Clinical response to intravenous thrombolysis depends on the extent of early ischemic changes on noncontrast computed tomography (NCCT). These are subtle markers of parenchymal damage and include (1) loss of delineation of the lentiform nucleus/caudate head/internal capsule, (2) sulcal effacement, and (3) loss of gray–white junction differentiation underlying the cortical sulci and insula.

Sulcal effacement on NCCT and concomitant preservation of the underlying gray–white matter junction (isolated sulcal effacement [ISE]) may not represent cytotoxic edema. We aimed to evaluate the frequency and significance of ISE in patients with large vessel occlusion acute ischemic stroke.

Methods—Consecutive acute ischemic stroke patients with middle cerebral artery M1 or internal carotid artery terminus occlusions who underwent computed tomography angiogram/perfusion followed by intra-arterial therapy were screened for ISE.

Results—Out of the 568 patients who underwent intra-arterial therapy between March 2011 and September 2014, 108 fulfilled inclusion criteria. ISE was present in 8 (7.4%) patients (age 55.7±10.5 years, 6 female, baseline National Institutes of Health Stroke Scale 16.1±3.8, 5 middle cerebral artery-M1, and 3 internal carotid artery terminus occlusions). Computed tomography angiogram revealed engorged/dilated leptomeningeal vessels obliterating the sulci within the areas of effacement, whereas computed tomography perfusion indicated normal-to-increased cerebral blood volume and prolonged $T_{\text{max}}$ in all patients. Modified treatment in cerebral ischemia (mTICI) 2b-3 reperfusion was achieved in all patients. Follow-up imaging confirmed no infarct in the ISE area in all patients, and 5 (62%) had modified Rankin Scale 0 to 2 at 3 months.

Conclusions—Sulcal effacement with preserved gray–white delineation is occasionally visualized in patients with proximal occlusion strokes, relates to robust leptomeningeal collaterals, and indicates preserved underlying parenchyma. ISE should not be used to exclude patients from thrombectomy. (Stroke. 2015;46:1704-1706. DOI: 10.1161/STROKEAHA.115.009304.)

Key Words: brain edema • ischemia • stroke • tomography, computed, scanners
leptomeningeal vessels obliterating the sulci within the areas of ISE in all patients.

Six of the 8 patients had technically adequate perfusion maps. All patients had prolonged $T_{\text{max}}$ within the ISE territories and regionally normal-to-increased cerebral blood volume. The cerebral blood flow patterns were variable. The mean ischemic core was $20.3\pm20.5$ cc (range 0–61), perfusion defect $144.0\pm53.3$ cc, and penumbral volume $106.0\pm56.9$ cc.

Cerebral angiography confirmed good collateral flow in all optimally studied patients. Complete and rapid collateralization of the vascular bed in the entire ischemic territory was seen in 3 patients with M1 occlusions. One patient had a proximal M1 occlusion partially obstructing the A1 segment of the anterior cerebral artery (with anterior communicating artery cross-flow and posterior cerebral artery leptomeningeal flow). One patient with MCA-M1 occlusion had a hypoplastic ipsilateral A1 segment of the anterior cerebral artery and 3 had internal carotid artery terminus occlusions.

**Discussion**

In this study of patients with large vessel occlusion AIS, we found sulcal effacement with underlying preservation of the gray–white differentiation to be indicative of brain tissue viability. This phenomenon is likely related to robust leptomeningeal collateral flow at the time of image acquisition.

von Kummer et al suggested that brain swelling without concomitant parenchymal hypopattenuation may represent a compensatory vasodilation in regions of low perfusion pressure. However, sulcal effacement continues to be broadly referred to as a marker of early ischemic changes and has been imprecisely used as a sign of parenchymal injury. Our findings reinforce the idea that sulcal effacement without hypoattenuation and with preserved gray–white junction represents viable tissue. The development of ISE happens before the occurrence of cytotoxic edema (leading to loss of gray–white differentiation) and of blood–brain barrier breakdown (leading to vasogenic edema and hypopattenuation). Two reports evaluated the presence of ISE in AIS patients. One found ISE in 13% (22/172) and the other in 20% (6/30) of patients, and both revealed uniformly increased cerebral blood volume. Most patients were managed conservatively (only 2 IAT), and only 35% to 50% had lack of infarct within the ISE territory on follow-up imaging. The frequency of ISE in our report compares favorably (7.4%), despite including only patients with confirmed large vessel occlusion. The CT perfusion patterns of our patients are comparable. We have demonstrated that the area with ISE was always preserved on follow-up imaging, which may relate to the fact that all of our patients were successfully reperfused.

This study has multiple limitations, mostly associated with the inherent challenges of retrospective analyses. Because of the small number of identified patients, group comparisons could not be performed and trends could not be characterized.

**Conclusions**

Sulcal effacement with preserved gray–white delineation is occasionally visualized in patients with proximal occlusion
is associated with robust leptomeningeal collateral flow, and indicates the presence of underlying preserved brain parenchyma. The presence of ISE should not be used to exclude patients from acute reperfusion therapy.

Disclosures
Dr Nogueira: Stryker (PI:Trevo-2 PI/DAWN Trials), Covidien (SWIFT/SWIFT-PRIME Steering Committee, STAR Trial Core-Laboratory), and Penumbra (3D Trial Executive Committee). The other authors report no conflicts.

References

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ACA indicates anterior cerebral artery; CBF, cerebral blood flow; CBV, cerebral blood volume; CTP, CT perfusion; ISE, isolated sulcal effacement; length, procedural length; MAP, mean arterial pressure; MCA, middle cerebral artery; mRS, modified Rankin Scale; mTICI, modified treatment in cerebral ischemia; onset-groin puncture, time interval between stroke-onset and groin puncture; PCA, posterior cerebral artery; NIHSS, National Institutes of Health Stroke Scale; site, intracranial occlusion site; IPA, tissue-type plasminogen activator; and TTP, time to peak.

*Perfusion scan limited by motion.
Sulcal Effacement With Preserved Gray–White Junction: A Sign of Reversible Ischemia
Diogo C. Haussen, Andrey Lima, Michael Frankel, Aaron Anderson, Samir Belagaje, Fadi Nahab, Srikant Rangaraju and Raul G. Nogueira

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