Secondary prevention of ischemic stroke relies on determination of stroke pathogenesis. Yet, conventional clinical, laboratory, and imaging investigations often fail to identify the cause of a stroke. 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, central nervous system vasculitis, and reversible cerebral vasoconstriction syndrome. 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 images of the intracranial vessels.
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) cardioembolic, (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 $\chi^2$ 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 cardioembolic embolism evident (n=8), cardioembolic 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>
<thead>
<tr>
<th>Subject No.</th>
<th>Age, y</th>
<th>Sex</th>
<th>Location of Occlusion</th>
<th>Stroke Classification</th>
<th>tPA</th>
<th>MT</th>
<th>Imaging Interval, d</th>
<th>Arterial Wall Thickening</th>
<th>Arterial Wall Enhancement</th>
<th>Location of Wall Abnormality</th>
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<td>M</td>
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<td>N</td>
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<td>Definite</td>
<td>M1</td>
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<td>M1, M2</td>
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<td>Possible</td>
<td>M2</td>
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<td>R PCA P2-3</td>
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<td>PCA P2-3</td>
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<td>Supra-aortic†</td>
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<td>M</td>
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<td>Supra-aortic†</td>
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<td>R MCA M1, M2</td>
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<td>Y</td>
<td>2</td>
<td>Definite</td>
<td>Definite</td>
<td>M1</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.
in 2 patients, and no enhancement in 6 patients. Figures 2 and 3 show representative images from patients treated with medical therapy alone.

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 mechanical thrombectomy is similar to the VW-MRI appearance of inflammatory conditions such as primary central nervous system vasculitis\(^5,7,8\) and drug-induced arteriopathy.\(^14\) Recognition of this similarity is important to avoid misinterpretation of the post-thrombectomy appearance of the arterial wall as evidence of underlying arteritis.

Histopathologic studies in swine and canine models have shown that mechanical thrombectomy injures the arterial wall. Gory et al\(^15\) 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 elastic lamina, and edema in the intimal and medial layers of the arterial wall. Nogueira et al\(^16\) 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 arterial wall thickening in these patients. An alternative explanation for arterial wall enhancement is leakage of gadolinium from vasa vasora; however, intracranial arteries normally lack vasa vasora.\(^18\) Histopathologic studies have also demonstrated intramural thrombus after stent retriever use,\(^15\) but we did not observe hyperintensity in the arterial wall on the nonenhanced T1-weighted vessel wall images to provide evidence of intramural blood product in this study.

One of the study subjects (subject 7) who underwent mechanical thrombectomy provided an opportunity to evaluate 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 M\(_2\) 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 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

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**Figure 2.** Effect of thromboembolism on the arterial wall (subject 12). A, Coronal image from a computed tomographic angiogram demonstrates a filling defect, consistent with occlusive thrombus, involving the right middle cerebral artery bifurcation and extending a short distance into the superior and inferior divisions (arrows). The patient was managed medically with IV tissue-type plasminogen activator. B, Time-of-flight magnetic resonance angiogram 4 days after symptom onset demonstrates recanalization of the previously occluded vessels. C and D, Contrast-enhanced T1-weighted vessel wall magnetic resonance images in the coronal plane show that the artery (arrows) has recanalized, with no evidence of arterial wall thickening or enhancement.

**Figure 3.** Effect of thromboembolism on the arterial wall (subject 8). A, Axial image from a computed tomographic angiogram demonstrates a filling defect, consistent with occlusive thrombus, in a left middle cerebral artery M1 branch (arrow). The patient was managed medically with IV tissue-type plasminogen activator. B, Computed tomographic angiogram 3 days after symptom onset demonstrates recanalization of the initially occluded vessel (arrow). C, Contrast-enhanced T1-weighted vessel wall magnetic resonance image in the axial plane shows that the artery (arrow) has recanalized, with no evidence of arterial wall thickening or enhancement. Inset box is a magnified view.
site of occlusion should at least raise the possibility of primary intracranial arteriopathy.

Acknowledgments
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Disclosures
None.

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