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**Case Report**

# Bilateral thalamic infarcts due to occlusion of artery of percheron - a rare vascular variant

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## ABSTRACT

The artery of percheron is a rare vascular variant, occlusion of which is uncommon and associated with bilateral paramedian thalamic infarcts with or without mesencephalic infarctions. We report a case of a 50 year-old male patient with acute bilateral thalamic infarcts. Patient presented with history of sudden onset of altered sensorium with episodes of restlessness, agitation and loss of consciousness. Non-contrast CT scan of head was suggestive of bilateral paramedian thalamic infarcts.

**Keywords:** Artery of percheron, Thalamic infarcts

## INTRODUCTION

The artery of Percheron was first described in 1973 by the French medical scientist Gerard Percheron.[1]

The arterial supply of thalamus and midbrain is complex and is provided by perforating branches from the posterior cerebral artery and the posterior communicating artery. Many numbers of significant variations in vascular supply of thalami and mesencephalon are seen. The thalamic vascular supply is classically categorized into four territories: anterior, paramedian, inferolateral and posterior.[2]

The Artery of Percheron is a rare vascular variant in which a single dominant thalamo-perforating artery arises from one P1 segment of PCA 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.[3]

## CASE REPORT

A 50 years male patient presented with history of sudden onset of altered sensorium lasting for 12 hours. This was accompanied with frequent episodes of restlessness, agitation and loss of consciousness.

Detailed history was taken and all possible causes were excluded (like trauma, headache, head injury, seizure, fever, jaundice, loose stools, constipation, breathlessness, distension of abdomen, swelling of feet, decreased urine output, any bleeding manifestations, snake bite, etc.).

Patient was smoker, and alcoholic. History of occasional drug abuse (cannabis) was present.

There was no significant past history and family history related to patients complains.

On Examination: Patient was confused, and was not well oriented to time, place and person. Patient was obeying commands and was not able to open eyes. Vitals were

normal. No other abnormal finding was found on general physical examination.

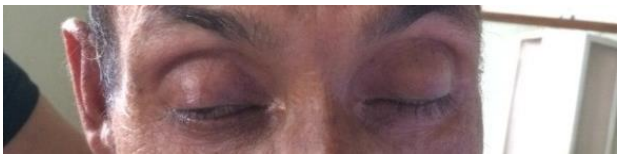

**Figure 1: Case image reveals that patient is not able to open eyes (Bilateral ptosis).**

No neurological deficit except bilateral ptosis was seen on detailed neurological examination. No abnormal finding was detected on systemic examination.

Investigations: Hb-16.1 g/dl, RBCs-5.02 million/microlitres, Hematocrit-47.6%, MCV-94.8 fl, MCH-32.8 pg, MCHC-33.8g/dl, Platelet count-1,65,000/microlitres, WBCs-12,300 , DLC (N-58%, L-36%, M-04%, E-02%, B-00%), ESR-28mm at 1st hour (westergren method), RBS-70mg/dl, Sodium-141.3mmol/L, Potassium- 4.18mmol/L, Chloride-107.9mmol/L, BUN-8mg/dl, Creatinine-0.9mg/dl, Bilirubin (Total-0.6mg/dl, Conjugated-0.13mg/dl), AST (SGOT)- 56U/L, ALT (SGPT)- 36U/L, ALP-88U/L, Protein (Total-5.7g/dl, Albumin-3.1g/dl)

Non-contrast CT scan of head was suggestive of bilateral thalamic infarct (Figures: 2a, 2b and 2c).

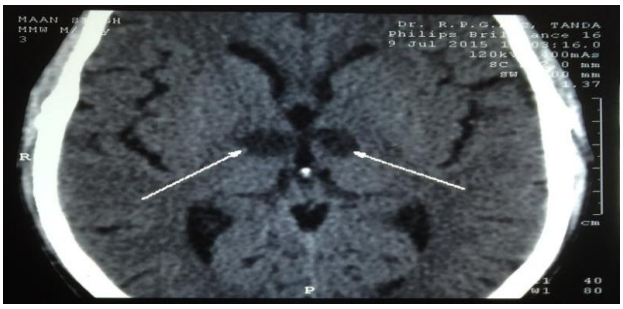

**Figure 2a: Axial CT section (magnified) at the level of thalami showing bilateral symmetrical paramedian infarcts (arrows).**

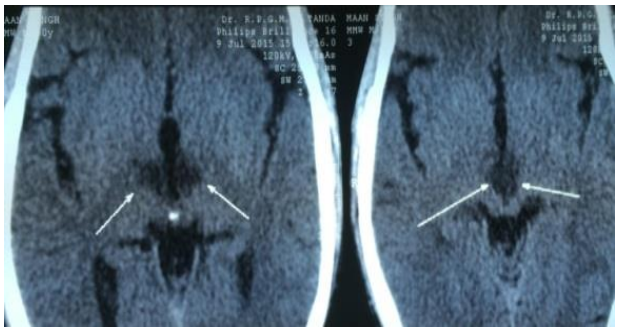

**Figure 2b: Axial CT sections show extension of infarct into bilateral cerebral peduncle and anterior mesencephalon (arrows).**

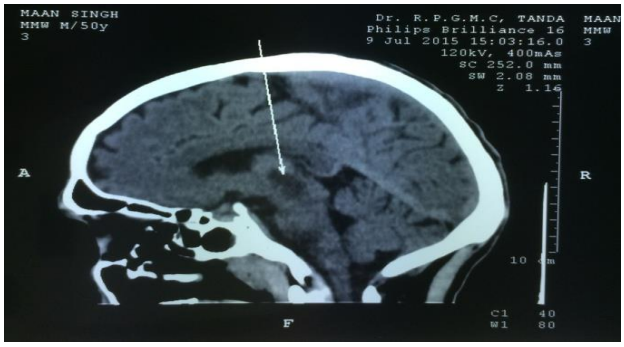


**Figure 2c: Sagittal CT section showing thalamic infarct extending into cerebral peduncle and midbrain (arrow).**

Patient was treated with aspirin, statins and supportive tharapy. Daily bedside follow was done. Frequent episodes of restlessness, agitation and loss of consciousness were noticed however patient was arousable and was obeying commands. Bilateral ptosis was persisting during patient's stay in hospital for one week. Rest of symptoms show gradual improvement during hospital stay. Patient lost to follow up thereafter.

## DISCUSSION

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.[4]

Bilateral thalamic infarcts are rare occurrences, accounting for 22 to 35% of all thalamic infarcts.[5,6] 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.[7]

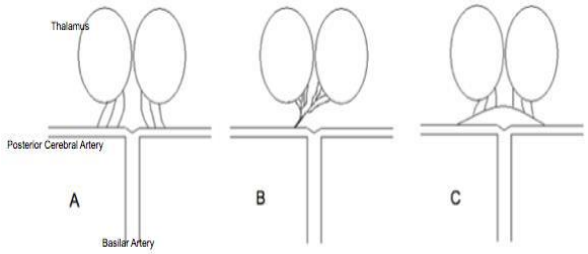


**Figure 3: Paramedian thalamo -mesencephalic arterial supply.**

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.[8] The medial dorsomedial nucleus contributes to autonomic control and emotions. The thalamus is also responsible for regulating consciousness, sleep and alertness.[9] When the thalamus is infarcted, patients may have symptoms including vertical gaze palsy, memory impairment, confusion and coma.

Historically, Percheron described three variations in the vascular supply to the paramedian thalami. Three types of paramedian thalamic-mesencephalic arterial supply described by Percheron (Figure 3):

- Type I - Most common variant, where a perforating artery arises from each P1 segment.
- Type II - The Artery of Percheron arises from one P1 segment and splits to supply the bilateral thalami and rostral midbrain.
- Type III - An arcade of perforating arteries arising from an artery bridging the bilateral P1 segments.

Occlusion of Artery of Percheron produces paresis of upward gaze, drowsiness & often abulia.[4]

If occluded, the Artery of Percheron is the only variant that results in bilateral paramedian thalamic infarcts, with or without midbrain involvement.[4,11] Although rare, the Artery of Percheron has been demonstrated on MRA.[12]

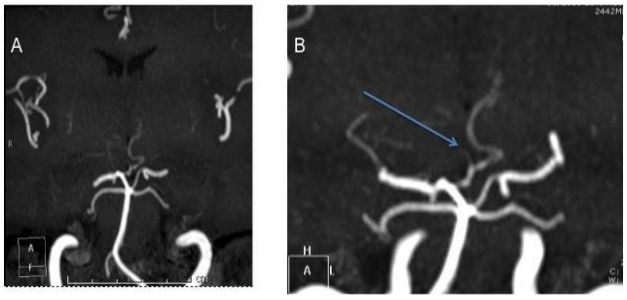


**Figure 4: Bilateral thalamic infarcts due to cardio-embolic occlusion of the Artery of Percheron. A: Coronal MRA image (3T MRI,TR 20, TE 3.45, source images 1.2mm thickness) of the posterior circulation showing a single branching vessel originating from the P1 segment of the right PCA which appears 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).**

The most common etiology of bilateral thalamic infarctions is cardio-embolism, as was diagnosed in this case.[6,13] Some of the risk factors of stroke causing thromboembolism include atherosclerosis, atrial fibrillation, ventricular wall aneurysms, right-to-left shunts as well as hyper-coagulable states, severely reduced left ventricular function, and vasoconstriction.[14] 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.[14] Treatment options include thrombolysis and medical therapy. Bilateral thalamic infarcts usually carry a favorable prognosis, although some patients experience persistent visual field deficits.[2,14]

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.[15] 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.[10]

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 hyper-intense 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 hyper-intense signal lesions on T2 weighted and FLAIR images and hypo-intense 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 hyper-intense 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.[15] Hemorrhagic conversion is common and venous sinus thrombosis may be visualized on magnetic resonance venography (MRV).[15] A filling defect may be seen on MRV or hyper-intense signal within the vessel on T1 weighted images.

## 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.

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