Radiology Case Reports 19 (2024) 1344–1350

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/radcr

Case Report

Bilateral thalamic infarcts: Percheron territory ☆

Ghita Lahnine, MD, Younes Abdourabbih, MD, Nizar El Bouardi, MD, Meryem Haloua, MD, Badreddine Alami, MD, My Youssef Alaoui Lamrani, MD, Meryem Boubbou, MD, Mustapha Maaroufi, MD*

Radiology Department, CHU Hassan II, Fès, Morocco

ARTICLE INFO

Article history:
Received 21 October 2023
Revised 11 December 2023
Accepted 12 December 2023

Keywords:
Artery of Percheron
Thalamus
Stroke
MRI

ABSTRACT

Blood supply to the human thalami is complex and multiple variants exist. The artery of Percheron is one of those variants and is characterized by a solitary arterial trunk that branches from one of the proximal segments of either posterior cerebral artery and supplies blood to the paramedian thalami. Its occlusion results in bilateral paramedian thalamic infarction sometimes extending to the midbrain. We report a case of bithalamic infarction secondary to occlusion of the artery of Percheron. We will illustrate the complex clinical symptomatology and underscore the role of imaging, especially MRI, for diagnosis.

Introduction

The thalami and the midbrain receive blood supply from the perforating branches of the posterior cerebral and communicating arteries. The thalami have blood supply categorized into 4 territories: anterior, paramedian, posterior, and inferolateral. The paramedian territories of the thalami are supplied by perforating thalamic arteries of posterior circulation known as paramedian arteries. There are 4 normal variants of neurovascular supply to the thalami and midbrain. The most common variant is variant I, which is where the perforating branches arise from the right and left posterior cerebral arteries (PCAs) individually [1]. Variant IIa is where the left P1 segment is the source of both paramedian arteries. In variant IIb, the perforating arteries arise from the artery of Percheron (AOP). In these variants, the AOP comes from a partof the posterior cerebral artery, namely, the P1 segment. The AOP supplies the paramedian thalami and the rostral midbrain. Variant III is known as an arcade variant. In this anatomical variant, the arcade gives off small perforating branches from one arterial arc. This arterial arc bridges the P1 segments and the PCAs together [1].

Occlusion of the AOP may lead to an infarction of the paramedian thalami and mesencephalon. The AOP variant is present in 4%-12% of the population. In 2 large stroke series studies, characteristic AOP infarct patterns constituted 0.1%-2% of all ischemic strokes, indicating that this type of ischemic stroke is quite rare [1].

☆ Competing Interests: The authors have no conflicts of interest to declare. All the co-authors have seen and agree with the contents of the manuscript and there is no financial interest to report. We certify that the submission is original work and is not under review at any other publication.

* Corresponding author.
E-mail address: lahnineghita@gmail.com (G. Lahnine).

https://doi.org/10.1016/j.radcr.2023.12.024

Observation

A 64-year-old patient was admitted to the emergency department for the management of a sudden coma, which started a day before his admission. He had no previous history of disease and did not smoke, or drink alcohol or illicit drugs. His vital signs were normal (blood pressure 120/70 mmHg, breath rate 18/minute, heart rate 78/minute, axillary temperature 37.5°C). There was otherwise no comitial seizure or trauma preceding the coma. The physical examination showed a GCS at 11. There was no asymmetric finding at "pierre marie et foix," and the tonus in both extremities was asymmetric.

All blood tests were normal, and illicit drug and toxic profiles were negative. The laboratory test results were as follows: complete blood cell count: hemoglobin 15.3, platelets 371; cholesterol profile: cholesterol 177 mg/dL, triglycerides 256, high-density lipoprotein (HDL) 35 mg/dL, low-density lipoprotein 85 mg/dL, ratio of cholesterol to HDL 5.

The patient underwent a brain CT scan with iodine contrast injection and showed a bilateral thalamic hypodensity, and annular enhancement after iodine injection (Fig. 1).

The patient benefited from a thoraco-abdomino-pelvic CT scan, which objectified the presence of a bilateral pulmonary embolism, proximal on the left, complicated by bilateral LI pulmonary infarction foci.

He has benefited from cardiac ultrasound, and the thrombophilia work up were all negative. Lower extremity venous Doppler was also performed showing no deep venous thrombosis (Fig. 2).

We evoked a probable diagnostic:

- Infection: Toxoplasmosis or tuberculosis
- Vascular: Percheron
- Tumor: Bilateral thalami glioma, metastasis

The patient underwent a lumbar puncture which came back negative. We completed with a cerebral MRI which objectified the presence of 2 bilateral thalamic lesions, extended to the right midbrain, well limited in rounded shape, described in T2 and flair hypersignal, containing hemorrhagic areas, with a restrictive shell in diffusion, and ring enhancement (Figs. 3 and 4).

Diagnosis retained: bithalamic ischemic stroke reminiscent of Percheron stroke probably due to Behçet type vasculitis.

The patient continued to improve. Within the first 48 hours, the patient was confused but awake and following minimal commands. She continued to show improvement and passed a speech and swallow test. Eventually, the patient was discharged to subacute rehab with neurological follow-ups and was given dual antiplatelet therapy with Aspirin and Plavix as per recommendations of neurosurgery with reevaluation for the need of dual antiplatelet in 4 weeks.

Discussion

Bithalamic infarct

Bilateral thalamic infarcts are rare presentations of stroke. They are the result of a complex combination of risk factors and a predisposing vessel distribution. The artery of Percheron, characterized by a single arterial trunk that irrigates both paramedian thalamic regions, can be occluded as a result of embolic diseases leading to bilateral paramedian thalamic infarcts. Clinical and image findings of this uncommon form of posterior circulation infarct are presented along with their anatomic and pathophysiologic correlates [2].

Epidemiology

Thalamic infarcts are very scarce. It is accounted to be 11% of all infarcts involving the vertebrobasilar region [3].

The AOP variant prevalence is estimated to occur in 4%-30% of the general population and accounts for between 0.1% and 2% of ischemic strokes [3].

Physiopathology

Strokes affecting both paramedian thalamic territories are unusual and may lead to a suspicion of an occlusion of a single arterial trunk known as the artery of Percheron. Although

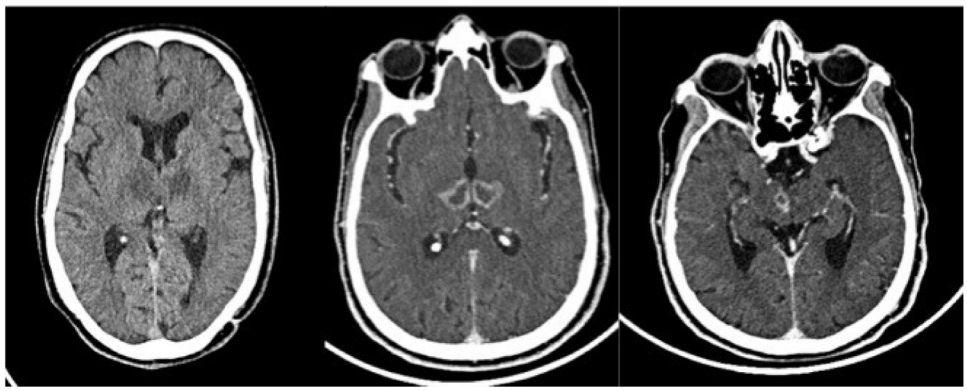

Fig. 1 – CT scan without and after injection of the PDC was carried out which objectified the presence of 2 hypodense lesions in spontaneous contrast at the level thalamic bilaterally and right midbrain enhanced in an annular fashion after injection of PDC, surrounded by a collar of perilesional edema.

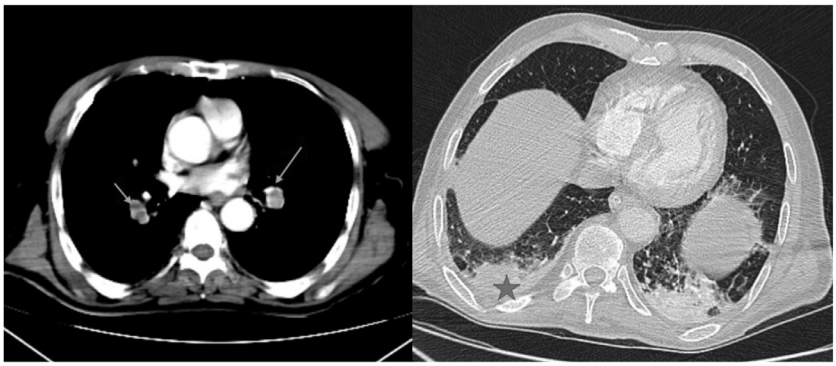

Fig. 2 – The patient benefited from a thoraco-abdomino-pelvic CT scan which objectified the presence of a bilateral pulmonary embolism, proximal on the left, complicated by bilateral LI pulmonary infarction foci.

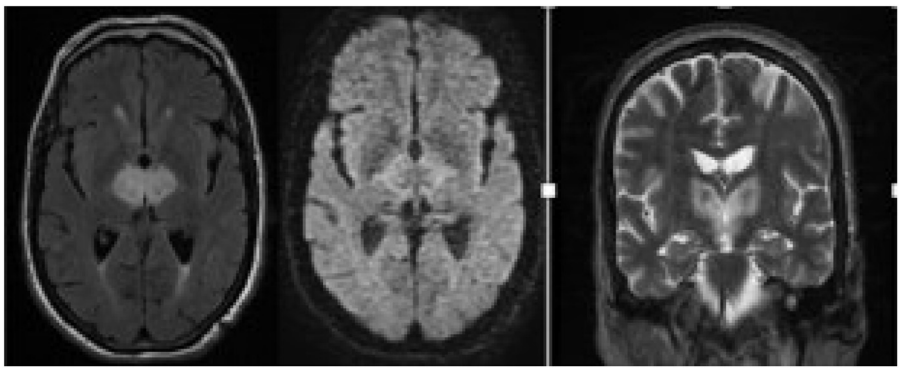

Fig. 3 – MRI in axial sections, flair, diffusion, and T2 sequences showing the presence of 2 moderate thalamic lesions, extended to the right midbrain, well limited in rounded shape, described in T2 and flair hypersignal, with a restrictive shell in diffusion.

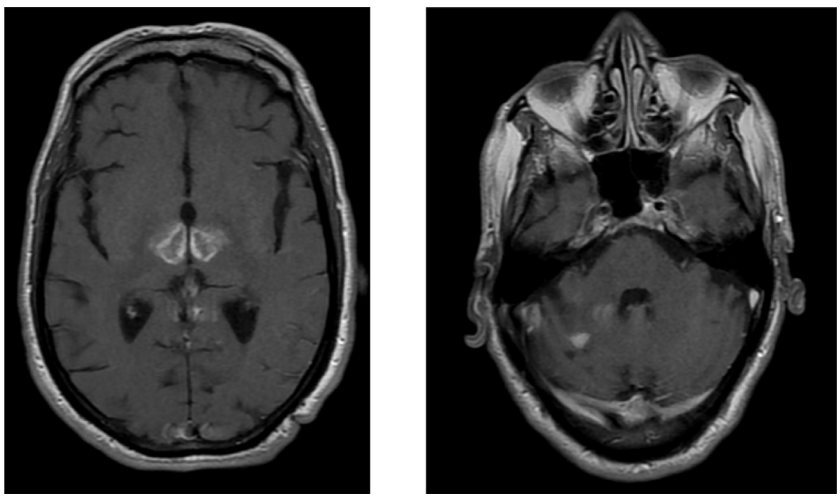

Fig. 4 – After injection of gadolinium, we note the presence of an annular enhancement.

not visible on angio-MRI, the presence of this anatomic variant must be suspected when bilateral symmetric paramedian thalamic infarcts are revealed on image studies in the context of a patent basilar artery and posterior cerebral arteries [2].

French neurologist Gerard Percheron described 4 variants of perforator supply of the thalamus and the rostral midbrain (Fig. 5). The third variant (Type IIb) encompasses the artery of Percheron [3].

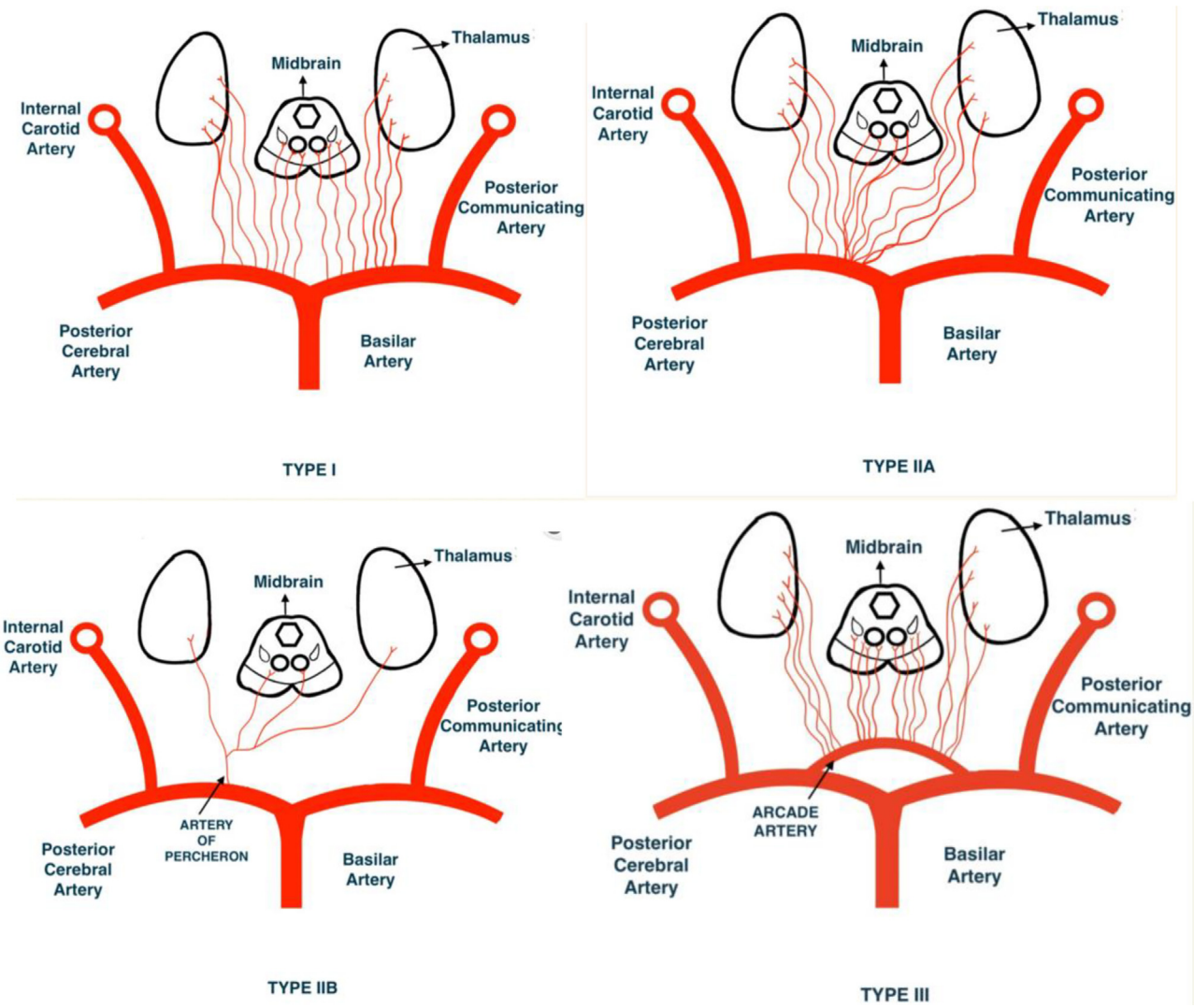

Fig. 5 – Type I variant of perforator supply of thalamus and the rostral midbrain. Type IIa variant of perforator supply of thalamus and the rostral midbrain. Type IIb variant of perforator supply of thalamus and the rostral midbrain. Type III variant of perforator supply of thalamus and the rostral midbrain.

An understanding of thalamic anatomy is important to explain the pathophysiology of bilateral paramedian thalamic infarction. Paramedian nuclei consist mainly of a dorsomedian nucleus and intralaminar nuclei. The intralaminar nuclei consist of parafascicular, centromedian, central medial, paracentral, and central lateral nuclei. Smaller nuclei of the "midline," such as the paraventricular, rhomboid, and reunions nuclei, are also included in the intralaminar group. Both nuclear groups are characterized by important and reciprocally activating connections with the anterior, orbitofrontal, and medial prefrontal cortices through the thalamic peduncles [4,5], thus explaining the neuropsychiatric impairment and the loss of self-activation characteristic of paramedian infarctions. The rostral midbrain can also be involved after occlusion of the artery of Percheron. The initial presence of right mydriasis, ptosis and exophthalmos are all suggestive of an effect at this level due to the periaqueductal gray matter being affected, where the third cranial nerve nuclei are located [2] (Fig. 6).

Clinical presentation

As result of the complexity of the thalamus function and anatomy, especially its irrigational variations previously mentioned, the clinical syndromes of these patients can differ depending on the sub-structure of the thalamus involved. There has been described an overlap with mesencephalic syndromes.

The classic syndrome of occlusion of the AOP presents with bilateral vertical gaze palsy in 65%, memory impairment (anterograde and retrograde amnesic syndrome) in 58%, confusion in 53% and coma in 42% of the patients [6].

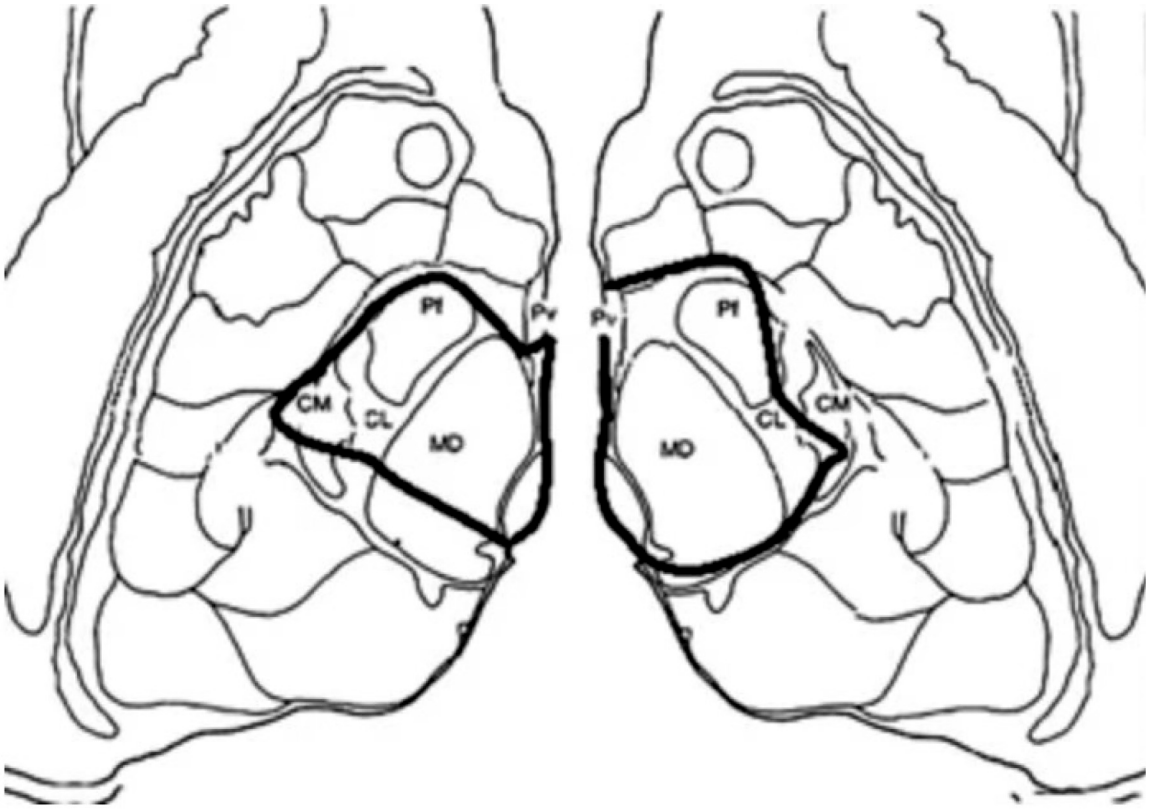

Fig. 6 – Schematic representation of an affected thalamic nuclei the black line limits the area of infarction in both paramedian regions. CM, centromedian; Pf, parafascicularis; Pv, paraventricular; CL, central lateral; MD, dorsomedialis.

Imaging findings

Brain MRI is the most specific imaging study for the diagnosis. The DWI sequence has a sensitivity of 100%, compared to CT scan which appears normal 50% of the time, making the MRI the best tool to establish an opportune diagnosis and the possibility to offer thrombolytic therapy [7].

A recent study identified four different MRI findings in case of Percheron's artery occlusion:

- Isolated bilateral paramedian thalamic involvement alone.
- Bilateral paramedian thalamic involvement associated mesencephalic impairment.
- Bilateral paramedian and anterior thalamic damage.
- Bilateral paramedian and anterior thalamic damage associated with a mesencephalic lesion.

The authors also insist on the "V sign" as a hyperintensity in axial sections in the FLAIR sequence or DWI along the pial surface of the midbrain, adjacent to the interpeduncular fossa with a sensitivity of up to 67% in patients with midbrain involvement.

The CT scan may show a bithalamic hypodensity; but this examination is most often in the acute phase [8].

The occlusion of the artery can be visualized by performing a selective arteriography with the possibility of performing intra-arterial thrombolysis arterial thrombolysis [9]. However, in case of suspected occlusion of Percheron's artery, conventional angiography is not indicated because the nonvisualization of the artery does not eliminate its existence, due to its possible occlusion and especially its small size [10].

To the best of our knowledge, the value of CT perfusion in acute Percheron stroke has not been evaluated. The advantage of CT perfusion is that it is able to delineate areas of the brain that may be salvaged by intervention (e.g., thrombolysis or clot retrieval), known as the penumbra, from the parts that are irrevocably destined to go into infarct regardless of therapy, known as the infarct core. What is more, identification of infracted areas is easier than with non-contrast head CT. The weakness of CT perfusion at the level of the midbrain is that we often come across artefacts that reduce the quality of the examination [11].

Differential diagnosis

Bilateral thalamic lesions are uncommon. These paired lesions have a limited differential diagnosis that includes metabolic and toxic processes, infection, vascular lesions, and neoplasia. The differential diagnosis can be further narrowed with the patient history, imaging characteristics, and presence or absence of lesions outside the thalami [12].

Primary neoplasm

Bilateral thalamic glioma is a rare neoplasm, usually a diffuse low-grade astrocytoma (World Health Organization grade II),

that occurs in both children and adults [13]. Bilateral thalamic glioma has a poor prognosis due to the location of the lesions [14]. Children typically have signs of increased intracranial pressure and movement disorders. Adults experience mental deterioration. Typically, expansion of both thalami is accompanied by abnormal hyperintensity on T2-weighted images and hypointensity on T1-weighted images that is not associated with contrast enhancement.

Hydrocephalus depends on the degree of mass effect. Diffusion is normal [12].

Metabolic and toxic disorders

Wernicke encephalopathy

Wernicke encephalopathy results from a deficiency of vitamin B1 and is frequently associated with alcohol abuse [15]. The classic clinical triad is ataxia, altered consciousness, and abnormal eye movements; however, the presentation is variable. Wernicke encephalopathy is a medical emergency managed with IV thiamine. T2-weighted MR images may show symmetric high signal intensity in the mammillary bodies, medial aspects of the thalami, tectal plate, periaqueductal gray matter, and dorsal medulla [16]. Contrast enhancement is variable. Thiamine is an osmotic gradient regulator, and deficiency can disrupt the blood–brain barrier, resulting in contrast enhancement [17]. Wernicke encephalopathy can have reduced diffusion owing to ischemia-like changes in the thalami that should be differentiated from true venous and arterial infarction [12].

Extrapontine myelinolysis

Osmotic myelinolysis accompanies rapid shifts in serum osmolality; the classic setting is the rapid correction of hyponatremia [18]. The classic lesion involves the central pons (central pontine myelinolysis). Other lesions affect the basal ganglia, thalami, and white matter (extrapontine myelinolysis). Acute T2 hyperintensity and T1 hypointensity occur in the affected regions. Contrast enhancement is uncommon, and reduced diffusion may be seen [12].

Vascular occlusion

Occlusion of cerebral venous

Typically results in bilateral symmetric involvement of the thalami and occasionally the basal ganglia. The causes include pregnancy, oral contraceptives, infection, trauma, and dehydration, but the cause is undetermined in 20%-25% of patients [19]. An abnormally hyperdense vein may be seen on CT scans, and corresponding T1 hyperintensity from clot in the venous may be seen on MR images. CT and MR venography show no areas of contrast enhancement or signal intensity in the deep venous sinuses. Diffusion-weighted imaging may show heterogeneous signal intensity [20]. Patchy contrast enhancement may be seen.

Infection

Toxoplasma

Cerebral infection by the protozoan toxoplasma gondii appears as multiple regions predominantly in the basal ganglia and at the corticomedullary junction [21]. The diagnosis is to be considered in immunocompromised patients in the presence of any neurological sign.

CT scan: Hypodense lesion with peripheral enhanced shell after PDC injection
MRI: Multiple and diffuse rounded lesions, significant perilesional swelling.
T1: hyposignal with nodular or annular enhancement
T1 after injection of gadolinium: eccentric target enhancement.
T2: appearance of a necrotic mass with concentric target.
Diffusion: low central signal of the necrosis and high ADC.

Treatment and prognosis

Thrombolysis is the treatment of choice when a diagnosis is made within the therapeutic window of 4.5 hours. Because of the nonlocalizing presentations associated with the change in mental status, many diagnoses are delayed and fall outside the therapeutic window. Prognosis can be favorable if the diagnosis is rapidly identified, and the patient receives appropriate treatment [22].

A series of 18 cases of paramedian infarction reported that 61% of them had favorable outcome and were able to perform activities of daily living without assistance [23]. One case report stated that there was significant improvement in the state of alertness after administering modafinil 100 mg twice a day [2].

Conclusion

AOP infarcts are rare and often misdiagnosed as the initial radiological evaluations are usually normal. Symptoms of AOP are different from other ischemic strokes and vary depending on the size and distribution of the infarct. A sudden onset alteration in sensorium with behavioral manifestation and abnormal eye movements are clues suggestive of an AOP infarction. A diffusion-weighted MRI of the brain in the early hours is the investigation of choice. A detailed evaluation of cardiac and arterial sources of embolism is recommended in all cases of AOP infarction [24].

Patient consent

Informed written consent was obtained from both patients for publication of the case report and all imaging studies.

REFERENCES

[1] Kichloo A, Jamal SM, Zain El-A, Wani F. Vipparala N Artery of Percheron infarction: a short review. J Investig Med High Impact 2019;7:1–6.

[2] López-Serna R, González-Carmona P, López-Martínez M. Bilateral thalamic stroke due to occlusion of the artery of Percheron in a patient with patent foramen ovale: a case report. J Med Case Rep 2009;3(1) Article ID 7392.
[3] Phate N, Pawar T, Andhale A, Singh RK, Talwar D, Acharya S, et al. Artery of percheron stroke: a case report with a diagnostic challenge. Cureus 2022;14(2):e21939.
[4] Siwek D, Pandya D. Prefrontal projections to the mediodorsal nucleus of the thalamus in the rhesus monkey. J Comp Neurol 1991;312:509–24. doi:10.1002/cne.903120403.
[5] Groenewegen H, Berendse H. The specificity of the "nonspecific" midline and intralaminar nuclei. Trends Neurosci 1994;17:52–6. doi:10.1016/0166-2236(94)90074-4.
[6] Monet P, Garcia PY, Saliou G, Spagnolo S, Desblache J, Franc J, et al. Bithalamic infarct: is there an evocative aspect? Radioclinical study (Article in French). Rev Neurol (Paris) 2009;165:178–84.
[7] Xu Z, Sun L, Duan Y, Zhang J, Zhang M, Cai X. Assessment of Percheron infarction in images and clinical findings. J Neurol Sci 2017;383:87–92.
[8] Caballero PEJ. Bilateral paramedian thalamic artery infarcts: reports of 10 cases. J Stroke Cerebrovasc Dis 2010;19:283–9.
[9] Kostanian V, Cramer SC. Artery of Percheron thrombolysis. AJNR Am J Neuroradiol 2007;28:870–1.
[10] Matheus MG, Castillo M. Imaging of acute bilateral paramedian thalamic and mesencephalic infarcts. AJNR Am J Neuroradiol 2003;24:2005–8.
[11] Lamot U, Ribaric I, Popovic KS. Artery of Percheron infarction: review of literature with a case report. Radiol Oncol 2015;49(2):141–6.
[12] Smith AB, Smirniotopoulos JG, Rushing EJ, Goldstein SJ. Bilateral thalamic lesions. Am J Roentgenol 2009;192:W53–62.
[13] Partlow GD, del Carpio-O'Donovan R, Melanson D, Peters TM. Bilateral thalamic glioma: review of eight cases with personality change and mental deterioration. Am J Neuroradiol 1992;13:1225–30.
[14] Di Rocco C, Iannelli A. Bilateral thalamic tumors in children. Childs Nerv Syst 2002;18:440–4.
[15] Ogershok PR, Rahman A, Nestor S, Brick J. Wernicke encephalopathy in nonalcoholic patients. Am J Med Sci 2002;323:107–11.
[16] Zuccoli G, Gallucci M, Capellades JL, Regnicolo B, Tumiati T. Cabada G, et al. Wernicke encephalopathy: MR findings at clinical presentation in twenty-six alcoholic and nonalcoholic patients. Am J Neuroradiol 2007;28:1328–1331.
[17] Doherty MJ, Watson NF, Uchino K, Hallam DK, Cramer SC. Diffusion abnormalities in patients with Wernicke encephalopathy. Neurology 2002;58:655–7.
[18] Laureno R, Karp BI. Myelinolysis after correction of hyponatremia. Ann Intern Med 1997;126:57–62.
[19] Masuhr F, Mehraein S, Einhaupl K. Cerebral venous and sinus thrombosis. J Neurol 2004;251:11–23.
[20] Yoshikawa T, Abe O, Hayashi N, Mori H, Yamada H, Aoki S, et al. Diffusion-weighted magnetic resonance imaging of dural sinus thrombosis. Neuroradiology 2002;44:481–8.
[21] Jha P, El-Feky M, Deng F, et al. Neurotoxoplasmosis. Reference article, Radiopaedia.org, https://doi.org/10.53347/rID-8614, accessed Jan 05, 2024.
[22] Donohoe C, Kiani Nia N, Carey P, Vemulapalli V. Artery of Percheron infarction: a case report of bilateral thalamic stroke presenting with acute encephalopathy. Case Rep Neurol Med 2022;2022:8385841.
[23] Xu Z, Sun L, Duan Y, Zhang J, Zhang M, Cai X. Assessment of Percheron infarction in images and clinical findings. J Neurol Sci 2017;383:87–92.
[24] Sasi S, Ahmed A, Yousuf W, Vattoth S. Artery of Percheron infarct: a rare presentation of acute ischemic stroke in a high-risk antiphospholipid syndrome patient. Acute Med 2020;3:46–52.