

When the Visible Thrombus Does Not Explain the Stroke

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Abstract

Imaging can uncover more than one vascular abnormality. The presence of a thrombus, plaque or stenosis does not prove that it caused a particular infarct. This paper proposes an anatomical-concordance method for interpreting complicated vascular studies, especially when an obvious neck-vessel lesion and a brain infarct occupy different circulation territories.

The anatomical error in the visible-clot story

A visible clot or stenosis is not automatically the culprit lesion: causal localization requires vascular anatomy, laterality and timing.

Arterial supply is a constrained network. The internal carotid artery generally supplies anterior circulation territories, whereas the vertebral and basilar system supports much of the posterior circulation. Variations such as a fetal-origin posterior cerebral artery complicate simplistic rules, but they do not erase the need to trace a plausible embolic route. A left common carotid lesion and bilateral paramedian thalamic infarcts, for example, should prompt investigation of posterior circulation mechanisms rather than an automatic causal label.

The interpretive sequence begins with lesion location and MRI diffusion pattern, then CTA or MRA vessel findings, vascular variants and cardiac sources. Artery of Percheron anatomy, when relevant, may explain bilateral paramedian thalamic injury despite the absence of a large visible occlusion. A separate carotid thrombus can remain clinically important even if it does not explain the observed infarcts. Two problems may coexist without one being the cause of the other.

A clot is evidence only when the pathway fits

Vascular territory reasoning has an especially important limitation: unusual arterial variants can permit routes that standard diagrams overlook. Thus a mismatch is a reason to revisit imaging, not an automatic proof of incidental disease. The artery of Percheron and fetal-origin posterior cerebral arteries provide contrasting examples of variant anatomy: one changes perforator supply, while the other can alter whether a posterior cerebral territory receives blood predominantly from the internal carotid. Accurate interpretation requires knowing which variant is actually present, not speculating that one might exist simply to rescue a preferred causal story.

When vascular imaging reveals a thrombus that lies outside the expected arterial supply of a brain lesion, anatomical mismatch becomes a scientific clue. It may indicate two separate disease processes, a coincidental finding, or an incomplete understanding of the lesion's origin. A clot in the

common carotid artery cannot directly account for an isolated bilateral paramedian thalamic infarct if the infarct is supplied by the posterior circulation. The appropriate response is not to discard either finding but to investigate them on their own terms.

Several scenarios require discrimination. An unusual posterior-circulation perforator anatomy could explain a focal imaging distribution; an embolus from the heart might travel to a posterior vessel; another vascular lesion may have been missed; and apparent acute tissue injury may reflect a different process. Clinical examination, diffusion-weighted MRI, angiographic anatomy and cardiac evaluation should be interpreted together. A negative study decreases some probabilities but does not prove the absence of all ischemia.

A thrombus in the common carotid artery may be clinically consequential without explaining bilateral paramedian thalamic infarcts supplied by a posterior-circulation perforator. Conversely an incidental old lacune may distract from a new arterial event. A rigorous paper must distinguish the coexistence of two vascular problems from evidence that one produced the other. The failure mode is diagnostic anchoring: the most striking abnormality in the report becomes the cause of every symptom.

Testing competing sources of neurological injury

This situation illustrates the distinction between “a risk factor present in the patient” and “the culprit lesion responsible for this episode.” Treating a conspicuous thrombus while failing to investigate the actual territory risks an incomplete secondary-prevention plan. Conversely, assuming an invisible embolus without adequate evidence can produce an equally misleading account. The paper's most valuable contribution is a structured approach to causal attribution when alarming findings coexist but do not align.

The trap of the dramatic finding. Clinical records often contain one striking abnormality that becomes the anchor for every later interpretation. A visible carotid thrombus is a serious finding, yet it cannot simply be assigned responsibility for a posterior-circulation thalamic lesion because both involve vessels. The question is how blood or an embolus could travel from the alleged source to the injured territory. Standard arterial anatomy makes some connections implausible and identifies others that need investigation.

Alternative explanations are not mutually exclusive. The patient may have two independent vascular conditions. A systemic tendency toward thrombosis could, in principle, contribute to both, but that is a distinct hypothesis requiring appropriate evidence. An embolic source in the heart could also produce lesions in more than one territory. Inflammatory vascular disease or an imaging interpretation error may be considered when the pattern warrants. The mere possibility of a systemic unifier does not prove one.

What the paper should insist upon. Retain the image finding, the clinical syndrome and the vascular territory as separate observations until a responsible mechanism connects them. If the imaging does not explain the exam, review timing, technique and alternative diagnoses. This principle guards against both premature reassurance and unwarranted diagnostic certainty. It also means that treating a coincidental abnormality should not end the search for the mechanism of the neurological event.

Construct vessel-territory diagrams for each proposed culprit and require the imaging chronology to fit. Repeat targeted studies when an acute lesion is small or early imaging is inconclusive. A true causal clot should connect anatomically to the affected region or plausibly generate an embolus along that circulation. When that route is absent, investigate parallel processes instead of forcing the data into one story.

The strongest discriminating workup asks whether the infarct configuration, vessel imaging and cardiac findings fit one route. If the pattern remains posterior-circulation specific with no supporting carotid-to-posterior pathway, a common carotid thrombus does not become the proven culprit merely because it looks alarming. It may still be an independent serious problem requiring management. Separating causal attribution from clinical importance is the paper's central discipline.

The neurological pathway must be drawn

A hypothetical patient has a left common-carotid thrombus and a bilateral paramedian thalamic infarct. Both findings are important, but the carotid abnormality belongs to the anterior circulation whereas the thalamic lesion may arise from a variant posterior perforator. Without an unusual connecting anatomical route, asserting that the carotid clot explains both thalami is not persuasive. The clinician still needs to investigate the carotid lesion because it may present a separate future risk. This distinction illustrates how good mechanistic research resists a tempting single-cause story without abandoning investigation. It also exposes a danger in retrospective metabolic explanations: a systemic risk factor could contribute to both abnormalities while neither lesion directly caused the other. Causal diagrams should therefore permit shared upstream susceptibility and independent proximal events. The evidence that would unite the findings would have to be anatomical, chronological or pathologically specific, not merely the observation that both happened in one body.

When two abnormal findings are independent

A causal workup can preserve the seriousness of an incidental thrombus without wrongly making it the source of every lesion. If brain MRI shows posterior-circulation infarction but angiography reveals an anterior-circulation abnormality, both deserve interpretation. They may share upstream risks such as hypertension or thrombophilia; they may be temporally unrelated; or another source may explain the injury. A serious article should insist that an investigator identify the exact vessel segment, arterial territory and chronology before assigning an embolic path. It should also distinguish a lesion that appears acute on imaging from one that is definitely responsible for the current symptoms. The more complicated the case, the more useful a two-column record becomes: directly observed findings on one side and causal inferences on the other. This separation prevents a compelling narrative from becoming a substitute for anatomical evidence.

Temporal and anatomical correlation are necessary but not always sufficient. A vascular abnormality seen after the event might represent an embolic source, incidental disease or a consequence of altered flow; detailed neuroradiology and stroke-neurology assessment are crucial. Overcalling causality can lead to wrong secondary prevention, while dismissing an incidental but dangerous thrombus can also be harmful.

The practical lesson is to write two sentences: what the imaging definitely shows, and which mechanism most plausibly explains the affected tissue. If those statements do not align, the case remains mechanistically unresolved. That is not diagnostic failure; it is diagnostic discipline. The paper is an educational framework and does not interpret any identifiable patient's records.

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.