Part of this article, especially about case 2 has been previously published by me as a single case report.

Encephalitis or Stroke? Thalamic stroke in covid era. Dubai Medical Journal. doi: 10.1159/000527054-november 2022.

Author contributions

Anandi Damodaran: Study design, data analysis and conceptual developments; Dawlat Hussien Sany: Data acquisition and data analysis; Bushra Hamed Ali Abuzayed: Data acquisition and data analysis; Devdutt Nayak Kotekar: Data analysis and conceptual development.

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Conflicts of Interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflicts of interest.

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