FLAIR Vascular Hyperintensity as a Marker of Leptomeningeal Collaterals in Subacute Stroke

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Purpose
FLAIR vascular hyperintensity during the acute stage of ischemic stroke has been described as an early finding associated with hypoperfusion. Proximal arterial hyperintensities may be caused by occlusive thrombus or slow antegrade flow, although the nature of more distal vascular hyperintensities remains unclear. We hypothesize that distal vascular hyperintensities on FLAIR sequences represent slow leptomeningeal collateral flow that may be observed into the subacute stage of ischemic stroke.

Materials & Methods
FLAIR images and CT angiography (CTA) source images of 28 patients (median age, 66 years, range, 22-89 years; 14 male, 14 female) with supratentorial ischemic stroke were reviewed. FLAIR and CTA were performed within 14 days of stroke onset in all cases. The average interval between the two imaging studies was 2.63 days. Two blinded reviewers noted the presence or absence of FLAIR vascular hyperintensities and graded the extent of these findings on a 4-point scale. Two blinded reviewers also independently rated the CTA source images with a standardized scale of leptomeningeal collaterals incorporating vessel patency.

Results
Distal FLAIR vascular hyperintensities were observed in 13/28 cases, predominantly located at the sylvian fissure and over the cerebral convexities. The extent of these hyperintensities was limited to a single focus in two cases, involved a limited area of distal vessels in seven cases, and extended across the cerebral hemisphere in four cases. The presence of a distal FLAIR vascular hyperintensity correlated with the presence of either a proximal vessel stenosis or occlusion ($r = 0.52$, $p < 0.001$), although a stronger correlation was noted with the presence of a proximal occlusion ($r = 0.65$, $p < 0.001$). The presence of a distal FLAIR vascular hyperintensity showed a strong correlation with the presence of leptomeningeal collaterals on CTA source images ($r = 0.71$, $P < 0.001$). The extent of FLAIR vascular hyperintensities, however, exhibited no relationship with respect to the degree of leptomeningeal collaterals. No correlation was noted between the presence of FLAIR vascular hyperintensity and age, gender, or time from symptom onset.
Conclusion
Distal FLAIR vascular hyperintensities may be observed into the subacute stage of ischemic stroke. The presence of these hyperintensities may be associated with a proximal vascular occlusion and preserved leptomeningeal collaterals. Recognition of distal FLAIR vascular hyperintensities may have therapeutic implications during the subacute period.

References