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Artery of Percheron Infarction Presenting as Bilateral Thalamic Stroke with Hemorrhagic Transformation: A Case Report highlighting diagnostic challenges

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Case Report

Keywords: Artery of Percheron, Bilateral thalamic infarction, Hemorrhagic transformation, Stroke, NIHSS, Posterior circulation, Atrial fibrillation, Neuroanatomy

Posted Date: April 19th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-9310453/v1

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Additional Declarations: No competing interests reported.

Abstract

Background: The artery of Percheron (AoP) is a rare anatomical variant in which a single perforating trunk arising from one P1 segment of the posterior cerebral artery supplies both paramedian thalami bilaterally. AoP occlusion accounts for approximately 0.1–2% of all ischaemic strokes and classically presents with altered consciousness, vertical gaze palsy, and memory impairment. Early CT findings are frequently non-specific, leading to diagnostic delay. Haemorrhagic transformation is a recognised but infrequently reported complication that may worsen prognosis and complicate anticoagulation decisions when a cardioembolic source is identified.

Case Presentation: A 63-year-old man with known hypertension and previously undiagnosed atrial fibrillation was found unresponsive at home and presented to our centre after an 11-hour delay. Despite initial profound unresponsiveness, his admission NIHSS was 3/42 and GCS was 15/15 with preserved orientation; however, upward vertical gaze palsy, anisocoria, and left ptosis were noted. Non-contrast CT revealed bilateral thalamic hypodensities. MRI demonstrated bilateral paramedian thalamic infarcts with restricted diffusion and blooming artefacts on susceptibility-weighted imaging consistent with haemorrhagic transformation. A concurrent left occipital infarct supported a cardioembolic mechanism. Haemorrhagic transformation precluded anticoagulation, and the patient was managed with antiplatelets, high-intensity statins, and blood pressure control. On day three, the patient’s neurological status deteriorated rapidly, requiring intubation. He was discharged against medical advice on day four and died the following day.

Conclusions: This fatal case illustrates the treacherous trajectory of AoP infarction, in which a deceptively low NIHSS score reflecting the well-documented insensitivity of this scale to posterior circulation deficits may mask the severity of bilateral thalamic injury. Haemorrhagic transformation complicated management by precluding anticoagulation for newly diagnosed atrial fibrillation. Clinicians should maintain a high index of suspicion for AoP infarction when bilateral thalamic lesions are encountered and recognise that the NIHSS alone is insufficient to gauge severity in thalamic and brainstem strokes.

Background

The thalamus has a complex and highly variable arterial supply derived primarily from perforating branches of the posterior circulation. Classically, four vascular territories are described: anterior (tuberothalamic artery), paramedian (thalamoperforating arteries), inferolateral (thalamogeniculate arteries), and posterior (posterior choroidal arteries) territories(Fig. 1). [1,2]

The paramedian thalamic region is supplied by thalamoperforating arteries arising from the P1 segment of the posterior cerebral artery (PCA). In a rare anatomical variant first described by Percheron, a single dominant perforating trunk arises from one P1 segment and supplies both paramedian thalami bilaterally, with or without extension to the rostral midbrain. This configuration is known as the artery of Percheron (AoP). [1]

AoP infarction accounts for approximately 0.1–2% of all ischemic strokes and up to 18% of thalamic infarcts.[2] We report a case of bilateral thalamic infarction with hemorrhagic transformation and an associated left occipital infarct .

Case Presentation

A 63-year-old right-handed male was found lying on the floor of his home at approximately 11:00 AM with decreased responsiveness. He was last known well at approximately 7 AM while performing routine morning activities. Relatives found him unconscious on the floor after noticing an overfilled rooftop water drum. He was able to grasp their hand weakly with his left hand but not the right. There was no history of fever, vomiting, abnormal body movements, urinary or fecal incontinence, or vision loss. He was initially evaluated at a local hospital and referred to our center, arriving at 6:00 PM.

Past medical history revealed hypertension under amlodipine 5mg for 12 years and Psychiatric illness under amitriptyline, escitalopram, olanzapine, haloperidol, propranolol, trihexyphenidyl. On arrival, ECG showed atrial fibrillation with rapid ventricular rate (160 bpm), previously undiagnosed. He consumed tobacco daily and alcohol occasionally. No history of diabetes, dyslipidemia, smoking, prior transient ischemic attack or stroke were present.

Despite decreased responsiveness at home, on arrival at the emergency department, The National Institutes of Health Stroke Scale (NIHSS) score on admission was 3/42, reflecting a mild neurological deficit and his GCS was E4V5M6 (15/15). His vital signs were: HR 95 bpm, BP 200/120 mmHg, RR 18/min, SpO 98% on room air, and temperature 98.5°F. He was well oriented to time, place, and person, and could follow verbal commands with preserved attention and memory.

Neurological examination revealed upward vertical gaze palsy, anisocoria (right pupil 2 mm, left pupil 4 mm, both reactive), and left eye ptosis with preserved extraocular movements bilaterally. No overt mutism or abulia was present. Motor examination showed symmetric bulk with normal tone; power was 4/5 in the right upper and lower limbs and 5/5 on the left. Deep tendon reflexes were 2 + throughout with bilateral downgoing plantar responses. Superficial, deep, vibration, and proprioceptive sensations were intact. Remaining cranial nerve examination was unremarkable, including intact facial sensation

and movement, normal hearing, and preserved gag reflex without tongue fasciculations. Cerebellar finger-nose testing and rapid alternating movements were normal; gait could not be assessed as the patient was bedridden. The combination of anisocoria and ptosis suggested likely involvement of the rostral midbrain in addition to paramedian thalamic infarction.

An urgent non-contrast CT head with 3D face reconstruction revealed bilateral thalamic hypodensities, raising concern for infarction versus infectious or metabolic etiologies. No fractures were noted. Subsequently, the MRI brain revealed bilateral thalamic infarcts showing T2/FLAIR hyperintensities (A&B) and T1 hypointensity (C). The lesions demonstrated restricted diffusion [DWI hyperintensity (D) with corresponding ADC hypointensity (E)] consistent with acute ischemic infarct. On SWI, areas of blooming artifacts were noted (F), suggestive of hemorrhagic transformation.(Fig. 2)

The patient was started on antiplatelets and high-intensity statin therapy along with blood pressure control. On the third day of admission, he developed acute deterioration in sensorium, became disoriented, and progressed to decreased consciousness. He was intubated and mechanically ventilated for airway protection. He was discharged against medical advice on day four. The family was contacted by telephone and they reported that the patient had died 1 day after discharge. No further clinical details regarding the terminal course were available, and post-mortem examination was not performed.

Discussion and Conclusions

Percheron described four variants of paramedian thalamic arterial supply [1]. Variant I: bilateral perforators from each PCA (most common); Variant IIa: asymmetric perforators from one PCA; Variant IIb (AoP): single trunk supplying both paramedian thalami; and Variant III: arterial arcade bridging P1 segments. Our case corresponds to Variant IIb (AoP).(Fig. 3) The image showing the four variants is taken with permission from Elsevier from the article by Ghita Lahnine et al.[3]

Because the AoP is a small perforator, it is rarely visualized on conventional angiography; thus diagnosis is primarily clinico-radiological [1].

Artery of Percheron infarction predominantly involves the paramedian thalamic nuclei including the intralaminar, anterior, and mediodorsal nuclei rather than the inferolateral territory containing the ventral posterolateral and posteromedial nuclei responsible for somatosensory relay. The intralaminar nuclei are integral to the ascending reticular activating system, explaining the characteristic alteration of consciousness. Paramedian infarcts frequently extend to the rostral midbrain, where vertical gaze centres including the rostral interstitial nucleus of the medial longitudinal fasciculus and interstitial nucleus

of Cajal are located, accounting for the vertical gaze palsy observed in our patient. This selective involvement of paramedian thalamic–mesencephalic networks produces the clinical constellation of altered consciousness, oculomotor abnormalities, and executive dysfunction, while primary sensory modalities are typically spared consistent with the intact sensory examination in this case.[1,4,5].

Hemorrhagic transformation in ischemic stroke mainly occurs in patients with advanced age, stroke severity (Higher NIHSS score), hypertension, hyperglycemia, poor collaterals, early infarction on brain imaging, low platelet count, use of antithrombotic drugs, and reperfusion therapy. [6] In our patient, advanced age and severely uncontrolled hypertension at presentation (200/120 mmHg) likely contributed to haemorrhagic conversion, which was identified on susceptibility-weighted imaging as blooming artefacts within the bilateral thalamic infarcts.

The fatal outcome in our patient likely resulted from a confluence of adverse prognostic factors. The admission NIHSS score of 3/42 appeared deceptively reassuring given his preserved consciousness and orientation; however, the NIHSS systematically underestimates posterior circulation strokes because it does not capture vertical gaze palsy, pupillary abnormalities, or thalamic cognitive dysfunction all of which were prominent features in this case.[7]The subsequent rapid deterioration on day three, progressing from full orientation to decreased consciousness requiring intubation, illustrates the unpredictable and treacherous clinical trajectory of AoP infarction, in which involvement of the intralaminar nuclei and reticular activating system can lead to abrupt loss of consciousness.Çetin et al. studied 10 patients with AoP infarction and found that all patients had cognitive impairment, consciousness and behavioral disorder (mutism, confusion, and loss of initiation) at the onset of stroke. Although the patients survived, 50% of them had persistent cognitive deficits at 12 months.[8]Notably, none of their patients had concurrent haemorrhagic transformation or newly diagnosed atrial fibrillation features that likely contributed to the more severe course and fatal outcome observed in our case.

A notable feature of this case is the significant delay between symptom onset and hospital arrival. The delay of 11 hours precluded any consideration of acute reperfusion therapies, including intravenous thrombolysis or endovascular intervention, which may have been relevant given the likely cardioembolic mechanism. In resource-limited settings such as Nepal, delayed presentation is common due to geographic barriers, initial evaluation at peripheral health facilities without neuroimaging capability, prolonged inter-hospital transfer times, and limited public awareness of stroke symptoms.

The identification of previously undiagnosed atrial fibrillation with rapid ventricular rate (160 bpm) on the admission ECG strongly suggests cardioembolic aetiology. The

concurrent left occipital infarct, involving a vascular territory distinct from the paramedian thalamic territory, further supports cardioembolism, as multifocal infarcts across different vascular distributions are characteristic of a proximal embolic source. In this context, long-term oral anticoagulation would constitute the standard of care for secondary stroke prevention. However, the early haemorrhagic transformation identified on susceptibility-weighted imaging precluded the safe initiation of anticoagulation during the acute phase, and the patient was managed with antiplatelets a suboptimal but pragmatically safer approach given the haemorrhagic complication.

The principal differential diagnoses of bilateral thalamic lesions include top of the basilar syndrome, which typically involves additional vascular territories with asymmetric distribution and visual field deficits; deep cerebral venous thrombosis, characterised by headache, seizures, and papilledema; infectious or metabolic encephalopathies, which do not respect vascular territories; and infiltrative neoplasm, which follows a gradual non-vascular pattern.[9]

This case has several limitations. First, although MRI of the brain was performed and confirmed bilateral paramedian thalamic infarction, CT angiography and MR angiography could not be obtained as the patient's family declined further imaging due to financial constraints. Consequently, the posterior circulation anatomy could not be directly visualised, and the diagnosis of AoP infarction remains clinico-radiological.

AoP infarction should be considered in any patient presenting with acute altered consciousness and bilateral thalamic lesions, particularly when accompanied by vertical gaze palsy and oculomotor abnormalities suggesting rostral midbrain involvement.

Abbreviations

ADC
Apparent Diffusion Coefficient
AJNR
American Journal of Neuroradiology
AoP
Artery of Percheron
BP
Blood Pressure
bpm
Beats Per Minute
COMSTH-IRC
College of Medical Sciences Teaching Hospital – Institutional Review Committee
CT

Computed Tomography
CTA
Computed Tomography Angiography
DWI
Diffusion-Weighted Imaging
ECG
Electrocardiogram
FLAIR
Fluid-Attenuated Inversion Recovery
GCS
Glasgow Coma Scale
MRA
Magnetic Resonance Angiography
MRI
Magnetic Resonance Imaging
NIHSS
National Institutes of Health Stroke Scale
P1
First Segment of the Posterior Cerebral Artery
PCA
Posterior Cerebral Artery
SWI
Susceptibility-Weighted Imaging

Declarations

Ethics approval and consent to participate

Our institution does not require ethical approval for reporting individual cases or case series.

Consent for publication

Written informed consent was obtained from next to kin of the patient for participation and publication of this case report and any accompanying data. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Availability of data and materials

Not applicable

Competing interests

The authors declared no competing interests with respect to the research, authorship, and/or publication of this article.

Funding

The authors received no financial support for the research, authorship, and/or publication of this article.

Authors’ contributions

PP and AKJ contributed to the clinical management of the patients and supervised the manuscript. ASK, UKS, RBK, AK, and SD contributed to the history taking, examination, monitoring of the patient’s condition, wrote the original draft of the manuscript. SJ did the imaging and interpreted the imaging findings. All authors read and approved the final manuscript.

Acknowledgements

Not applicable.

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Figures

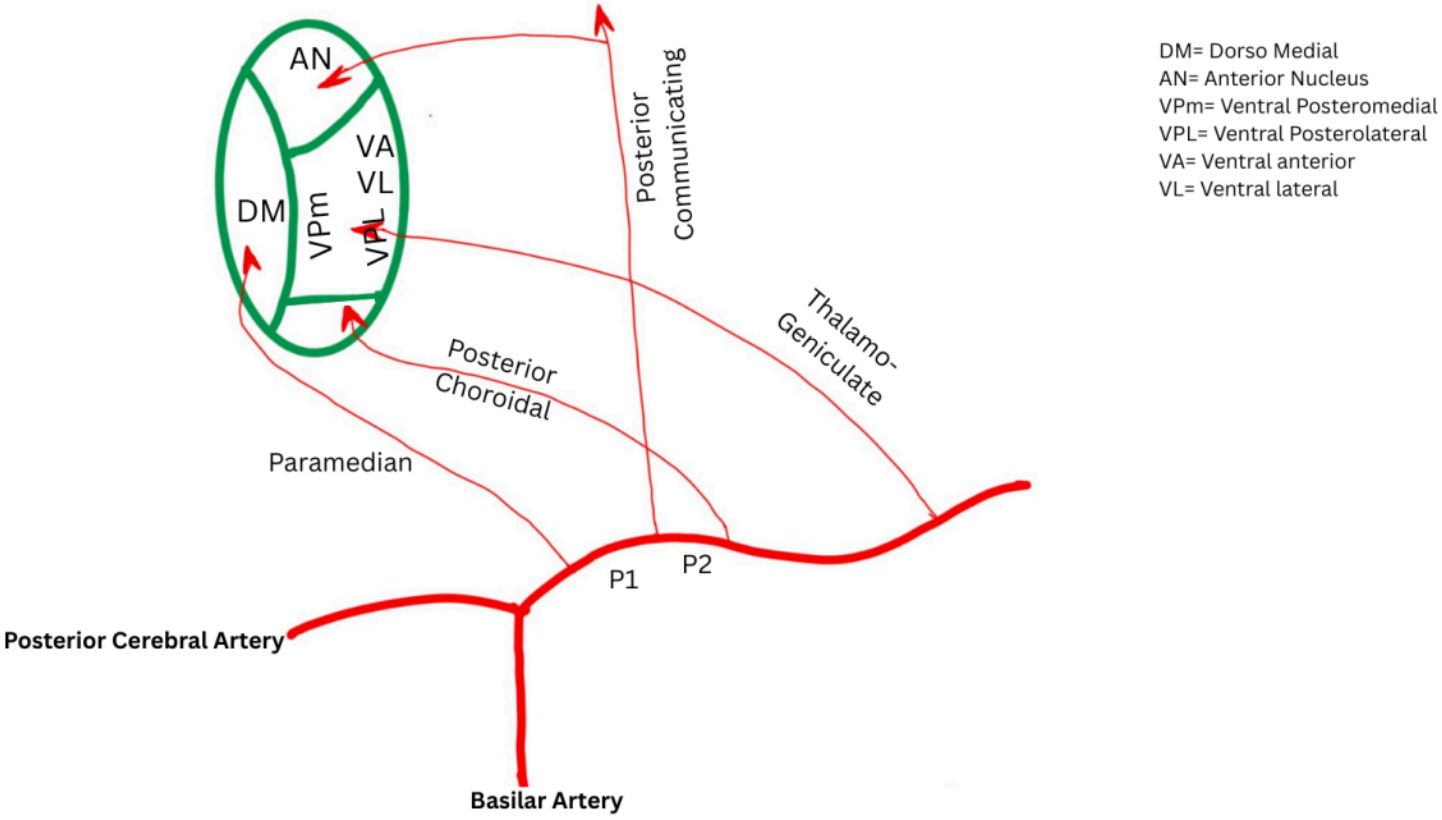


Figure 1

Showing four vascular territories of the thalamus

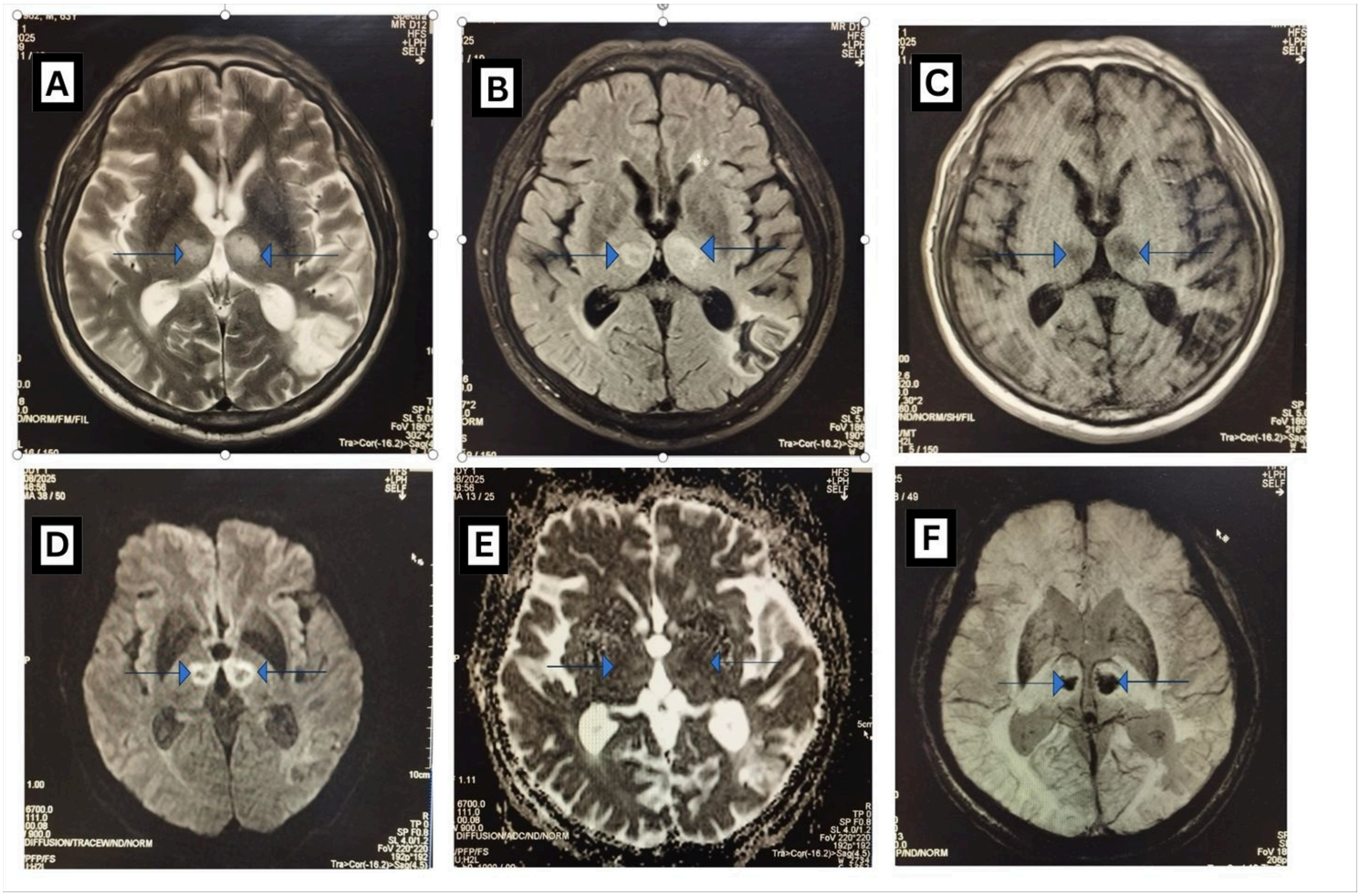


Figure 2

MRI brain reveals bilateral thalamic infarcts showing T2/FLAIR hyperintensity (A&B) and T1 hypointensity (C). The lesions demonstrate restricted diffusion [DWI hyperintensity (D) with corresponding ADC hypointensity (E)] consistent with acute ischemia. On SWI, areas of blooming artifact are noted (F), suggesting hemorrhagic transformation

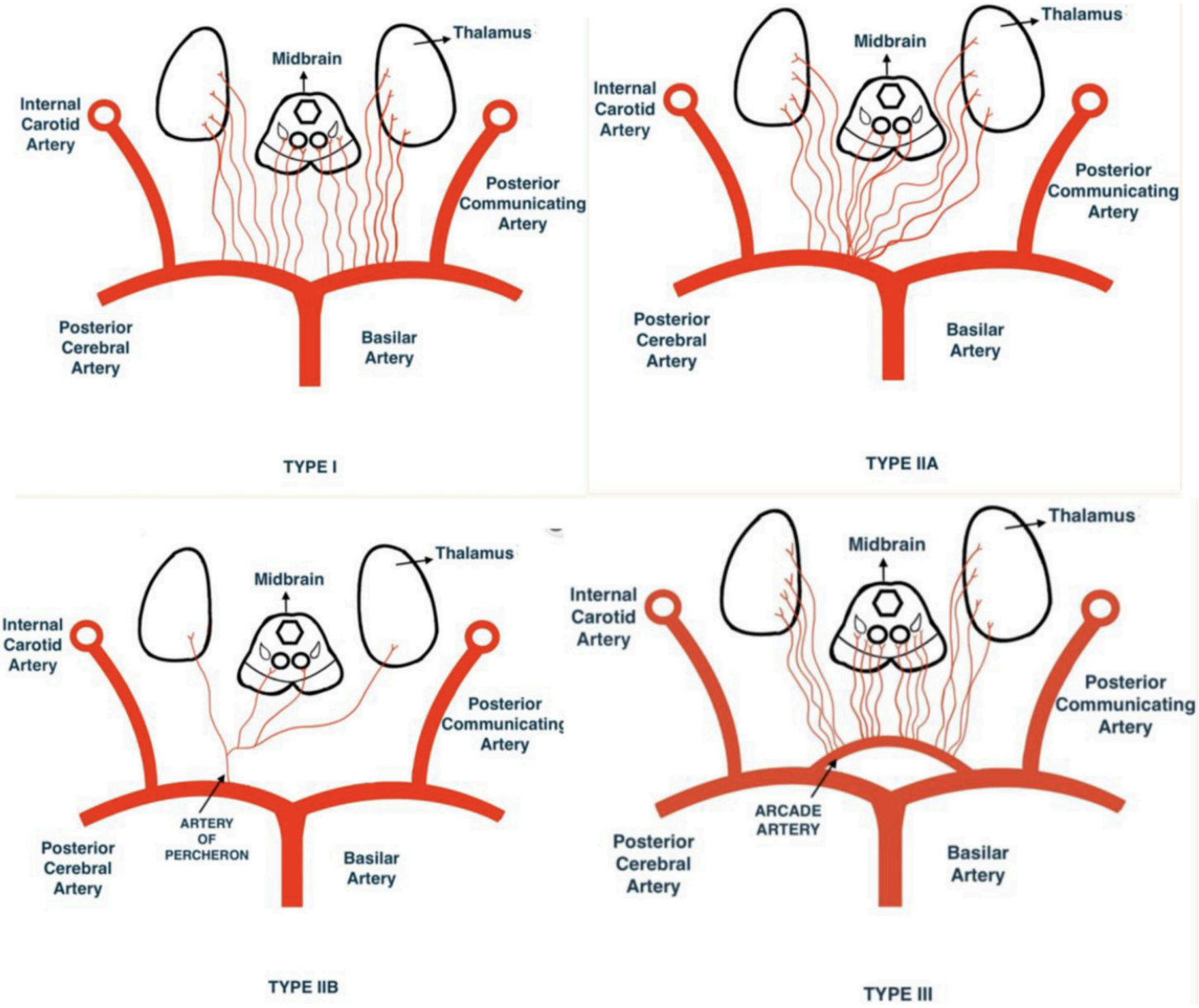


Figure 3

Four variants of paramedian thalamic arterial supply.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.Reprinted from Radiology Case Reports, Vol 19, Ghita Lahnine et al., Bilateral thalamic infarcts: Percheron territory, Pages 1344-1350, Copyright (2024), with permission from Elsevier