Sulcal Effacement With Preserved Gray–White Junction
A Sign of Reversible Ischemia

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Background and Purpose—Sulcal effacement with preserved underlying gray–white matter junction (isolated sulcal effacement [ISE]) in acute ischemic stroke may not represent irreversible parenchymal injury. 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_{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

Clinical response to intravenous thrombolysis depends on the extent of early ischemic changes on noncontrast computed tomography (NCCT).1 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.2,3

Sulcal effacement on NCCT and concomitant preservation of the underlying gray–white matter junction (isolated sulcal effacement [ISE]) may not represent cytotoxic edema.1,4,5 We aimed to evaluate the frequency and significance of ISE in patients with large vessel occlusion acute ischemic stroke (AIS) undergoing intra-arterial therapy (IAT).

Methods

Inclusion and Exclusion Criteria

We performed a retrospective review of patients consecutively treated with IAT for AIS between March 2011 and September 2014 in a tertiary academic center. Individuals with middle cerebral artery (MCA) M1 or internal carotid artery terminus occlusions who had baseline CT angiogram/CT perfusion and follow-up magnetic resonance imaging or NCCT were included. This study was approved by the local Institutional Board Review.

Image Analysis

The baseline NCCTs (axial) were reviewed independently by 2 reviewers for asymmetry in hemispherical sulci, followed by a consensus read. Patients with previous strokes were excluded. NCCT was used to grade ASPECTS.6 CT angiograms were evaluated and leptomeningeal vascularity graded as less, equal, or greater than the contralateral unaffected side.7 CT perfusion was used to estimate the ischemic core lesion and perfusion deficit (RAPID, iSchemaView Inc, CA). Follow-up magnetic resonance imaging (preferred) or NCCT were used to evaluate the fate of the ISE area. Hemorrhagic transformation was graded by European Cooperative Acute Stroke Study (ECASS) and reperfusion by modified treatment in cerebral ischemia (mTICI) criteria.8,9

Results

Out of 568 patients who underwent IAT for AIS within the study period, 108 fit inclusion criteria. Fifteen patients had asymmetrical sulci and preserved gray–white delineation; however, 7 were excluded because of previous strokes, leading to 8 (7.4%) patients with ISE for the primary
analysis (Figure). Mean age was 55.7±10.5 years, baseline SBP 134.0±13.7 mm Hg, and National Institutes of Health Stroke Scale 16.1±3.8 (Table). Mean ASPECTS (not including ISE as abnormal) was 7.0±1.8. A total of 33 cortical regions had ISE (mean of 4.1 per patient). Mean time from last-known-normal to groin-puncture was 519.1±248.3 minutes and procedural length 96.0±40.6 minutes. Stentretrivers (5), thromboaspiration (2), or a combination (1) were used, leading to 100% mTICI 2b-3 reperfusion. Seven patients had a follow-up magnetic resonance imaging and 1 NCCT (median interval from IAT, 35 hours). No patients developed infarct in the area of ISE on follow-up imaging. Five (62%) achieved modified Rankin Scale 0–2 at 3 months.

Isolated Sulcal Effacement
ISE isolated to the MCA was noted in 4 patients, MCA/posterior cerebral artery in 2, and anterior cerebral artery/MCA/posterior cerebral artery in 2. CT angiogram revealed engorged/dilated 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±20.5 cc (range 0–61), perfusion defect 144.0±53.3 cc, and penumbral volume 106.0±56.9 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 hypoattenuation 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 occurs before the occurrence of cytotoxic edema (leading to loss of gray–white differentiation) and of blood–brain barrier breakdown (leading to vasogenic edema and hypoattenuation).

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
strokes, 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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**Table. Clinical and Radiological Variables of Patients With ISE**

<table>
<thead>
<tr>
<th>Age/Sex</th>
<th>NIHSS</th>
<th>MAP</th>
<th>IV IPA</th>
<th>ISE Area</th>
<th>CTP</th>
<th>Onset-Groin Puncture</th>
<th>Length</th>
<th>Site</th>
<th>Tandem</th>
<th>mTICI</th>
<th>Bleed</th>
<th>mRS</th>
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<tr>
<td>76F</td>
<td>19</td>
<td>69</td>
<td>+</td>
<td>MCA</td>
<td>↑↑</td>
<td>CBV ↑↑ CBF ↑↑ TTP</td>
<td>270 m</td>
<td>M1</td>
<td>–</td>
<td>3</td>
<td>–</td>
<td>2</td>
</tr>
<tr>
<td>64F</td>
<td>12</td>
<td>84</td>
<td>+</td>
<td>MCA</td>
<td>↑</td>
<td>CBV ↑ CBF ↑↑ TTP</td>
<td>865 m</td>
<td>M1</td>
<td>–</td>
<td>3</td>
<td>–</td>
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<tr>
<td>49M</td>
<td>12</td>
<td>94</td>
<td>–</td>
<td>MCA/PCA</td>
<td>↑</td>
<td>CBV ↑↑ CBF ↑↑ TTP</td>
<td>520 m</td>
<td>M1</td>
<td>–</td>
<td>3</td>
<td>PH2</td>
<td>1</td>
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<tr>
<td>38F</td>
<td>19</td>
<td>89</td>
<td>+</td>
<td>MCA/PCA</td>
<td>*</td>
<td>CBV ↑↑ CBF ↑↑ TTP</td>
<td>569 m</td>
<td>M1</td>
<td>–</td>
<td>3</td>
<td>–</td>
<td>4</td>
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<tr>
<td>55M</td>
<td>21</td>
<td>113</td>
<td>–</td>
<td>MCA/PCA</td>
<td>↑</td>
<td>CBV ↑↑ CBF ↑↑ TTP</td>
<td>580 m</td>
<td>M1</td>
<td>–</td>
<td>2b</td>
<td>H2</td>
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<tr>
<td>60F</td>
<td>11</td>
<td>89</td>
<td>–</td>
<td>ACA/MCA/PCA</td>
<td>↑↑</td>
<td>CBV ↑↑ CBF ↑↑ TTP</td>
<td>375 m</td>
<td>M1</td>
<td>–</td>
<td>2b</td>
<td>H2+SAH</td>
<td>3</td>
</tr>
<tr>
<td>51F</td>
<td>20</td>
<td>90</td>
<td>–</td>
<td>ACA/MCA/PCA</td>
<td>*</td>
<td>CBV ↑↑ CBF ↑↑ TTP</td>
<td>863 m</td>
<td>M1</td>
<td>–</td>
<td>2b</td>
<td>–</td>
<td>1</td>
</tr>
</tbody>
</table>

ACA indicates anterior cerebral artery; CBF, cerebral blood flow; CBV, cerebral blood volume; CTP, CT perfusion; ICA, internal carotid artery; 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.
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