

Bilateral thalamic infarcts due to occlusion of the Artery of Percheron and discussion of the differential diagnosis of bilateral thalamic lesions

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ABSTRACT

The Artery of Percheron is a rare vascular variant in which a single dominant thalamoperforating artery arises from one P1 segment and bifurcates to supply both paramedian thalami. Occlusion of this uncommon vessel results in a characteristic pattern of bilateral paramedian thalamic infarcts with or without mesencephalic infarctions. We report a case of a 31-year-old man with acute bilateral thalamic infarcts and a truncated Artery of Percheron demonstrated on magnetic resonance angiography (MRA). Occlusion of the vessel was presumably due to embolism from a patent foramen ovale that was subsequently closed. The case presentation is followed by a discussion of bilateral paramedian thalamic infarcts including the causes and clinical presentation. The differential diagnosis of vascular and nonvascular etiologies of bilateral thalamic lesions is also discussed.

CASE REPORT

CASE REPORT

A 31-year-old man presented to the emergency department six days after experiencing visual, speech and gait disturbances. Over those six days, his symptoms improved and his sole remaining complaint was impaired vision. The only abnormality on the physical and complete neurological examinations was dysarthria. All laboratory blood tests were negative, including a profile for hypercoagulable state. He had a history of viral meningitis and seizures as a child. However, an audiovisual electroencephalogram showed no abnormalities.

Non-contrast magnetic resonance imaging (MRI) of the brain was performed and demonstrated areas of hyperintense signal in both thalami, left greater than right, on axial diffusion-weighted images [Figure 1]. Restricted diffusion was confirmed on the apparent diffusion coefficient (ADC) map. In addition, areas of increased signal corresponding to the abnormalities on diffusion-weighted images were present on fluid-attenuated inversion recovery (FLAIR) sequences [Figure 2]. Corresponding hypointense signal was demonstrated in these areas on T1 weighted images [Figure 3].

These findings were consistent with subacute infarcts in the bilateral paramedian thalami. An MRA of the head was performed, demonstrating a single common trunk arising from the left P1 segment with double-branching vessels distally (Figure 4). The diagnosis of occlusion of the vascular variant known as the Artery of Percheron was made. Although the branch supplying the right thalamus appeared more truncated compared to the left, the area of infarct in the left thalamus was more prominent compared to the area of infarct in the right thalamus. This may be due to the limitations of imaging in the case of very small vessels, or it may reflect recanalization of the branches by the time the patient presented with subacute infarcts in the bilateral thalami.

To further investigate the cause of stroke in this young patient, a transesophageal echocardiogram was obtained. A patent foramen ovale (PFO) was seen on transesophageal echo (TEE) and the patient subsequently underwent catheter-based PFO closure [Figure 5]. The patient was discharged following closure of the PFO with improving symptoms. Long term follow up information was not available.

DISCUSSION

Bilateral thalamic infarcts are rare occurrences, accounting for 22 to 35% of all thalamic infarcts [2,3]. The thalami contain nuclei that integrate cortical function and serve as pathway of communication across the cerebral cortex and midbrain. The medial and lateral geniculate nuclei are involved with visual and auditory function [4]. The pulvinar and lateral dorsomedial nuclei also participate with visual functions. The ventral posterior lateral and ventral posteromedial thalamic nuclei transmit somatosensory information. Motor signals travel through the ventral lateral and ventral anterior nuclei. [5]. The medial dorsomedial nucleus contributes to autonomic control and emotions. The thalamus is also responsible for regulating consciousness, sleep and alertness [6]. When the thalamus is infarcted, patients may have symptoms including vertical gaze palsy, memory impairment, confusion and coma. The patient's presenting symptoms discussed above included visual, speech and gait disturbances. A classic history of altered level of consciousness was not described, possibly due to the patient's late presentation for medical evaluation. Usually the vascular anatomy of the thalamus is from dual arterial contribution from both anterior and posterior intracranial circulations. The anterior thalamus is supplied by the thalamotuberal arteries arising from the posterior communicating artery via the anterior circulation. The paramedian thalamic and rostral midbrain territories are supplied by thalamoperforators, arterial branches of the P1 segments of the posterior cerebral arteries. Historically, Percheron described three variations in the vascular supply to the paramedian thalami. Type I is the most common variant, where a perforating artery arises from each P1 segment [7]. Type III was described as an arcade of perforating arteries arising from an artery bridging the bilateral P1 segments [7]. Type II, the Artery of Percheron, arises from one P1 segment and splits to supply the bilateral thalami and rostral midbrain [Figure 6].

If occluded, the Artery of Percheron is the only variant that results in bilateral paramedian thalamic infarcts, with or without midbrain involvement [8, 1]. Although rare, the Artery of Percheron has been demonstrated on MRA before [9].

The most common etiology of bilateral thalamic infarctions is cardioembolism, as was diagnosed in this case [3,10]. Some of the risk factors of stroke causing thromboembolism include atherosclerosis, atrial fibrillation, ventricular wall aneurysms, right-to-left shunts as well as hypercoagulable states, severely reduced left ventricular function, and vasoconstriction [11]. The mean age and sex predilection of bilateral thalamic infarcts secondary to occlusion of the Artery of Percheron are unknown due to its rarity. However, 58% of posterior cerebral artery infarcts affect men at a mean age of 61.5 years [11]. Treatment options include thrombolysis and medical therapy. Bilateral thalamic infarcts usually carry a favorable prognosis, although some patients experience persistent visual field deficits [2,11].

Other vascular etiologies that can cause bilateral thalamic infarctions are considered in this patient. Firstly, top of the

basilar syndrome, usually due to embolic disease, should be suspected in all patients who show the classic triad for this syndrome including complex ocular symptoms, impaired consciousness (agitation, memory dysfunction, coma) and long tract neurological signs [12]. The distal basilar artery bifurcates into the bilateral posterior cerebral arteries that supply branches to the posterior thalami, geniculate bodies and cerebral peduncle. The distal basilar artery also supplies the superior cerebellar arteries as well as branches to rostral pontine, median, paramedian, and lateral pontine perforators [7]. When the basilar artery is occluded, there are typically infarcts of not only the bilateral thalami but the posterior cerebral, superior cerebellar artery and pontine territories as well. Thrombosis of the basilar artery may appear as hyperintense signal within the vessel on T1 weighted images with absence of a flow void on T2 weighted images. If an MRA is performed a filling defect may be seen within the distal basilar artery. Infarcts associated with top of the basilar syndrome demonstrate restricted diffusion as well as hyperintense signal lesions on T2 weighted and FLAIR images and hypointense signal lesions on T1 weighted images. In this case, the basilar artery and posterior cerebral arteries are patent on MRA excluding the diagnosis of basilar artery occlusion.

In cases of bilateral thalamic infarcts, apart from the top of the basilar syndrome, deep cerebral venous thrombosis should be taken into consideration. Rarely, venous sinus thrombosis can present with isolated bilateral thalamic infarcts from occlusion of the internal cerebral veins, most notably the straight sinus. Signs and symptoms typically result from increases in intracranial pressure and include headache, vomiting and papilledema. Seizure, aphasia and focal neurologic deficits may ensue as a result of infarction. There are many predisposing factors, including hypercoagulability from oral contraceptives, pregnancy, malignancy, sinus and mastoid infections, inflammatory processes and mechanical compression. The cause is idiopathic in 25% of cases. The radiographic tests of choice for the diagnosis of deep central vein thrombosis are magnetic resonance imaging and magnetic resonance venography images. Isolated involvement of the deep central venous system (the internal cerebral veins, Vein of Galen, and straight sinus) presents as bilateral infarcts and hyperintense T2 and FLAIR signal involving the thalami. Additionally, the bilateral basal ganglia may also be affected by venous hypertension or infarction. Most of the time, deep central venous sinus thrombosis occurs as an extension of widespread superficial dural sinus thrombosis with involvement of the thalami, basal ganglia and cerebral cortex [12]. Hemorrhagic conversion is common and venous sinus thrombosis may be visualized on magnetic resonance venography (MRV) [12]. A filling defect may be seen on MRV or hyperintense signal within the vessel on T1 weighted images. In our case, the signal changes are confined to the bilateral thalami and patent flow voids are seen within the internal cerebral veins and venous sinuses on MRI.

Nonvascular etiologies such as metabolic and toxic processes, infection and neoplasm may also present with bilateral thalamic lesions and can mimic bilateral thalamic infarctions on imaging. Patient history, specific imaging

characteristics and the presence or absence of lesions outside of the thalami aid in narrowing the differential diagnosis [13]. In addition, there can be subtle differences in the MR appearance of these nonvascular causes that can help in differentiating these vascular and nonvascular etiologies.

Wernicke's encephalopathy results from a dietary vitamin B1 deficiency in patients with malnutrition or malabsorption and is frequently associated with chronic alcohol abuse. This neurologic disorder is classically characterized by ataxia, altered consciousness, anterograde amnesia and ocular dysfunction. MRI findings are represented by symmetric hyperintensity on T2-weighted and fluid-attenuated inversion recovery (FLAIR) images and corresponding hypointensity or no abnormalities on T1-weighted images within the medial thalami. The thalamic lesions can show restricted diffusion mimicking infarction on imaging. However, in addition to bilateral thalamic lesions, similar MR signal changes are seen in the periaqueductal gray, mamillary bodies, tectal plate and surrounding the third ventricle [12,4]. Following contrast, T1 weighted images may demonstrate enhancement of these anatomic structures, most notably the mamillary bodies which may be atrophied. A thorough review of the patient's history, physical examination and MRI of the brain is crucial in excluding Wernicke's encephalopathy from bilateral thalamic infarctions.

Next, extrapontine myelinolysis, resulting from the rapid correction of hyponatremia can also present with bilateral thalamic lesions. Cases have been reported in patients with chronic renal insufficiency, diabetes, liver disease, the syndrome of inappropriate antidiuretic hormone and disequilibrium syndrome [4]. Clinically, patients may exhibit spastic hemiparesis, pseudobulbar palsy, an altered level of consciousness or coma [12]. Isolated involvement of the bilateral thalami in cases of extrapontine myelinolysis is rare. Lesions from osmotic myelinolysis typically involve the putamen, caudate nucleus, internal, external and extreme capsules, lateral geniculate bodies and white matter as hyperintense signal on T2 weighted and FLAIR images and hypointense signal on T1 weighted images, possibly demonstrating restricted diffusion. Extensive involvement of these structures that are in addition to the bilateral thalamic signal changes help to differentiate extrapontine myelinolysis from bilateral thalamic infarcts.

Wilson's disease, an autosomal recessive disorder of copper metabolism, is another metabolic disorder that can cause hyperintense T2 and FLAIR signal lesions and hypointense T1 signal lesions within the bilateral thalami [4]. In the early phase of Wilson's disease, restricted diffusion may be seen within the thalami mimicking acute infarctions. Clinically, symptoms of Wilson's disease are usually detected in young adults and may include dysarthria, dystonia, tremors, ataxia, Parkinsonism and psychiatric manifestations [12]. Hypointense T2 and FLAIR signal lesions, likely due to paramagnetic properties of deposited copper deposition may also be noted in the lentiform nucleus as well as the thalami [4]. Additional lesions may be seen in the caudate nuclei, putamen, globus pallidus and pons with atrophy of the caudate nuclei and brainstem [14,4]. The patient's history, clinical

findings and extent of MR changes outside of the thalami aid in excluding Wilson's disease in our case.

Creutzfeldt-Jakob disease is a neurodegenerative disease due to the presence of prion proteins that may present clinically with rapidly progressive dementia, myoclonus and neurologic dysfunction [12]. Patients usually present with this disorder in the seventh decade of life [4]. There are four types of the disease including sporadic, iatrogenic, familial and variant forms [15]. Lesions associated with Creutzfeldt-Jakob disease in the brain have hyperintense T2 and FLAIR signal in the putamen, caudate nuclei, periaqueductal gray as well as in both thalami, aiding in the correct diagnosis [15]. Additional findings may include cerebral atrophy and symmetric hyperintense T2 and FLAIR signal in the occipital cortex and white matter. The "hockey stick sign", or symmetric hyperintense T2 and FLAIR signal in the bilateral pulvinar and dorsomedial nuclei of the thalami, is a classic finding in variant Creutzfeldt-Jakob disease, the rare infectious form. There is no evidence of restricted diffusion in the case of Creutzfeldt-Jakob disease. The absence of the pulvinar sign, lack of additional MR signal changes and the presence of restricted diffusion help to exclude the variant form of Creutzfeldt-Jakob disease in our case.

Bilateral thalamic glioma, a rare low-grade astrocytoma, can cause expansion of the thalami with hyperintense T2 and FLAIR signal and isointense T1 signal, sometimes resulting in hydrocephalus. These tumors are confined within the thalami and do not enhance. Although the distribution of a bilateral thalamic glioma may be similar to bilateral thalamic infarcts, the absence of restricted diffusion and correlation with clinical symptoms can lead to the correct diagnosis. Patients usually present as children or young adults with symptoms including personality changes or dementia and face a poor prognosis due to the tumor's deep location [12].

Conclusion

Bilateral thalamic infarcts are rarely encountered. Involvement of the paramedian thalamic territories is unusual and raises the suspicion of occlusion of a single arterial trunk known as the Artery of Percheron. Due to the small size of this artery, MRA evaluation is limited and therefore a review of the vascular and nonvascular causes of bilateral thalamic lesions should be made. Careful evaluation of the patient's history, clinical presentation together with imaging findings facilitates in making the correct diagnosis.

TEACHING POINT

Occlusion of a vascular variant known as the Artery of Percheron results in bilateral paramedian thalamic infarcts with or without midbrain involvement. The most common cause is cardioembolism. This type of infarct has a favorable prognosis.

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FIGURES

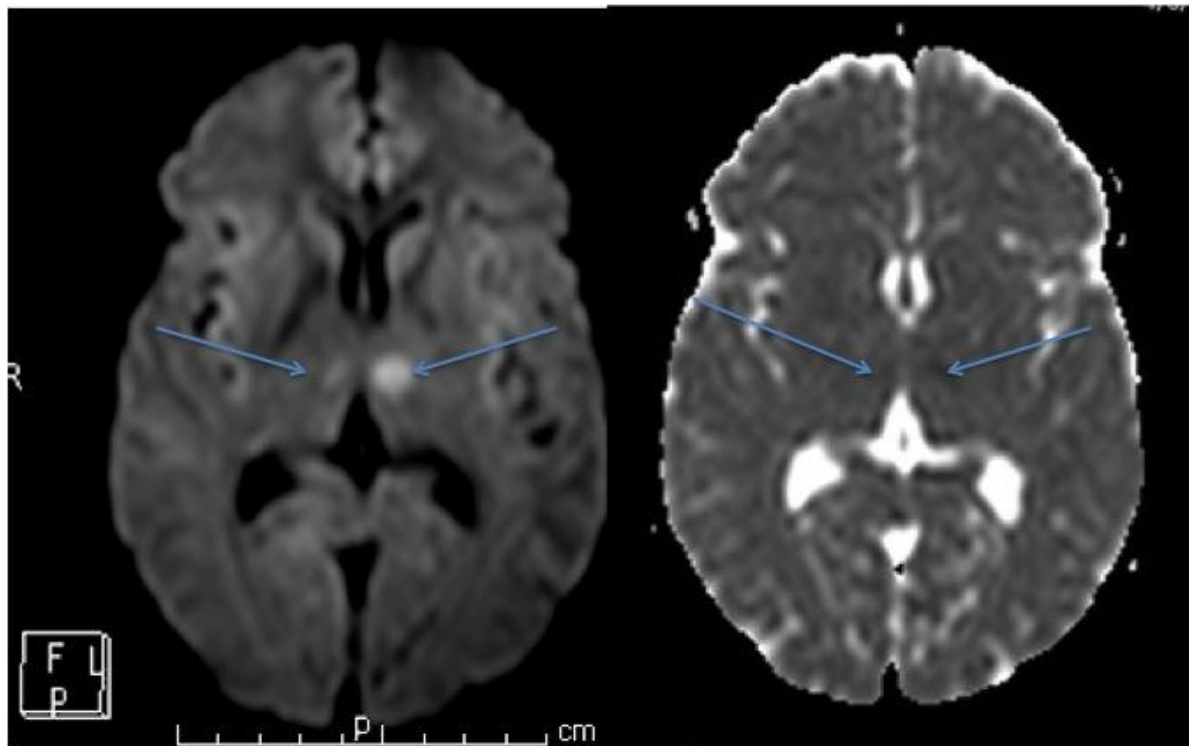


Figure 1: 31 year old man with bilateral thalamic infarcts due to cardioembolic occlusion of the Artery of Percheron. A: Diffusion weighted axial image (3T MRI, TR 2907.79, TE 68, 5mm slice thickness) showing hyperintense signal in bilateral thalami in the areas of infarct consistent with infarct (arrows). B: Axial ADC map (3T MRI, TR 2907.79, TE 68, 5mm slice thickness) shows decreased signal in bilateral thalami in the areas of infarct.

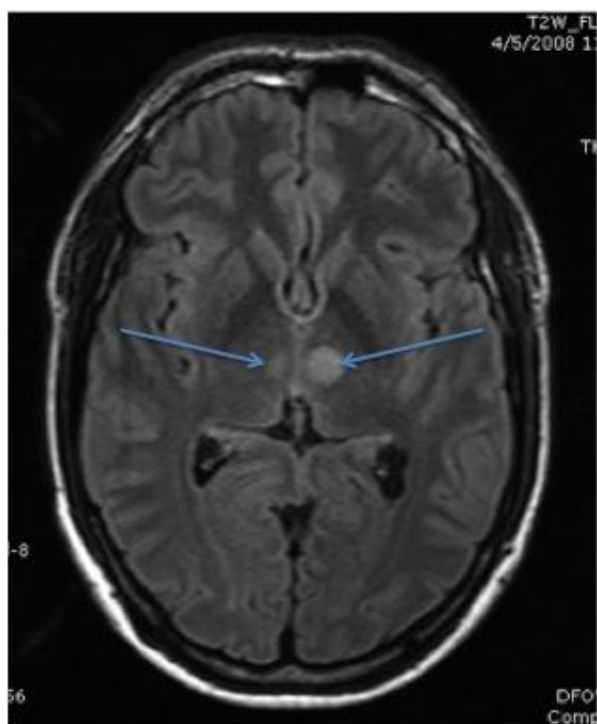


Figure 2: 31 year old man with bilateral thalamic infarcts due to cardioembolic occlusion of the Artery of Percheron. Axial FLAIR image (3T MRI, TR 11000, TE 125, 5mm slice thickness) demonstrating hyperintense signal in the bilateral thalami corresponding to areas of infarct (arrows).

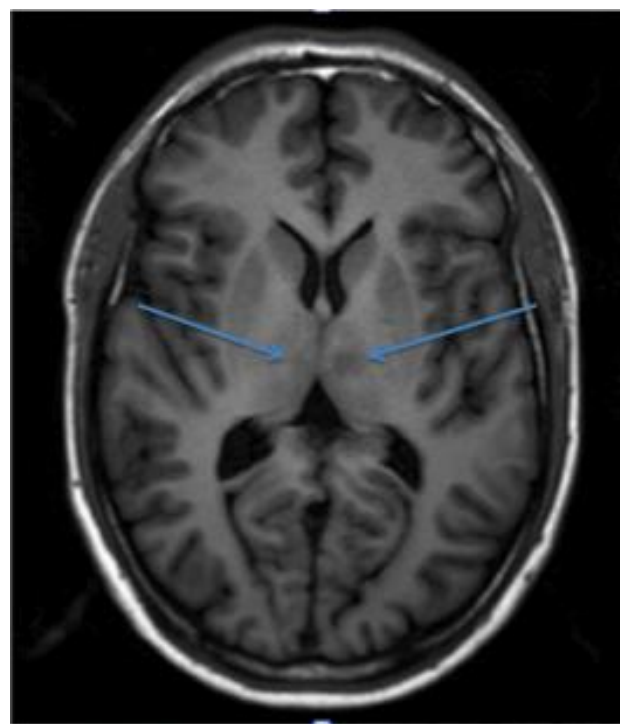


Figure 3: 31 year old man with bilateral thalamic infarcts due to cardioembolic occlusion of the Artery of Percheron. T1 weighted image (3T MRI, TR 2000, TE 10, 5mm slice thickness) showing hypointense signal within the bilateral thalami in the areas of infarct (arrows).

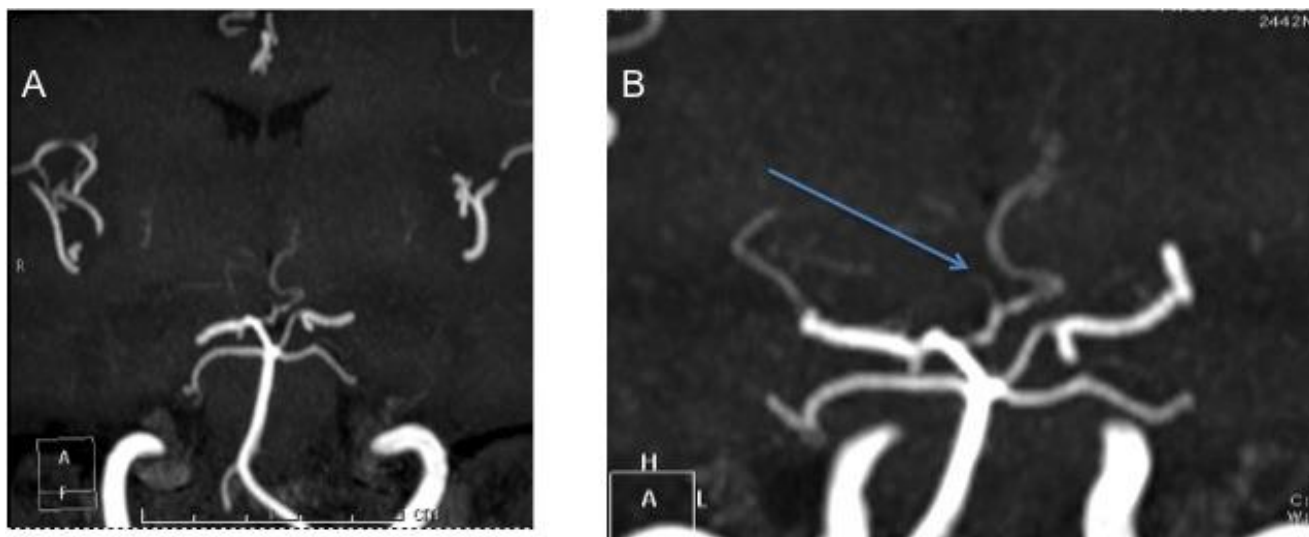


Figure 4: 31 year old man with bilateral thalamic infarcts due to cardioembolic occlusion of the Artery of Percheron. A: Coronal MRA image (3T MRI, TR 20, TE 3.45, source images 1.2mm thickness) of the posterior circulation showing a single branching vessel originating from the P1 segment of the right PCA which appears to supply both thalami. B: Zoomed image demonstrating single branching vessel originating from the P1 segment of the right PCA which appears to supply both thalami (arrow).

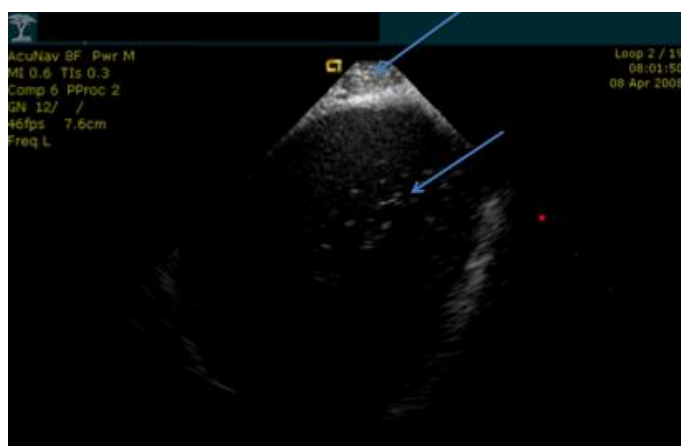


Figure 5 (left): 31 year old man with bilateral thalamic infarcts due to cardioembolic occlusion of the Artery of Percheron. Transesophageal echocardiographic image using a Philips IE33 ultrasound machine with 5 MHz transducer showing air bubbles on both sides of the interatrial septum (arrows), proving the presence of a patent foramen ovale.

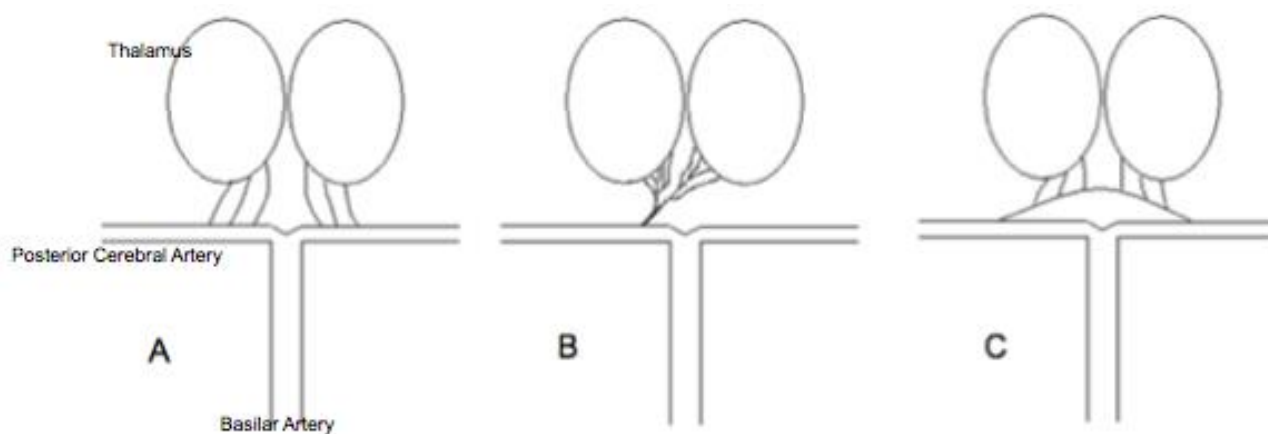


Figure 6: Three types of paramedian thalamic-mesencephalic arterial supply described by Percheron. A) Type I - Most common variant, where a perforating artery arises from each P1 segment. B) Type II - The Artery of Percheron arises from one P1 segment and splits to supply the bilateral thalami and rostral midbrain. C) Type III - An arcade of perforating arteries arising from an artery bridging the bilateral P1 segments.

Etiology	Thromboembolism
Incidence	22 to 35% of thalamic infarcts
Gender	Cases of posterior cerebral artery infarcts affect males 58% of the time, females 42%
Age	Mean age 61.5 years in cases of posterior cerebral artery infarct
Risk Factors	Patent foramen ovale, atherosclerosis, hypercoagulable state, atrial fibrillation, intracardiac clot, severely reduced left ventricular function (or regional wall akinesia), intracardiac clots, right-to-left shunts, wall aneurysms, and stroke related to cardiac procedures, vasoconstriction
Treatment	Thrombolysis, medical therapy
Prognosis	Favorable, possible visual field deficits
Findings on Imaging	Hypointense T1 signal, hyperintense T2 signal and restricted diffusion in areas of infarct

Table 1: Summary table for occlusion of the Artery of Percheron

	MRI T1	MRI T2/FLAIR	MRI DWI/ADC	MRA/MRV	Pattern of contrast enhancement
Artery of Percheron Occlusion	Hypointense signal in areas of infarct	Hyperintense signal in areas of infarct	Restricted diffusion in case of acute or subacute infarct	Rare Possible filling defect in single vessel arising from unilateral P1 segment on MRA	No enhancement
Top of the Basilar Syndrome	Hypointense signal in areas of infarct	Hyperintense signal in areas of infarct	Restricted diffusion in case of acute or subacute infarct	Filling defect on MRA in distal basilar artery	No enhancement
Deep Cerebral Venous Thrombosis	Hypointense signal in areas of infarct	Hyperintense signal in areas of infarct	Restricted diffusion in case of acute or subacute infarct	Filling defect on MRV, most commonly in straight sinus	No enhancement
Wernicke's Encephalopathy	Hypointense signal lesions	Hyperintense signal lesions	Possible restricted diffusion	No characteristic findings	Lesions may enhance
Extrapontine Myelinolysis	Hypointense signal lesions	Hyperintense signal lesions	Possible restricted diffusion	No characteristic findings	Lesions show variable enhancement
Wilson's Disease	Hypointense signal lesions	Hyperintense signal lesions	Possible restricted diffusion in early phase	No characteristic findings	No enhancement
Creutzfeldt-Jakob Disease	Hypointense signal lesions	Hyperintense signal lesions	No restricted diffusion	No characteristic findings	No enhancement
Bilateral Thalamic Glioma	Isointense lesions	Hyperintense signal lesions	No restricted diffusion	No characteristic findings	No enhancement

Table 2: Differential diagnosis table bilateral thalamic lesions

Function	Thalamic Nuclei
Visual	Medial Geniculate, Pulvinar, Lateral Dorsomedial
Auditory	Lateral Geniculate
Somatosensory	Ventral Posteromedial, Ventral Posterolateral
Motor	Ventral Lateral, Ventral Anterior
Autonomic control and emotion	Medial Dorsomedial

Table 3: Functions of Thalamic Nuclei

ABBREVIATIONS

MRA = magnetic resonance angiography
MRI = magnetic resonance imaging
ADC = apparent diffusion coefficient
FLAIR = fluid=attenuated inversion recovery
TEE = transesophageal echo
PFO = patent foramen ovale
MRV = magnetic resonance venography
DWI = diffusion weighted images

KEYWORDS

Artery of Percheron; bilateral thalamic infarcts; patent foramen ovale

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