Radiology

REVIEWS AND COMMENTARY • REVIEW

Essentials for Interpreting Intracranial Vessel Wall MRI Results: State of the Art

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Intracranial vessel wall (VW) MRI has become widely available in clinical practice, providing multiple uses for evaluation of neurovascular diseases. The Vessel Wall Imaging Study Group of the American Society of Neuroradiology has recently reported expert consensus recommendations for the clinical implementation of this technique. However, the complexity of the neurovascular system and caveats to the technique may challenge its application in clinical practice. The purpose of this article is to review concepts essential for accurate interpretation of intracranial VW MRI results. This knowledge is intended to improve diagnostic confidence and performance in the interpretation of VW MRI scans.

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Learning Objectives:

After reading the article and taking the test, the reader will be able to:

- Develop a vessel wall (VW) MRI protocol that can identify intracranial vasculopathies and distinguish pathologic wall features from artifacts
- Identify reasons for flow artifacts and describe techniques to confirm lumen patency
- Specify reasons for VW enhancement and its mimics and define techniques for distinguishing them

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Vessel wall (VW) MRI has emerged as an effective method with which to characterize pathologic features involving the VW and detect lesions that cause low-grade stenosis and may elude angiographic detection (1,2). The clinical importance of characterizing components of atherosclerotic plaque and looking beyond the lumen for low-grade stenosis using VW MRI has previously been shown for carotid atherosclerotic disease (3–5). Recently, its application has been extended to evaluate intracranial vascular disease by characterizing intracranial lesions (eg, atherosclerotic plaque [6–9], vasculitis [10], Moyamoya disease [11]), detecting angiographically occult atherosclerotic plaques (2,12), and guiding biopsies of inflamed vessels (1,13). The ability to achieve these applications has been largely driven by the optimization of three-dimensional high-spatial-resolution VW MRI imaging (1,7).

Although three-dimensional (3D) VW MRI has enabled a significant advancement in the diagnosis and care of patients with cerebrovascular disease, the interpretation of VW MRI scans is not a simple task, given the small size of these lesions, the complexity of imaging features, and the caveats of this technique. Furthermore, administration of gadolinium-containing contrast agents has proven to be essential in the detection and characterization of inflammatory vasculopathies on VW MRI scans, but reasons for enhancement on VW MRI scans extend beyond inflammation (9,13). These challenges may complicate the use of VW MRI in clinical practice (1,14).

There are several technical considerations that should be addressed for accurate implementation and interpretation of VW MRI scans of the intracranial circulation, including blood flow suppression, spatial resolution, cerebrospinal fluid (CSF) signal suppression, and acquisition time. Understanding these concepts and the reasons for gadolinium enhancement on VW MRI scans is essential for accurate interpretation of VW MRI findings. We describe these considerations and how they can account for common reasons for misinterpretation of 3D high-spatial-resolution VW MRI examinations in the evaluation of intracranial vasculopathies. We elaborate on the causes of these misinterpretations and offer solutions. Our specific recommendations for avoiding interpretative errors are summarized in Table 1.

Abbreviations

CSF = cerebrospinal fluid, DIR = double inversion recovery, ICA = internal carotid artery, MRA = MR angiography, 3D = three-dimensional, 2D = two-dimensional, TOF = time of flight, VW = vessel wall

Summary

Awareness of common reasons for errors in the interpretation of intracranial vessel wall MRI examinations, including the challenges of differentiating between causes of abnormal wall thickening and enhancement and distinguishing these changes from artifacts that frequently arise in clinical applications, is essential for the diagnostic confidence and performance of radiologists interpreting these examinations.

Essentials

- Accurate interpretation of intracranial vessel wall (VW) MRI findings in clinical practice requires understanding the conditions of adequate flow suppression, optimal spatial resolution, and reasons for enhancement after administration of a gadolinium-containing contrast agent.
- Slow or disturbed flow patterns can create artifacts mimicking plaque, and contrast-enhanced MR angiography should be used, when available, to confirm lumen patency.
- Acquisition of VW MRI scans in multiple orientations could avoid misinterpretation due to inadequate blood flow suppression since flow suppression depends on the orientation of the vessel relative to the frequency-encoding direction of VW MRI.
- Enhancement does not always reflect an underlying vasculopathy, and false-positive diagnoses can occur with leptomeningeal enhancement, luminal thrombus, microhemorrhages with surrounding enhancement, parenchymal enhancement from subacute ischemic stroke, or a vascular plexus at the skull base.
- Even at the maximum achievable spatial resolution for 3-T intracranial VW MRI, radiologists should be aware that artifactual concentric or eccentric wall thickening and incomplete discrimination of vessels from adjacent structures are often encountered and can mimic atherosclerotic plaque.

VW MRI Imaging Technique

Evolution of VW Imaging Techniques from Carotid to Intracranial Vessels

Black-blood MRI was established as a reliable and accurate method with which to characterize carotid artery atherosclerotic plaque size and composition by studies driven by access to endarterectomy specimens for histologic correlation (5,15,16). The delineation of the lipid-rich necrotic core and overlying fibrous cap and the identification of intraplaque hemorrhage and calcification can be achieved by multicontrast MRI, which distinguishes features based on T1, T2, and proton density–weighted images and time-of-flight (TOF) MR angiography (MRA) (17,18). Gadolinium-based contrast enhancement improves tissue characterization (3) and can be used to identify markers of VW inflammation, such as neovascularization (19,20). Extending these techniques to evaluate intracranial vessels is not straightforward, given the small size and tortuous course of these vessels. Carotid artery VW MRI was largely based on standard two-dimensional (2D) black-blood sequences, which are prone to partial volume artifacts for small structures, such as intracranial vessels, amplified by nonorthogonal section orientations that are a consequence to the inherent curving course of these vessels. Adding to the challenge of 2D imaging of intracranial vessels is the wide-ranging number of potential sites for lesions to occur and the limited examination time for covering these sites.

The 3D acquisitions enable high isotropic resolution that can minimize the overestimation of wall thickness as a consequence of the tortuosity and small size of intracranial vessels; however, 3D techniques suffer from long imaging times and suboptimal flow suppression (21). For example, double inversion recovery (DIR) techniques (16,22) typically employed in 2D acquisitions generally are inadequate for suppressing flow in 3D acquisitions because of the relatively thick reinversion pulse required. The more recent implementation of a variable flip angle refocusing pulse to turbo spin echo enables a longer echo train length that improves flow suppression without compromising signal and at relatively short imaging times (23). In fact, this variable flip angle turbo spin-echo technique has been shown to have higher signal-to-noise ratio efficiency and stronger black-blood effects compared with conventional 3D turbo spin-echo sequences (23,24). Experience with extranial carotid atherosclerotic plaque characterization by 2D DIR MRI has offered great insight into interpretations of 3D VW MRI scans of intracranial vessels given the relative scarcity of pathologic specimens to correlate with intracranial imaging findings.

Blood Flow Suppression

Blood flow suppression is generally achieved by inflow blood saturation, blood signal nulling by an inversion preparation pulse, or dephasing of the moving spins (eg, motion-sensitized driven equilibrium) (22,25,26). Among these techniques, DIR (16,22) has been most commonly used for 2D black-blood MRI. The first 180° pulse is not section selective and inverts all spins within the imaging volume, with the inversion time set to null the signal of inflowing blood. The second 180° pulse is section selective, thereby effectively canceling the inversion of spins in that section back to the z direction, mitigating the T1 weighting of the VW and thereby improving its signal-to-noise ratio.

The primary mechanism for blood flow suppression on 3D VW MRI scans is intravoxel dephasing of moving blood proton spins. Blood with laminar flow and acceleration traversing the magnetic field can lead to widespread phase dispersion with consequent signal attenuation. Low-flip-angle refocusing pulses promote simulated echoes, which store magnetization along the longitudinal axis and exhibit a complicated phase evolution between the longitudinal and transverse planes that results in signal loss (23,27).

Flow suppression is a function of blood velocity and is more effective when the velocity is more than 5 cm/sec (27). Blood velocity often has a laminar distribution, with the slowest flow adjacent to the VW where inadequate flow suppression may occur when flow is reduced sufficiently. Disordered velocity distributions, such as disturbed or recirculating flow, can potentiate incomplete signal suppression at the periphery of the lumen, which can mimic eccentric VW thickening (ie, plaque) (Fig 1) or concentric VW thickening (ie, circumferential plaque or vasculitis) (Fig 2). Common sites for flow artifacts on VW MRI scans include the genu of the petrous segment of the internal carotid artery (ICA) and juxtasellar cavernous segment of the ICA, as

Table 1: Recommendations for Avoiding Errors in Interpretations

Artifact and Misinterpretation	Solution	Corresponding Figure No.
Incomplete blood flow suppression mimicking atherosclerotic plaque	CE MR angiography to confirm lumen patency	1
Incomplete blood flow suppression mimicking circumferential wall thickening	CE MR angiography to confirm lumen patency	2
Incomplete blood flow suppression due to sequence orientation	Acquire 3D VW MRI scans in multiple orientations to increase the number of vessel segments parallel to the frequency-encoding direction	3
Vessel crossing through leptomeningeal enhancement mimicking vasculitis	Evaluate reconstructions of 3D VW MRI scans in multiple obliqe images	6
Thrombus with recanalization mimicking vasculitis	May be unavoidable, highlights importance of biopsy confirmation	7
Microhemorrhage with surrounding enhancement mimicking perivascular enhancement with vasculitis	Distinguish single focus versus linear distribution of signal loss central to enhancement to differentiate microhemorrhage from vascular inflammation, respectively*	8
Parenchymal enhancement from subacute ischemic stroke mimicking vasculitis	Review diffusion-weighted images (prior to and at the time of examination, when available) to help confirm enhancement from subacute ischemia	9
Venous plexus enhancement of the vertebral artery mimicking vasculitis or atherosclerotic plaque	Reconstruction of VW MRI scans to depict vessels in the long axis to help confirm enhancement from vascular plexus at the skull base; consider 2D DIR short-axis section for higher-spatial-resolution evaluation	10
Focal benign nodular lesion at the foramen magnum mimicking atherosclerotic plaque	Reconstruction of VW MRI scans to depict vessels in the long axis to help confirm enhancement from the vascular plexus at the skull base; look for continuous connection between lesion and intradural vein or venous plexus at skull base	11
Venous plexus enhancement surrounding the petrous ICA mimicking vasculitis	Look for ipsilateral carotid stenosis	11
Venous enhancement mimicking vasculitis	CE MR angiography (or 3D postcontrast T1-weighted brain imaging) reconstructed to confirm lumen patency	12
Inadequate spatial resolution and venous enhancement mimicking atherosclerotic plaque	Acquire 2D DIR images through short axis of vessel	5

Note.— CE = contrast enhanced, DIR = double inversion recovery, ICA = internal carotid artery, 3D = three-dimensional, 2D = two-dimensional, VW = vessel wall.

* Perivascular enhancement in cerebral amyloid angiopathy may be noninflammatory (13).

these segments are curved and have relatively large diameters, making them prone to recirculating flow. Other factors predisposing an individual to flow artifacts include slow flow within an aneurysm or dilated artery or slow flow in the setting of proximal or distal stenosis or occlusion. Slow flow also can be a consequence of edema, such as in the setting of cerebral infarction (28). Awareness of the potential for inadequate flow suppression in this setting is critical to avoid the temptation to misdiagnose this as a vasculopathy ascribed to the stroke.

Flow suppression is more pronounced in vessels with small diameters (ie, flow artifacts are more common in larger vessels). This can be explained by the voxel sensitivity function, which defines the relation between tissue structure and signal intensity imaged in a voxel (27,29). In most MRI applications, tissue structure is much larger than voxel size, and MRI signal intensity is not affected by its position relative to the voxel. However, for a small structure with subvoxel dimensions, MRI signal intensity depends on its position within the voxel. A vessel with a large diameter relative to the voxel dimensions will have blood protons located in the center of a voxel that produce a strong signal, based on the voxel sensitivity function, which is less efficiently suppressed compared with protons in a small vessel with subvoxel dimensions positioned near the edge or corner of a voxel (29). Considering that the small size of intracranial vessels may approximate subvoxel dimensions, blood signal is often adequately suppressed.

Although confirming lumen patency on TOF MRA scans can help avoid misinterpreting a flow artifact as plaque, it is important to recognize that the same hemodynamic mechanisms (eg, slow or disturbed flow) produce artifacts with both techniques (ie, TOF MRA and VW MRI scans). For this reason, gadolinium-enhanced MRA can help confirm lumen patency (Figs 1, 2).

Blood flow suppression depends on the orientation of the vessel relative to the orientation of the 3D VW MRI acquisition, and

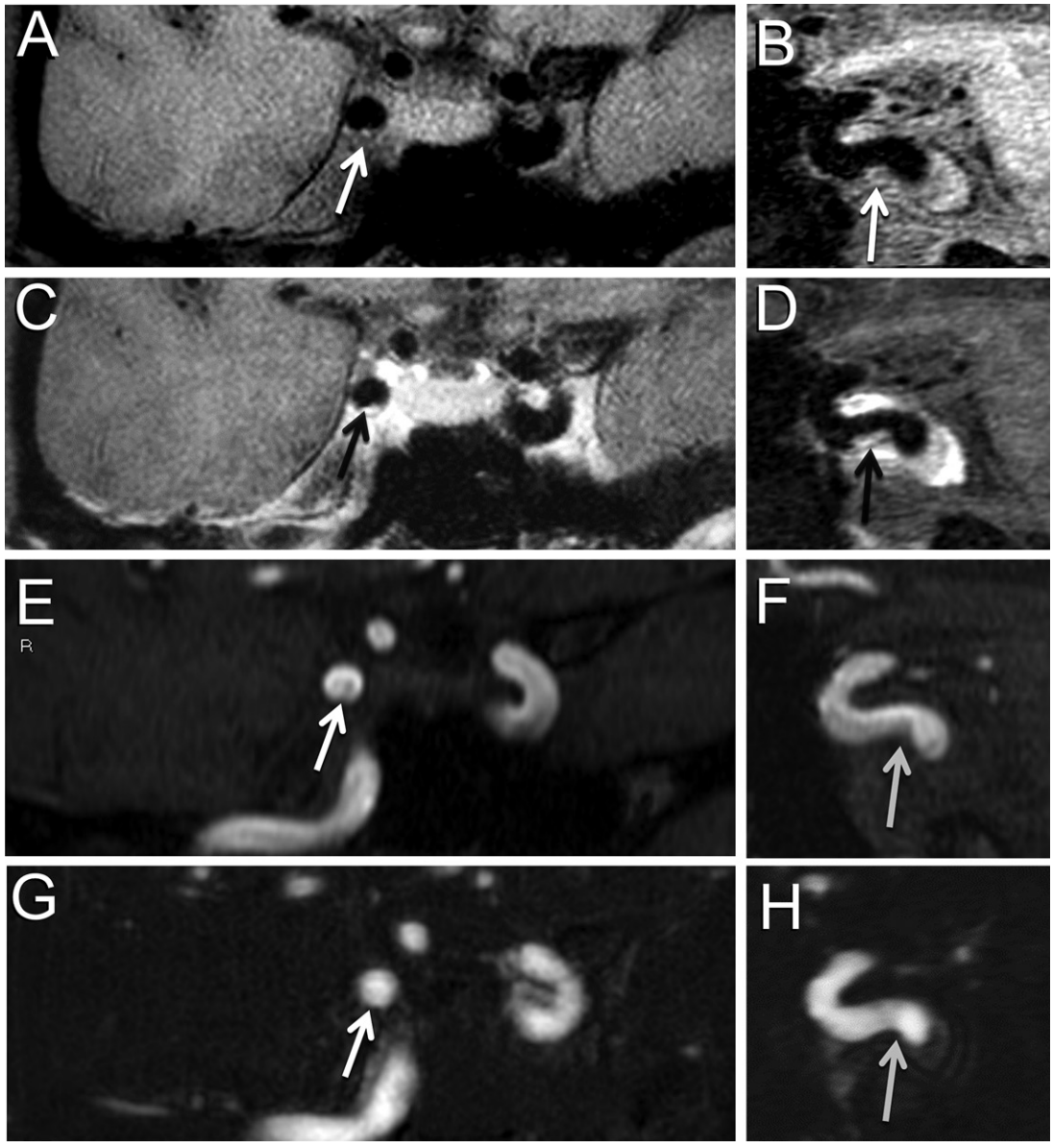

Figure 1: Incomplete blood flow suppression mimicking atherosclerotic plaque in a 53-year-old woman with a history of vision loss. **(A)** Coronal three-dimensional vessel wall MRI scan acquired through the short axis of the cavernous segment of the right internal carotid artery with **(B)** a long-axis reconstruction and **(C, D)** repeated MRI scans after administration of a gadolinium-containing contrast agent show what appears to be focal eccentric thickening with avid enhancement along the inferior wall suggesting plaque (arrow). **(E, F)** Reconstructions of a time-of-flight MR angiography (MRA) scan matching the short-axis **(E)** and long-axis **(F)** orientations shown above show similar but more subtle artifacts due to flow disturbances (arrow). **(G, H)** Contrast-enhanced MRA reconstructions matched with **E** and **F** reveal lumen patency without irregularity, enabling confirmation of the suspected wall thickening as artifactual (arrow).

is most effective when flow (vessel orientation) is parallel to the readout frequency-encoding direction (27). Frequency encoding gradient pulses used in the direction of blood flow can induce strong nonnull gradient moments that result in more signal attenuation in the readout direction than the phase- or section-encoding directions. To avoid misinterpretation due to sequence orientation artifacts, we suggest acquiring 3D VW MRI scans in multiple orientations (Fig 3) to increase the number of vessel segments parallel to the frequency-encoding direction. Flow suppression can be further optimized in small perforator vessels (ie, lenticulastraite arteries) by reducing the turbo factor and using a shorter echo time, as shown in Figure 3. This optimizes intravoxel dephasing for small vessels but not large vessels, so flow artifacts are more likely in larger vessels with these parameter changes. Flow suppression in small vessels (ie, perforator branches) has also been described by using extended phase graph simulations (30).

Blood flow suppression on 3D VW MRI scans can be further improved by using preparation pulse modules such as motion-sensitized driven equilibrium, which uses flow-sensitive dephasing gradients to suppress moving spins (26). This technique has been used to image brain aneurysms (31) but with noted signal-to-noise ratio reduction (26). Delay alternating with nutation for tailored excitation preparation uses nonselective low flip angle pulse trains interleaved with gradient pulses that enable a

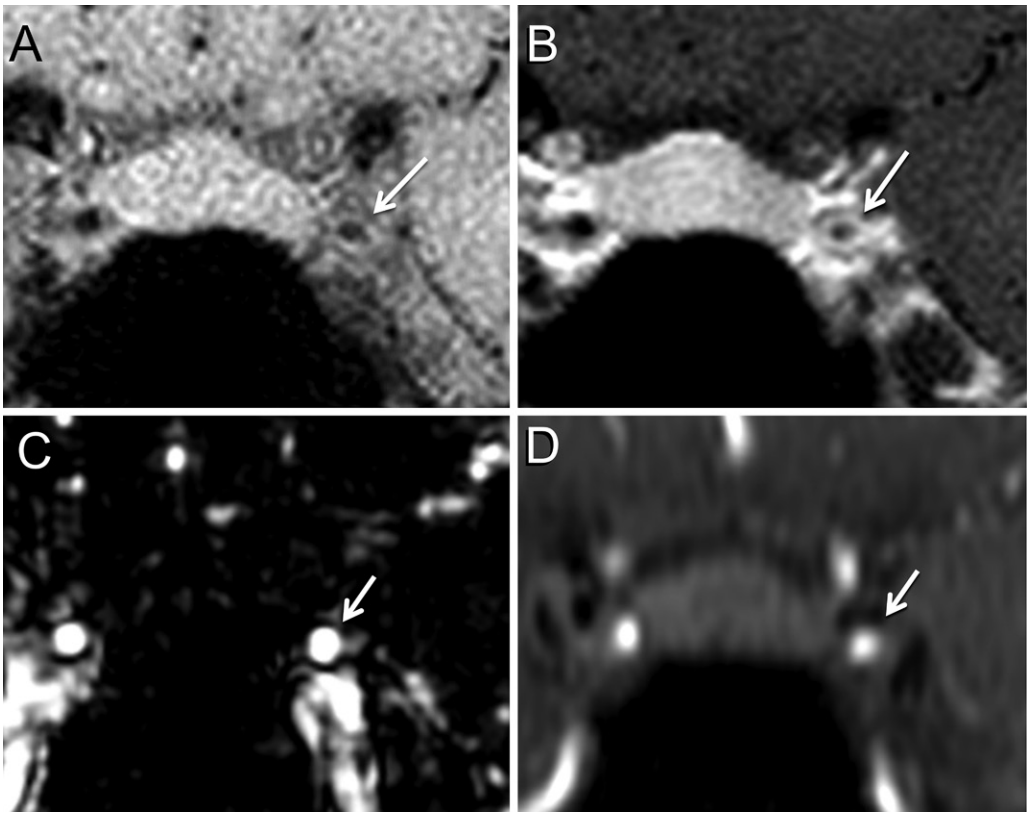

Figure 2: Incomplete blood flow suppression mimicking circumferential wall thickening in a 34-year-old woman with a history of stroke and bilateral distal internal carotid artery (ICA) obstruction. **(A, B)** Circumferential wall thickening is suggested at the cavernous segment of the left ICA (arrow) on **A** pre- and **B** postcontrast coronal vessel wall MRI scans with enhancement on **B**. **(C)** Apparent wall thickening and enhancement are artifactual and are confirmed via the widely patent enhanced lumen (arrow) on the arterial phase contrast-enhanced MR angiographic image and related to slow flow from hemodynamic impairment at the ipsilateral carotid terminus. **(D)** Reconstruction of the time-of-flight MR angiogram at this location shows a patent lumen (arrow), though the caliber is mildly reduced compared with that seen at contrast-enhanced MR angiography because of reduced signal from slow flow at the lumen periphery.

spoiling effect so flowing spins cannot achieve a steady state (32). It offers excellent blood suppression and tissue preservation and has been implemented for intracranial VW MRI (33–35). However, since these techniques are still not widely available, further studies are needed to assess VW MRI scans acquired with and without preparation modules in clinical practice.

CSF Signal Suppression

Delineation of the intracranial arterial VW on VW MRI scans can be optimized by suppressing CSF signal surrounding these vessels to highlight the outer wall. The use of radial ordering modulation, in which the center of the k-space is sampled at the beginning of the echo train (23), can minimize T2 weighting of the image, thereby darkening the signal intensity of the surrounding CSF. CSF signal intensity can also be attenuated by selecting a short repetition time (range, 800–1000 msec) owing to the long T1 value of CSF; however, signal loss from T1 weighting of the VW is an untoward consequence that limits the ability to resolve the thin walls of intracranial arteries. Delay alternating with nutation for tailored excitation VW MRI can suppress the signal of blood and CSF (32–34); however, its effect is diminished when CSF flow is less than 0.1 cm/sec, which is commonly encountered where CSF surrounds the middle cerebral arteries potentially mimicking atherosclerotic plaque formation (32). Addition of a flip-down radiofrequency pulse module to 3D VW MRI is another technique used to improve CSF signal suppression (36,37). It employs a positive 90° radiofrequency pulse at the end of the echo train to tip the transverse magnetization into the negative longitudinal plane, thereby suppressing the transverse magnetization and minimizing signal of tissues with long T2 values (eg, CSF) (Fig 4).

Spatial Resolution

Partial volume averaging due to inadequate spatial resolution is frequently encountered with intracranial VW MRI, especially considering the range of wall thicknesses encountered in the intracranial circulation (normal thicknesses range from 0.3 to 0.6 mm [38]) relative to the optimal acquired resolution (0.5-mm isotropic resolution at 3 T [1,7]). For this reason, a field strength of at least 3 T is considered necessary for adequate intracranial VW MRI (1). Intracranial plaque components might be identifiable as they are in extracranial carotid plaque by VW MRI under ideal conditions, particularly if the vessel is large (eg, intracranial ICA, vertebral or basilar arteries). However, more often intracranial plaques are too small for compositional characterization because of their small size relative to the achievable

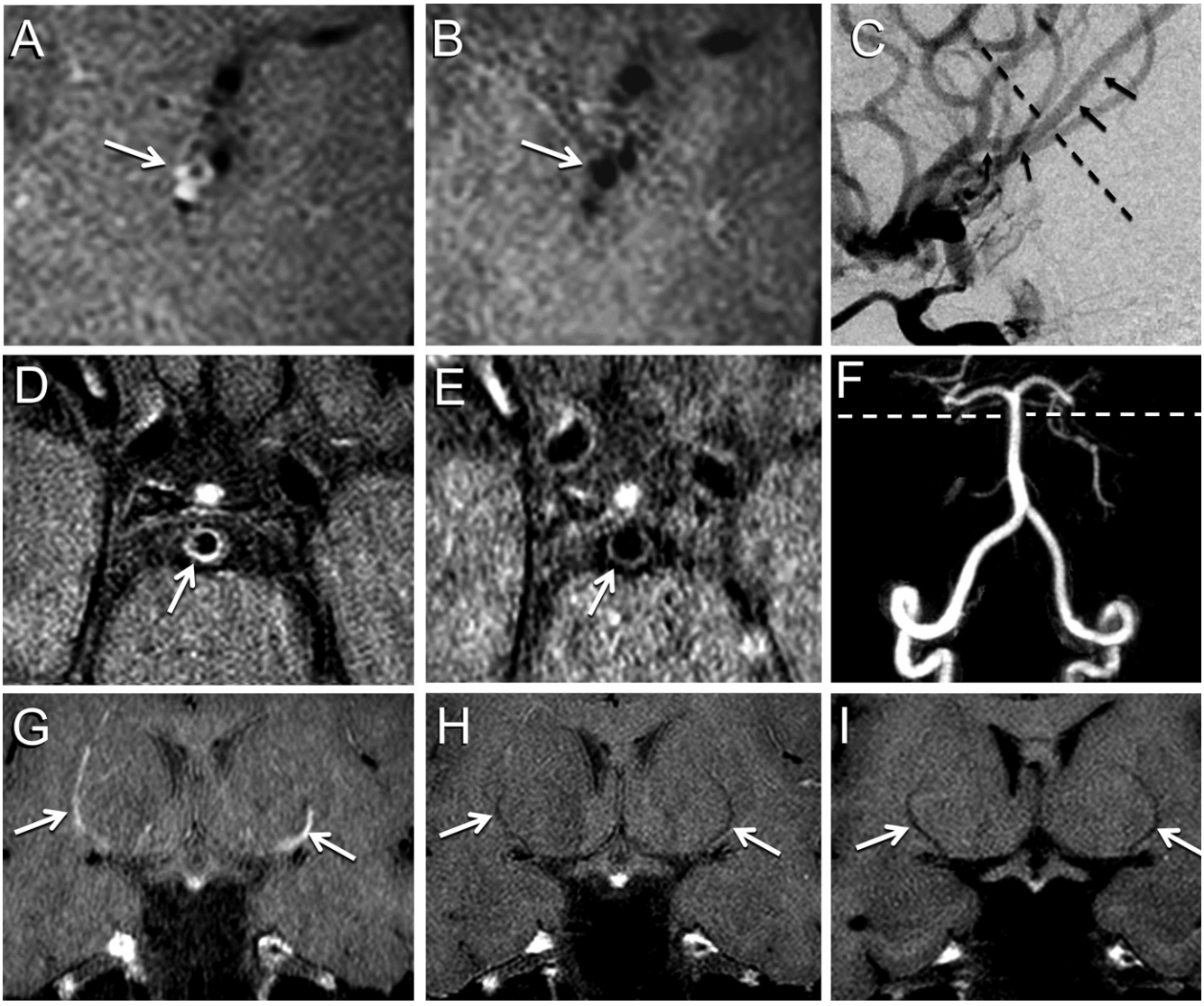

Figure 3: The effect of sequence orientation relative to vessel orientation for adequate blood flow suppression. **(A)** Coronal three-dimensional (3D) vessel wall (VW) MRI scan acquired through the short axis of a left middle cerebral artery (MCA) branch shows circumferential enhancement as might be seen with vasculitis (arrow) in a 32-year-old woman with a history of stroke. **(B)** Enhancement disappears when the VW MRI scan is acquired in an axial orientation where the same MCA segment is now imaged in its long axis (ie, parallel to frequency-encoding direction) (arrow). This image is reconstructed so the vessel is depicted in its short axis matching its orientation in **A**. **(C)** Catheter angiography enables confirmation that this MCA segment is widely patent (arrows). Dashed line indicates location of the MRI section. **(D, E)** In a second patient (32-year-old woman with stroke), artifactual wall enhancement and thickening are seen involving the basilar artery (arrow) on an axial 3D VW MRI scan **(D)** but are not seen on the coronal VW MRI scan **(E)** (arrow) (section reconstructed to match the orientation shown in **D**). **(F)** Time-of-flight MR angiogram shows the patent basilar artery. Dashed line indicates location of the MRI section. **(G)** In a third patient (56-year-old woman with intracranial stenoses), axial 3D VW MRI scan acquired through the short axis of lenticulostriate perforating arteries shows luminal signal intensity (arrows) in this coronal reconstruction. **(H)** Signal intensity is suppressed when the 3D VW MRI scan is acquired in a coronal orientation and the vessels are imaged in their long axes (arrows). **(I)** Flow suppression in these small vessels is optimized by using a reduced turbo factor and shorter echo time (arrows).

resolution (Fig 5). The consequential exaggeration of the true wall thickness can be misinterpreted as atherosclerotic plaque or vasculitis, especially if there is corresponding enhancement. Adequate resolution is also needed to resolve small adjacent vessels, especially cerebral veins and arteries that frequently course together in the Sylvian fissure (Fig 5). Artifactual enhancement in small veins due to slow flow can easily be mistaken as eccentric arterial wall enhancement suggesting atherosclerotic plaque when imaged with inadequate resolution. Application of a 2D DIR section through the short axis of a vessel can help resolve its structure because of the higher in-plane resolution of the 2D acquisition (eg, 300 ≥m at 3 T) if there is not much change in the lesion morphology along the section direction.

Acqustion Time

Although 3D VW MRI has proven to be a high signal-to-noise ratio efficiency technique, a typical high-spatial-resolution VW MRI acquisition (0.5-mm isotropic resolution) with whole-brain coverage (acquired in the sagittal orientation) can take more than 10 minutes (36). A coronal orientation using a slab-selective excitation (6.4- to 10-mm-thick slab) can reduce the imaging time to 5–8 minutes while still

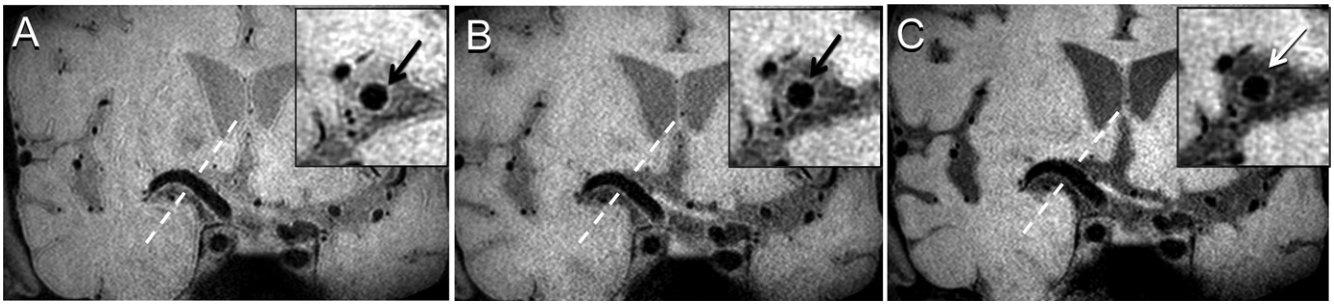

Figure 4: Cerebrospinal fluid (CSF) signal suppression on three-dimensional (3D) vessel wall (VW) MRI scans in a 77-year-old healthy male volunteer. **(A)** A 3D VW MRI scan (repetition time msec/echo time msec, 2000/37) shows poor contrast between CSF signal and the VW. **(B)** CSF signal is attenuated by reducing the repetition time to 1000 msec but at the cost of reduced signal of the VW. **(C)** CSF signal is further suppressed by using an antidriven equilibrium post pulse while maintaining the repetition time at 2000 msec to mitigate signal loss of the VW. All images were acquired with the same acquired resolution and scan times.

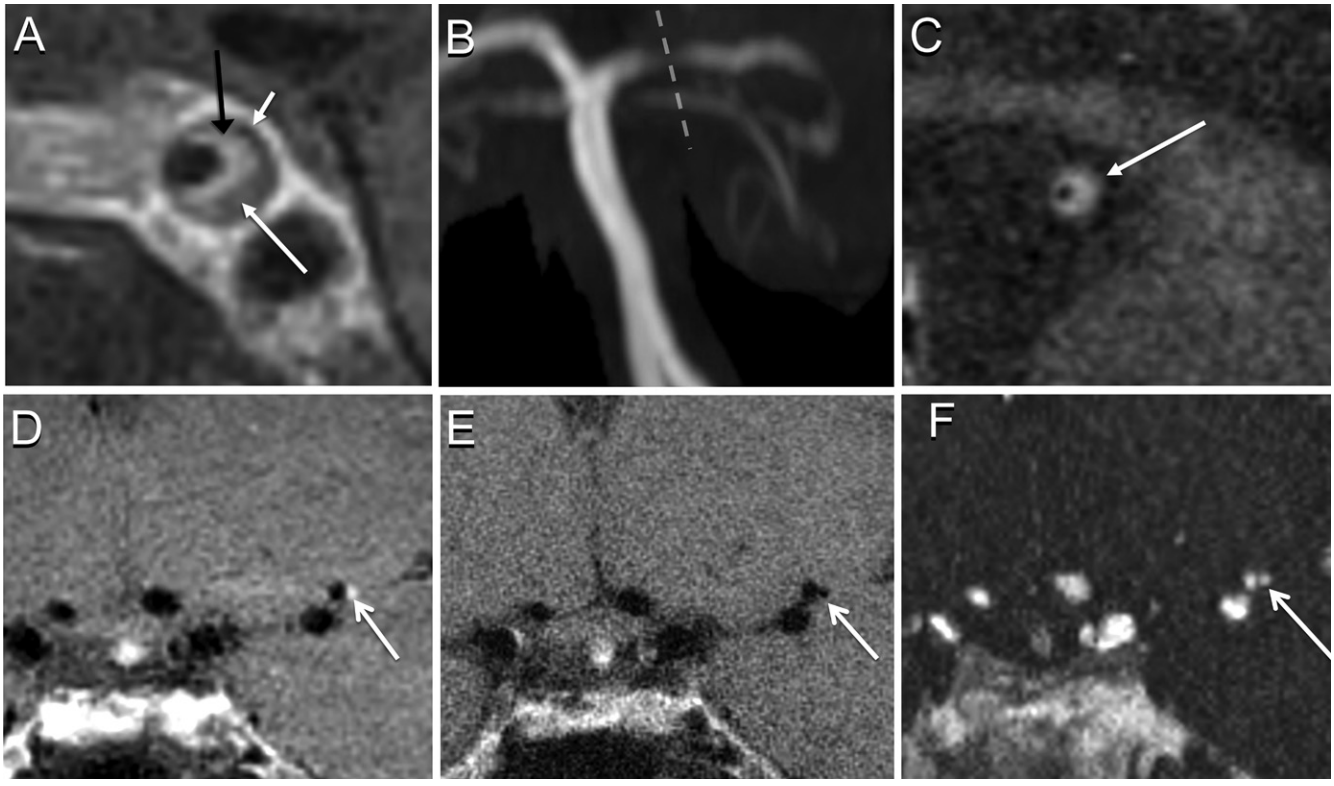

Figure 5: Resolution constraints in characterizing intracranial vessels. **(A)** Three-dimensional (3D) vessel wall (VW) MRI scan of a cavernous segment of a left internal carotid artery (ICA) shows characteristic features of an atherosclerotic plaque with a contrast-enhancing fibrous cap (black arrow), hypoenhancing lipid core (long white arrow), and small focus of hypointense calcification (short white arrow). **(B, C)** For comparison, VW MRI of a P1 segment of the left posterior cerebral artery as prescribed on a time-of-flight MR angiography (line in **B**) in a different patient shows an enhancing eccentric atherosclerotic plaque **(C)**. Both plaques were imaged using an acquired resolution of 0.5 mm^3, but the internal components of the posterior cerebral artery plaque are too small to characterize (maximum thickness, ≤1 mm) in contrast to the large cavernous plaque (maximum thickness, approximately 3 mm). **(D)** In a 52-year-old woman with clinical concern for intracranial vasculitis, focal enhancement along the lateral wall of the M2 segment of the left middle cerebral artery on a coronal postcontrast VW MRI scan cannot be distinguished from the arterial wall, raising suspicion for plaque (arrow; acquired resolution, 0.5 mm^3). **(E, F)** A two-dimensional black-blood MRI scan **(E)** acquired in the same patient and at the same location as **D** shows a patent lumen of a small vein near the M2 segment (arrow, 0.35 × 0.35 × 2 mm), also seen on the venous phase contrast-enhanced MR angiographic image **(F)**.

covering the major intracranial vessels, making it more feasible in clinical practice. Recent advances in compressed sensing reconstruction enables accelerated MRI by nonlinear iterative reconstruction of sparsely under-sampled k-space data (39). Compressed sensing has been widely implemented in different clinical applications. It has also been used to accelerate carotid plaque imaging (40) and, more recently, for intracranial VW MRI (41), allowing for a substantial reduction in imaging time. Currently, compressed sensing is available for clinical application with Food and Drug Administration approval for some vendor platforms (ie, MRI scanners from Philips Healthcare) (Fig E1 [online]).

Vessel Wall Enhancement

Vessel wall enhancement varies depending on the underlying vasculopathy, and the relative sparsity of vasa vasorum in

intracranial arteries under normal conditions and its development in the setting of intracranial vasculopathy is thought to largely contribute to these variations (42). Other factors that are thought to contribute to abnormal enhancement are related to the level of inflammatory activity because of increased endothelial permeability and neovascularity (9,13,43). This enhancement is characteristically transient and is associated with the active phase of inflammation (44). The degree of enhancement may be attenuated by steroid therapy, so current medications should be reviewed when VW MRI is considered to assess for vasculitis.

The 3D isotropic VW MRI sequence can be optimized for T1 weighting to highlight contrast enhancement by selecting a short repetition time as can be done for CSF signal suppression (see the CSF Signal Suppression section). This will reduce the signal of the VW, but this may not be an important limitation if detection of contrast enhancement is the primary clinical objective. Because the null point of blood changes after administration of a gadolinium-containing contrast agent, the inversion time should be adjusted for 2D DIR MRI (250 msec at 3 T) (19). However, flow suppression on 3D VW MRI scans does not depend on the T1 signal of blood, so no parameter changes are required. Postcontrast VW MRI examination should be adequate for most clinical applications after a 5-minute delay following contrast agent administration (9,13). The 2D DIR can be helpful to corroborate VW enhancement when suspected on 3D VW MRI scans.

Enhancement Mimicking Vasculopathy

Leptomeningeal Enhancement

Vasculitis affecting the central nervous system requires prompt diagnosis and care. The 3D VW MRI scans have been used to identify vascular inflammation based on the presence of segmental concentric enhancement of the VW and circumferential perivascular enhancement after a vessel segment (13) (Fig 6). However, it is important to recognize that blood vessels coursing through leptomeningeal enhancement, commonly seen in the setting of inflammatory diseases, can result in a false-positive appearance of vasculitis with perivascular inflammation (Fig 6). Awareness of anatomic structures and scrutiny of the appearance on multiple oblique images reconstructed from 3D VW MRI scans are essential to correctly evaluate VW enhancement.

Thrombus with Recanalization

When interpreting postcontrast VW MRI scans for suspected inflammatory vasculopathy, a potentially challenging task is differentiation of an inflamed vessel from the enhancement of an organized thrombus. Because the lumina of very small vessels are not always seen on VW MRI scans, small-vessel vasculitis might be suspected based on a linear configuration of enhancement (13). However, this appearance can result from an arterial thrombus that has organized (Fig 7). The organization and recanalization of a thrombus often involve the growth

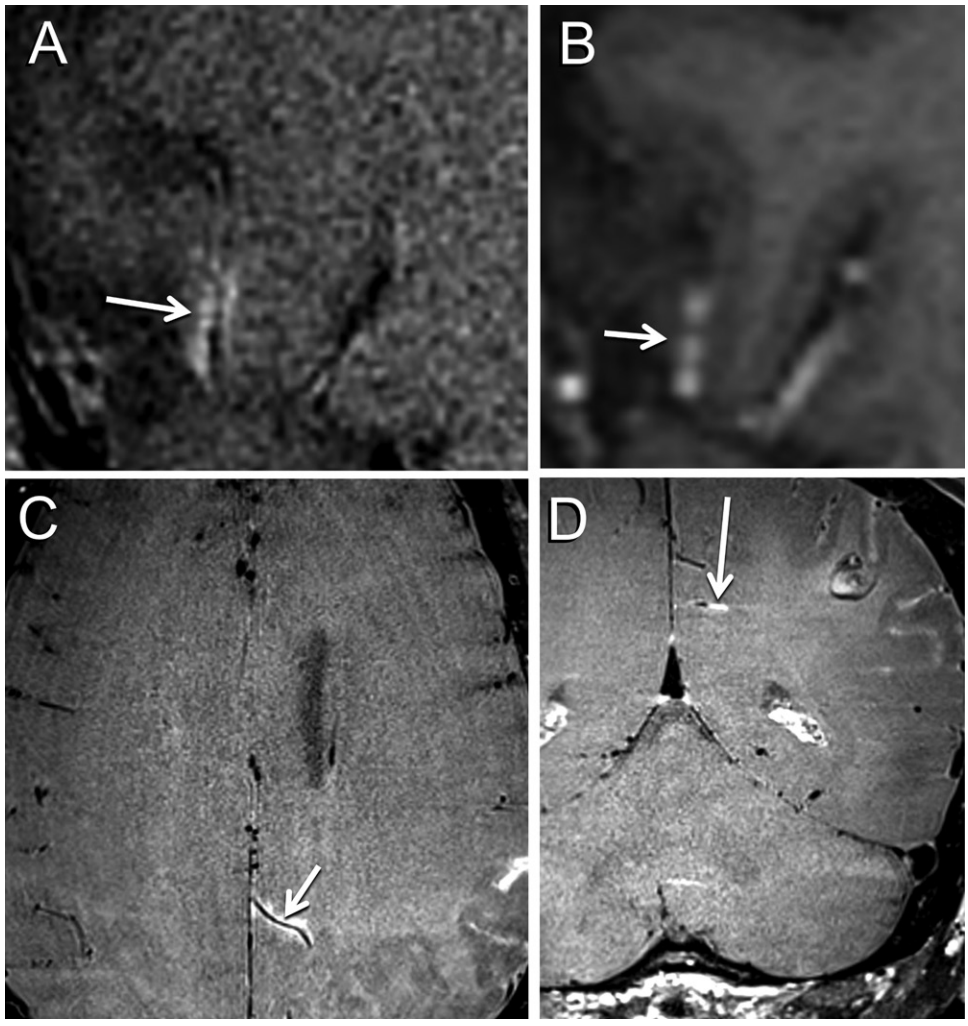

Figure 6: Biopsy-proven intracranial vasculitis compared with leptomeningeal enhancement mimicking this diagnosis on three-dimensional (3D) vessel wall (VW) MRI. **(A, B)** Coronal reconstructions of a 3D VW MRI scan in a 34-year-old man with biopsy-proven vasculitis show concentric segmental enhancement of the VW and periadventitial parenchyma involving a branch of the right middle cerebral artery (arrow in **A**), with corresponding irregular luminal enhancement on 3D postcontrast T1-weighted brain image (arrow in **B**). **(C)** In contrast, postcontrast axial VW MRI in a 69-year-old woman with left parietal intracerebral hemorrhage and prior stroke shows prominent perivascular enhancement surrounding a vessel in the left inferior parietal lobe (arrows). **(D)** Postcontrast coronal VW MRI scan shows enhancement is leptomeningeal, confined to a sulcus oriented along an axial plane, and does not involve brain parenchyma.

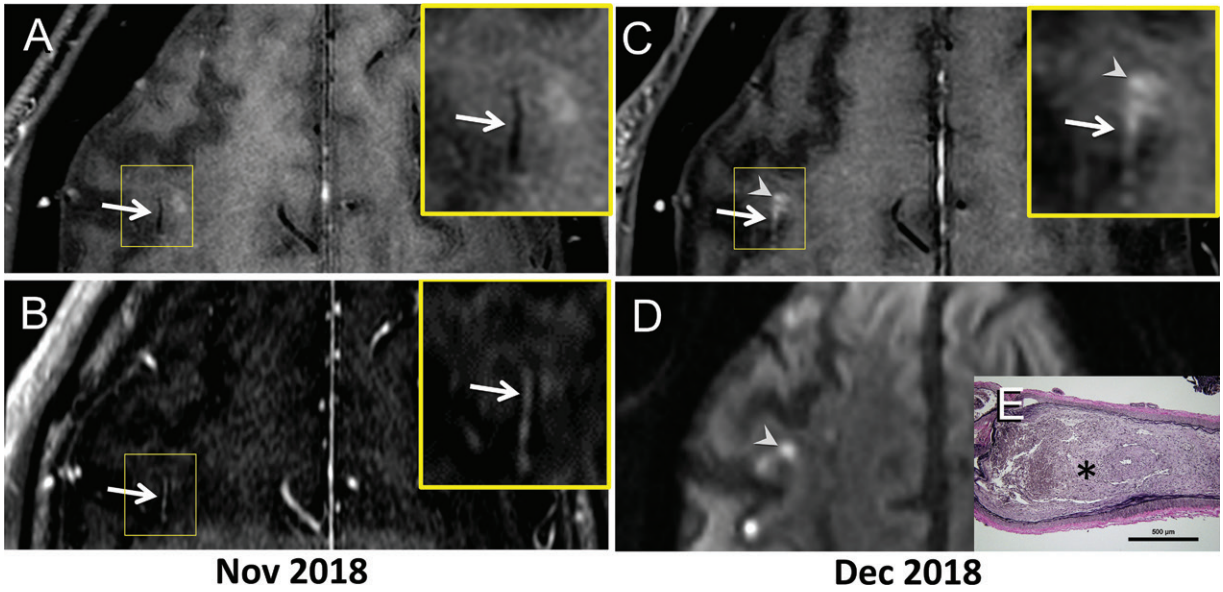

Figure 7: Thrombus with recanalization mimicking vasculitis in a 44-year-old man with a history of multiple prior strokes. **(A, B)** Normal appearance of a small vessel in the right middle frontal gyrus (arrow) on axial postcontrast vessel wall (VW) MRI scan **(A)** and contrast-enhanced MR angiogram **(B).** Insets show magnified views. (Original magnification, ×2.5.) **(C, D)** Repeat imaging performed 1 month later shows abnormal enhancement involving the vessel (arrow) on postcontrast VW MRI scan **(C)**. Inset shows a magnified view. The vessel leads to a focus of parenchymal enhancement (arrowhead in **C**) that corresponds to a focus of restricted diffusion on the diffusion-weighted image (arrowhead in **D**), suggesting a subacute infarct related to flow impairment in the depicted vessel. **(E)** The vessel was sampled for biopsy via open craniotomy, and the pathologic specimen revealed a partly recanalized luminal thrombus (*), without evidence of abnormal changes in the underlying VW. (Hematoxylin-eosin stain; original magnification, ×40.)

of capillary-sized channels (ie, immature microvessels) through the fibrin-rich thrombus for continuity of blood flow (45–47). These recanalized channels irrigate the thrombus and lead to enhancement after contrast material administration.

Microhemorrhages with Surrounding Enhancement Mimicking Perivascular Inflammation

Cerebral microhemorrhages are small chronic deposits of blood and are typically caused by structural abnormalities of small vessels in the brain. Postmortem studies have shown an increase in the density of inflammatory cells near microbleed lesions (48,49). This inflammatory response surrounding microhemorrhages, however, may resemble an inflammatory process involving the VW, especially since the signal loss from the microbleed can resemble the flow-suppressed lumen on VW MRI scans. Microhemorrhages can be distinguished from vasculitis on susceptibility-weighted images by identifying a single focus of signal loss central to the enhancement reflecting the ferromagnetic composition of a microhemorrhage (Fig 8) in contrast to a linear distribution of signal loss as seen with a vessel lumen. The interpretation of vascular inflammation in the setting of microhemorrhages can be even more challenging in patients with cerebral amyloid angiopathy. Histologic evaluation of a biopsy specimen of a vessel showing wall and perivascular enhancement on VW MRI scans in a patient with cerebral amyloid angiopathy revealed only VW thickening with amyloid deposition but no inflammation (13). The reason for enhancement was not clear and could relate to β-amyloid toxicity in the VW, but radiologists should be aware of the potential for this false-positive finding (49).

Parenchymal Enhancement of Subacute Ischemic Stroke

Parenchymal contrast enhancement is often detected a few days after the onset of an ischemic stroke due to leakage of contrast material across the disrupted blood-brain barrier (50,51). Enhancement from a subacute ischemic lesion can resemble inflammatory enhancement of a small parenchymal vessel (Fig 9). Diffusion-weighted imaging (prior to and at the time of the examination when available) can help confirm enhancement from subacute ischemia. This can be especially helpful when identifying a biopsy target for small-vessel vasculitis associated with infarctions (13).

Vascular Plexus Enhancement

Vessel wall enhancement not attributable to vasculopathic changes can also be identified due to enhancement of the vasa vasorum or a vascular plexus. Vaso vasorum is present in extracranial arteries but is not typically present in intracranial vessels in healthy young adults and children (42). However, vasa vasorum can communicate with extracranial vascular plexuses and follow arteries as they enter the intracranial compartment, frequently involving the proximal portions of the V4 segments of the vertebral arteries (52). This can lead to concentric arterial wall thickening and enhancement that can mimic the appearance of vasculitis or atherosclerotic plaque (52) (Fig 10). Reconstruction of VW MRI scans to depict vessels in their long-axis view rather than in their straight short-axis view can help confirm if enhancement might be due to the vascular

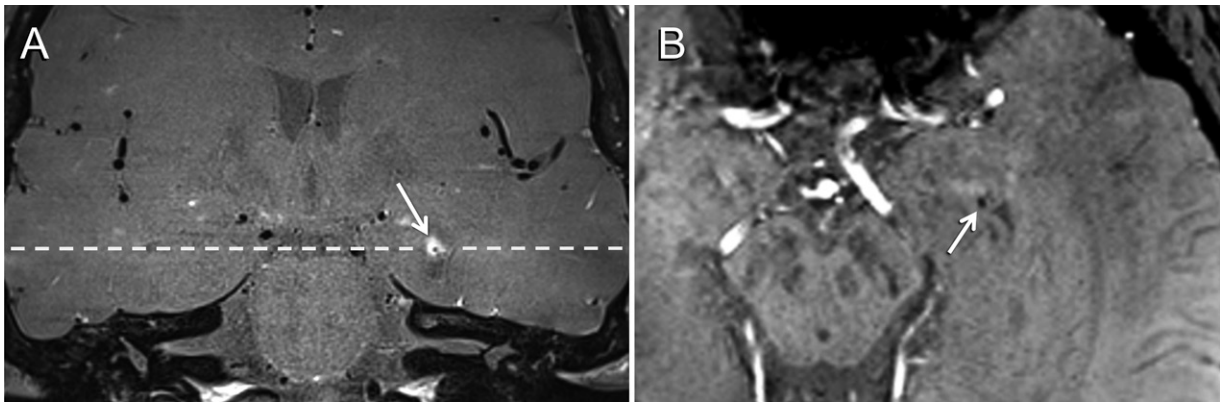

Figure 8: Microhemorrhage with surrounding enhancement mimicking perivascular enhancement with vasculitis in a 34-year-old man with multiple small strokes. **(A)** Coronal postcontrast vessel wall (VW) MRI scan shows prominent enhancement surrounding a tiny hypointense focus in the left temporal lobe (arrow). **(B)** This corresponds to a punctate hypointense focus on the susceptibility-weighted image, suggesting a microhemorrhage (arrow). Enhancement and hypointensity did not show a linear configuration on the VW MRI scan or susceptibility-weighted image, respectively, as would have been expected if it had been a flow-suppressed vessel with surrounding inflammation.

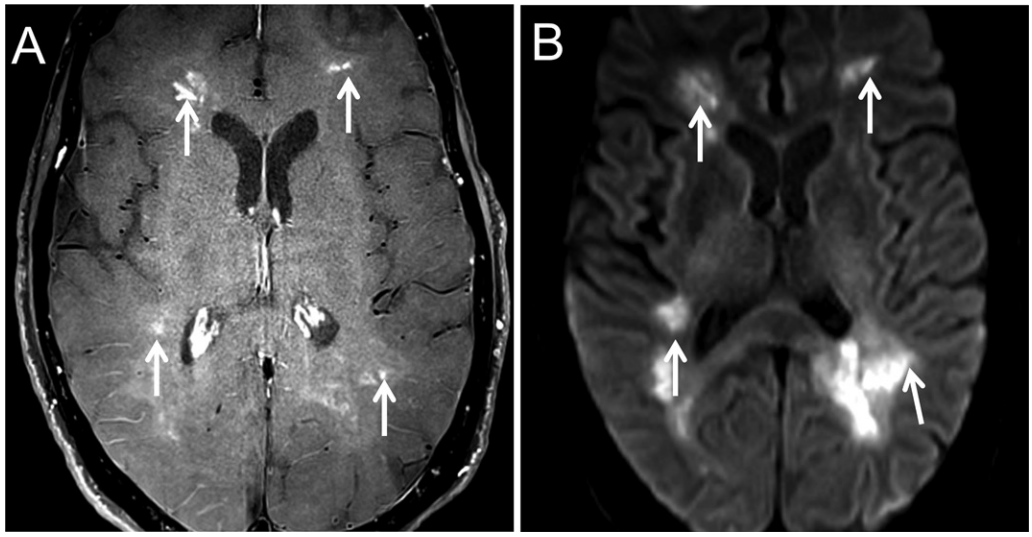

Figure 9: Parenchymal enhancement from subacute ischemic stroke mimicking vasculitis in a 63-year-old woman with a history of intravascular lymphoma. **(A)** Postcontrast vessel wall (VW) MRI scan shows patchy parenchymal enhancement, some with a linear configuration, in the frontal and parietal lobes (arrows). **(B)** Enhancement corresponds to foci of restricted diffusion (arrows) on diffusion-weighted images.

plexus at the skull base. Acquisition of a short-axis 2D DIR image through the vessel in question often helps to exclude true disease. A common cause of vascular enhancement that is often misinterpreted as vasculitis or atherosclerotic plaque is the venous enhancement that surrounds a partially collapsed petrous carotid artery segment when there is ipsilateral hemodynamic impairment. This reflects a compensatory expansion of the venous plexus within the petrous canal (53) (Fig 10).

Focal nodular thickening and enhancement from a varix or ganglion may also be seen at the cisterna magna (54) and can be misdiagnosed as eccentric atherosclerotic plaque when contiguous with the V4 segment of the vertebral artery (Fig 11). A continuous connection is often seen between the lesion and an intradural vein leading to the venous plexus at the skull base where the vertebral artery penetrates the dura.

In comparison with arteries of comparable size, veins have a higher density of vasa vasorum related to more hypoxic blood within the lumina (55) that could contribute to wall enhancement in the absence of underlying disease (42). Inadequate suppression of slow flow along the lumen periphery could contribute even more to the appearance of abnormal wall enhancement in veins (Fig 12).

Protocol Recommendations for Clinical Application

Given the unique histoanatomy of intracranial arteries and the diseases specific to these vessels along with the artifacts frequently encountered when imaging these small and tortuous structures, a protocol should be designed that considers these conditions and is tailored to the clinical concern and region of interest while considering the time burden for clinical implementation (Table 2). Results are acquired that corroborate abnormalities suspected on VW MRI scans and might add specificity to its interpretation. For example, when considering the diagnosis of

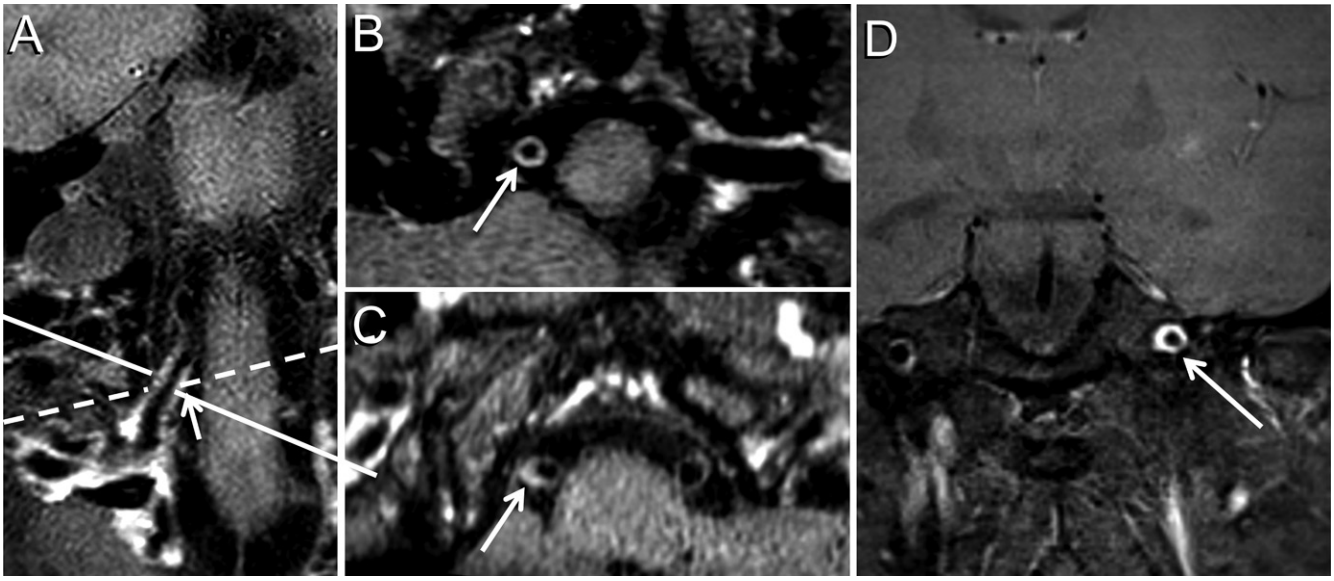

Figure 10: Venous plexus enhancement mimicking vasculitis or atherosclerotic plaque. **(A)** Coronal long-axis postcontrast vessel wall (VW) MRI scan of the right vertebral artery in a 32-year-old woman without cardiovascular risk factors shows thick enhancement along the wall from the extradural venous plexus that follows the vessel as it enters the intracranial compartment. **(B)** Short-axis reconstructed image (acquired resolution, 0.5 mm^3) through the right vertebral artery (solid line in **A**) shows concentric thickening and avid enhancement due to partial volume averaging of the wall and plexus (arrow in **B**). **(C)** Oblique orientation of the reconstructed image through the right vertebral artery (dashed line in **A**) exaggerates the lateral wall thickness of the artery (arrow in **C**), resembling eccentric wall thickening as seen with plaque. Venous plexus enhancement surrounding the petrous internal carotid artery (ICA) mimicking vasculitis in a 16-year-old girl suspected of having focal cerebral arteriopathy. **(D)** Postcontrast VW MRI scan shows partial collapse of the left ICA due to severe stenosis at its terminus, resulting in compensatory expansion of the venous plexus within the petrous carotid canal surrounding the vessel and resembling vasculitis (arrow).

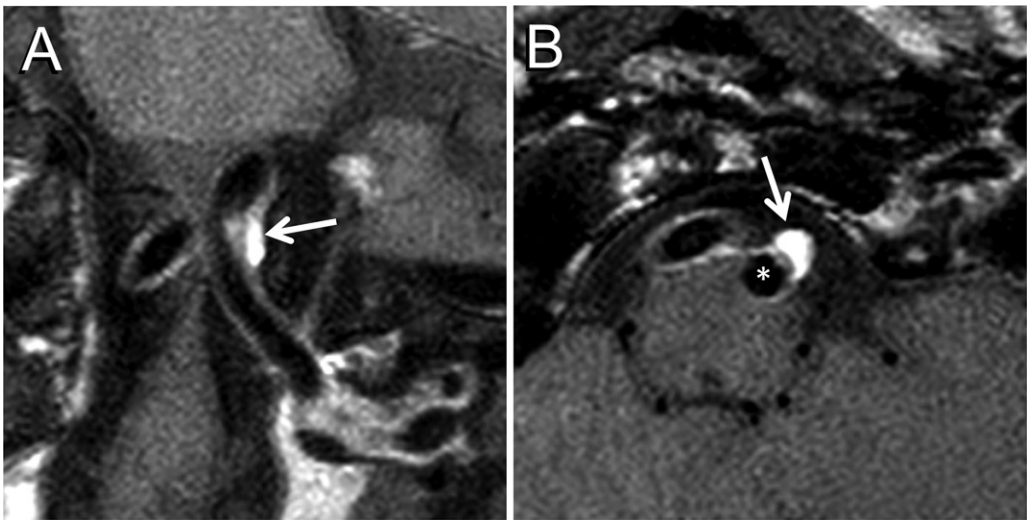

Figure 11: Focal benign nodular lesion at the foramen magnum mimicking atherosclerotic plaque in a 56-year-old woman with distal left internal carotid artery and middle cerebral artery stenoses. **(A)** Coronal postcontrast vessel wall MRI scan shows an enhancing lesion (arrow) along the lateral margin of the left vertebral artery. **(B)** Lesion (arrows) can be distinguished from the arterial wall on a short-axis reconstructed image.

vasculitis, susceptibility-weighted imaging can be useful to improve confidence that linear enhancement on 3D VW MRI scans represents an inflamed vessel (13). If the susceptibility-weighted imaging sequence displays the arterial circulation as hyperintense, it can also be used to differentiate between arteries and veins when characterizing small-vessel vasculitis and it can help to confirm mural calcification in larger arteries. This could support atherosclerotic plaque over a vascular plexus when encountered in the proximal V4 segment of the vertebral artery (Fig 10). Although magnetization-prepared rapid gradient-echo sequence is useful for evaluating intraplaque hemorrhage and calcification in extracranial carotid atherosclerosis, it is not used as commonly for intracranial vascular imaging in part because of challenges with luminal signal suppression that can obscure the relatively thin arterial wall. During contrast material administration, we recommend performing contrast-enhanced MR angiography to confirm lumen patency since flow artifacts narrowing the lumen on VW MRI scans are often mirrored on TOF MRA images (Figs 1,

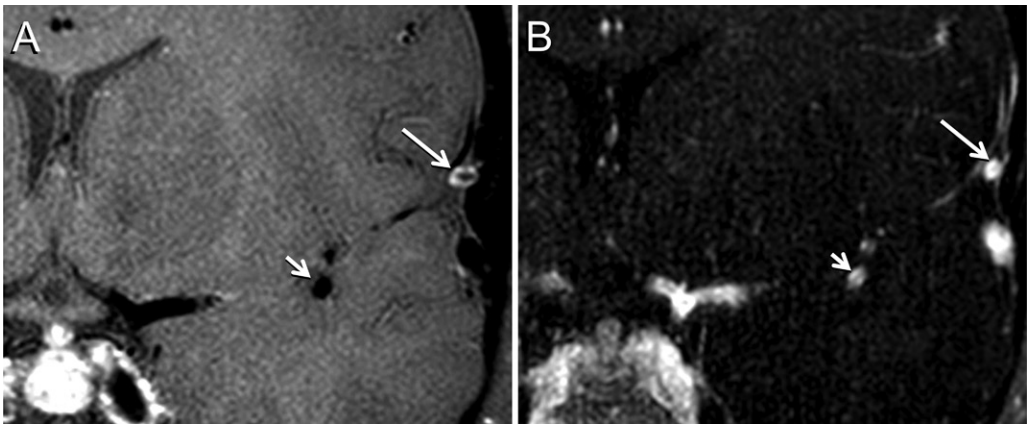

Figure 12: Venous enhancement mimicking vasculitis in a 31-year-old woman with a history of left-sided neck pain. Concentric wall enhancement shown on vessel wall MRI scan can be seen in a normal cortical vein (long arrow in **A**) and on a venous phase contrast-enhanced MR angiographic image (long arrow in **B**), whereas no wall enhancement is expected in the arterial wall (short arrow, M2 segment of left middle cerebral artery) under normal conditions.

Table 2: Protocol Recommendations

Sequence	Orientation	Justification
3D time-of-flight MR angiography	Axial	Survey for intracranial lesions, identify T1 hyperintense blood (IPH or dissection), serve as a scout to orient the 3D VW MRI acquisition
Diffusion-weighted images	Axial	Correlate with plaque enhancement when evaluating for culprit lesions, distinguish small vessel vasculitis from subacute infarctions (Fig 9), assess downstream impact of vascular lesions
3D VW MRI (precontrast)	Coronal (depends on vessels of interest)	Confirm blood products; when diagnosing small-vessel vasculitis, ischemic changes can appear mildly hyperintense on 3D VW MRI scans due to intrinsic proton density signal making periadventitial enhancement difficult to distinguish once contrast material is administered
Susceptibility-weighted imaging	Axial	Identify vessels for correlation with VW MRI scans to diagnose small-vessel vasculitis, distinguish small arteries from veins*
CE MR angiography	Coronal	Confirm lumen patency (Figs 1, 2, 5, 7, 12)
3D VW MRI	Coronal and axial (depends on vessels of interest)	Identify vascular lesion, add specificity to angiographic narrowing and distinguish vasculopathies
2D DIR	Short axis	Confirm enhancement detected on 3D VW MRI scans, confirm lumen patency

Note.— CE = contrast enhanced, DIR = double inversion recovery, IPH = intraplaque hemorrhage, 3D = three-dimensional, 2D = two-dimensional, VW = vessel wall.

* Vendor dependent (ie, possible if gradients are set to display the arterial and venous circulation as bright and dark, respectively).

2). These artifacts are particularly dangerous since their identification on both the VW MRI and TOF MRA scans might increase the interpreter's confidence that true narrowing exists. The contrast-enhanced MRA source images are sufficient to confirm lumen patency, so venous contamination should not limit its value. We suggest both pre- and postcontrast 3D VW MRI be performed; however, the precontrast acquisition may be dispensable depending on the application, and postcontrast images acquired in multiple orientations should be prioritized to maximize flow suppression of vessels coursing in orthogonal directions (Fig 3). Multiple orientations of the precontrast 3D VW MRI scans should be considered when evaluating dissection, in which case precontrast images are likely to be more helpful than postcontrast images; otherwise, the time demand of multiple precontrast 3D VW MRI acquisitions is hard to justify. In fact, the administration of contrast material may not be needed at all to evaluate dissection and should be questioned if TOF MRA and precontrast 3D VW MRI appear to be adequate. The coronal orientation is our preferred standard orientation since it captures most of the middle cerebral artery and the proximal anterior and posterior cerebral artery branches, but this should be tailored depending on the vessels of interest. The 2D DIR sequence is a useful technique to confirm lesions suspected on 3D VW MRI scans.

Summary

The accurate interpretation of intracranial three-dimensional vessel wall MRI scans in clinical practice relies on an ability to recognize pitfalls that arise from incomplete flow suppression, insufficient spatial resolution, or misinterpretation of enhancement, and an understanding of the requirements for

technical adequacy. These preconditions highlight the need for optimized sequences appropriate to the diagnosis in question and adequate training to identify pitfalls and interpret examination results correctly.

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