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Case Series

Imaging of Bilateral Thalamic Infarcts (Percheron Territory) and Differential Diagnosis (About Six Cases)

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Abstract

The artery of Percheron is a rare anatomic variant of the paramedian (or thalamo-perforating) artery that represents a solitary trunk which provides bilateral arterial supply to the rostral midbrain and paramedian thalamus. The complex anatomy causes a large clinical variability which makes the clinical diagnosis of Percheron occlusion very difficult and often delayed and made outside the window of thrombolytic treatment for ischemic stroke. It is characterized in imaging with a paramedian bithalamic infarcts, that can be associated to a mesencephalic infarction. Even though the usual imaging results are habitually acknowledged, only a limited number of case series and case reports of artery of Percheron infarction have been disclosed. We report a series of six cases that presented with bilateral thalamic infarcts. The objective of this study is to illustrate the thorough and precise imaging spectrum of artery of Percheron infarction and its wide differential diagnosis.

Keywords: *Artery of Percheron; Thalamus; Infarction; Imaging; Magnetic Resonance Imaging*

Abbreviations

AOP: Artery of Percheron; PCA: Posterior Cerebral Artery; MRI: Magnetic Resonance Imaging; CT: Computed Tomography; GCS: Glasgow Coma Scale; ECG: Electrocardiogram; CTA: Computed Tomography Angiography; PComA: Posterior Communicating Artery; FLAIR: Fluid-Attenuated Inversion Recovery; TG: Triglycerides; HDL: High-Density Lipoprotein; NIHSS: National Institutes of Health Stroke Scale; LP: Lumbar Punction; GRE: Gradient Echo; CTA: Computed Tomography Angiography; ACT: Anti-Coagulant Therapy; APT: Anti-Platelets Therapy; IVT: Intra-Venous Thrombolysis; DWI: Diffusion Weighted Imaging; DSA: Digital Subtraction Angiography; SPECT: Single-Photon Emission Computed Tomography; CNS: Central Nervous System

Introduction

The medial part of the thalami is supplied by thalamo-perforating arteries that originate from the P1 segment of the posterior cerebral artery (PCA) [1]. The artery of Percheron (AOP) is a rare variant which is characterized by vascularizing the medial part of both thalami, and its blockage results in bilateral paramedian thalamic infarction with or without involvement of the middle brain [1]. The classic clinical

presentation of AOP infarction includes altered consciousness, memory impairment and supranuclear vertical gaze palsy [1]. However, an early diagnosis is sometimes challenging due the wide range of clinical symptoms, and is critical for selecting appropriate treatment options [1]. The gold standard imaging technique for AOP infarction is magnetic resonance imaging (MRI) [2]. It allows visualization for the acute phase of the infarction [3]. We conducted a retrospective case study displaying all the different imaging characteristics of six patients who experienced AOP infarction and were hospitalized at university hospital center Hassan II at the radiology department.

Materials and Methods

We collected, by browsing the electronic clinical charts and the Stroke Unit records, 1,400 acute ischemic stroke cases between January 2023 and January 2024, from which six cases of AOP infarction were found. We included the patients whose brain MRI or CT visioned bilateral ischemic lesions affection the medial thalamus. We reexamined sociodemographic (age and gender), clinical and radiological (admission severity and symptoms, and short-term and long-term prognoses) features from the individual electronic charts. This study was approved by institutional ethics committee. Informed consent was waved due to its retrospective nature.

Results

Case presentation 1

A 68-year-old woman patient was admitted to our emergency department with the sudden onset of impaired consciousness (Glasgow Coma Scale (GCS) graded to 8), which had started 20 hours before. The neurological examination showed a symmetrical drop of all 4 limbs, an abolished horizontal oculocephalic reflex as well as osteotendinous reflexes, without any facial paralysis. The cardiological examination was normal with a regular sinus rhythm in the electrocardiogram (ECG). The patient's medical history included hypertension and diabetes mellitus.

A brain CT (Figure 1) scan indicated a paramedian bithalamic hypodensity, a cortico-subcortical left occipital hypodensity and a right lenticular lacuna. Brain CT angiogram (CTA) of the intracranial vessels showed a filling defect in the first portion of the left PCA and hypoplasic posterior communicating arteries (PComA). CTA of the supra-aortic trunks found a left carotidian bulb plaque without significant stenosis. The atheromatous etiology was retained for this patient.

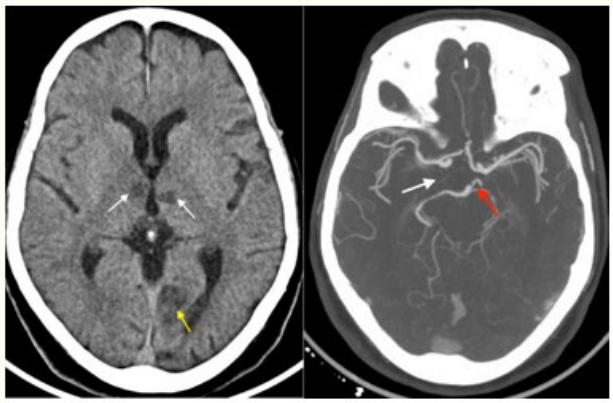

Figure 1: *Patient 1: Brain CT contrast enhanced: (a) and CTA (b) in an axial plane:*

(a) Shows paramedian bithalamic hypodensity (yellow arrow) and a cortico-subcortical left occipital hypodensity (green arrow), that are not modified after injection of contrast

(b) Shows a filling defect in the first portion of the left PCA (red arrow) and hypoplasic PComAs (blue arrow).

The patient was determined to be ineligible for thrombolytic therapy due to exceeding the thrombolytic treatment window. She was given anticoagulants and antiplatelet drugs. After three days, the patient's level of awareness had significantly improved, with a GCS scale at 15. A CT-scan was realized 3 days afterwards and showed a stability of the paramedian bithalamic hypodensity.

Case presentation 2

A 76-year-old male patient presented with the abrupt onset of altered consciousness with a GCS scale graded at 10. A neurological examination on admission revealed a vertical gaze palsy and a preserved oculo-cephalic reflex. The rest of the neurological investigation was normal. He didn't have any notable previous history. The cardiological examination showed a regular sinus rhythm in the ECG, a mild supraventricular hyperexcitability with a single non-sustained supraventricular tachycardia in the Holter-ECG.

A brain CT scan revealed no abnormal high or low-density shadows. Brain CTA of the intracranial vessels showed a permeable vertebro-basilar trunk and an hypoplastic P1 bilaterally. CTA of the supra-aortic trunks found a left common carotidian plaque extending to the internal carotid without significant stenosis. The atheromatous etiology was retained for this patient.

A brain MRI (Figure 2) taken on the same day showed paramedian bithalamic high signal intensity in Fluid-attenuated inversion recovery FLAIR and T2 sequences, these regions were not restrictive in diffusion.

The patient then beneficiated of a thrombolysis (3.5cc of METALYSE). He stayed afterwards stationary in the neurological examination, the family reported confusional episodes with behavioral problems (aggressiveness), anterograde amnesic disorder, hypersomnia and a Parinaud syndrome.

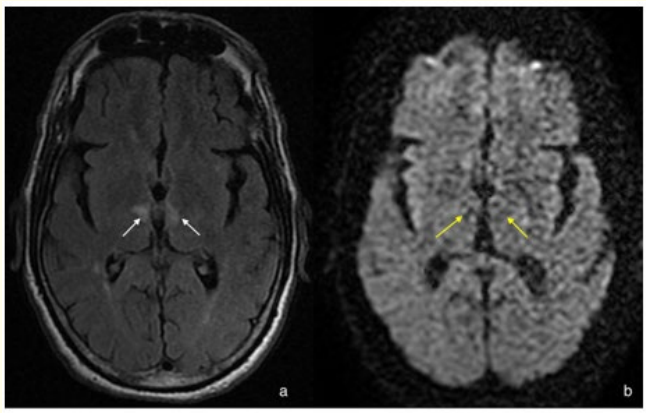

Figure 2: *Patient 2: Brain MRI in axial section in FLAIR sequence (a) and diffusion (b) shows high signal intensity in bilateral paramedian thalamic in FLAIR sequences (white arrow) that show no restriction in diffusion (yellow arrow).*

Case presentation 3

A 51-year-old female was admitted for acute impairment of consciousness with a GCS scored at 9 seven and half hours after the onset the clinical disorder. A neurological examination revealed no abnormality, besides a left anisocoria. The patient's medical history included hypertension, diabetes mellitus, a non-documented rheumatic pathology, and a poorly followed-up goiter. A cardiological examination showed a regular sinus rhythm in the ECG with extrasystoles. Lipid profile showed increased triglycerides levels.

A brain CT scan indicated paramedian bithalamic hypodensity, a CTA of the intracranial vessels showed a permeable Willis polygon with a left fetal type PCA. CTA of the supra-aortic trunks found a bilateral common carotidian plaque extending to the internal carotid without

significant stenosis. The atheromatous etiology was retained for this patient. He then beneficiated of a curative dose of anticoagulants. A clinical control afterwards still showed hypersomnia and retrograde memory disorders.

Case presentation 4

64-year-old patient with an unremarkable medical history was admitted at the emergency department for the management of a sudden coma, since one day. The physical examination showed à GCS scaled at 11, his arterial blood pressure was normal 110/80 mmgh. The neurological examination revealed a National Institutes of health stroke scale (NIHSS) graded at 19. There was no asymmetric finding at "pierre marie et foix", and their tonus in both extremity was asymmetric. A cardiological examination showed a regular sinus rhythm in the ECG.

A brain CT scan with iodine contrast injection (Figure 3) showed bilateral thalamic hypodensity with an annular enhancement after iodine injection.

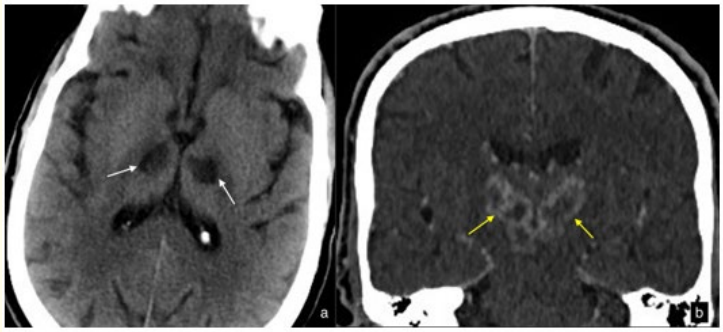

Figure 3: *Patient 4: Brain MRI in axial section in FLAIR sequence (a) and diffusion (b) shows high signal intensity in bilateral paramedian thalamic in FLAIR sequences (white arrow) that show no restriction in diffusion (yellow arrow).*

The patient underwent a lumbar puncture (LP) that came back negative. We completed with a cerebral MRI (Figure 4) which objectified bilateral thalamic lesions, extending to the right midbrain, well limited in rounded shape, described in hypersignal in T2 and FLAIR sequences, containing hemorrhagic areas, with a restrictive shell in diffusion, and a ring enhancement after injection of gadolinium.

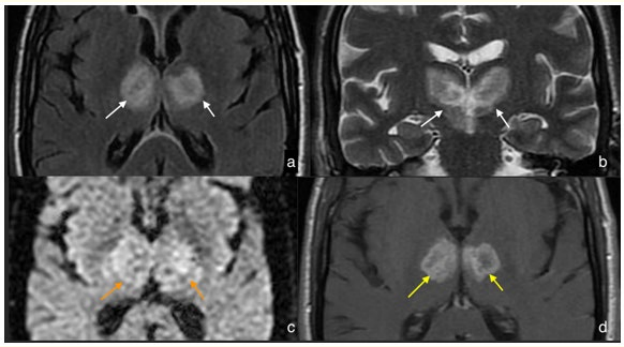

Figure 4: *Patient 4: Brain CT scan in axial planes (a), contrast enhanced CT (b) and brain MRI in axial planes in FLAIR sequence (c) and after injection of Gadolinium (d) objectified the presence of bilateral thalamic and midbrain hypodensity in spontaneous contrast that appear hyperintense in FLAIR sequences and show an annular enhancement.*

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

Taking into consideration the result of a thoraco-abdomino-pelvic CT and the aspect in the brain MRI, we evoked AOP infarction, and the embolic etiology was retained.

The patient then beneficiated of curative dose of anticoagulants. He stayed afterwards stationary in the neurological examination with confusional episodes.

Case presentation 5

A 22-year-old female patient, without any notable previous history, presented with the abrupt onset of altered consciousness with a GCS scale graded at 13. A neurological examination on admission was normal.

A brain CT scan revealed no abnormal high or low-density shadows. A cerebral MRI (Figure 5) objectified the presence of two bilateral thalamic lesions, described in T2 and FLAIR hypersignal, with a restrictive shell in diffusion.

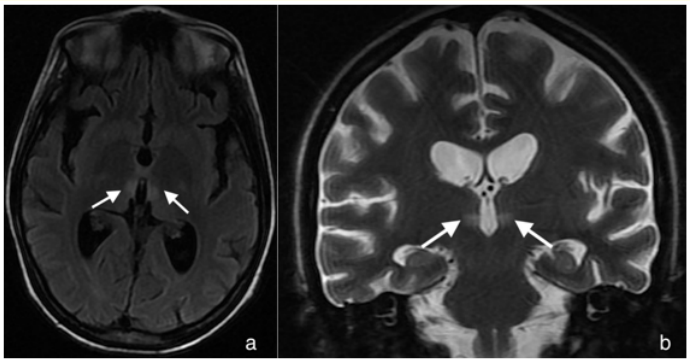

Figure 5: *Patient 5: MRI in axial sections, FLAIR (a) and T1 after injection of gadolinium (b) shows high signal in bilateral paramedian thalamic (green arrow) in FLAIR sequences (a), that are enhanced after injection of gadolinium (b).*

She was given a curative dose of anticoagulants, and after a week, her level of awareness had significantly improved with a GCS scale at 15.

The patient was afterwards diagnosed with lupus.

Case presentation 6

42-year-old female with an unremarkable medical history was admitted at the emergency department for the management of an acute impaired consciousness that appeared 6 hours before. The physical examination showed à GCS scaled at 9, his arterial blood pressure was normal 100/80 mmgh. A cardiological examination showed a regular sinus rhythm in the ECG.

A brain CT scan with iodine contrast injection was normal.

A brain MRI (Figure 6) demonstrate bithalamic paramedian lesions described in high signal in FLAIR and T2, restrictive in diffusion and show a mild enhancement.

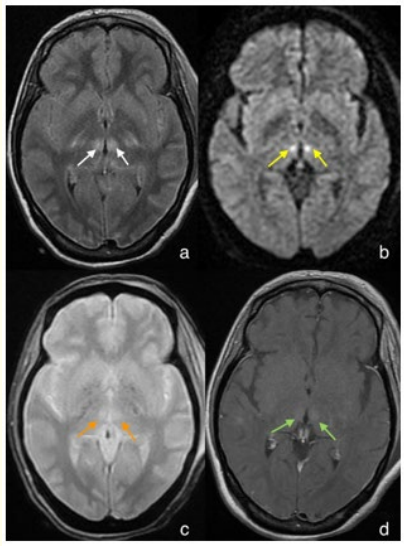

Figure 6: *Patient 6: Brain MRI in axial planes in FLAIR sequence (a), diffusion (b), Echo Gradient T2 (c) and T1 after injection of Gadolinium (d) demonstrate bithalamic paramedian lesions described in high signal in FLAIR, restrictive in diffusion and show a mild enhancement.*

	Age	Gender	Clinical exam	Delay	CT/MRI	CTA	Etiology	Treatment	Evolution
Cas 1	68	F	Impaired consciousness (GCS 8), symmetric drop of four limbs, abolished reflexes	20h	Paramedian bithalamic hypodensity, cortico-subcortical left occipital hypodensity, right lenticular lacuna (Figure 1)	Filling defect in the first portion of the PCA, hypoplasic posterior communicating arteries, left carotidian bulb plaque without significant stenosis	Atheromatous	ACT, APT	Improvement in the level of consciousness (GCS 15)
Cas 2	76	M	Impaired consciousness (GCS 10), vertical gaze palsy	3h	CT: Paramedian bithalamic hypodensity, MRI: Paramedian bithalamic high signal intensity, not restrictive in diffusion (Figure 2)	Permeable vertébro-basilar trunk, hypoplasic P1 bilaterally, left common internal carotidian plaque without significant stenosis	Atheromatous	IVT (3.5 cc of METALYSE)	No improvement in the level of consciousness, confusional episodes with behavioral problems, anterograde amnesic disorder

Cas 3	51	F	Impaired consciousness (GCS 9), left anisocoria	7.5h	CT: Paramedian bithalamic hypodensity	Permeable Willis polygone, left fetal type PCA	Atheromatous	ACT	Hypersomnia, retrograde memory disorders
Cas 4	64	M	Impaired consciousness (GCS 11)	24h	CT: Paramedian bithalamic hypodensity, with annular enhancement after iodine injection, MRI: paramedian bithalamic hyperintensity in T2 and FLAIR sequences, extending to the midbrain, with a restrictive shell in diffusion and annular enhancement after injection of Gadolinium (Figure 3 and 4)	Permeable Willis polygone, bilateral carotidian bulb plaque without significant stenosis	Embolic	ACT	No improvement in the level of consciousness, confusional episodes
Cas 5	22	F	Impaired consciousness (GCS 13)	4h	CT: normal, MRI: paramedian bithalamic hyperintensity in T2 and FLAIR, restrictive in diffusion (Figure 5)		Lupus	ACT	Improvement in the level of consciousness (GCS 15)
Cas 6	42	F	Impaired consciousness (GCS 9)	6h	CT: normal, MRI: paramedian bithalamic hyperintensity in T2 and FLAIR, restrictive in diffusion (Figure 6)				Death

Table 1: *Sociodemographic, clinical and radiological features of five cases of acute stroke due to the artery of Percheron occlusion in CHU Hassan II University Hospital of Fez.*

Discussion

Anatomic base

For the thalamus and midbrain, four neurovascular anatomical variations have been documented [2]. The Artery of Percheron, designated as Type IIb, is described as a single arterial trunk that originates from the P1 portion of the PCA, and supplies the bilateral paramedian thalamic regions and rostral midbrain [2]. (Figure 7).

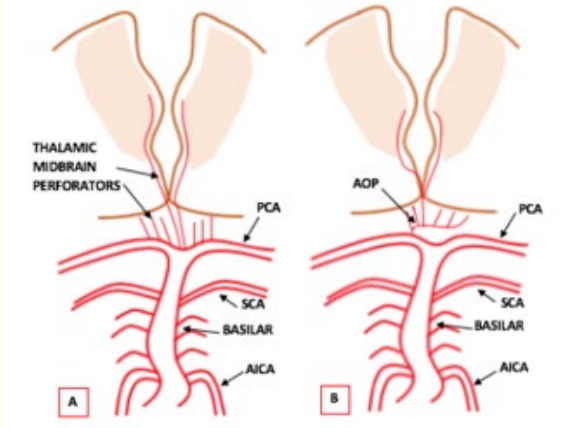

Figure 7: *Typical anatomy representing paired thalamic and midbrain perforating arteries. B, AOP rising as a solitary trunk from P1 supplying the both paramedian thalami and rostral midbrain (illustrating the figure from Lazzaro., et al [4]).*

Clinical presentation

A bilateral paramedian thalamic infarction is characterized by three symptoms: memory impairment, vertical gaze palsy, and altered mental status [2]. Vertical gaze palsies typically indicate mesencephalic participation, but they have also been perceived in cases with no midbrain lesions [4]. More severe memory impairments are linked to anterior thalamic involvement [2]. There have also been reports of other symptoms, including apathy, improper social behavior, and significant cognitive impairment [2].

In our series, all six patients presented with an acute impaired consciousness, and one of them with vertical gaze palsy. All of the patients kept a hypersomnia and two of them a memory disorder even at a distance of the infarction.

Imaging

In neuroimaging, four patterns of AOP infarction, consistent with well-known variants in the paramedian artery [4], have been defined so far, and bilateral paramedian thalamic region involvement is shared with each pattern [2]. Up to 43% of cases typically involve bilateral paramedian thalami and the midbrain [2].

Due to the difficulty of visualization of the AOP infarction in the hyperacute phase, its diagnosis is habitually made in the late phase [3]. On the other hand, no research has been done on how long it takes for an AOP infarction to fully manifest itself on diffusion weighted imaging (DWI) [3]. ADC and diffusion weighted images are now considered the "gold standard" for identifying and managing AOP infarction, especially in its initial phases, thanks to advancements in MRI technology [3]. Usual AOP infarction patients' brain MRIs show symmetrical long T1 and T2 signals in the paramedian bithalamic regions, a FLAIR high signal, a high DWI signal and a low apparent diffusion coefficient (ADC) signal in the initial phase [3]. According to Lazzaro., *et al*. (2010), 67% of cases with AOP infarction

demonstrated a high signal V-shaped intensity in FLAIR and DWI sequences in the pial surface of the midbrain in the interpeduncular fossa [3]. This suggested a connection between midbrain involvement and bilateral paramedian thalamus infarction [3].

A pathologic DWI and normal results on T2-weighted imaging suggests an acute stroke, much like it does in other locations in the brain [5]. The opportunity for thrombolysis is closed if the lesions are visible on T2 [5]. On CT images we can perceive a paramedian bithalamic hypodensity that might not exist during the acute phase [6]. The lesions appear six hours on average after the onset of clinical symptoms [6]. CT scan is however important to rule out cerebral bleeding at admission [6].

As with all strokes, non-invasive vascular imaging (CT angiography or angio-MRI) of the cerebral vessels and supra-aortic trunks is still required as part of the etiological work-up [7]. It should be done concurrently and will help rule out basilar trunk obstruction given the often-severe consciousness disruptions linked to Percheron's artery occlusion [7].

The AOP is so small in diameter that digital subtraction angiography (DSA) and magnetic resonance angiography (MRA) are unable to detect it, or even confirm its stenosis or occlusion [6].

Additionally, it has been reported that the role of the neuronal nuclei in the median parathalamic area can be assessed by single photon emission computed tomography (SPECT) of cerebral perfusion [3].

Nonetheless, a bilateral paramedian thalamic infarction may be associated with a concomitant embolism in two different paramedian arteries as part of a basilar trunk termination syndrome (fracturing of the embolus at the basilar trunk termination) [3-6].

All of our patients beneficiated of a CT scan, four of which presented a paramedian bithalamic hypodensity. Four of the patients had a CTA for intra-cranial vessels and supra-aortic trunks. A brain MRI was realized for four patients which revealed a high signal in paramedian bithalamic in FLAIR and T2 sequences, restrictive in diffusion for three of them.

Differential diagnosis

Bithalamic lesions have a broad imaging differential that involves the top of the basilar syndrome, hypoxic injury, thiamine deficiency, infections, demyelination and spongiform encephalopathy [8]. The patient's history, imaging features, and the existence or absence of lesions outside the thalami can all be used to restrict the differential diagnosis [8].

Primary neoplasm

Glioma: Bilateral thalamic glioma is a rare tumor, typically a diffuse low-grade astrocytoma (grade II according to the WHO) [9]. Classically, there is no enhancement seen in these lesions [10]. However, in high-grade lesions (grades III and IV), we observe an enhancement, hyperperfusion, central necrosis, and bleeding. Hypercellular tumors exhibit diffusion restriction [11].

Lymphoma: The thalami are frequently affected by primary central nervous system (CNS) lymphoma, an uncommon type of non-Hodgkin lymphoma [12]. High attenuation lesions, strongly enhanced, are visible on a CT scan [12]. Lesions on the MRI are hypointense to isointense to grey matter on T2W and T1W imaging with restricted diffusion [12]. In immunocompetent individuals, they show a homogeneous enhancement, but exhibit a central necrosis and ring enhancement in immunocompromised patients [12].

Metastasis

Patients who have several lesions and a known primary tumor should be suspected of having metastatic lesions [13]. They may exhibit distinct patterns of enhancement and be hemorrhagic, necrotic, or both [13].

Metabolic and toxic disorders

Wernicke encephalopathy, also known as alcoholic encephalopathy, is due to vitamin B1 deficiency [14]. On the MRI, we can find lesions contiguous to the third ventricle, the periaqueductal area and the tectal plate, the medial thalami, hypothalamus, mammillary bodies, and the nucleus of cranial nerve XII, that are described as a hyperintensity on T2WI and FLAIR sequences, a hypointensity in T1 sequences, and show a gadolinium enhancement [14] (Figure 8).

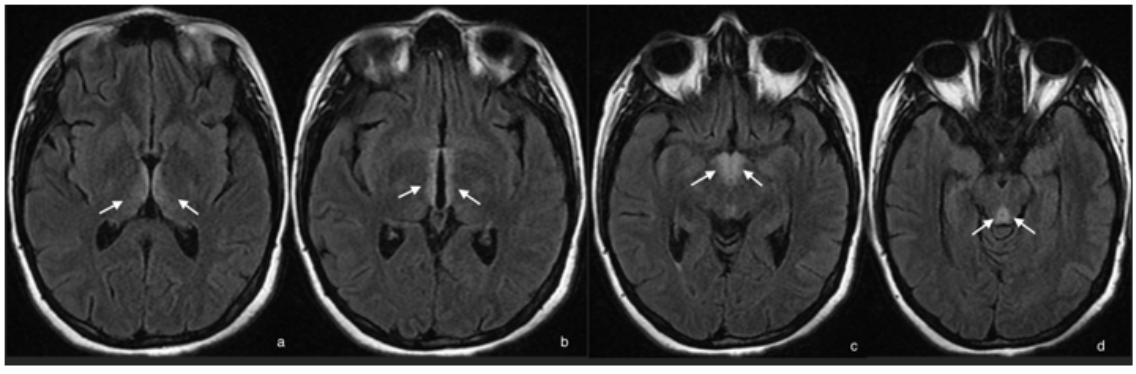

Figure 8: *MRI in axial sections in Flair sequence shows bilateral thalamic hyperintensity (a) around the third ventricle (b) in the mamillary bodies (c) and periaqueductal grey matter (d) concordant with a Wernicke's encephalopathy.*

Extrapontine myelinolysis occurs during a quick correction of hyponatremia and can affect the thalamic regions [15]. Within 24 hours after the onset of clinical symptoms, diffusion restriction is the earliest and only anomaly that has been recorded [15]. The FLAIR/T2-hyperintense signal emerges afterwards, followed by the T1-weighted signal alteration [15].

Wilson's disease: is an autosomal recessive disorder that generates a copper buildup in the liver and brain [13]. Copper deposits show as hypointense on T2WI and gradient echo (GRE) in the thalamus and lentiform nucleus on the MRI [13]. On T2WI and FLAIR, a high signal intensity is typically observed at the putamen's outer rim [13].

Vascular

Deep venous thrombosis (Figure 9): A bithalamic paramedian lesion can be caused by thrombosis of the internal cerebral veins and Rosenthal basal veins [16].

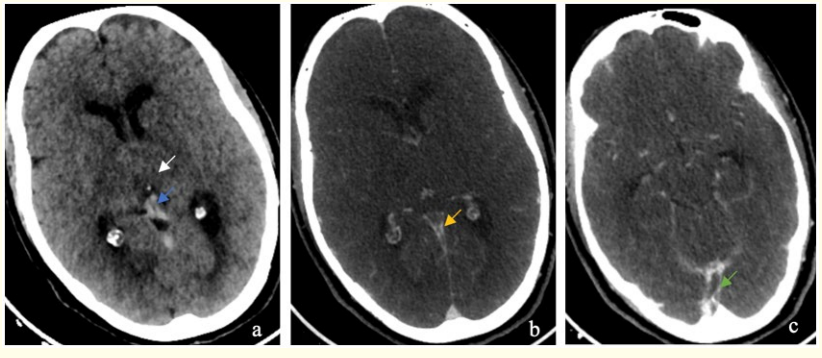

Figure 9: *Non contrast enhanced brain CT (a) and contrast enhanced CT (c) in axial planes show bithalamic paramedian hypodensity (white arrow) internal cerebral veins that appear spontaneously hyperdense (blue arrow), with an heterogenous enhancement (yellow arrow) extending to the straight sinus (green arrow), correlated to a deep venous thrombosis.*

Hypoxia/ischemia (Figure 10): Ischemic lesions in low-output situations are mainly found at specific sites called the watershed areas [13]. These areas are situated amongst two contiguous arterial territories that may involve the thalami [13]. CT and MRI results are similar to those found in ischaemia caused by arterial blockage, but the location of the lesions does not correspond to an arterial territory [13]. The "reversal sign," in which white matter exhibits greater attenuation than grey matter on CT, can be seen in cases of severe hypoxia [13].

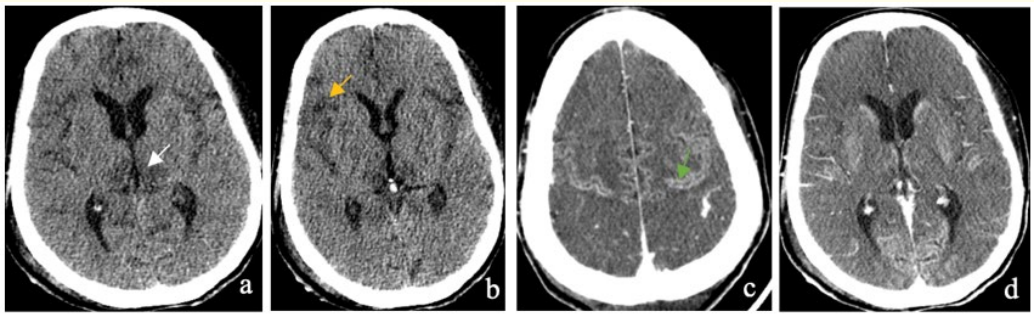

Figure 10: *Non contrast enhanced brain CT (a) and (b) and contrast enhanced CT (c) and (d) in axial planes show bithalamic paramedian hypodensity (white arrow) and a temporal hypodensity (yellow arrow). We can also see a bilateral frontal gyral enhancement (green arrow). This imaging findings correspond to a hypoxic ischemic encephalopathy.*

Infection

Many infectious agents, including bacterial (*Mycoplasma pneumoniae, Mycobacterium tuberculosis*) and viral (influenza A, parainfluenza), have a predilection for the thalamus [13].

The Creutzfeldt-Jacob illness, more precisely the pulvinar, as seen by MRI images that show T2WI/FLAIR hyperintensity and diffusion restriction of grey matter, which includes the cortex, thalami, and basal ganglia [17]. It is possible to find the "hockey stick sign" or "pulvinar sign," which is characterized by hyperintensity involving the bilateral dorsomedial and pulvinar thalamic nuclei on FLAIR sequences [17]. White matter is usually not engaged, and there is traditionally no enhancement [17].

Epilepsy

MRI could manifest anomalies in the thalamus, such as T2WI hyperintensity and diffusion restriction in the early postictal phase, following the status epilepticus, as well as in the event of a unique episode or cluster of epilepsy [18]. Most of these anomalies can be reversible [13].

Acute necrotizing encephalopathy of childhood

Acute necrotizing encephalopathy in children is characterized in neuroimaging with multiple symmetrical lesions in the thalami displaying T2 prolongation (Figure 11), often coupled with lesions in the cerebellum, putamina, periventricular white matter, and brain stem tegmentum [19].

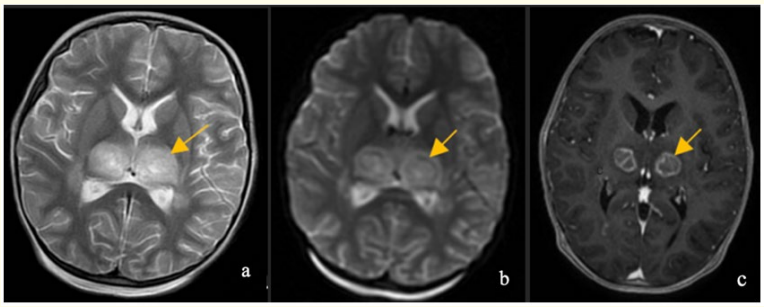

Figure 11: *MRI in axial sections in T2 (a), diffusion (b) and T1 3D C+ (c) sequences shows bilateral thalamic lesions (yellow arrow) described in hypersignal in T2 sequences, presenting a restriction in diffusion and describing the trilaminar appearance (b) and a peripheral enhancement after injection of gadolinium (c).*

Conclusion

It is unknown how often Percheron arteries arise as these vessels are typically not seen on angiography scans. The posterior cerebral fossa's numerous blood supply variations and the prevalence of P1 segments aplasia could suggest that the Percheron artery is not a particularly rare variation. We have to question if strokes in these regions are genuinely extraordinary or significantly underdiagnosed since they are rarely identified. When a patient experiences an abrupt onset of neurological deficit in one of the identified regions, we must diagnose a Percheron artery stroke and evaluate the possibility of thrombolytic therapy. Even though AOP infarction's characteristic imaging results are consistently identifiable, the majority of investigations have only detailed a small number of individual cases, and as far as we are aware, a thorough evaluation of its radiographic spectrum has not been recorded. It is essential to identify an AOP infarction in order to avoid unnecessary procedures and to manage the proper time-sensitive guidance.

Conflict of Interest

We have no known conflict of interest to disclose.

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