Scientific Research Publishing

World Journal of Neuroscience, 2025, 15(4), 344-355
https://www.scirp.org/journal/wjns
ISSN Online: 2162-2019
ISSN Print: 2162-2000

FLAIR Vascular Hyperintensities (Slow Flow) and Early Therapeutic Triage in Ischemic Stroke: A Case Series

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How to cite this paper: Agba, L., Soro, D., Awidina-Ama, A., Ngoujo, L., Yaiche, H. and Heinzleif, O. (2025) FLAIR Vascular Hyperintensities (Slow Flow) and Early Therapeutic Triage in Ischemic Stroke: A Case Series. *World Journal of Neuroscience*, **15**, 344-355.
https://doi.org/10.4236/wjns.2025.154027

Received: May 20, 2025
Accepted: November 4, 2025
Published: November 7, 2025

Abstract

Introduction: FLAIR vascular hyperintensities (FVH), also known as "slow flow" signs, reflect arterial hemodynamic slowing downstream of stenosis or occlusion. In hyperacute ischemic stroke, FVH can precede parenchymal diffusion-weighted imaging (DWI) abnormalities and serve as an indirect marker of hypoperfusion. Their integration into the clinical-radiological assessment may influence thrombolysis decisions, particularly when perfusion imaging is unavailable. **Methods:** We conducted a prospective descriptive series of three hyperacute ischemic strokes managed within an MRI-first pathway in a tertiary center. Clinical data, imaging findings (FLAIR, DWI, T2*), and treatment details were recorded. FVH were qualitatively assessed for extent and distribution. The main outcome was early clinical improvement (NIHSS variation) and absence of hemorrhagic transformation on 24-hour MRI. **Results:** All patients presented with disabling deficits despite low-to-moderate NIHSS scores (4 - 10). Initial MRI performed within 2 hours of symptom onset showed prominent FVH without DWI lesions (n = 2) or with minimal changes (n = 1). No thrombus was detected on T2*. All received intravenous thrombolysis (alteplase 0.9 mg/kg) within 2 h 30 min. Follow-up MRI at 24 hours revealed no infarction or hemorrhagic conversion. Clinical improvement was significant in all cases (median NIHSS decrease: −6 points), with resolution or marked reduction of deficits at discharge. **Conclusion:** In ultra-early ischemic stroke, FVH represent an operational marker of hypoperfusion that may guide thrombolysis, particularly when perfusion imaging is not accessible. The FVH-DWI mismatch can substitute for the perfusion-diffusion mismatch in identifying salvageable tissue. In resource-limited settings, especially in sub-Saharan Africa, recognizing FVH could expedite therapeutic decisions and optimize out-

comes. Larger studies with standardized FVH quantification are needed to confirm their prognostic value.

Keywords

Ischemic Stroke, FLAIR Vascular Hyperintensity, Slow Flow, Thrombolysis, FVH-DWI Mismatch

1. Introduction

Stroke remains a leading cause of mortality and morbidity worldwide; cerebral ischemia alone accounts for roughly 60% - 70% of all strokes, with a growing burden in low- and middle-income countries [1]. This underscores the need for rapid and effective management from the hyperacute phase, in which diagnosis relies on brain magnetic resonance imaging (MRI), particularly FLAIR and diffusion-weighted imaging (DWI) sequences [2]. The "time is brain" paradigm reminds us that each lost minute costs neurons and quality-adjusted life years, hence the need to promptly identify patients eligible for reperfusion. The benefits of intravenous alteplase, the most widely used thrombolytic, are greater the earlier treatment is administered and remain demonstrated up to 4 h 30 min after symptoms onset, across ages and severities [3] [4]. Guidelines from the American Heart Association/American Stroke Association (AHA/ASA) and the European Stroke Organisation (ESO) govern intravenous thrombolysis (IVT) and emphasize assessing the disabling nature of symptoms, particularly when the NIHSS is low. Recent trials suggest that in minor, non-disabling strokes, IVT offers no advantage over aspirin or dual antiplatelet therapy (DAPT) [5] [6]. On early MRI, FLAIR vascular hyperintensities (FVH), also termed the "hyperintense vessel sign" or "slow flow", reflect low-velocity arterial blood in sulcal vessels, typically distal to a proximal stenosis/occlusion and/or via collateral back-filling. FVH can appear before any parenchymal lesion, and their extension beyond DWI lesion limits (FVH-DWI mismatch) acts as a penumbra surrogate when perfusion imaging is unavailable [7]-[9]. In resource-limited settings, notably sub-Saharan Africa where IVT remains unevenly deployed and emergent perfusion imaging is rarely accessible, recognizing FVH can help secure and accelerate reperfusion decisions. This motivates the present study, whose general objective is to report a case series of hyperacute cerebral ischemia treated with IVT prompted by slow-flow signs.

2. Methods

2.1. Setting, Design, and Study Period

Prospective, descriptive, monocentric case series conducted in the neuro-ICU/stroke unit (USINV) of Saint-Germain-en-Laye/Poissy Hospital over calendar year 2020.

2.2. Study Population

Eligible patients were those admitted under a thrombolysis alert for a focal neurological deficit compatible with acute ischemic stroke and imaged immediately with a stroke MRI protocol including at minimum DWI/ADC and FLAIR.

2.3. Inclusion Criteria

Patients were included if they had:

1) slow-flow (FVH) on FLAIR, with FVH-DWI mismatch when present, confirmed by joint reading (neuroradiologist + stroke neurologist);

2) complete early clinical data, including admission NIHSS.

2.4. Exclusion Criteria

Patients were excluded if they had intracranial hemorrhage on initial imaging, a non-vascular mimic explaining the focal deficit, or uninterpretable/absent key MRI sequences.

3. MRI Acquisition Parameters and FVH Definition

All MRIs were performed on a 1.5 T Siemens scanner (Magnetom Avanto, Siemens Healthineers, Erlangen, Germany) using the institutional acute-stroke protocol. The sequences included DWI (b = 0/1000 s/mm^2, slice thickness = 5 mm, TR/TE = 3400/90 ms), ADC maps, T2* GRE (slice thickness = 5 mm), and FLAIR (TR/TE = 9000/120 ms, inversion time = 2500 ms, slice thickness = 5 mm, matrix 256 × 192).

3.1. Operational Definitions

FVH were defined as linear or serpentine hyperintensities along cortical sulci, following arterial courses distal to a stenosis or within collateral territories, reflecting local hemodynamic slowing, visible on at least two consecutive slices.

A FVH-DWI mismatch was defined when the spatial extent of FVH exceeded the limits of any diffusion lesion or when FVH were present despite a negative DWI.

3.2. Data Handling

Given the small sample size, we performed a descriptive analysis (narrative presentation and summary Table 1).

3.3. Ethics

Written informed consent was obtained from all patients or their legal representatives for participation and the publication of anonymized data and images, in accordance with the Declaration of Helsinki. The study protocol received approval from the Institutional Ethics Committee (Approval No. 2020-NEURO-FVH-01).

Table 1. Summary of the three cases.

Parameter	Case 1	Case 2	Case 3
Age (years)/Sex	62/M	68/F	58/M
Past medical history	Hypertension, dyslipidemia, active smoking	Hypertension, type 2 diabetes, dyslipidemia	Hypertension, severe obstructive sleep apnea, migraine with aura
Initial deficit	Left upper limb weakness 3/5 (MRC), left central facial palsy, mild dysarthria	Right hemiparesis 2/5 with contralateral hypoesthesia, mixed aphasia	Left hemiparesis 2/5 with hypoesthesia and subtle spatial neglect
Initial NIHSS	4	10	6
Time to initial MRI	1 hour	1 h 45 min	1 h 15 min
FVH (location)	Right fronto-opercular (M3)-perisylvian	Right perisylvian	Right perisylvian
Initial DWI lesion	None	None	None
Visible thrombus (T2*)	Yes (right M1-M2 junction)	No	No
Onset-to-needle time	1 h 30 min	2 h 15 min	1 h 55 min
Thrombolytic agent	Alteplase (0.9 mg/kg)	Alteplase (0.9 mg/kg)	Alteplase (0.9 mg/kg)
NIHSS at 24 h	2	4	3
Hemorrhagic transformation	None	None	None
Day-7 recovery	Partial (left upper limb 4/5)	Not specified (discharged early)	Near-complete (left upper limb 4/5)

Note: Abbreviations: FVH = FLAIR vascular hyperintensity (slow flow); DWI = diffusion-weighted imaging; T2* = gradient-echo; NIHSS = National Institutes of Health Stroke Scale; MRC = Medical Research Council.

4. Results

4.1. Case 1

Right-handed 62-year-old man admitted to the emergency department at 14:30 for a sudden-onset isolated left upper-limb motor deficit that began 30 minutes earlier. This was the third episode of the same kind, after two similar events 7 weeks and 1 year previously. Comorbidities included well-controlled arterial hypertension on amlodipine 10 mg/day, dyslipidemia on atorvastatin 10 mg/day, and active smoking estimated at 13 pack-years. There was no history of diabetes, cardioembolic heart disease, or established stroke.

The initial examination at 14:45 found an alert patient (Glasgow 15), normothermic (37.2˚C), with WHO grade III hypertension (BP 188/115 mm Hg in the left arm and 190/115 mm Hg in the right arm), heart rate 72 bpm in sinus rhythm, and oxygen saturation 98% on room air. Capillary glucose was 5.2 mmol/L. Neurological assessment revealed an isolated left upper-limb motor deficit graded 3/5 on the MRC scale, associated with left central facial palsy and mild dysarthria,

with no other focal neurological abnormality, corresponding to an initial NIHSS score of 4. Brain MRI with the emergency neurovascular protocol performed at 15:00 (*i.e.*, 1 hour after symptom onset) showed:

- No parenchymal abnormality on DWI or FLAIR;
- A linear vascular hyperintensity of the right fronto-opercular artery on FLAIR, consistent with slow flow;
- A marked hypointensity measuring 3 mm on the M3 segment of the right middle cerebral artery on *T*2 *gradient-echo**, confirming the presence of an in situ arterial thrombus (see Figure 1).

Intravenous thrombolysis with alteplase was initiated at 15:30 (total dose 0.9 mg/kg), within the recommended time window. The clinical course was marked by progressive improvement with partial motor recovery to 4/5 by the 6th hour post-thrombolysis, then an NIHSS of 2 at 24 hours. The 24-hour follow-up MRI confirmed the absence of hemorrhagic transformation and persistent partial patency of the right middle cerebral artery.

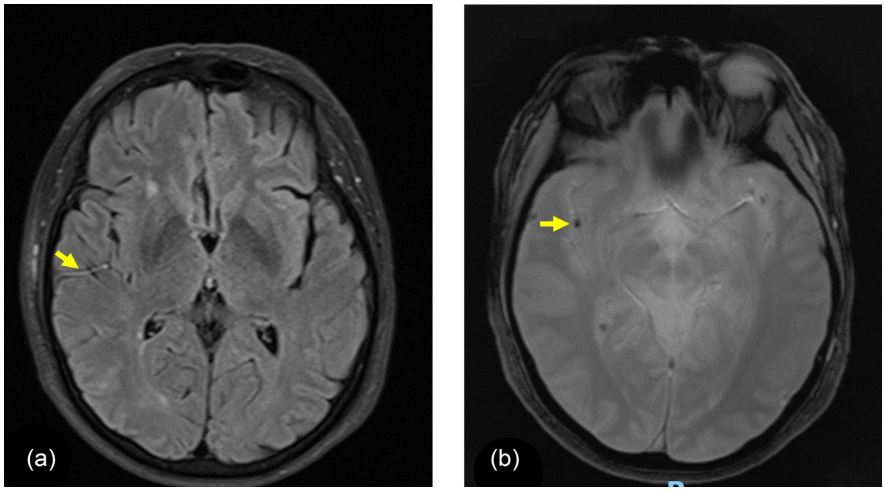

Figure 1. Axial brain MRI performed 1 hour after symptom onset. (a) FLAIR sequence shows a linear hyperintensity along the right fronto-opercular cortical artery (yellow arrow), consistent with a vascular hyperintensity due to slow or stagnant flow in the M3 segment territory of the middle cerebral artery. No parenchymal FLAIR signal abnormality is detected at this stage; (b) Corresponding axial T2* gradient-echo sequence demonstrates a focal, well-defined hypointense signal (yellow arrow) located at the right M1-M2 junction, indicating the presence of acute intraluminal thrombus.

4.2. Case 2

Right-handed 68-year-old woman with a history of arterial hypertension treated with irbesartan 150 mg/day, type 2 diabetes on metformin, and dyslipidemia controlled with rosuvastatin 10 mg/day, admitted emergently for sudden-onset right hemiparesis associated with aphasia. The initial examination 50 minutes after symptom onset showed normal consciousness (Glasgow 15), blood pressure 170/90 mm Hg, and a regular heart rate of 72/min. Neurological assessment revealed right hemiparesis predominating in the upper limb (2/5 on the MRC scale) with con-

tralateral hypoesthesia, associated with mixed aphasia with expressive predominance. The remainder of the physical examination was unremarkable. The initial NIHSS score was 10. Brain MRI performed 1 h 45 min after onset showed a right perisylvian vascular hyperintensity on FLAIR, suggesting marked hemodynamic slowing, without parenchymal diffusion abnormality and no visible thrombus on T2* (see Figure 2).

The patient underwent intravenous thrombolysis (IVT) with alteplase administered 2 h 15 min after symptom onset. The course was marked by progressive improvement, with an NIHSS of 4 at 24 hours. The 24-hour control MRI confirmed the absence of established ischemic lesion and no hemorrhagic transformation.

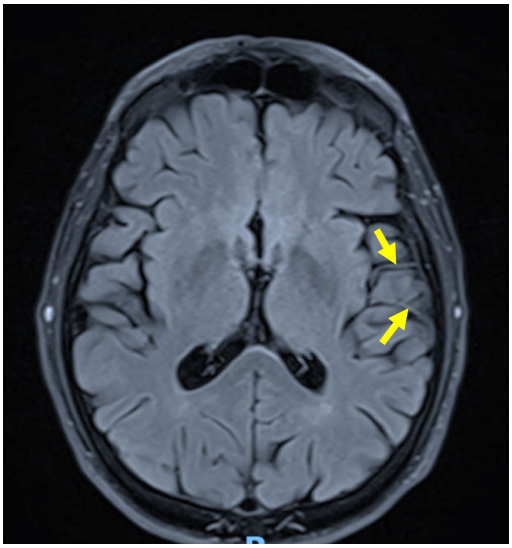

Figure 2. Axial FLAIR MRI demonstrates linear hyperintense signal abnormalities along the cortical and subcortical branches of the left middle cerebral artery (yellow arrows), consistent with FLAIR vascular hyperintensities. These findings are suggestive of slow flow secondary to hypoperfusion in the left MCA territory.

4.3. Case 3

A right-handed 58-year-old man, a company director, presented with sudden-onset left hemibody weakness while at work. He was brought by EMS to the neurovascular emergency department 45 minutes after symptom onset. His medical history included severe obstructive sleep apnea (AHI 45/hour) on nocturnal ventilation and migraine with aura, as well as hypertension treated with candesartan 150 mg/day. On admission under a thrombolysis alert, the examination found an alert patient (Glasgow 15) with obesity (BMI 34 kg/m^2) of marked android type (waist circumference 118 cm) and clinical signs suggestive of severe OSA (short neck with 44 cm neck circumference, narrow oropharynx Mallampati grade IV). Hemodynamic parameters showed arterial hypertension 178/95 mm Hg in both arms without significant asymmetry, with a regular heart rate 64/min in sinus rhythm (Figure 3).

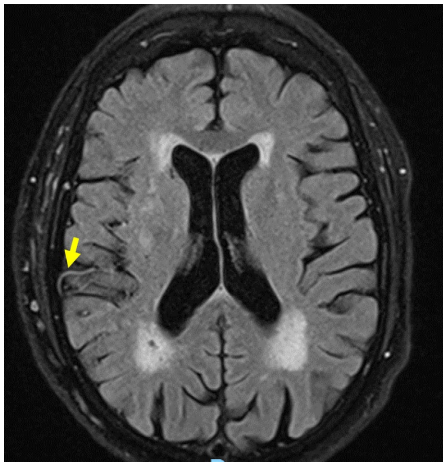

Figure 3. Axial FLAIR MRI demonstrates a linear, serpiginous hyperintensity within the right perisylvian cortical sulci (yellow arrow), consistent with FLAIR vascular hyperintensity (FVH). This appearance indicates slow flow in distal right MCA (M2/M3) branches.

Neurological examination revealed left hemiparesis predominating in the upper limb (2/5 on the MRC scale) associated with ipsilateral hypoesthesia and subtle spatial neglect, without language disturbance or cranial nerve involvement (NIHSS 6). A comprehensive cardiovascular examination noted a grade 1/6 systolic murmur at the aortic area without radiation, symmetric peripheral pulses, and no carotid or vertebral bruits on cervical auscultation. There were no signs of deep vein thrombosis in any limb. Pulmonary examination showed a slight decrease in breath sounds at the bases with oxygen saturation 94% on room air, while the endocrine examination noted mild acanthosis nigricans with no other signs of endocrinopathy.

The initial emergency workup included capillary glucose 5.8 mmol/L, an ECG showing sinus rhythm with no repolarization abnormalities, and standard laboratory tests demonstrating a normal complete blood count, serum creatinine 98 µmol/L, negative cardiac enzymes, and a normal electrolyte panel.

Emergency brain MRI (1 h 15 min after symptom onset) demonstrated a right perisylvian vascular hyperintensity on FLAIR, without parenchymal diffusion abnormality and no visible thrombus on T2*. Management consisted of intravenous thrombolysis (IVT) with alteplase administered 1 h 55 min after symptom onset, with a favorable course marked by improvement to NIHSS 3 at 24 hours and near-complete motor recovery by day 7 (4/5 in the left upper limb). Carotid-vertebral duplex subsequently proved normal, while follow-up respiratory polygraphy confirmed persistent severe OSA despite nocturnal ventilation, requiring optimization of CPAP settings.

5. Discussion and Literature Review

We have reported a prospective series of three cases of hyperacute ischemic stroke managed within an MRI-first pathway. The limitations of our study lie in the small sample size, the absence of systematic perfusion imaging, and the non-standard-

ized quantification of slow flow, which does not allow statistical analyses. Another limitation concerns the absence of 90-day functional follow-up using the modified Rankin Scale (mRS), as all patients were discharged early and could not be reached afterwards. Early clinical recovery and 24-hour MRI findings therefore served as short-term outcome measures. However, it has several strengths: clinico-radiological coherence (concordant FVH), hyperacute time window, and favorable outcomes after IVT, in line with certain publications [10] [11]. This study also allowed us to present a literature review focused on slow flow and its role in therapeutic decision-making.

5.1. Definition of Slow Flow

Also referred to as "FLAIR vascular hyperintensities" (FVH), slow flow reflects arterial hemodynamic slowing (reduced anterograde flow and/or retrograde collateral filling) distal to a stenosis/occlusion; it is frequent in large-vessel or distal occlusions and regresses after reperfusion [7] [8]. Quantitatively, Kufner *et al.* showed that slow flow is associated with wider perfusion deficits, more severe hypoperfusion, and greater infarct growth, which is more pronounced in the absence of reperfusion [10]. By contrast, recent work by Legrand *et al.* in 2023 emphasizes that the raw extent of slow flow does not always faithfully reflect collateral quality without considering DWI; hence the need for an integrated FLAIR-and-diffusion reading [11].

5.2. The FVH-DWI "Mismatch": A Surrogate of Penumbra

When FVH (slow flow) extend beyond the limits of a DWI lesion (or when no lesion is present), the FVH-DWI mismatch aligns with the perfusion diffusion mismatch and identifies patients likely to benefit from recanalization (IVT) [4] [11]. In large-vessel occlusions, Legrand *et al.* also report that the quality of this mismatch seems a better surrogate of collateral status than the simple sum of FVH [11]. Several series and meta-analyses confirm the prognostic value of these slow-flow signs (and their scores) in patients treated with reperfusion therapies, including endovascular treatment and/or IVT (EVT/IVT) [12] [13].

5.3. Negative DWI, Early Symptoms, and the IVT Decision

The meta-analysis by Edlow *et al.* [14], extending the seminal observations of Oppenheim *et al.* [15], reminds us that a negative DWI does not exclude ischemic stroke in the very first hours, particularly in posterior circulation or minor presentations; slow flow then constitutes a hemodynamic indicator of hypoperfusion. More recently, in 2023, Zhu *et al.* [16] showed that a proportion of patients treated with IVT remain DWI-negative on follow-up, with good clinical outcomes, underscoring the value of indirect indices such as slow flow to avoid delaying decision-making. The cases we report are consistent with the literature, since the early decision was based on clinical-slow-flow concordance despite an initially negative or minimal diffusion signal in some patients.

5.4. Low NIHSS: Distinguishing "Minor Non-Disabling" from "Low but Disabling"

The 2019 AHA/ASA guidelines discourage IVT for non-disabling symptoms with NIHSS ≤ 5, but allow it for disabling deficits (aphasia, upper-limb hemiparesis, neglect) despite a low NIHSS (≤5) [5]. The PRISMS trial conducted by Khatri *et al.* in 2018 showed no benefit of alteplase versus aspirin in minor non-disabling stroke [17], whereas the ARAMIS trial by Chen *et al.* [6] demonstrated the non-inferiority of dual antiplatelet therapy compared with IVT in those same non-disabling profiles; the decision should therefore remain personalized, integrating clinical assessment and imaging, according to the conclusions of Chen *et al.* and Ferrari *et al.* [6] [18]. The cases we report clearly illustrate presentations with low NIHSS and disabling symptoms (patients 1 and 3) and concordant slow-flow findings, justifying IVT, which was followed by a favorable outcome without early hemorrhagic transformation.

5.5. Thrombolytic Agent: Alteplase or Tenecteplase?

Until recently, including during the study period, alteplase at a dose of 0.9 mg/kg was the standard for IVT in neurovascular practice. The 2023 ESO recommendations accept tenecteplase (TNK) at 0.25 mg/kg as a safe and effective alternative within 4 h 30 min of symptom onset. Several 2024-2025 syntheses tend to consider TNK (0.25 mg/kg) a preferable option in practice (single bolus, simplified logistics) [19]-[21]. All patients in our series were treated with alteplase, which does not diminish the central point: regardless of the agent, integrating slow flow and diffusion (DWI) is helpful for decision-making, especially when perfusion imaging is not available [22].

5.6. Distal Occlusions (M2/M3, ACA) and "Slow Flow"

As noted by Ahn *et al.* [23], FVH are particularly visible in distal territories (M2/M3, perisylvian/ACA) due to retrograde collateral flow; they anchor clinical-imaging concordance when DWI is negative or minimal. The FVH distribution seen in our cases fits this distal profile and was helpful to localize risk territory and guide management quickly.

6. Conclusion

In hyperacute ischemic stroke, FVH (or slow flow) provide an operational marker of hypoperfusion that may precede parenchymal DWI changes. Integrated with the assessment of disabling symptoms, FVH strengthen IVT decisions and offer a penumbra surrogate via the FVH-DWI mismatch when perfusion imaging is unavailable. In resource-limited settings, notably sub-Saharan Africa where IVT remains unevenly deployed and emergent perfusion imaging is rarely accessible, recognizing FVH can secure and accelerate therapeutic decisions. Prospective studies with standardized FVH quantification and angio-perfusion correlations should refine their place in reperfusion algorithms and interactions with agent choice (al-

teplase vs tenecteplase).

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

[1] Feigin, V.L., Brainin, M., Norrving, B., Martins, S.O., Pandian, J., Lindsay, P., *et al.* (2025) World Stroke Organization: Global Stroke Fact Sheet 2025. *International Journal of Stroke*, **20**, 132-144. https://doi.org/10.1177/17474930241308142

[2] Sanossian, N., Saver, J.L., Alger, J.R., Kim, D., Duckwiler, G.R., Jahan, R., *et al.* (2009) Angiography Reveals That Fluid-Attenuated Inversion Recovery Vascular Hyperintensities Are Due to Slow Flow, Not Thrombus. *American Journal of Neuroradiology*, **30**, 564-568. https://doi.org/10.3174/ajnr.a1388

[3] Saver, J.L. (2006) Time Is Brain—Quantified. *Stroke*, **37**, 263-266. https://doi.org/10.1161/01.str.0000196957.55928.ab

[4] Legrand, L., Tisserand, M., Turc, G., Naggara, O., Edjlali, M., Mellerio, C., *et al.* (2015) Do FLAIR Vascular Hyperintensities Beyond the DWI Lesion Represent the Ischemic Penumbra? *American Journal of Neuroradiology*, **36**, 269-274. https://doi.org/10.3174/ajnr.a4088

[5] Powers, W.J., Rabinstein, A.A., Ackerson, T., Adeoye, O.M., Bambakidis, N.C., Becker, K., *et al.* (2019) Guidelines for the Early Management of Patients with Acute Ischemic Stroke: 2019 Update to the 2018 Guidelines for the Early Management of Acute Ischemic Stroke: A Guideline for Healthcare Professionals from the American Heart Association/American Stroke Association. *Stroke*, **50**, e344-e418. https://doi.org/10.1161/str.0000000000000211

[6] Chen, H., Cui, Y., Zhou, Z., Zhang, H., Wang, L., Wang, W., *et al.* (2023) Dual Antiplatelet Therapy vs Alteplase for Patients with Minor Nondisabling Acute Ischemic Stroke. *Journal of the American Medical Association*, **329**, Article 2135. https://doi.org/10.1001/jama.2023.7827

[7] Zeng, L., Chen, J., Liao, H., Wang, Q., Xie, M. and Wu, W. (2021) Fluid-Attenuated Inversion Recovery Vascular Hyperintensity in Cerebrovascular Disease: A Review for Radiologists and Clinicians. *Frontiers in Aging Neuroscience*, **13**, Article 790626. https://doi.org/10.3389/fnagi.2021.790626

[8] Karadeli, H.H., Giurgiutiu, D., Cloonan, L., Fitzpatrick, K., Kanakis, A., Ozcan, M.E., *et al.* (2016) FLAIR Vascular Hyperintensity Is a Surrogate of Collateral Flow and Leukoaraiosis in Patients with Acute Stroke Due to Proximal Artery Occlusion. *Journal of Neuroimaging*, **26**, 219-223. https://doi.org/10.1111/jon.12274

[9] Kamr, W.H., Almalki, M.S., Saadawy, A.M.I. and El-Tahan, A. (2021) FLAIR Vascular Hyperintensity, an Early Sign of Stroke (Case Report). *Egyptian Journal of Radiology and Nuclear Medicine*, **52**, Article 243.

[10] Kufner, A., Galinovic, I., Ambrosi, V., Nolte, C.H., Endres, M., Fiebach, J.B., *et al.* (2015) Hyperintense Vessels on FLAIR: Hemodynamic Correlates and Response to Thrombolysis. *American Journal of Neuroradiology*, **36**, 1426-1430. https://doi.org/10.3174/ajnr.a4320

[11] Legrand, L., Le Berre, A., Seners, P., Benzakoun, J., Ben Hassen, W., Lion, S., *et al.* (2023) FLAIR Vascular Hyperintensities as a Surrogate of Collaterals in Acute Stroke: DWI Matters. *American Journal of Neuroradiology*, **44**, 26-32. https://doi.org/10.3174/ajnr.a7733

[12] Legrand, L., Turc, G., Edjlali, M., Beaumont, M., Gautheron, V., Ben Hassen, W., *et al.* (2019) Benefit from Revascularization after Thrombectomy According to FLAIR Vascular Hyperintensities–DWI Mismatch. *European Radiology*, **29**, 5567-5576. https://doi.org/10.1007/s00330-019-06094-y

[13] Wang, C., Wang, C. and Ni, Y. (2024) FLAIR Vascular Hyperintensity Is Associated with Functional Outcome in Patients with Ischemic Stroke Receiving Endovascular Treatment: A Meta-Analysis. *Frontiers in Neurology*, **15**, Article 1497504. https://doi.org/10.3389/fneur.2024.1497504

[14] Edlow, B.L., Hurwitz, S. and Edlow, J.A. (2017) Diagnosis of DWI-Negative Acute Ischemic Stroke. *Neurology*, **89**, 256-262. https://doi.org/10.1212/wnl.0000000000004120

[15] Oppenheim, C., Stanescu, R., Dormont, D., *et al.* (2000) False-Negative Diffusion-weighted MR Findings in Acute Ischemic Stroke. *American Journal of Neuroradiology*, **21**, 1434-1440.

[16] Zhu, C., Qin, W., Xu, J. and Hu, W. (2023) Perfusion Deficits in Thrombolysis-Treated Acute Ischemic Stroke Patients with Negative or Positive Diffusion-Weighted Imaging. *BMC Neurology*, **23**, Article No. 380. https://doi.org/10.1186/s12883-023-03427-9

[17] Khatri, P., Kleindorfer, D.O., Devlin, T., Sawyer, R.N., Starr, M., Mejilla, J., *et al.* (2018) Effect of Alteplase vs Aspirin on Functional Outcome for Patients with Acute Ischemic Stroke and Minor Nondisabling Neurologic Deficits. *Journal of the American Medical Association*, **320**, 156-166. https://doi.org/10.1001/jama.2018.8496

[18] Ferrari, J., Reynolds, A., Knoflach, M. and Sykora, M. (2021) Acute Ischemic Stroke with Mild Symptoms—To Thrombolyse or Not to Thrombolyse? *Frontiers in Neurology*, **12**, Article 760813. https://doi.org/10.3389/fneur.2021.760813

[19] Alamowitch, S., Turc, G., Palaiodimou, L., Bivard, A., Cameron, A., De Marchis, G.M., *et al.* (2023) European Stroke Organisation (ESO) Expedited Recommendation on Tenecteplase for Acute Ischaemic Stroke. *European Stroke Journal*, **8**, 8-54. https://doi.org/10.1177/23969873221150022

[20] Wang, L., Hao, M., Wu, N., Wu, S., Fisher, M. and Xiong, Y. (2024) Comprehensive Review of Tenecteplase for Thrombolysis in Acute Ischemic Stroke. *Journal of the American Heart Association*, **13**, e031692. https://doi.org/10.1161/jaha.123.031692

[21] Muir, K.W., Ford, G.A., Ford, I., Wardlaw, J.M., McConnachie, A., Greenlaw, N., *et al.* (2024) Tenecteplase versus Alteplase for Acute Stroke within 4·5 H of Onset (AT-TEST-2): A Randomised, Parallel Group, Open-Label Trial. *The Lancet Neurology*, **23**, 1087-1096. https://doi.org/10.1016/s1474-4422(24)00377-6

[22] Song, L., Lyu, C., Shen, G., Guo, T., Wang, J., Wang, W., *et al.* (2019) Application of FLAIR Vascular Hyperintensity-Dwi Mismatch in Ischemic Stroke Depending on Semi-Quantitative Dwi-Alberta Stroke Program Early CT Score. *Frontiers in Neurology*, **10**, Article 994. https://doi.org/10.3389/fneur.2019.00994

[23] Ahn, S.J., Suh, S.H., Lee, K.-Y., Kim, J.H., Seo, K.-. and Lee, S. (2015) Hyperintense Vessels on T2-PROPELLER-FLAIR in Patients with Acute MCA Stroke: Prediction of Arterial Stenosis and Perfusion Abnormality. *American Journal of Neuroradiology*, **36**, 2042-2047. https://doi.org/10.3174/ajnr.a4423