

Perivascular and Subarachnoid Fluid-Attenuated Inversion Recovery Hyperintensities Related to Delayed Gadolinium Leakage After Stroke

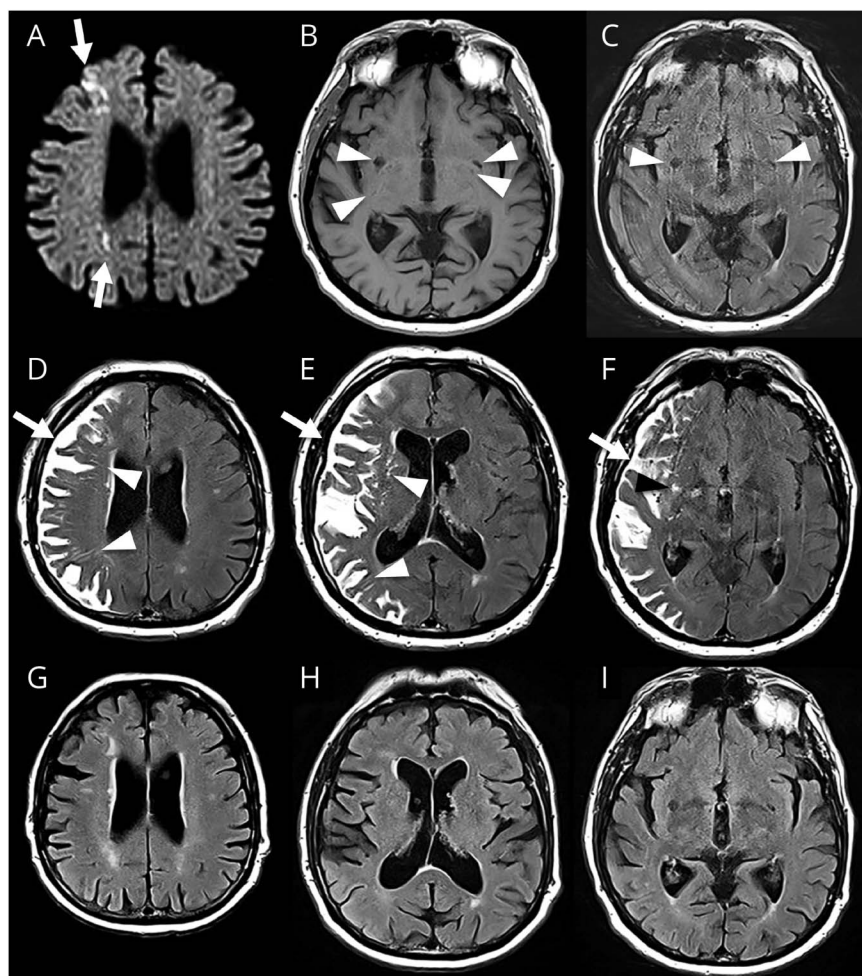
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Figure Imaging Results



Initial MRI (gadolinium-injected for magnetic resonance angiography) shows right-sided infarction (A, diffusion-weighted imaging) and perivascular spaces (PVS) (B, T1-weighted imaging; C, fluid-attenuated inversion recovery [FLAIR]). (D–F) Four hours later, MRI shows ipsilateral subarachnoid (arrows) and deep and subcortical PVS (arrowheads) gadolinium leakage seen as FLAIR hyperintensities. (G–I) Complete resolution of subarachnoid/perivascular FLAIR abnormalities was observed 10 days later.

A 74-year-old man presented with right watershed infarction (initial gadolinium-injected MRI, Figure). Renal function was normal. MRI 4 hours later showed ipsilateral perivascular spaces (PVS) and subarachnoid fluid-attenuated inversion recovery (FLAIR) hyperintensities,

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probably related to stroke-associated delayed gadolinium leakage (Figure). MRI after 10 days showed complete resolution of subarachnoid/perivascular abnormalities (Figure).

Stroke-related subarachnoid and ocular gadolinium leakage on FLAIR probably represents blood–brain/ocular barrier disruption.^{1,2,e1} An earlier report described PVS gadolinium leakage on MRI performed >1 month after stroke.^{e1} The glymphatic system (playing a role in CSF–interstitial fluid interchange) may be involved in stroke-related blood–brain barrier leakage observed in the subarachnoid space and PVS.

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Name	Location	Contribution
Larisa Nistorec, MD	Department of Neurology, Nîmes University Hospital, France	Drafting/revision of the manuscript for content, including medical writing for content
Dimitri Renard, MD	Department of Neurology, Nîmes University Hospital, France	Drafting/revision of the manuscript for content, including medical writing for content
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2. Warach S, Latour LL. Evidence of reperfusion injury, exacerbated by thrombolytic therapy, in human focal brain ischemia using a novel imaging marker of early blood-brain barrier disruption. *Stroke*. 2004;35(11 suppl 1):2659-2661. Supplemental data (eReference 1) is available at: links.lww.com/WNL/B552

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