Intracranial arterial wall imaging using high-resolution 3-tesla contrast-enhanced MRI

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ABSTRACT

Background: Conventional arterial imaging focuses on the vessel lumen but lacks specificity because different pathologies produce similar luminal defects. Wall imaging can characterize extracranial arterial pathology, but imaging intracranial walls has been limited by resolution and signal constraints. Higher-field scanners may improve visualization of these smaller vessels.

Methods: Three-tesla contrast-enhanced MRI was used to study the intracranial arteries from a consecutive series of patients at a tertiary stroke center.

Results: Multiplanar T2-weighted fast spin echo and multiplanar T1 fluid-attenuated inversion recovery precontrast and postcontrast images were acquired in 37 patients with focal neurologic deficits. Clinical diagnoses included atherosclerotic disease (13), CNS inflammatory disease (3), dissections (3), aneurysms (3), moyamoya syndrome (2), cavernous angioma (1), extracranial source of stroke (5), and no definitive clinical diagnosis (7). Twelve of 13 with atherosclerotic disease had focal, eccentric vessel wall enhancement, 10 of whom had enhancement only in the vessel supplying the area of ischemic injury. Two of 3 with inflammatory diseases had diffuse, concentric vessel wall enhancement. Three of 3 with dissection showed bright signal on T1, and 2 had irregular wall enhancement with a flap and dual lumen.

Conclusions: Three-tesla contrast-enhanced MRI can be used to study the wall of intracranial blood vessels. T2 and precontrast and postcontrast T1 fluid-attenuated inversion recovery images at 3 tesla may be able to differentiate enhancement patterns of intracranial atherosclerotic plaques (eccentric), inflammation (concentric), and other wall pathologies. Prospective studies are required to determine the sensitivity and specificity of arterial wall imaging for distinguishing the range of pathologic conditions affecting cerebral vasculature. *Neurology*® 2009;72:627–634

GLOSSARY

CTA = computed tomographic angiography; **DSA** = digital subtraction angiography; **FIESTA** = fast imaging employing steady state acquisition; **FLAIR** = fluid-attenuated inversion recovery; **FRFSE** = fast recovery fast spin echo; **ICA** = internal carotid artery; **MCA** = middle cerebral artery; **MR** = magnetic resonance; **MRA** = magnetic resonance angiography; **TE** = echo time; **TI** = inversion time; **TR** = recovery time.

Many modalities exist for imaging the lumina of blood vessels, including conventional CT and magnetic resonance (MR) angiography. However, these approaches have a limited ability to differentiate vascular pathologies, because different pathologies can produce the same luminal defects. Direct imaging of the blood vessel wall offers the potential to discriminate between these pathologies. Characterization of atherosclerotic plaque using MRI is already well established.[1-3] High-resolution MR studies of the extracranial carotid arteries have identified features of atherosclerotic plaque that may convey increased risk of thromboembolic events[1] even in cases with less than 50% luminal stenosis.[2] Extracranial large artery plaques have been reported to cause asymmetric wall thickening with enhancement.[4] In contrast, extracranial vasculitis has been reported to show circumferential thickening and enhancement on MRI.[5]

Supplemental data at www.neurology.org

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Intracranial arterial wall imaging poses a greater challenge given the smaller size and relatively deep location of the target vessels. Three-tesla (T) MRI has been used to characterize carotid atherosclerosis[1,6] and dissection,[7] as well as extracranial vessel wall imaging to assess giant cell arteritis.[8,9] Although the need to look "beyond the lumen" has been increasingly recognized in assessing the extracranial vasculature, it has not yet been systemically applied to multiple intracranial vascular pathologies using high-resolution 3T MRI. To make this assessment, we applied high-resolution 3T MRI in a consecutive case series from a tertiary stroke center with the goal of identifying the unique imaging appearance of intracranial vessel pathologies.

METHODS

Hypotheses. Based on our understanding of the morphology and pathophysiology of the three major disorders affecting the wall blood of vessels, we developed the following imaging hypotheses:

- Atherosclerotic plaques show eccentric irregular wall thickening; gadolinium enhancement of the plaque correlates with plaque instability.
- Vasculitis produces smooth circumferential concentric wall thickening with diffuse gadolinium enhancement of the inflamed wall.
- Dissection shows eccentric wall thickening with T1 bright wall components representing intramural hematoma.

Subjects. This study was a retrospective analysis of consecutive patients presenting to neurology or neurosurgery at a tertiary care center (Toronto Western Hospital) with focal neurologic symptoms for which the treating clinicians requested additional information about the intracranial vasculature, beyond conventional angiographic techniques (computed tomographic angiography [CTA], magnetic resonance angiography [MRA], or digital subtraction angiography [DSA]). All patients had to have no contraindications to MRI and be medically stable to be in the MR scanner for 30 to 90 minutes without general sedation. The sequences were applied, when requested, beginning in August 2005, and scans were collected until the beginning of the analysis period in September 2007. Where patients presented under acute stroke protocols, plain CT brain scans had been performed. All participants also had routine MRI pulse sequences (fluid-attenuated inversion recovery [FLAIR], T2-weighted, and diffusion-weighted images) and at least one method of vessel lumen assessment (MRA, CTA, or DSA) before the wall imaging protocol. When possible (in 34 of 37 people), wall imaging was performed during the acute hospitalization, within days to weeks of the presenting symptoms. The three patients who did not have acute wall imaging returned for follow-up at 2 months (table e-1 on the *Neurology*® Web site at www.neurology.org, case 11), 3 months (case 32), and 5 months (case 37) after symptom onset.

Institutional ethics approval. All MR images were acquired using vendor-supplied sequences approved for clinical use. To correlate the MR findings with patient demographics and clinical diagnoses, a retrospective chart review and retrospective detailed imaging review was undertaken. Hospital research ethics board approval was obtained for a systematic, retrospective chart and imaging review of all patients who had the 3T wall imaging protocol performed (Toronto Western Hospital Research Ethics Board).

Imaging protocol and analysis. Patients were scanned on a 3T MRI system (HDX platform, GE Healthcare, Milwaukee) using an eight-channel head coil. All sequences applied were standard, approved, vendor-supplied pulse sequences. No new experimental sequences were pioneered in this study. The "protocol" described in this study refers to the implementation of a standardized set of sequences and the disciplined application of those sequences (e.g., with specific axial, coronal, and sagittal sampling of the site of intracranial stenosis). The number of pulse sequences used for 3T wall imaging was reduced over the 2 years that the protocol was performed. Initially, multiple sequences were applied: T1 spin echo (repetition time [TR]/echo time [TE] 450/21, slice thickness 3 mm, 384 × 384 matrix), T1 FLAIR (TR/TE/inversion time [TI] 2108/12/860, slice thickness 3 mm, 384 × 384 matrix), three-dimensional fast imaging employing steady state acquisition (FIESTA) (TR/TE 6/2.5, slice thickness 0.8 mm, 256 × 256 matrix), and T2 fast recovery fast spin echo (FRFSE) (TR/TE 3450/92, echo train length 17, slice thickness 3 mm, 512 × 512 matrix). All T1 FLAIR and T2 FRFSE sequences used a parallel factor of 2. Field of view was 16 to 22 cm. The ability to distinguish the vessel wall from the lumen, as well as the presence of flow artifacts, was assessed for each of the sequences. It was observed that T1 FLAIR improved visualization of blood vessel walls via its black blood characteristics including the absence of blood signal and decreased sensitivity to flow artifacts. The T1 spin echo sequences, which had these effects, were replaced with multiplanar T1 FLAIR, either with or without fat saturation, but always with and without gadolinium. The MRI technologist, in consultation with the neuroradiologist, selected the best combination of acquisition orientations and target vessels. Three-dimensional FIESTA was found to be more susceptible to artifacts than T2, without providing additional information. These were thus eliminated from the protocol.

The final common "wall protocol" consisted of a minimum of seven sequences on the 3T MRI: 1) axial T2-weighted images; precontrast 2) axial, 3) sagittal, and 4) coronal T1 FLAIR images; and postcontrast 5) axial, 6) sagittal, and 7) coronal T1 FLAIR images. All sequences had to be monitored for quality to ensure that the orientation was successful to capture the affected artery at the site of stenosis on at least one slice both parallel and perpendicular to a site of abnormality. Where no clear focal abnormality was noted on baseline angiography (MRA, CTA, or DSA), the acquisitions were targeted to ensure sampling of the clinically suspected vessel (e.g., the left middle cerebral artery [MCA] in a patient presenting with an aphasic TIA).

Imaging analysis was performed on a radiology information system–picture archiving and communication system and, if necessary, multiplanar reformatting was performed. At least one method of conventional luminal imaging (MRA, CTA, or DSA) was available for all patients.

Retrospective visual analysis of all imaging information from each case was performed to describe any cerebral pathology, to identify intracranial and extracranial sites of stenosis, to evaluate focal wall thickening, and to assess postcontrast enhancement, as well as to categorize any enhancement as either concentric or eccentric. All imaging analysis was performed by a neuroradiologist (D.J.M.) blinded to the final clinical diagnosis.

Table Patient demographics by final clinical diagnoses

Diagnosis	No. of patients	Sex, F:M	Average age, (min–max), y
Intracranial atherosclerotic stroke	13	3:10	64 (44–80)
Stroke with extracranial source	5	2:3	60 (32–84)
Vasculitic/Inflammatory diseases	3	3:0	43 (18–66)
Dissection	3	1:2	56 (49–60)
Other vascular lesions (3 aneurysms, 1 cavernoma)	4	4:0	48 (23–65)
Moyamoya syndrome	2	0:2	38 (30–46)
No final clinical diagnosis	7	2:5	50 (30–79)
Total	37	15:22	55 (18–84)

Wall thickening and enhancement patterns were categorized by visual inspection. Enhancement was considered concentric if it was circumferential and uniform (defined specifically as width of the thinnest wall segment at least 50% or more of the thickest segment). In contrast, eccentric enhancement was defined as either clearly limited to one side of the vessel wall (e.g., not 360-degree circumferential enhancement) or, where circumferential enhancement was noted, the thinnest part of the wall enhancement was estimated to be less than 50% of the thickest point (figure e-4). More precise measurements are not feasible based on the limits of resolution of the imaging.

Case histories. Charts were reviewed for demographic data, medical history (including cerebrovascular risk factors), presenting symptoms and physical findings (blood pressure, height, weight, neurologic deficits), laboratory results, and imaging findings.

Diagnostic categorization. Subjects were classified as "atherosclerotic" if they had two or more vascular risk factors: men older than 50 years, women older than 60 years, hypertension, hypercholesterolemia, diabetes, overweight or obesity, smoking, or calcified intracranial arterial plaques on plain CT head. Subjects were classified as "inflammatory" if they had one or fewer vascular risk factors with blood tests or prior diagnoses consistent with inflammatory diseases (lupus, erythrocyte sedimentation rate or C-reactive protein elevations, antinuclear antigen, or anti–phospholipid antibody positivity). Subjects whose workup discovered a carotid or cardiac source for their presenting symptoms were classified as "extracranial source." Those subjects who had structural pathology diagnosed on conventional angiographic techniques (MRA, CTA, or DSA) were categorized as either "dissection," "structural (aneurysm/cavernoma)," or "moyamoya." Subjects were classified as "no final clinical diagnosis" if they met none, or more than one, of the above criteria.

RESULTS Thirty-seven patients underwent the vessel wall protocol. Demographics by final clinical diagnoses are shown in table. Individual case data are available in table e-1.

Wall imaging patterns. ***Stroke with intracranial atherosclerosis.*** All 13 of the patients with stroke/TIA and intracranial atherosclerotic disease showed focal luminal narrowing of intracranial vessels on MRA. Twelve had diffusion-weighted abnormalities suggestive of recent cerebral infarction, whereas 1 patient with transient focal symptoms had no changes on diffusion-weighted imaging. The narrowed areas on conventional luminal studies corresponded to focal areas of thickened wall with narrowed lumen seen on T1 FLAIR (precontrast) and T2-weighted images (figure 1). Of these 13 patients, 12 had focal areas of eccentric wall enhancement in the intracranial vessel supplying the territory of acute infarct (figures 1, 2, and e-1). Enhancing plaques were visualized in the relevant major branches of the circle of Willis, including the MCA (figure 1), anterior cerebral artery (figure e-1), and basilar artery (figure 2). Most patients with enhancement (10 of 12) had enhancement only in the vessel supplying the area of acute infarction, even with multiple areas of wall thickening and luminal stenosis indicative of plaques elsewhere (figure e-2). Two patients had multiple vessels with focal eccentric enhancement, not all of which had evidence of ischemic injury in the territory they supplied (figure e-3). Atherosclerotic enhancement was typically irregular and eccentric (figures 1 and 2). Circumferential, but still eccentric, enhancement was occasionally seen (figure e-4). One patient had a thickened wall with luminal stenosis but no enhancement; that patient had wall imaging only at follow-up, 5 months postinfarct (figure e-5).

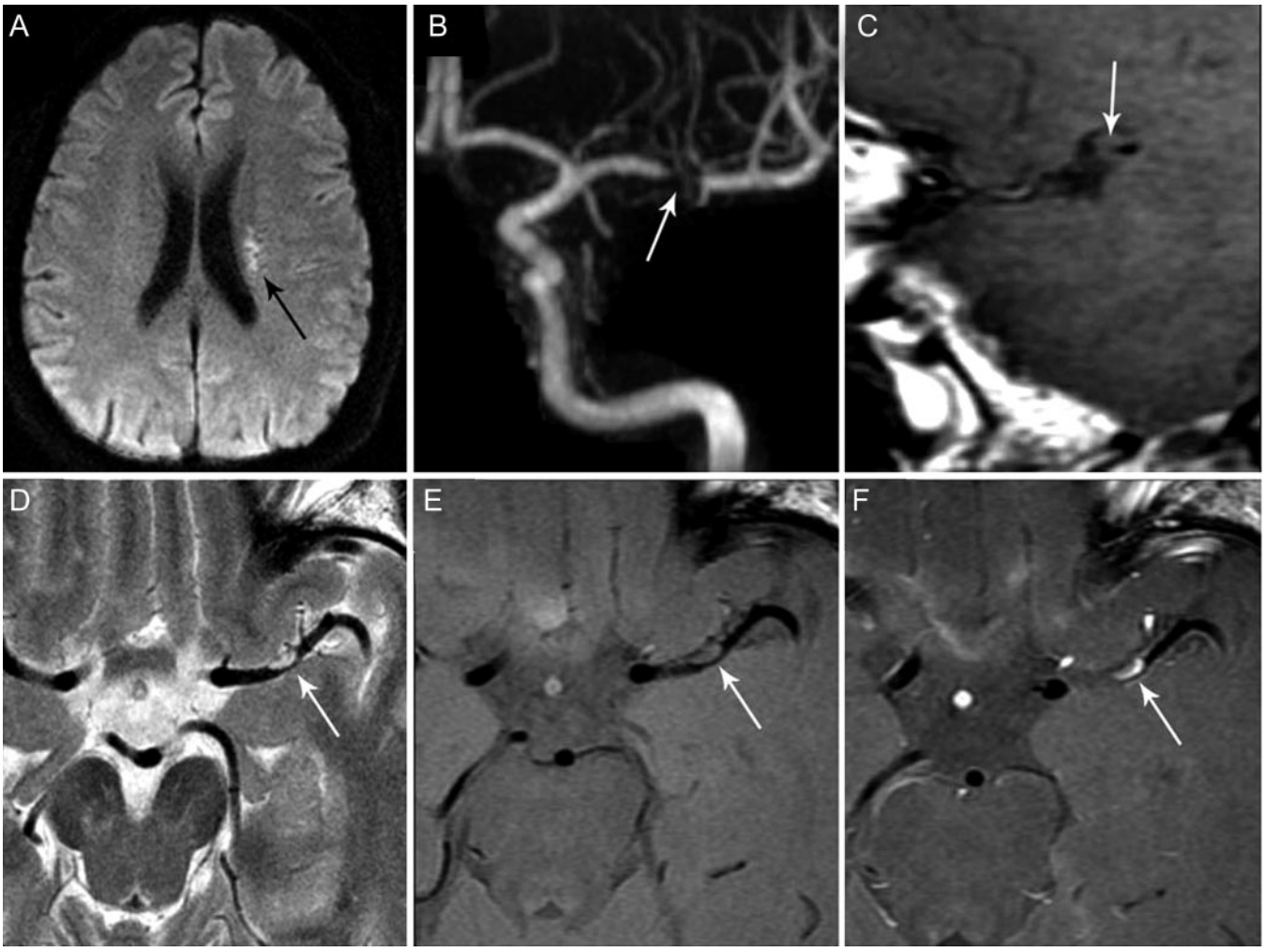
Extracranial source of stroke. In the five patients with an extracranial source of stroke, none showed intracranial wall enhancement.

Inflammatory disease. In contrast to the pattern seen in patients with atherosclerotic risk factors, one patient with biopsy-proven giant cell arteritis (figure 3) showed a smooth, diffuse, concentric pattern of enhancement. The same pattern of smooth, concentric enhancement was seen in another patient with systemic vasculitis, as well as one patient with drug-induced vasculopathy. These patterns are difficult to discern on conventional 1.5T sequences. One patient with systemic lupus and amaurosis fugax showed no intracranial wall enhancement of the circle of Willis vessels. Notably, the ophthalmic artery was not well visualized; vessels of this caliber are likely at the limit of the resolving power of the 3T system.

Intracranial dissection. Intracranial dissection had a similar pattern to atherosclerosis, including eccentric wall thickening with enhancement. Distinguishing features included T1 bright wall elements on nonenhanced T1 FLAIR sequences (indicating methemoglobin in the arterial wall), as well as visualization of a false lumen (figure 4).

Other vascular lesions. In three patients with intracranial aneurysms, a thin wall with smooth, circumferential, concentric enhancement was noted (figure e-6). The prognostic significance of aneurysmal wall

Figure 1 Focal, eccentric enhancement of the left middle cerebral artery wall



A 57-year-old man with untreated hypertension presented with recurrent right hemiplegia and dysarthria (table e-1, case 4). Axial diffusion-weighted image (A) shows an acute infarct (arrow) in the left corona radiata. Time-of-flight magnetic resonance angiogram maximum-intensity projection image (B) demonstrates focal stenosis (arrow) in the left middle cerebral artery (MCA). Sagittal T1 fluid-attenuated inversion recovery (FLAIR) image (C) shows eccentric thickening (arrow) of the anterosuperior wall of the left MCA. Axial T2 fast recovery fast spin echo image (D) shows focal eccentric wall thickening (arrow) of the left M1 segment of the MCA. Axial T1 FLAIR images before (E) and after (F) gadolinium show an eccentrically enhancing plaque (arrow) on the anterior wall of the left MCA. This is likely representing an inflamed active plaque ("hot plaque").

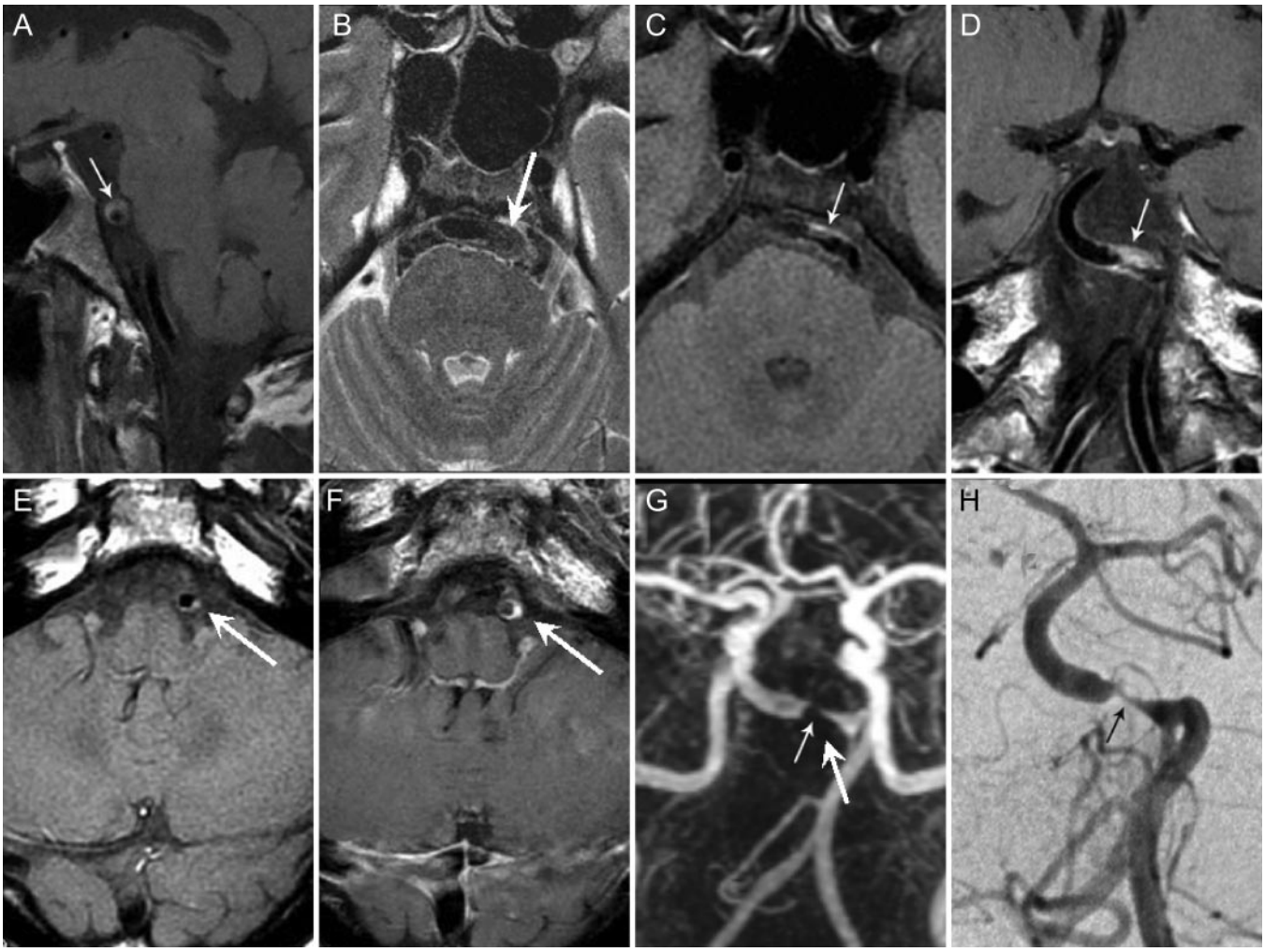
enhancement patterns is uncertain. One patient who was initially thought to have an aneurysm underwent wall imaging and was found to have a nonenhancing cavernous angioma.

Moyamoya syndrome. Two patients with moyamoya syndrome had severely narrowed or occluded MCA branches and extensive collateralization. One presented with hemorrhage, and the other presented with hemodynamic symptoms. Neither patient showed wall thickening or enhancement of the circle of Willis vessels (including the distal internal carotid artery).

No final clinical diagnosis. In the seven people with no final clinical diagnosis, three had acute infarcts on diffusion-weighted imaging. All seven had complex case histories with either risk factors for multiple types of disease or insufficient risk factors for any specific disease (table e-1). Two showed eccentric wall enhancement, two showed concentric enhancement, one had a mixed pattern, and two had thickened walls without enhancement; both of these had been imaged months after acute infarctions.

DISCUSSION The assessment of the wall of intracranial vessels is limited by several factors. Conventional imaging sequences (T1, T2, FLAIR), even at 3 T, do not render these vessels with sufficient detail to fully assess the vessel wall. Sequence optimization using higher spatial resolution is required. Increasing spatial resolution by decreasing slice thickness and decreasing in-plane voxel size is limited by the signal-to-noise ratio, which is already deficient for small vessels using a 1.5T scanner. One prior study used MRI to examine the wall in atherosclerosis[10]; how-

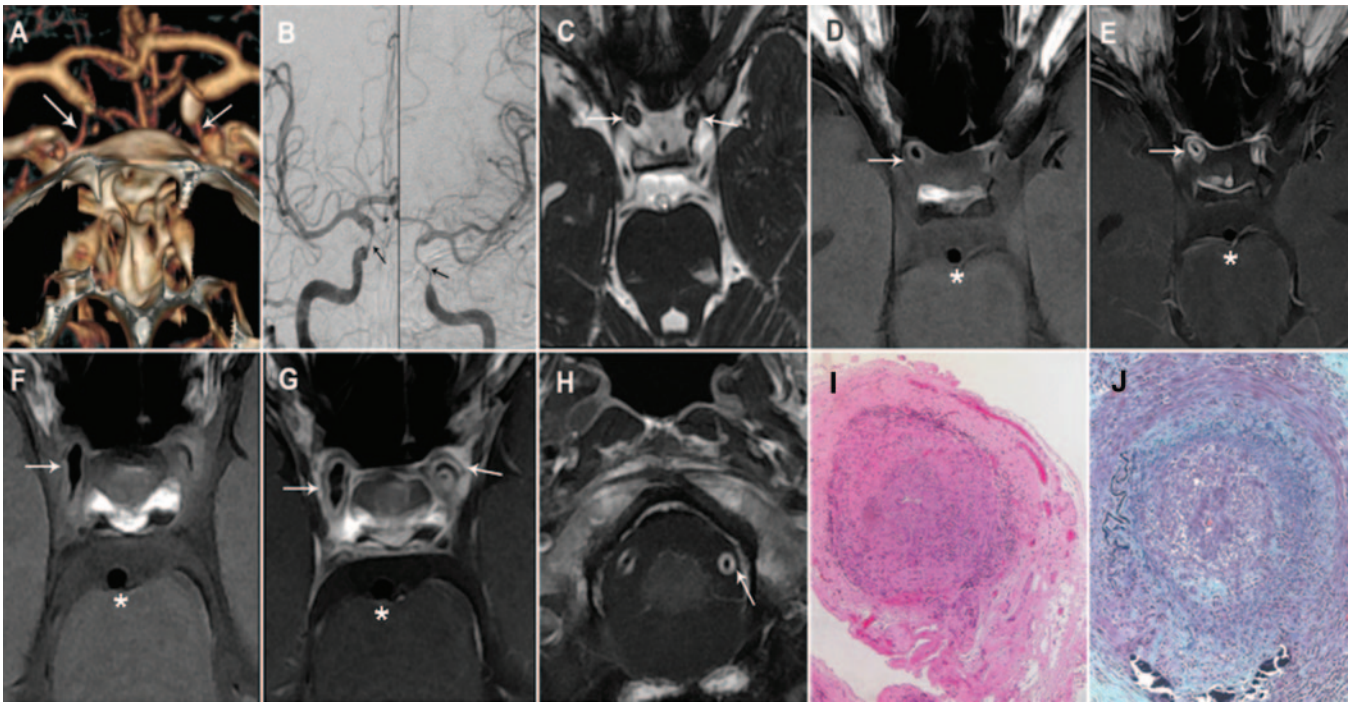
Figure 2 **Focal eccentric enhancement of the basilar artery wall**



A 61-year-old man presented with recurrent vertebrobasilar TIAs and small strokes in the posterior circulation on diffusion imaging (not shown) (table e-1, case 5). Sagittal T1 fluid-attenuated inversion recovery (FLAIR) image (A) shows eccentric thickening (arrow) and high signal in the wall of a short segment of the tortuous basilar artery. Axial T2-weighted image (B) shows thickening of the wall (arrow) with iso to low signal. On a pregadolinium T1 axial image with fat saturation (C), minimal high signal intensity is noted in the arterial plaque (arrow). A coronal T1 FLAIR postgadolinium image (D) shows a larger area of eccentric enhancement (arrow) in the mid basilar artery. Axial T1 FLAIR precontrast (E) shows eccentric wall thickening of the basilar artery. The eccentric enhancement of the basilar artery wall is best appreciated on the corresponding axial T1 FLAIR postcontrast image (F). Gadolinium dynamic contrast enhanced magnetic resonance angiogram maximum-intensity projection image (G) and digital subtraction angiogram (H) demonstrate a corresponding area of severe stenosis (arrow) in the mid basilar artery (compare G and H with D).

ever, this study used 1.5T MRI, which cannot achieve sufficient spatial resolution to assess the intracranial wall. The improved signal-to-noise provided by 3T MRI is therefore used to achieve higher spatial resolution. However, examinations still require targeting the vessel of interest, because the intracranial vessels are smaller, are more variable in their distribution than the extracranial carotid arteries, and require longer acquisition times. This can be time-consuming for the radiologist, technologist, and patient. Finally, it is difficult to validate radiologic findings of intracranial arteries because, unlike the case of imaging the carotid bifurcation where endarterectomy is often performed, pathology is rarely obtained from the intracranial vessels. In the current study, we used the demonstrated advantages of 3T MRI[11,12] to overcome these limitations and demonstrate pathology within the wall of the smaller caliber intracranial vessels. The use of T1 FLAIR sequences, acquired in multiple slice dimensions precontrast and postcontrast, as well as high-resolution T2 images, has provided adequate signal to resolve the smaller vessels. It was chosen for its superior block blood characteristics, where flowing blood returns no signal. The eccentric wall enhancement observed in intracranial atherosclerosis is consistent with findings using 3T MRI of atherosclerotic plaques in carotid arteries,[6] femoral arteries,[3] and the abdominal aorta,[3] all of which have been validated with histologic correlations. Although most of our atherosclerotic plaque cases show homogeneous enhancement within the plaque, case 4 may be an ex-

Figure 3 Biopsy-confirmed giant cell arteritis with smooth concentric middle cerebral artery enhancement



A 67-year-old woman presented with multiple TIAs followed by headache and multiple focal neurologic deficits, progressing to decreased level of consciousness (table e-1, case 9). Diffusion images (not shown) revealed dozens of small cortical and subcortical areas of restriction. Reformatted CT angiogram (A) shows bilateral narrowing (arrows) of the cavernous and supraclinoid internal carotid arteries (ICAs). Catheter digital subtraction angiogram images (B, combined right and left injections) confirm these findings (black arrows). Axial fast imaging employing steady state acquisition images (C) at the level of the cavernous sinuses show wall thickening (arrow) of bilateral cavernous ICAs. Axial T1 fluid-attenuated inversion recovery (FLAIR) (D and F) demonstrates mural thickening (arrow) of both cavernous ICAs with luminal narrowing. After gadolinium administration, corresponding axial T1 FLAIR images (E and G) show smooth, concentric wall enhancement (arrows) of the cavernous ICAs. Similar enhancement (arrow) is also noted in both vertebral arteries (H). Note the normal-appearing, nonenhancing basilar artery (starred, D–G). Temporal artery biopsy (I and J) shows destruction of the internal elastic lamina with inflammatory cells (predominately lymphocytic with occasional multinucleated giant cells) in the media and adventitia. There were also luminal occlusion and fibrin platelet plugs in the vasa vasorum confirming the diagnosis of giant cell arteritis.

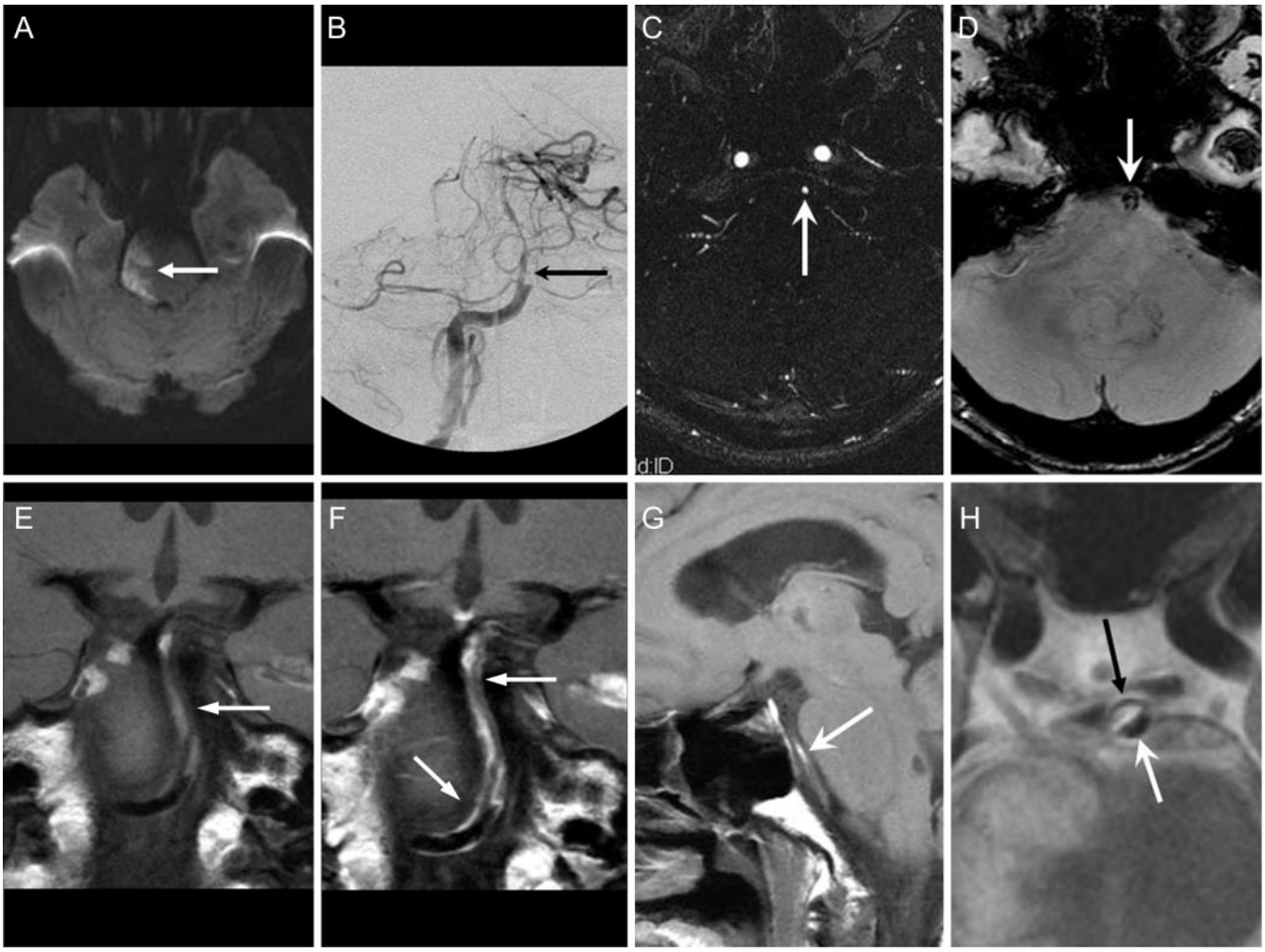
ample of an atherosclerotic plaque with fibrous cap enhancement superficial to a lipid core.

In studies of extracranial vessel imaging with pathologic confirmation, inflammatory conditions are associated with concentric, circumferential wall thickening and enhancement,[5] whereas atherosclerotic disease is frequently eccentric.[4] In our study, we found similar patterns in the intracranial vessels of patients with atherosclerotic and inflammatory disease (biopsy confirmed in one case) confirming our hypotheses. The enhancement patterns were consistent and largely confined to the vessels supplying the area of acute infarction. In some cases, remote vessels also enhanced, raising concern of active or "hot" plaque that might be at risk of future thromboembolic events. It remains to be determined whether enhancement within intracranial atherosclerotic plaques can be used to select patients at high risk of recurrent ischemic stroke.

None of the three individuals who had wall imaging months after their acute events showed wall enhancement, and none of the patients with extracranial sources for their strokes showed intracranial wall enhancement. In addition, all 12 patients with atherosclerotic disease imaged acutely showed enhancement, and some had enhancement in vessels that had not yet shown signs of infarction. This suggests that enhancement may reflect active plaques or "plaques at risk." Studies with carotid wall imaging have shown similar correlations between plaque rupture and symptomatic presentations.[13] The concept of using imaging to identify vulnerable plaques is well established.[14] Inflammation in chronic atherosclerotic plaques plays a role in destabilizing the plaque.[15] Denuded vascular endothelium with inflammatory components or neovascularization is most likely to enhance and also likely serve as a focus for clot formation and embolization.[14] It is therefore not surprising that similar enhancement patterns can be seen in intracranial vessels using higher-resolution scanners and optimized acquisitions. It remains to be seen whether current treatments (e.g., statin loading, anti-inflammatory therapies) can alter the enhancement pattern and affect clinical outcome.

Our dissection cases also confirmed the hypothesis that eccentric bright T1 elements (higher signal

Figure 4 Basilar artery dissection



A 60-year-old man with neck pain after a rugby injury presented with ataxia and left-sided weakness (table e-1, case 13). Diffusion-weighted imaging (A) showed restricted diffusion in the pons, and conventional angiography was interpreted as a dissection of the basilar artery (B). Contrast-enhanced magnetic resonance angiogram (C, axial slice) also shows a severely narrowed lumen of the basilar artery at the level of the midpons (level shown by arrow on slice G). At the same level, blooming artifact can be seen on the gradient echo T2* images (D, arrow). T1 fluid-attenuated inversion recovery (FLAIR) image without contrast (E) shows bright signal consistent with hematoma in the wall (arrow), with narrowed lumen. Postcontrast T1 FLAIR (F) shows additional enhancement and thickening of the wall throughout the length of the basilar artery (arrows) and into the vertebral arteries bilaterally (slices not shown). Sagittal T1 FLAIR precontrast images also show clot extending the length of the basilar artery (G). High-resolution T2-weighted images show a widened basilar artery with two lumens (H, black and white arrows) and an intervening flap. Full characterization of intracranial wall pathology may be able to reduce the need for diagnostic angiography for the diagnosis of intracranial dissections, as has been the case for extracranial dissections.

intensity than brain) in the vessel wall indicate methemoglobin. It remains to be seen whether T1 bright wall elements alone are specific for dissection or whether they may also be seen in vulnerable atherosclerotic plaque with hemorrhage.

Using 3T MRI to image intracranial vessel walls addresses several technological concerns, but it also has its own limitations. First, the acquisitions are time-consuming and require cooperative patients with minimal movement. Second, to identify vessels of interest (and aid in the diagnostic workup of patients), baseline imaging (e.g., acute stroke protocols, CTA, MRA, or DSA) had already been performed. Wall imaging thus requires additional imaging sessions, which add expense and imaging time. Third, proper vessel targeting requires interaction between an experienced and motivated MR technologist and a neuroradiologist to monitor the sequence acquisitions and ensure adequate coverage of the vessels. Given limited 3T MRI availability, these labor-intensive studies are not routinely available at most centers. Fourth, this study was entirely retrospective and reviewed consecutive patients. Patients were selected to receive these additional imaging sequences based entirely on clinical requests by physicians for more information about the intracranial process. The population is thus biased toward the more complicated patients in whom clinicians or radiologists wanted further clarification about their disease. This selection bias means that our cohort may not be rep-

resentative of the full population of patients presenting to a neurologist or neurosurgeon with focal neurologic symptoms. The results presented here are hypothesis generating and must be interpreted with caution. Large, longitudinal, prospective studies of a broader cohort of patients using wall imaging are required to determine the sensitivity, specificity, and predictive values of this technique.

AUTHOR CONTRIBUTIONS

Data acquisition, statistical analysis, and manuscript writing were performed by R.H.S.

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