ABSENCE OF FLAIR HYPERINTENSE VESSEL SIGN IN INTERNAL CAROTID ARTERY OCCLUSION: A CASE REPORT AND SYSTEMATIC LITERATURE REVIEW

Adi Kuncoro[1], Ahmad Sobri Muda[2*], Mah Sin Yeat[2,3], Anas Tharek[2], Mohd Hanif Bin Amran[2,4], Nurain Mahfar[5], Noor Hayatul Al Akmal Noralam[2]

[1]Radiology Department, Universitas Brawijaya, Malang, Indonesia
[2]Radiology Department, Hospital Pengajar Universiti Putra Malaysia, Selangor, Malaysia
[3]Radiology Department, Pantai Hospital Ayer Keroh, Melaka, Malaysia
[4]Radiology Department, Avisena Specialist Hospital, Shah Alam, Selangor, Malaysia
[5]Radiology Department, KPJ Damansara, Petaling Jaya, Malaysia

***Corresponding author:**
Ahmad Sobri Muda, Radiology Department, Hospital Pengajar Universiti Putra Malaysia, Selangor, Malaysia

Email: asobri@upm.edu.my

DOI: https://doi.org/10.32896/cvns.v7n2.25-37
Received: 30.05.2025
Revised: 15.06.2025
Accepted: 28.06.2025
Published: 30.06.2025

ABSTRACT

Large vessel occlusions (LVOs) are commonly associated with distinct pathognomonic radiological features, one of which is the fluid attenuation inversion recovery (FLAIR) hyperintense vessel sign (FHVS). This sign indicates an alteration in blood flow dynamics within the affected artery. In most cases, the presence of arterial occlusion is accompanied by FHVS. However, there are exceptions wherein FHVS is absent despite the presence of an occlusion. This case report presents a patient with a complete right internal carotid artery (ICA) occlusion who exhibited relatively mild clinical deficits and limited infarction volume, alongside a notable absence of FHVS. This finding offers an opportunity to explore the pathophysiological mechanisms that might explain the absence of FHVS in such cases and its potential relationship with collateral circulation. The case report is followed by a systematic literature review that supports the phenomenon, that is, the absence of FHVS in cases of internal carotid artery occlusion.

Keywords: Large vessel occlusion, Fluid-attenuated inversion recovery, FLAIR hyperintense vessels sign, Endovascular mechanical thrombectomy, Thrombolysis, Cerebral infarction

INTRODUCTION:

The FLAIR hyperintense vessel sign (FHVS) has emerged as an essential radiological finding in the diagnosis of acute ischemic stroke. To understand the underlying basis of FHVS, it is crucial to consider both hemodynamic principles and magnetic resonance imaging (MRI) physics. Under normal conditions, flowing blood typically appears as a signal void on FLAIR sequences, due to the "flow void" phenomenon. This occurs because fast-moving blood does not provide sufficient time for proton relaxation, resulting in an absence of signal within the blood vessels. In contrast, when an occlusion occurs in a proximal vessel, blood flow velocity diminishes significantly, allowing for the accumulation of signals within the affected vessel, which manifests as FHVS on FLAIR sequences [1].

Despite the established correlation between large vessel occlusions and the presence of FHVS, some cases show the absence of this radiological sign, even when there is clear arterial occlusion [2,3]. This article aims to investigate a particular case where a complete right ICA occlusion occurred without the expected FHVS, highlighting the potential mechanisms behind this discrepancy. This article may provide valuable insights into the role of collateral circulation and other compensatory mechanisms in maintaining cerebral perfusion in the face of significant vascular occlusion.

CASE REPORT:

A 66-year-old female patient, with a known history of atrial fibrillation and undergoing treatment with amiodarone, presented initially with complaints of blurry vision at. 11.00 am, which was followed by involvement in a motor vehicle accident approximately one hour later. An initial examination by an ophthalmologist and a subsequent CT scan at KPJ Puteri Hospital did not reveal any signs of intracranial hemorrhage. The patient was then referred to Hospital Sultan Abdul Aziz Shah for further management.

Approximately 12 hours after the onset of symptoms, the patient was evaluated in the emergency department and a brain magnetic resonance imaging (MRI) with vessel wall imaging was performed using a 3T MRI scanner (scan parameters: TR 8000 ms, TE 125 ms, slice thickness 5 mm, interslice gap 1 mm). At that time, she exhibited signs of drowsiness and slurred speech, with a National Institutes of Health Stroke Scale (NIHSS) score of 12/42. Her pre-stroke Modified Rankin Scale (mRS) score was recorded as 0/5. A magnetic resonance imaging (MRI) scan was performed

The MRI revealed several findings: Restricted diffusion in the right temporal lobe, including the hippocampus, temporal stem, parahippocampal gyrus, and fusiform gyrus, as well as in the right thalamus, all showing corresponding FLAIR hyperintensity, shown in Figure 1. A complete occlusion of the intracranial segment of the right ICA was observed, with a complete loss of flow signal on magnetic resonance angiography (MRA). Despite the complete occlusion of the right ICA, there was no evidence of distal FHVS in the affected hemisphere, illustrated in Figure 2. There was also an absence of stagnation of black blood distal to the occlusion site. A patchy and modest-sized prominent vessel sign was also observed in the susceptibility-weighted imaging (SWI). Cerebral angiography was performed, confirming the total occlusion of the right ICA. The angiogram also showed substantial cross-filling from the contralateral circulation, shown in Figure 3. Following these findings, endovascular mechanical thrombectomy was performed, achieving complete recanalization of the right ICA and the right anterior cerebral circulation, conforming to the grade III thrombolysis in cerebral infarction (TICI) scale, seen in Figure 4. This procedure resulted in a rapid reversal of the stroke-

related symptoms. Post-procedural CT imaging revealed no intracranial haemorrhage, as observed in Figure 5.

METHODOLOGY:

A literature search was performed on 5th of May, 2025 with key words "Absence of FLAIR Hyperintense Vessel Sign AND Internal Carotid Artery" using PubMed and Science Direct databases. The search was limited to case reports and research articles available in English reporting cases investigated to current standards from 2020 until 2025, which displays the absence of FLAIR hyperintense vessel in internal carotid artery occlusion. PubMed yielded 1 article, and Science Direct yielded 124 articles; 6 additional articles were found through alternate sources. 4 articles were relevant, and 1 article was inaccessible. Eventually, 3 articles were included in the systematic literature review, illustrated in Figure 6.

DISCUSSION:

The 3 eligible articles are summarized in Table 1. We reported 3 articles that consisted of 1 research article and 2 case reports that display an absent FLAIR hyperintense vessel sign despite the presence of internal carotid artery occlusion. 2 articles display patients with stroke, and 1 article displays a patient with giant cell arteritis that causes an internal carotid stenosis [4–6].

The FLAIR hyperintense vessel sign (FHVS) typically manifests as a serpentine or linear hyperintensity along the affected arterial segment, visible on FLAIR sequences. The pathophysiological basis of FHVS is rooted in the interplay between hemodynamic alterations and the MRI signal characteristics. Under normal physiological conditions, blood flowing through arteries generates a signal void on FLAIR imaging due to the "flow void" phenomenon. This occurs because of the rapid movement of blood, which prevents the formation of a spin echo necessary for signal generation. As a result, arteries with normal blood flow appear hypointense on FLAIR sequences [1,3,7].

When an arterial occlusion occurs, blood flow within the vessel slows significantly, allowing for the detection of a hyperintense signal during MRI pulse sequences. This phenomenon is referred as the FLAIR hyperintense vessel sign. The presence of FHVS in acute stroke patients is clinically significant, as it serves as a radiological marker of proximal large vessel occlusions, providing valuable clinical information. Studies have shown that the presence of FHVS indicates the occurrence of slow antegrade or retrograde flow within the distal artery. FHVS also provides important information regarding patient management. A study stated that FHVS is a significant predictor of poor prognosis in acute ischemic stroke caused by unilateral internal carotid artery occlusion or stenosis that is managed without recanalization within 72 hours of the onset, which correlates with a higher baseline National Institute of Health Stroke Scale (NIHSS) score. A separate study also suggests that FHVS may be a significant clinical factor in determining whether tissue salvage can be expected through recanalization, as it predicts the patient's collateral status. The comprehension of FHVS also provides a monumental insight into the management of the patient, particularly the recanalization of the occluded vessel. A study revealed that endovascular recanalization decreases the postoperative FHVS, which indicates an improved hemodynamic status [1,3,7 –10].

The development of collateral circulation is a compensatory mechanism in response to large vessel occlusion. Collateral circulation helps to maintain cerebral perfusion when a major artery is occluded. Several types of collateral circulations are recognized, based on their origin and functional capacity. Collaterals can arise from the Circle of Willis (CoW), microvascular intracranial collaterals (including leptomeningeal and subcortical collaterals), and extracranial sources of

blood flow. Functionally, collaterals are classified into primary, secondary, and tertiary categories. Primary collateral systems can immediately redirect blood flow in the event of vessel occlusion, with the Circle of Willis serving as the primary compensatory pathway. Secondary collaterals, such as leptomeningeal collaterals, become activated in response to more significant or prolonged vascular compromise. Tertiary collaterals are newly formed or matured vessels that further assist in sustaining cerebral perfusion during ongoing ischemia [11–13]. Another finding that we can observe is the presence of a prominent vessel sign. The presence of the prominent vessel sign (PVS) is commonly attributed to an elevated oxygen extraction fraction (OEF). In the setting of acute ischemic stroke, a substantial reduction in cerebral blood flow prompts the affected brain tissue to increase its OEF. This results in a higher concentration of deoxyhemoglobin within the venous and capillary systems. Due to its paramagnetic properties and high magnetic susceptibility, deoxyhemoglobin becomes more conspicuous on susceptibility-weighted imaging (SWI), thereby manifesting as the PVS. Some literature states that prominent vessel sign reflects a poorly established collateral, while our case displays prominent vessel signs despite the presence of a presumably well-established collateral [14].

In this case report, the absence of FHVS in the presence of a complete ICA occlusion can likely be explained by the presence of a robust collateral circulation, particularly from the contralateral circulation. The angiogram confirmed abundant crossflow through the Circle of Willis and leptomeningeal collaterals, which supplied blood to the affected areas of the brain, thereby preventing the expected FHVS. The minimal infarction seen on MRI further supports the role of collateral circulation in preserving brain tissue despite significant occlusion of the right ICA. This suggests that the patient's brain was sufficiently perfused by the collateral network, preventing the buildup of ischemic changes that would typically lead to the development of FHVS. The narration above is also supported by a similar case report and research that also displays the absence of FHVS in patients with internal carotid artery occlusions.

In our case, following successful thrombectomy, which restored the patency of the right ICA, the collateral circulation diminished, and the previously abundant contralateral flow was no longer necessary. This observation supports the notion that collateral systems adapt to the restoration of normal blood flow and can be transient. The absence of FHVS during acute ischemic stroke has been reported in other instances, particularly in cases with effective collateral circulation, as rapid blood flow velocity in the collateral pathways may prevent the formation of a detectable hyperintense signal on FLAIR imaging.

CONCLUSION:

This case report and systematic literature review underscore the complex relationship between large vessel occlusion, the FLAIR hyperintense vessel sign, and the collateral circulation system. The absence of FHVS in this case, despite a complete right ICA occlusion, illustrates the crucial role of compensatory collateral blood flow. This phenomenon provides valuable insight into the pathophysiology of acute ischemic stroke, where the effectiveness of collateral circulation can directly influence the radiological presentation and clinical outcome. Understanding the mechanisms behind FHVS and its connection with the collateral circulation is essential for improving the management and prognostication of patients with large vessel occlusions, particularly in the context of acute stroke care.

DATA AVAILABILITY:

Further information regarding the data used for this work can be obtained from the corresponding author upon reasonable

request.

FUNDING:
This work received no external funding.

CONFLICT OF INTEREST:
The authors have no conflicts of interest to declare and is in agreement with the contents of the manuscript.

REFERENCES:

1. Azizyan A, Sanossian N, Mogensen MA, Liebeskind DS. Fluid-attenuated inversion recovery vascular hyperintensities: An important imaging marker for cerebrovascular disease. American Journal of Neuroradiology 2011;32:1771–5. https://doi.org/10.3174/ajnr.A2265.
2. Salahuddin H, Saherwala A, Pinho MC, Moore W, Castonguay A, Khan NI, et al. Association of distal hyperintense vessel sign and recurrent stroke in patients with symptomatic intracranial stenosis. Journal of Stroke and Cerebrovascular Diseases 2022;31. https://doi.org/10.1016/j.jstrokecerebrovasdis.2022.106616.
3. Kashiwazaki D, Yamamoto S, Akioka N, Hori E, Shibata T, Kuwayama N, et al. High-intensity vessel sign on fluid-attenuated inversion recovery imaging: a novel imaging marker of high-risk carotid stenosis-a MRI and SPECT study n.d. https://doi.org/10.1007/s00701-020-04408-4/Published.
4. Lyu J, Hu J, Wang X, Bian X, Wei M, Wang L, et al. Association of fluid-attenuated inversion recovery vascular hyperintensity with ischaemic events in internal carotid artery or middle cerebral artery occlusion. Stroke Vasc Neurol 2023;8:69–76. https://doi.org/10.1136/svn-2022-001589.
5. Yao A, Wang B, Jia L. Asymptomatic severe occlusion of the internal carotid artery (ICA) in a Chinese patient with multiple cerebrovascular and cardiac stenoses. Interdiscip Neurosurg 2017;9:80–1. https://doi.org/10.1016/j.inat.2017.04.001.
6. Kikumoto M, Aoki S, Shiga Y, Kinoshita N, Nezu T, Ueno H, et al. Giant cell arteritis with internal carotid artery occlusion in the absence of typical clinical features. Internal Medicine 2021;60:1293–7. https://doi.org/10.2169/internalmedicine.5592-20.
7. Nam KW, Kwon HM, Park SW, Lim JS, Han MK, Lee YS. Distal hyperintense vessel sign is associated with neurological deterioration in acute ischaemic stroke. Eur J Neurol 2017;24:617–23. https://doi.org/10.1111/ene.13259.
8. Xiang W, Wei H, Liang Z, Zhang M, Sun Z, Lv Y, et al. FLAIR vascular hyperintensity combined with asymmetrical prominent veins in acute anterior circulation ischemic stroke: prediction of collateral circulation and clinical outcome. Eur J Med Res 2023;28. https://doi.org/10.1186/s40001-023-01445-4.
9. Karadeli HH, Giurgiutiu DV, Cloonan L, Fitzpatrick K, Kanakis A, Ozcan ME, et al. FLAIR Vascular Hyperintensity is a Surrogate of Collateral Flow and Leukoaraiosis in Patients With Acute Stroke Due to Proximal Artery Occlusion. Journal of Neuroimaging 2016;26:219–23. https://doi.org/10.1111/jon.12274.
10. Liu W, Yin Q, Yao L, Zhu S, Xu G, Zhang R, et al. Decreased hyperintense vessels on FLAIR images after endovascular recanalization of symptomatic internal carotid artery occlusion. Eur J Radiol 2012;81:1595–600. https://doi.org/10.1016/j.ejrad.2011.04.026.
11. Maguida G, Shuaib A. Collateral Circulation in Ischemic Stroke: An Updated Review. J Stroke 2023;25:179–98. https://doi.org/10.5853/jos.2022.02936.
12. Kimmel ER, Al Kasab S, Harvey JB, Bathla G, Ortega-Gutierrez S, Toth

G, et al. Absence of Collaterals is Associated with Larger Infarct Volume and Worse Outcome in Patients with Large Vessel Occlusion and Mild Symptoms. Journal of Stroke and Cerebrovascular Diseases 2019;28:1987–92. https://doi.org/10.1016/j.jstrokecerebrovasdis.2019.03.032.

13. Connolly F, Röhl JE, Lopez-Prieto J, Danyel LA, Schreiber SJ, Valdueza JM. Pattern of Activated Pathways and Quality of Collateral Status in Patients with Symptomatic Internal Carotid Artery Occlusion. Cerebrovascular Diseases 2020;48:244–50. https://doi.org/10.1159/000504663.

14. Jiang H fei, Zhang Y qun, Pang J xia, Shao P ning, Qiu H cheng, Liu A fei, et al. Factors associated with prominent vessel sign on susceptibility-weighted imaging in acute ischemic stroke. Sci Rep 2021;11. https://doi.org/10.1038/s41598-021-84269-8.

FIGURE LEGEND:

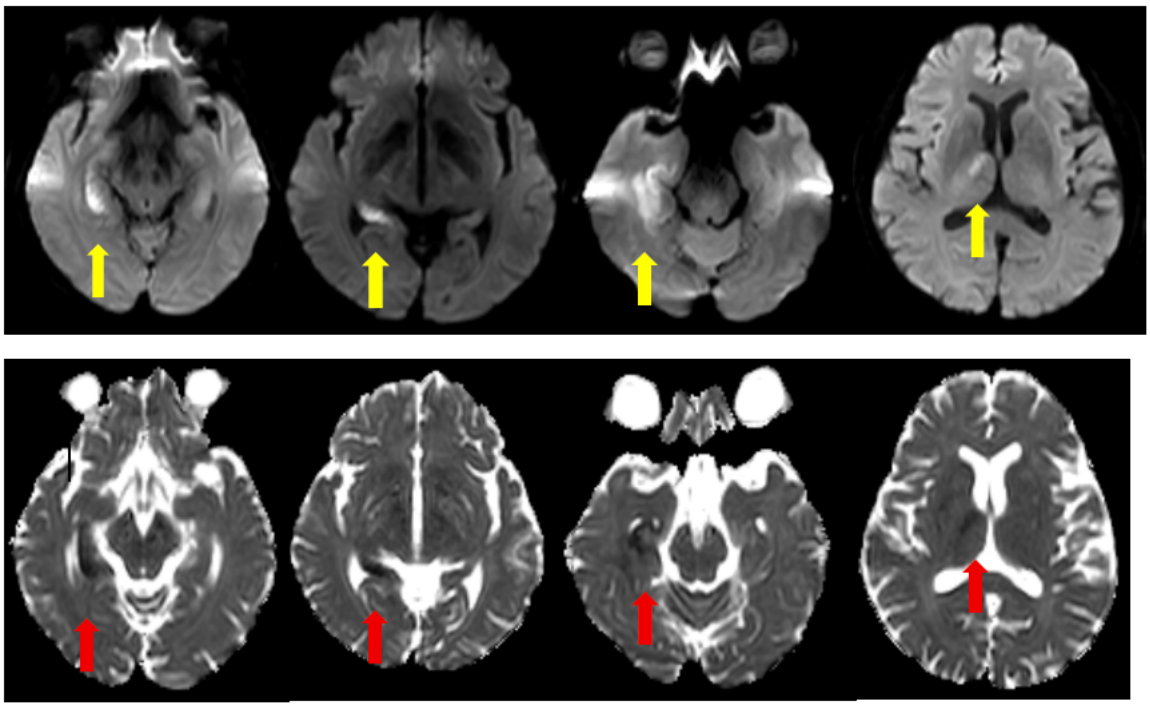

Figure 1: Areas of acute infarctions evidenced by hyperintense signals on DWI (green arrows) and hypointense signals on ADC (red arrows)

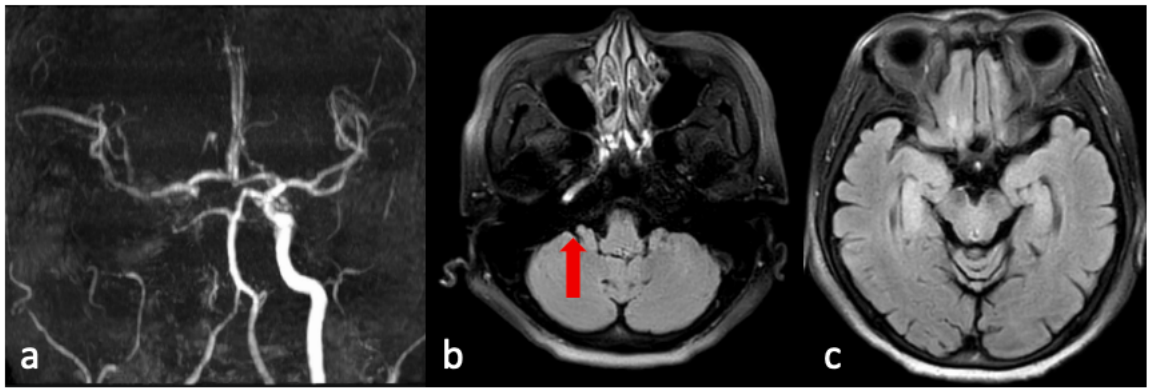

Figure 2: A complete occlusion of the intracranial segment of Right ICA with presence of right ACA and MCA signals were observed (a). Correspondingly, FHVS was seen at the right ICA proximal to the occlusion, however absent FHVS distal to the occlusion (b, c).

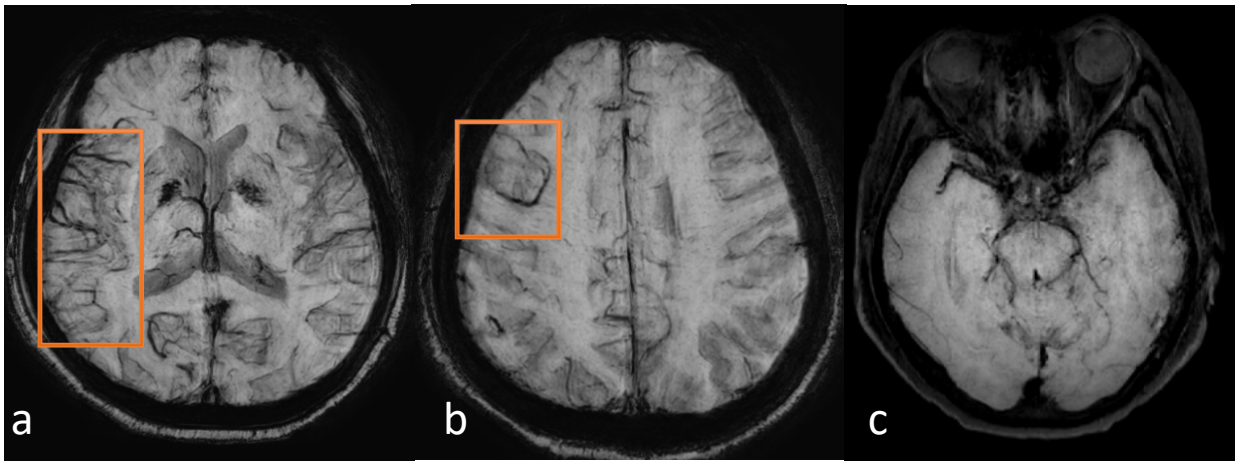

Figure 3: Prominent vessel signs were present at the right frontoparietal region on SWI sequence (a,b, within orange box) and an absence of stagnation on the black blood (c).

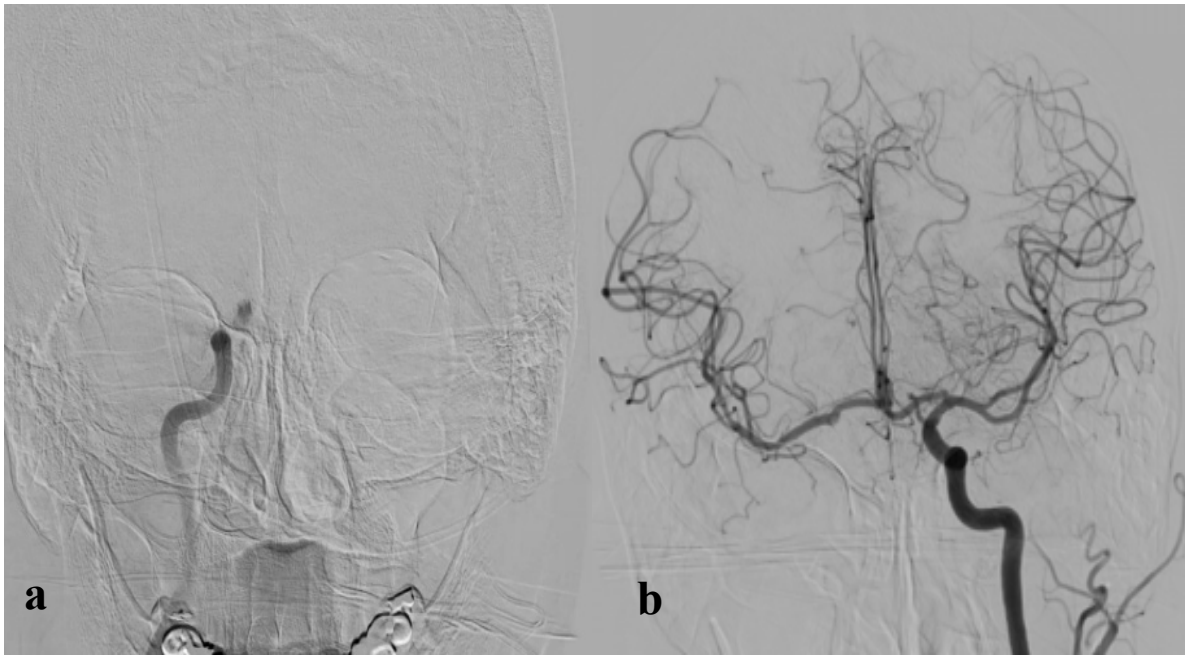

Figure 4: Angiography revealed a total occlusion of the right internal carotid artery (a), and an abundant crossflow from the contralateral circulation (b)

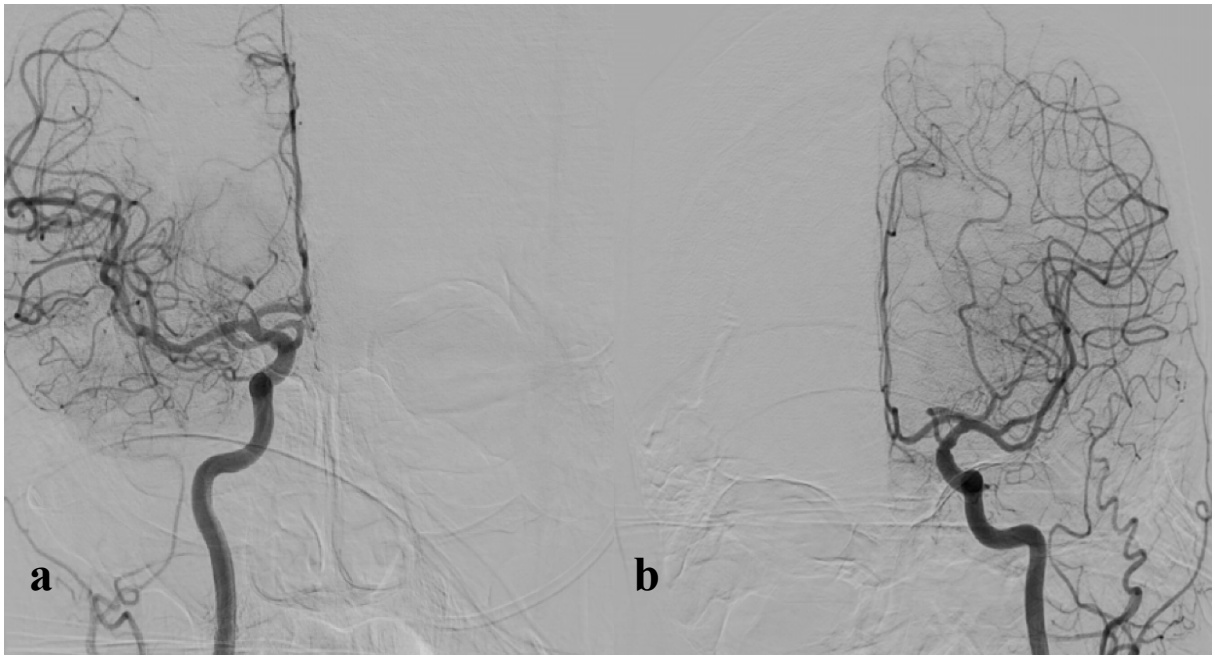

Figure 5: Post-thrombectomy cerebral angiography showed recanalization of the right ICA occlusion (a) and the disappearance of the previously abundant contralateral flow (b)

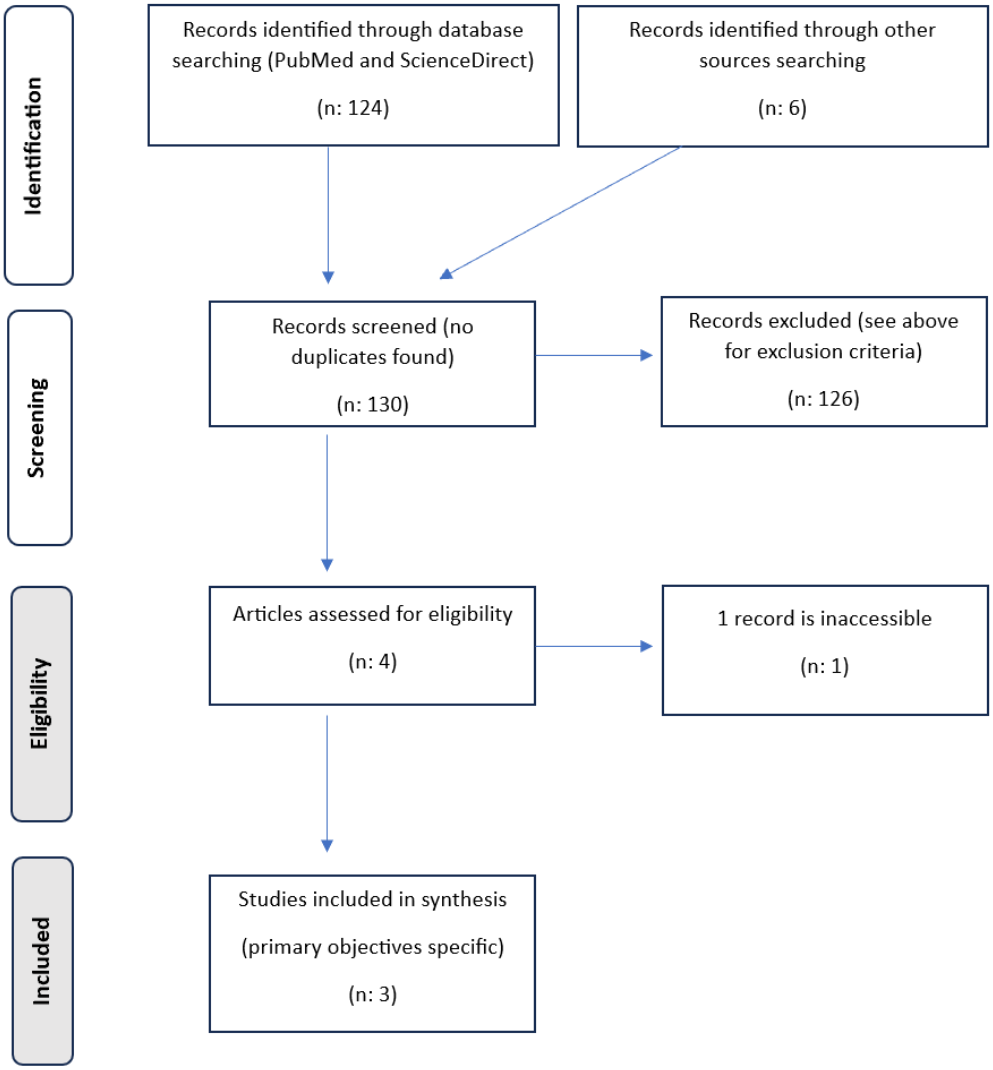

Figure 6: PRISMA Flow Diagram

TABLE LEGEND:

Table 1: Summary of eligible articles

Study	Patient Demographics	FHVS Absence
Kikumoto et al.,	A 65-year-old man with left ICA occlusion caused by giant cell arteritis (GCA); presented only with mild headache and transient visual disturbance	**Yes.** FHVS was absent in the MRI examination
Yao et al.,	A 72-year-old man with chronic right ICA and vertebral artery occlusions had only mild, brief symptoms, despite having longstanding hypertension (>20 years) and severe coronary artery disease.	**Yes.** FHVS was Absent – MRI only showed old lacunar infarcts with no acute ischemia
Lyu et al.,	Retrospective study of 147 patients (mean age 60) with 149 chronic occlusions of the ICA or MCA; 37 had no ischemic symptoms ("asymptomatic" occlusion) and 112 had recent ischemic events ("symptomatic" occlusions)	**Yes.** FLAIR vascular hyperintensity **(**FVH) evaluated by FVH-ASPECTS (0–7). Higher FVH-ASPECTS scores indicate symptomatic occlusions (recent strokes) while low or absent (score 0) are common in asymptomatic occlusions