Vessel Wall Magnetic Resonance Imaging in Acute Ischemic Stroke
Effects of Embolism and Mechanical Thrombectomy on the Arterial Wall

Sarah Power, MB, PhD; Charles Matouk, MD; Leanne K. Casaubon, MD; Frank L. Silver, MD; Timo Krings, MD, PhD; David J. Mikulis, MD; Daniel M. Mandell, MD, PhD

Background and Purpose—The aim of the study was to determine the effects of thromboembolism and mechanical thrombectomy on the vessel wall magnetic resonance imaging (VW-MRI) appearance of the intracranial arterial wall.

Methods—This was a cross-sectional study of consecutive patients with acute intracranial arterial occlusion who underwent high-resolution contrast-enhanced VW-MRI within days of stroke presentation. For each patient, we categorized arterial wall thickening and enhancement as definite, possible, or none using contralateral arteries as a reference standard. We performed $\chi^2$ tests to compare the effects of medical therapy and mechanical thrombectomy.

Results—Sixteen patients satisfied inclusion criteria. Median time from symptom onset to VW-MRI was 3 days (interquartile range, 2 days). Among 6 patients treated with mechanical thrombectomy using a stent retriever, VW-MRI demonstrated definite arterial wall thickening in 5 (83%) and possible thickening in 1 (17%); there was definite wall enhancement in 4 (67%) and possible enhancement in 2 (33%). Among 10 patients treated with medical therapy alone, VW-MRI demonstrated definite arterial wall thickening in 3 (30%) and possible thickening in 2 (20%); there was definite wall enhancement in 2 (20%) and possible enhancement in 2 (20%). Arterial wall thickening and enhancement were more common in patients treated with mechanical thrombectomy than with medical therapy alone ($P=0.037$ and $P=0.016$, respectively).

Conclusions—Mechanical thrombectomy results in intracranial arterial wall thickening and enhancement, potentially mimicking the VW-MRI appearance of primary arteritis. This arterial wall abnormality is less common in patients with arterial occlusion who have been treated with medical therapy alone. (Stroke. 2014;45:2330-2334.)

Key Words: infarction ■ magnetic resonance angiography ■ magnetic resonance imaging ■ secondary prevention ■ stroke ■ thrombectomy ■ vasculitis

Secondary prevention of ischemic stroke relies on determination of stroke pathogenesis.1 Yet, conventional clinical, laboratory, and imaging investigations often fail to identify the cause of a stroke.2,3 High-resolution contrast-enhanced vessel wall magnetic resonance imaging (VW-MRI) is an emerging method of differentiating among intracranial causes of stroke such as intracranial atherosclerosis,4,6 central nervous system vasculitis5,7,8 and reversible cerebral vasoconstriction syndrome.8 However, it is not known whether thromboembolism may itself injure the wall of an intracranial artery and mimic the VW-MRI appearance of primary intracranial arteriopathy. Similarly, it is not known whether mechanical thrombectomy alters the VW-MRI appearance of the arterial wall. The purpose of this study was to determine the effects of embolism and mechanical thrombectomy on the appearance of the arterial wall.

Methods

Patients
This was a cross-sectional study of consecutive patients with acute ischemic stroke secondary to intracranial large artery occlusion who underwent contrast-enhanced VW-MRI within days of stroke presentation. The arterial occlusions were diagnosed using computed tomographic angiography at presentation, and VW-MRI was performed days later as part of a secondary prevention clinical imaging protocol. Subjects were retrospectively identified from a prospectively maintained database of intracranial VW-MRI cases. The study was approved by the Research Ethics Board of the University Health Network.

VW-MRI Protocol
Magnetic resonance imaging was performed using a Signa HDx 3.0-T scanner with an 8-channel head coil (GE Healthcare, Milwaukee, WI). The VW-MRI protocol included a time-of-flight magnetic resonance angiogram of the circle of Willis and T1-weighted black blood vessel...
wall sequences (single inversion recovery-prepared, 2-dimensional fast spin echo acquisition with field of view=22x22 cm, acquired matrix=512x512, slice thickness=2 mm, total slab thickness=3 cm, and TR/TI/TE=2263/860/13 ms) before and after intravenous administration of gadolinium. The same scan parameters were used for the nonenhanced and enhanced sequences. Imaging was monitored by a neuroradiologist to target the vessel that was occluded on the initial computed tomographic angiogram. The vessel wall sequences were run in both short- and long-axis planes through each targeted artery. The total magnetic resonance imaging time for each patient was 1 hour.

Classification of Acute Ischemic Strokes

We performed a detailed chart review for each case to categorize the cause of stroke. Strokes were categorized as (1) cardioaortic embolism, (2) supra-aortic large artery atherosclerosis, (3) other, or (4) undetermined using the Causative Classification System for Ischemic Stroke.9–11

Image Interpretation and Analysis

A neuroradiologist, blinded to clinical data including patient management, reviewed the initial computed tomographic angiograms and the VW-MRI. All the VW-MRI studies were deemed acceptable for inclusion. Using the magnetic resonance angiogram, we categorized the lumen as complete recanalization, partial recanalization, or no recanalization. Using the nonenhanced T1-weighted vessel wall sequence, we recorded whether there was hyperintensity within the arterial wall, that is, evidence of intramural blood product.12 For each patient, we categorized arterial wall thickening and arterial wall enhancement as definite, possible, or none using the contralateral arteries as a reference standard. The magnetic resonance imaging of arterial wall thickening and enhancement categories were assigned based on all of the nonenhanced and contrast-enhanced vessel wall cross-sectional images corresponding with vessel occlusion on computed tomographic angiography. We performed χ² tests to compare the incidence of possible/definite arterial wall thickening and wall enhancement in patients treated with mechanical thrombectomy versus medical therapy alone.

Results

There were 16 subjects; 8 were women; median age was 58.5 years (interquartile range, 18.5 years). The Table provides the demographic, clinical, and MRI details for each subject.

Stroke etiologies for the 16 patients were cardioaortic embolism evident (n=8), cardioaortic embolism possible (n=3), supra-aortic large artery atherosclerosis evident (n=2), and undetermined despite extensive investigation (n=3). The majority of patients (n=6) in the cardioembolic embolism evident category had either chronic or paroxysmal atrial fibrillation. The source of embolism for the patients in the large artery atherosclerotic evident category was ipsilateral carotid bulb atherosclerotic plaque.

The arterial occlusions were located in the anterior circulation in all patients except one who had a posterior cerebral artery (P2-3 segment) occlusion. Twelve (75%) patients received intravenous tissue-type plasminogen activator. Six patients (38%) underwent mechanical thrombectomy using a stent retriever. The stent retriever device was either a Solitaire AB (Covidien, EV3 Neurovascular, Irvine, CA) or Trevo (Stryker Neurovascular, Mountain View, CA), with a single pass of the device in each case.

Median time from symptom onset to VW-MRI was 3 days (interquartile range, 2 days). Magnetic resonance angiography demonstrated complete recanalization in 14 patients and partial recanalization in 2 patients. One patient with partial recanalization (subject 5) had a linear branching region of hyperintensity within

Table. Patient Demographics and VW-MRI Findings in 16 Patients With Large Artery Occlusive Stroke

<table>
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<th>Subject No.</th>
<th>Age, y</th>
<th>Sex</th>
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<th>Stroke Classification</th>
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<th>MT</th>
<th>Imaging Interval, d</th>
<th>Arterial Wall Thickening</th>
<th>Arterial Wall Enhancement</th>
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<td>Y</td>
<td>2</td>
<td>Definite</td>
<td>Definite</td>
<td>M₂</td>
</tr>
</tbody>
</table>

Imaging interval is time in days from symptom onset to vessel wall imaging. Cause of stroke as determined by Causative Classification System for Ischemic stroke.9–11 F indicates female; L, left side; M, male; MCA, middle cerebral artery; MT, mechanical thrombectomy; PCA, posterior cerebral artery; R, right side; T, carotid terminus; tPA, tissue-type plasminogen activator; and VW-MRI, vessel wall magnetic resonance imaging.

*Cardioaortic embolism possible.
†Cardioaortic embolism evident.
‡Supra-aortic large artery atherosclerosis evident.
retriever in place, there is antegrade filling of a left M2 superior
segment, and across the carotid terminus occlusion. With the stent
deployed from the superior division of MCA, through the M1 seg-
ment image in the short axis through the MCA shows that the


demonstrated intramural thrombus after stent retriever use, 15 but

Figure 1. Effect of mechanical thrombectomy on the arterial wall
(subject 10). A. Catheter angiography (anterioposterior view)
demonstrates occlusion of the left carotid terminus and middle
Cerebral artery (MCA) M1 segment. A stent retriever has been
deployed from the superior division of MCA, through the M1 seg-
ment, and across the carotid terminus occlusion. With the stent
retriever in place, there is antegrade filling of a left M1 superior
division branch. B. Angiogram after stent retrieval demonstrates
recanalization of the carotid terminus and left MCA. C. Contrast-
enhanced T1-weighted vessel wall magnetic resonance images
in the axial plane show a flow void in the left MCA lumen consist-
tent with recanalization, and smooth thick enhancement of the
left MCA M1 segment arterial wall (2 arrows) compared with a the
normal (barely perceptible) right MCA arterial wall (single arrow).
D. Contrast-enhanced T1-weighted vessel wall magnetic reso-
nance image in the short axis through the MCA shows that the
arterial wall thickening and enhancement are concentric.

Among the 6 patients treated with mechanical thrombectomy,
VW-MRI demonstrated definite arterial wall thickening
in 5 patients and possible wall thickening in 1 patient.
VW-MRI demonstrated definite arterial wall enhancement
in 5 patients and possible wall enhancement in 2 patients.
Figure 1 shows a representative case of a patient treated with
mechanical thrombectomy.

Among the 10 patients treated with medical therapy alone,
VW-MRI demonstrated definite arterial wall thickening in 3 pa-
tients, possible wall thickening in 2 patients, and no wall
thickening in 5 patients. VW-MRI demonstrated definite wall
arterial wall enhancement in 2 patients, possible enhancement
in 2 patients, and no enhancement in 6 patients. Figures 2
and 3 show representative images from patients treated with med-
ical therapy alone.

Figure 1 shows a representative case of a patient treated with
mechanical thrombectomy. Nogueira et al16 reported similar findings with
the Trevo stent retriever. There is some limited histological
data from humans after thrombectomy as well.17 Endothelial
denudation may result in increased endothelial permeability
to intravenous gadolinium and account for the arterial wall
enhancement in patients who have undergone mechanical
thrombectomy. Arterial wall edema may account for the arte-
tial wall thickening in these patients. An alternative explana-
tion for arterial wall enhancement is leakage of gadolinum
from vasa vasora; however, intracranial arteries normally
lack vasa vasora.18 Histopathologic studies have also dem-
onstrated intramural thrombus after stent retriever use,19 but
we did not observe hyperintensity in the arterial wall on the
nonenhanced T1-weighted vessel wall images to provide evi-
dence of intramural blood product in this study.

One of the study subjects (subject 7) who underwent
mechanical thrombectomy provided an opportunity to eval-
uate the differential effects of stent deployment and stent
retrieval on the arterial wall. In this subject, the thrombus had
a Y-configuration, extending into 2 middle cerebral artery M2
branches, but the stent retriever was deployed in just one of
these occluded branches. Both branches were recanalized at
the end of the procedure and both demonstrated pronounced

Arterial wall thickening (definite or possible) was more
common in patients treated with mechanical thrombectomy
than with medical therapy alone (100% versus 50%; P=0.037).
Similarly, arterial wall enhancement was more common in
patients treated with mechanical thrombectomy than with
medical therapy alone (100% versus 40%; P=0.016).

Discussion
This was a cross-sectional study to determine the effects of
thromboembolic occlusion and mechanical thrombectomy
on the VW-MRI appearance of the intracranial arterial wall.
Obtaining VW-MRI within days of recanalization, we found
that there can be smooth concentric arterial wall thickening
and enhancement at the site of a recent arterial occlusion. This
arterial wall abnormality is common among patients treated
with mechanical thrombectomy and less common among
those treated with medical therapy alone.

The pattern of concentric arterial wall thickening and
enhancement we observed in patients treated with mecha-
nical thrombectomy is similar to the VW-MRI appearance
of underlying arteritis.

Histopathologic studies in swine and canine models have
shown that mechanical thrombectomy injures the arterial
wall. Gory et al15 studied a variety of thrombectomy devices,
including the Solitaire FR stent retriever (Covidien, EV3
Neurovascular, Irvine, CA), and found that thrombectomy
causes endothelial denudation, disruption of the internal elas-
tic lamina, and edema in the intimal and medial layers of the
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the lumen at the middle cerebral artery bifurcation on nonenhanced
T1-weighted vessel wall images, consistent with residual nonoc-
clusive luminal thrombus. The other patient with residual narrow-
ing (subject 1) had multiple atherosclerotic risk factors (smoking,
hypertension, dyslipidemia, and diabetes mellitus) despite his
young age, the residual luminal narrowing involved a short seg-
ment of the middle cerebral artery trunk, and the VW-MRI findings
were different from all other cases in that there was focal arterial
wall enhancement at the site of a recent arterial occlusion. This
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arterial wall thickening and enhancement. We postulate that the arterial wall abnormality in the second branch reflects a shearing force on the arterial wall due to pulling occlusive thrombus from the lumen even without deployment of the device itself in the vessel.

Does thromboembolism itself result in a VW-MRI abnormality? Among 10 patients treated with medical therapy alone, only 2 had definite arterial wall thickening and enhancement, and the abnormality in 1 of these patients may have been secondary to an underlying intracranial atherosclerotic plaque. Therefore, it seems that one should consider the possibility of primary intracranial arteriopathy in patients with recent intracranial arterial occlusion who have evidence of arterial wall abnormality, with the knowledge that thromboembolism itself may occasionally result in VW-MRI abnormality. With further refinement of VW-MRI techniques, more subtle thromboembolism-induced arterial wall abnormalities may also become evident.

Toward our goal of using intracranial VW-MRI to differentiate among stroke etiologies in the time period when decisions about secondary prevention are made, we have studied patients in the days after ischemic stroke. However, from a pathophysiology perspective, longer-term follow-up VW-MRI may offer additional insight into the arterial response to mechanical thrombectomy. A recent retrospective study on the long-term angiographic effects of mechanical thrombectomy found that a small proportion (3.4%) of treated arteries developed new stenosis after a median of 3 months, presumably a consequence of arterial wall injury at the time of thrombectomy.19

We were not able to obtain histopathologic correlation for the VW-MRI findings in the study as all patients survived. Particularly for the one subject in whom we suspected intracranial atherosclerotic plaque, we could not be completely certain of this diagnosis without a pathological specimen, so we did not censor the subject from the data analysis. However, if this subject did have an intracranial atherosclerotic plaque accounting for the observed arterial wall thickening and enhancement, this would strengthen rather than weaken the observed difference in VW-MRI findings between the mechanical thrombectomy and medical therapy subgroups.

Conclusions
High-resolution intracranial VW-MRI can demonstrate arterial wall thickening and enhancement at the site of recent thromboembolic arterial occlusion. This arterial wall abnormality is common in patients who have had mechanical thrombectomy using a stent retriever and less common in patients treated with medical therapy alone. Recognition of the post-thrombectomy appearance is important to avoid misinterpretation of the arterial wall abnormality as evidence of underlying arteritis. Conversely, for patients treated with medical therapy alone, pronounced arterial wall abnormality at the
site of occlusion should at least raise the possibility of primary intracranial arteriopathy.

Acknowledgments
Dr Mandell gratefully acknowledges support from the Association of University Radiologists’ General Electric Radiology Research Academic Fellowship.

Disclosures
None.

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