

The Artery of Percheron: The Stroke Easily Missed

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Abstract

Bilateral paramedian thalamic infarction can follow occlusion of a single variant perforating artery arising from the posterior cerebral circulation: the artery of Percheron. Because the presentation can include altered alertness, memory disturbance, eye-movement abnormalities and sometimes unusual behavior, the syndrome can initially be mistaken for a diffuse toxic, psychiatric or epileptic process.

Why a tiny perforator matters so much

Artery-of-Percheron infarction is an instructive case where a small arterial event can cause extensive disturbances of consciousness and memory while remaining difficult to see early.

The thalamus participates in ascending arousal networks, memory circuitry and sensorimotor integration. Symmetric injury can therefore produce a perplexing syndrome without a dramatic unilateral cortical deficit. Standard noncontrast CT may be unrevealing early. MRI diffusion-weighted imaging is often more informative, although interpretation requires attention to timing and artifacts. Conventional large-vessel angiography frequently cannot directly show a tiny perforating artery.

This case illustrates why symptom-based reassurance can be hazardous. A patient with unexplained somnolence, confusion, vertical gaze palsy or fluctuating consciousness after an abrupt event warrants serious consideration of posterior circulation ischemia, among other emergencies. The occlusion may already have occurred even if no large visible thrombus appears on CTA. Diagnostic reasoning should combine time course, examination and lesion-specific imaging.

Bilateral thalamic circuits and unusual presentations

The published imaging series describes several patterns of bilateral paramedian thalamic infarction with and without rostral midbrain involvement. These variants explain why the bedside phenotype is not uniform: some patients present chiefly with impaired alertness, others with ocular motor findings or memory disturbance. Diagnostic delay can be aggravated when a patient is considered intoxicated, postictal or psychiatrically disturbed before posterior circulation ischemia is considered. The disease is rare, however, and broad emergency differential diagnosis still includes metabolic and venous causes. The best lesson is pattern recognition combined with disciplined exclusion, not fixation on a dramatic uncommon diagnosis.

The artery of Percheron is an anatomical variant in which a single perforating trunk can supply paramedian territories of both thalami. Occlusion can produce bilateral paramedian thalamic infarcts, sometimes with rostral midbrain involvement. Altered arousal, memory, vertical gaze and behavior

may dominate, making the presentation less recognizable than the classic unilateral weakness of a middle-cerebral-artery stroke. The small size of the culprit perforator also helps explain why routine angiography may not directly show its occlusion.

The anatomical inference is pattern-based rather than an absolute proof of vessel visibility. Symmetric thalamic injury can also arise from deep cerebral venous thrombosis, toxic-metabolic states, infection and other conditions; these alternatives have distinguishing imaging and clinical features. Diffusion-weighted MRI can clarify early ischemic changes, while attention to venous imaging and metabolic context may be needed in atypical presentations. The diagnosis requires compatibility between the tissue lesion and its vascular supply, not just recognition of two bright spots.

One perforating trunk can supply both paramedian thalami, sometimes involving the rostral midbrain. Because thalamic circuits regulate arousal and cognition, lesion volume alone badly predicts clinical seriousness. Early MRI, especially diffusion imaging in difficult posterior-circulation presentations, can still be imperfect. The provocative lesson for diagnostic reasoning is that a negative initial scan does not erase a coherent focal neurological syndrome; neither should every unexplained episode be renamed a hidden infarct.

Localizing the lesion despite uncertain early imaging

A more developed clinical essay should show why this variant matters to decisions. Treatable vascular emergencies must be recognized in time even when the patient's chief complaint is sleepiness or confusion. Later rehabilitation may need to address cognitive and behavioral consequences that are underestimated by simple motor scores. The vascular variant is compelling precisely because it overturns the expectation that clinically important stroke must present as hemiparesis.

The behavioral presentation. Bilateral thalamic injury may alter arousal, memory formation, orientation and eye movements with relatively subtle limb weakness. A patient can therefore appear unusually sleepy, confused or psychiatrically disturbed rather than exhibiting the classic signs that trigger stroke recognition. This does not mean that every new confusion episode is a thalamic infarct; it means that a strategic lesion can yield disproportionate cognitive symptoms and deserves anatomical consideration.

The imaging differential. The distribution and character of the lesions matter. Arterial ischemia should show a plausible pattern and diffusion characteristics, while venous congestion may involve additional structures and different imaging findings. Toxic or metabolic injury can also affect the two thalami. Neurological examination, vascular and venous imaging where indicated, metabolic testing and clinical chronology help separate these processes. The artery of Percheron is often not directly visualized, so anatomy is inferred cautiously rather than declared on the basis of one negative vessel scan.

The larger lesson. Rare vascular arrangements teach a general principle: the severity of an event cannot be judged from lesion volume alone. Strategic circuits make small infarcts clinically important. Follow-up may need attention to sleep-wake regulation, memory and executive function that standard motor rehabilitation overlooks. Recognition of the possibility must accelerate appropriate emergency

assessment, not encourage a purely theoretical search while reversible ischemia goes untreated.

A convincing case combines onset pattern, examination, diffusion abnormalities, vascular territory and follow-up imaging. Compare cases with bilateral thalamic lesions from venous thrombosis, metabolic causes or inflammatory disorders. Territorial fit and temporal evolution matter more than speculation about a narrowly “missed” artery that cannot be directly visualized.

A useful educational case exercise would present the initial symptoms, early CT, diffusion MRI and vascular anatomy in stages, asking what information changed the probability of an artery of Percheron infarct. The answer depends on imaging and anatomy rather than on a claim that bilateral confusion is pathognomonic. Recognition matters because time-sensitive stroke systems should be engaged while the diagnosis is clarified.

How a missed lesion changes the research question

The presentation of bilateral thalamic dysfunction can be dominated by sleepiness, confusion, eye-movement abnormalities or memory disturbance instead of the expected one-sided motor deficit. Those features can prompt a metabolic or psychiatric workup even as a vascular insult evolves. A normal or equivocal early study is therefore not grounds for closing a plausible vascular investigation. Yet the reverse mistake is equally costly: patients with metabolic encephalopathy or deep venous thrombosis can also exhibit bilateral thalamic abnormalities. The paper should compare these mechanisms using diffusion and vascular imaging, lesion distribution, onset and accompanying signs. A credible arterial hypothesis predicts restricted lesions conforming to a perfusion territory, whereas a venous or inflammatory mechanism may show a different pattern. Early diagnostic humility is not vague caution; it is the procedural discipline of keeping a serious, time-sensitive possibility alive while evaluating the features that could prove it wrong. That is more useful than declaring every imaging-negative symptom a hidden stroke.

The danger of declaring a negative scan conclusive

Bilateral paramedian thalamic infarction offers a lesson about early diagnostic uncertainty. A patient's presenting behavior may resemble toxic encephalopathy, sleep disorder or psychiatric disturbance, especially if motor deficits are modest. An early scan or routine vascular study may fail to display a small perforating artery, and this must not be overinterpreted as proof that no ischemic mechanism exists. However, the same distribution also requires consideration of venous or nonvascular injury when imaging characteristics suggest those processes. The proposed diagnostic pathway should therefore emphasize the evolving neurological examination and anatomic imaging correlations rather than any single test. Later cognition, memory and alertness can require long-term rehabilitation even if the infarct volume seems small. The unusual vascular anatomy is not a curiosity detached from clinical care; it shows why strategic location and the time to recognition may matter far more than lesion size.

Bilateral thalamic lesions also have alternatives: deep cerebral venous thrombosis, metabolic injury, Wernicke encephalopathy, infection and inflammatory disease. An imaging pattern must therefore be interpreted, not merely recognized. Calling every bilateral thalamic abnormality “Percheron” would repeat the same one-diagnosis error the paper seeks to correct.

The institute's clinical argument is modest but consequential: uncommon vascular anatomy deserves a place in emergency differential diagnosis because delay can cost treatment opportunity. This educational discussion supports specialist evaluation and acute stroke systems; it does not recommend a home treatment or symptom-based substitute for imaging.

Sources and research leads

1. [Artery of Percheron Infarction: Imaging Patterns and Clinical Spectrum](#)
2. [Navigating the clinical landscape of artery of Percheron infarction: A systematic review](#)

Additional clinical guideline: [2026 AHA/ASA Guideline for the Early Management of Acute Ischemic Stroke](#)

Editorial status: Third-pass conceptual research manuscript. The argument has been individually strengthened and shared boilerplate removed. Original reference leads were retained; a complete independent source-by-source scholarly verification has not been performed. No new clinical trial data are presented.