Conventional and high-resolution vessel wall MRI of intracranial aneurysms: current concepts and new horizons

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Intracranial aneurysms are heterogeneous in histopathology and imaging appearance. The biological behavior of different types of aneurysms is now known to depend on the structure and physiology of the aneurysm wall itself in addition to intraluminal flow and other luminal features. Aneurysm wall structure and imaging markers of physiology such as aneurysm wall enhancement have been assessed in many prior investigations using conventional-resolution MRI. Recently, high-resolution vessel wall imaging (HR-VWI) techniques with MRI have been introduced. Reports of findings on high-resolution imaging have already emerged for many types of aneurysms demonstrating detailed characterization of wall enhancement, thickness, and components, but many questions remain unexplored. This review discusses the key HR-VWI literature to date. Aneurysm wall findings on conventional-resolution MRI are also discussed as these may help one understand the potential utility and findings on HR-VWI for various aneurysm types. The authors have illustrated these points with several examples demonstrating both features already described in the literature and novel cases demonstrating the potential for future clinical and research applications.


KEY WORDS aneurysm wall; high-resolution vessel wall imaging; vascular disorders; intracranial

Intracranial aneurysms have a wide range of etiologies, pathological characteristics, imaging appearances, natural histories, and treatment options. Two recent developments have the potential to greatly advance the understanding and radiological assessment of intracranial aneurysms: 1) increased focus on aneurysm wall pathology, structure, and behavior, and 2) implementation of high-resolution vessel wall imaging (HR-VWI) techniques in clinical practice. HR-VWI complements the traditional luminal evaluation of intracranial aneurysms. However, the primary vessel wall pathology, and therefore HR-VWI appearance, varies drastically among the different types of aneurysms.

For HR-VWI, higher field strengths of at least 3T are advantageous due to the need for submillimeter spatial resolution and the resultant signal demands. Various techniques now exist in clinical practice, designed to provide optimal spatial resolution and to sharply delineate vessel walls between flowing blood and CSF; these include both 2D and 3D techniques. Two-dimensional techniques are widely available and can be readily implemented. Two-dimensional imaging in a plane perpendicular to the plane of the lumen is typically the most important imaging plane. This allows for accurate assessment of the lesion effects on the lumen and lesion morphology, better estimation of lesion and wall thickness, and the avoidance of...
volume averaging artifacts. Three-dimensional HR-VWI techniques have recently increased in utilization for vessel wall imaging. Advantages of 3D techniques include higher through-plane spatial resolution, with increased brain coverage, and the ability to perform isotropic acquisitions. Of the 3D techniques available, variable refocusing flip angle (VRFA) techniques are the most extensively studied and now predominate many clinical practices (sampling perfection with application optimized contrast using different flip angle evolutions [SPACE], Siemens Healthcare; CUBE, GE; and volumetric isotropic turbo spin echo acquisition [VISTA], Philips Healthcare) as they provide improved blood suppression in a shortened scan time relative to other 3D and 2D techniques. Three-dimensional VRFA techniques have been performed using T1, proton density (PD), and T2 contrast weightings for various applications, with each providing specific lesion characteristics that may help in disease differentiation and characterization. The utilization of multiple contrast weightings may be advantageous for differentiation of intracranial vasculopathies, although not yet extensively studied in aneurysms.

A variety of blood and CSF suppression techniques can be used to enhance visualization of the vessel wall. CSF suppression techniques include antide drive and delayed alternating withination for tailored excitation (DANTE), while blood suppression techniques include motion-sensitive driven equilibrium (MSDE), DANTE, double inversion recovery (DIR), quadruple inversion recovery (QIR), and magnetization-prepared inversion recovery (MPIR). While some suppression techniques are used with 3D HR-VWI (MSDE, DANTE, antide drive), others can only be used with 2D techniques (DIR, QIR). The precise suppression techniques used may not be salient to the clinician, but it is important to recognize that some are considered research, rather than FDA-approved product methods. Additionally, HR-VWI can be performed with small field-of-view (FOV) images (on the order of 16 cm × 16 cm) to achieve exquisite detail but with limited coverage of the head or whole-head FOV images (on the order of 24 cm × 24 cm). Because a wide variety of techniques exist, it is important to understand those available and used at one’s institution.

The HR-VWI used at our institution and demonstrated here is a 3D PD SPACE technique. Images are acquired with a 16-cm FOV. This technique allows 0.25 mm × 0.25 mm interpolated in-plane pixel size with a slice thickness of 0.5 mm and can be acquired in any imaging plane. Such a focused technique only covers a 6-cm slab of brain unless scan time is increased, so the radiologist and clinicians must work closely with the technologists to ensure the aneurysm is fully imaged. T1- and T2-weighted images can also be obtained. In this review, conventional MRI refers broadly to more typical MRI sequences used routinely in clinical practice such as spin echo T1-weighted images at whole-brain FOV without special parameters to optimize visualization of vessel or aneurysm walls. Although the spatial resolution of these techniques varies considerably, they are typically on the order of several millimeters in slice thickness.

There are many prior studies assessing the histopathology and imaging characteristics of aneurysm walls on conventional-resolution MRI, but data assessing the utility of HR-VWI are emerging. Therefore, expected findings on HR-VWI are in part extrapolated from current knowledge of pathology and conventional-resolution MRI. Compared with conventional-resolution MRI, HR-VWI has the potential to identify smaller or more subtle areas of signal change or enhancement, more precisely delineate aneurysm wall thickness, and provide a more precise representation of the pathological composition. Some of these features could serve as imaging biomarkers to predict the biological behavior of aneurysms, identify a culprit aneurysm in the setting of subarachnoid hemorrhage (SAH), and potentially direct optimum treatment.

In this review, we highlight key studies to date addressing aneurysm vessel wall histopathology and findings on conventional-resolution MRI. This information is extrapolated to discuss potential future utility of HR-VWI. Additionally, specific literature assessing the utility of HR-VWI for some aneurysm features has emerged and is reviewed. We have illustrated these points with several examples demonstrating both features already described in the literature and novel cases demonstrating the potential for future clinical and research applications. The key vessel wall imaging findings by aneurysm type are outlined in Table 1.

**Saccular Aneurysms**

The most common form of intracranial aneurysm is the saccular type, present in nearly 3% of the general population. Currently, aneurysm size, location, history of prior rupture, hypertension, and ethnicity are the primary indicators used to direct treatment and predict future rupture. Additionally, evidence indicates that vessel wall structural and inflammatory change is associated with, and likely precedes, rupture.

Recent studies suggest that circumferential wall enhancement on HR-VWI can identify unstable or ruptured saccular aneurysms (Fig. 1). The pathological basis for vessel wall enhancement has been presumed to represent inflammation and/or proliferation of vasa vasorum. Hu et al. provide limited evidence for this, demonstrating lymphocyte and phagocyte wall invasion on histological analysis of 1 ruptured and 1 unruptured saccular aneurysm that both demonstrated wall enhancement, but other mechanisms of enhancement remain possible. Edjlali et al. reported that circumferential wall enhancement was present on HR-VWI in 27 (87%) of 31 unstable aneurysms—including 16/17 (94%) ruptured, 5/5 (100%) changing morphology, or 6/9 (66%) symptomatic—and 28.5% of stable (any other) aneurysms. Overall, 27 (55%) of all aneurysms (stable or unstable) that had enhancement were unstable while 55 (93%) of all aneurysms that lacked enhancement were stable. Hu et al. studied 30 aneurysms with HR-VWI with correlation to clinical status, reporting wall enhancement in 12 (100%) of 12 potentially unstable aneurysms, including 6/6 (100%) ruptured, 1/1 (100%) changing morphology, 4/4 (100%) with symptoms due to mass effect, and 1 with symptoms in the setting of a ruptured contralateral mirror aneurysm. Overall, 12 (86%) of
TABLE 1. Summary of typical vessel wall imaging findings and the clinical significance by aneurysm type

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<th>Aneurysm Type</th>
<th>Key Aneurysm Wall Imaging Findings</th>
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<tr>
<td>Saccular</td>
<td>Lack of enhancement on HR-VWI appears to be a strong indicator of an asymptomatic aneurysm w/ stable morphology. Limited data suggest that most symptomatic or morphologically changing aneurysms demonstrate circumferential wall enhancement. Nearly all ruptured aneurysms demonstrate circumferential enhancement or focal enhancement on HR-VWI.</td>
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| Multiple aneurysms in the setting of SAH | Several cases reported indicating the enhancement on HR-VWI usually identifies the culprit aneurysm.  
Aneurysm after endovascular treatment | Frequent enhancement that can temporarily increase after treatment and can persist for years. Enhancement can persist w/ or w/o a remnant. Enhancement is typically smooth & circumferential, but can be nodular. Most studies employ conventional-resolution MRI rather than HR-VWI. |
| Partially thrombosed          | An onion-skin pattern w/ peripheral T1 hyperintensity is common w/in the thrombosed portion. Absent wall enhancement on conventional-resolution MRI is associated w/ decreased growth rates. Decreased enhancement after treatment on conventional-resolution MRI is associated w/ aneurysm shrinkage. The intramural thrombosed component is well delineated from the lumen on HR-VWI. |
| Chronic fusiform              | Enhancement is frequent on conventional-resolution MRI & is associated w/ aneurysm growth. Intramural thrombus & peripheral T1 hyperintensity consistent w/ repeated intramural hemorrhage is associated w/ aneurysm growth. Current literature has employed conventional-resolution MRI. |
| Acute dissecting              | Degree of aneurysmal dilatation, intramural hematoma, & wall enhancement decrease w/ time w/ HR-VWI. Some aneurysmal dilatation & wall enhancement can persist in the chronic phase. |
| Blood blister                 | A focal hematoma may be identified on the outer surface in the setting of rupture on HR-VWI. Enhancement is variable but can be present in actively enlarging aneurysms based on scant data. |
| Oncotic                       | Variable appearance of thrombosed components and variable enhancement on HR-VWI; the significance of enhancement needs further study. HR-VWI delineates the lumen and thrombosed components in both treated & untreated aneurysms. |
| Mycotic                       | Circumferential enhancement on conventional-resolution MRI is reported in several patients w/ different underlying infectious etiologies; the significance of enhancement & patterns of enhancement on HR-VWI need further study. |

14 of aneurysms with wall enhancement were ruptured, growing, or associated with symptoms and only 2 (11%) of 18 aneurysms without these associated features demonstrated wall enhancement. There is conflicting evidence on the relationship of aneurysm wall enhancement and aneurysm size. Edjlali et al. found no relationship between enhancement and size in aneurysms ranging from 4 to 8 mm,11 and Hu et al. found no relationship between aneurysms ranging in size from 3.4 to 55.4 mm.22 In contrast, Liu et al. found that the frequency of wall enhancement is positively correlated with aneurysm size in 61 unruptured aneurysms ranging in size from 2.9 to 30.5 mm (odds ratio 2.46 per mm increase in size).33 The reasons for this lack of agreement are not entirely clear, although it could be explained by the inclusion of ruptured aneurysms that may demonstrate enhancement regardless of size in the studies by Edjlali et al. and Hu et al.11,22 compared with inclusion of only unruptured aneurysms in the study by Liu et al.33 There are only limited data assessing the significance of the magnitude of aneurysm wall enhancement. Naga-hata et al. found that only 4.8% of unruptured saccular aneurysms demonstrate strong enhancement and 82% demonstrated no enhancement.43 Two of 4 of these unruptured aneurysms with strong enhancement were considered unstable, 1 symptomatic, and 1 growing.43 Omakada et al. reported a higher degree of enhancement on HR-VWI measured quantitatively, but compared ruptured aneurysms to unruptured aneurysms in two pooled groups.48 By this method, the degree of enhancement is expected to be higher in the ruptured group partly due to a presumed higher percentage of aneurysms with wall enhancement and does not directly demonstrate that the degree of enhancement in ruptured aneurysms is greater than that in unruptured enhancing aneurysms. Additionally, peri-aneurysmal inflammatory change is expected to increase in the setting of rupture, which likely increased the magnitude of enhancement in these 2 studies compared with nonruptured aneurysms.

Beyond vessel wall enhancement, HR-VWI can characterize architectural details of aneurysms such as aneurysm wall thickness. Correlation of HR-VWI estimation and microscopic evaluation has shown that aneurysm wall thickness can be estimated on imaging when it exceeds imaging spatial resolution.54 However, aneurysm wall thickness is often below the spatial resolution of current vessel wall imaging techniques (0.02–0.5 mm).27 Kleinloog et al. conclude that the thickness of such thin-walled aneurysms can be estimated by the signal intensity on non-enhanced T1-weighted vessel wall images at 7 T, but only 2 aneurysms included in this study had histopathological confirmation of wall thickness.27 Such reliance of signal intensity on wall thickness would raise the possibility that the visually perceived degree of enhancement of the aneu-
Aneurysms wall varies with wall thickness, but this possibility has not been specifically assessed to our knowledge.

In summary, the current literature suggest that saccular aneurysms with wall enhancement on HR-VWI are frequently unstable (changing morphology, symptomatic, or ruptured) and that more urgent treatment may be reasonable whereas the preponderance of saccular aneurysms without wall enhancement appears to be stable and that less urgent treatment or observation may be reasonable. However, these assertions are based on retrospective data that include relatively large percentages of ruptured aneurysms, which selects for aneurysms with wall enhancement, with relatively fewer data on unstable unruptured aneurysms. HR-VWI features of unstable aneurysms may prove to be variable, because several histopathological forms of aneurysm walls associated with rupture are described with varying patterns of smooth muscle cell infiltration, wall thickness, presence of associated organizing luminal thrombus, and wall cellularity.

Aneurysms in the Setting of SAH

In addition to predicting natural history, HR-VWI can provide useful information in the setting of acute SAH. In a study of 61 patients with aneurysmal SAH, Nagahata et al. demonstrated that strong wall enhancement is 95.2% specific for rupture but with a sensitivity of only 73.8% and that any (strong or faint) wall enhancement was 98.6% sensitive, but had limited specificity of 81.9%. Interestingly, the authors found that focal strong enhancement at the aneurysm apex was associated with the point of rupture as noted during surgery in nearly 50% of such cases. This finding suggests that the mechanism of focal enhancement in ruptured aneurysms may involve physical disruption or a robust local inflammatory reaction at the rupture site.

The findings by Nagahata et al. raise the possibility that enhancement could help identify the ruptured aneurysm when multiple aneurysms are present. A case series including 3 patients with multiple aneurysms provides initial evidence that HR-VWI can identify the culprit aneurysm in the setting of acute SAH. This observation is important because the distribution of SAH is not highly predictive of aneurysm location. This information could also triage acute treatment toward the culprit aneurysm. Overall, it is not surprising that ruptured aneurysms demonstrate frequent enhancement, because inflammatory cells are isolated from ruptured aneurysms.

HR-VWI could potentially provide insight into the source of nonaneurysmal SAH as well. In a study of 11 patients with angiogram-negative nonperimesencephalic SAH, Coutinho et al. reported focal abnormalities on HR-VWI in the basilar artery wall in 2 patients (19%). This was postulated to represent a tiny thrombosed aneurysm,
loculated extramural blood, or a ruptured blood-blister aneurysm.

The utility in patients with a perimesencephalic pattern is less certain. The origin of perimesencephalic subarachnoid blood remains unknown, although it has been suggested it may result from a small thrombosed and obliterated aneurysm, basilar artery dissections, or venous variants. It is possible that HR-VWI could identify the site of a small aneurysm or subtle basilar artery dissection, and shed light on the mechanisms of perimesencephalic hemorrhages, but this remains undemonstrated. In fact, a series of 7 patients with perimesencephalic patterns had basilar arteries assessed at 7T, but the artery was normal in all cases without evidence of dissection or intramural hematoma.

The cumulative literature assessing HR-VWI in the setting of SAH shows that it can help identify a culprit aneurysm when multiple aneurysms are present, but there is not strong evidence to direct use in the setting of non-aneurysmal SAH.

Aneurysm Follow-Up After Endovascular Treatment

A few studies have evaluated the incidence of vessel wall enhancement on conventional-resolution MRI after endovascular treatment. Su et al. retrospectively studied the rate of wall enhancement and surrounding brain edema in 132 aneurysms after endovascular treatment. Eighty-five aneurysms (64.4%) had wall enhancement, which was significantly more likely with larger aneurysms or those that were embedded in the brain with surrounding edema. Interestingly, steroid use was not associated with a decreased rate of enhancement. While the association with coil packing density with vessel wall enhancement did not reach statistical significance in this study, an earlier study by this group reported that coil packing density was associated with a greater likelihood of vessel wall enhancement. This aneurysmal enhancement can either temporarily increase following treatment, presumably due to an inflammatory response to heal the aneurysm or to isolate the coils, or can be steady and consistent over time. After flow-diverter treatment, vessel wall enhancement may be observed with or without an aneurysm remnant and with or without prior rupture with prior coil embolization. We have observed that a non-thrombosed aneurysm after flow-diverter treatment with wall enhancement may progress to complete aneurysm occlusion (Fig. 2). McGuinness et al. reported that vessel wall enhancement was smooth and circumferential in 8 (36%) of 22 patients and focal and nodular in 3 (14%) of 22 patients with flow-diverter–treated aneurysms imaged 2–10 days after the procedure. These studies conclude that conventional-resolution MRI vessel wall enhancement is a common posttreatment finding, can persist for years, and should be considered an expected posttreatment finding. However, these studies did not include routine pretreatment MRI examination.
tions, and the rate and significance of pretreatment vessel wall enhancement remains undefined. Histopathological studies of coiled aneurysms reveal increasing inflammatory cell infiltration during the 1st month following coil embolization followed by aneurysm dome neovascularization, which could account for early and persistent enhancement, respectively. Although reports of HR-VWI findings in treated aneurysms are currently lacking, different aneurysms with near-contemporaneous treatment in the same patient may demonstrate different levels of wall enhancement on HR-VWI (Fig. 2).

**Partially Thrombosed Aneurysms**

Partially thrombosed aneurysms constitute a unique aneurysm class. The thrombosed component consists histopathologically of repeated subadventitial hemorrhages within the wall itself rather than the lumen and therefore demonstrates unique vessel wall findings on MRI. In contradistinction to saccular aneurysms, this class most commonly presents with symptoms due to mass effect.

On conventional-resolution MRI, the mural thrombus has been described as either homogeneous or heterogeneous, often demonstrating an “onion skin” pattern of thrombus of varying ages. Several reports described T1 hyperintensity in the peripheral thrombus, distant from the lumen, consistent with the mechanism that acute hemorrhage preferentially occurs in the subadventitia from the vasa vasorum. Martin et al. reported that thrombosed components consistently had stable signal intensities in 9 aneurysms followed over time, including peripheral T1 hyperintensity, which suggests a constant remodeling process with ongoing intramural hemorrhage.

Over time, the thrombosed portion may enlarge independently from the lumen, including with stable or decreased lumen size. The thrombosed component can also enlarge after complete luminal occlusion. Therefore, it is important to assess the size and characteristics of both the thrombosed portion and lumen during image interpretation. HR-VWI nicely depicts both the thrombosed and luminal components (Fig. 3). We have observed that aneurysm walls may demonstrate asymmetrical enhancement isolated to noncalcified regions, but the significance of this pattern is undetermined (Fig. 4).

Peripheral vessel wall enhancement has also been reported in the majority of partially thrombosed aneurysms using conventional-resolution MRI or CT. Roccatagliata et al. reported aneurysm wall enhancement in 63.2% of 22 aneurysms on conventional-resolution MRI and considered this to be a surrogate of inflammation. In a small series, Iihara et al. found that among 10 partially thrombosed aneurysms with wall enhancement, decreased enhancement in the postoperative setting in 5 cases (50%) was significantly associated with a reduction in aneurysm size. At the same time, 3 aneurysms with both absent wall enhancement and absent surrounding T2 FLAIR hyperintensity had decreased growth potential prior to treat-
Aneurysm vessel wall imaging

Chronic Fusiform Aneurysms

Intracranial fusiform aneurysms may be subclassified in several ways. Broadly, these may be categorized as atherosclerotic or nonatherosclerotic. While advanced atherosclerosis can result in a fusiform-shaped artery, nonatherosclerotic fusiform aneurysms are more common. Nonatherosclerotic fusiform aneurysms can be categorized as chronic or acute dissecting, which are discussed separately. The classification scheme is important because the findings on HR-VWI should differ by category.

The common histopathological feature of chronic fusiform aneurysms is damaged or frankly disrupted internal elastic lamina, which could be observed with or without intimal thickening or associated atherosclerotic plaque. Mizutani et al. identified distinct subtypes including segmental ectasia and chronic dolichoectatic dissecting aneurysms. Segmental ectasias are asymptomatic fusiform enlargements with stretched or fragmented internal elastic lamina without associated thrombus; these are believed to have a benign clinical course. Chronic dolichoectatic dissecting aneurysms demonstrate fragmented internal elastic lamina with intimal thickening and an organized thrombus; these frequently enlarge with time and are symptomatic.

Nakatomi et al. reported conventional-resolution imaging findings of 16 fusiform aneurysms, 8 with histopathological correlation. In this study, vessel wall enhancement was present in 5 (100%) of 5 aneurysms within histopathologically confirmed neoangiogenesis within the intima and was noted in 9 (56%) of 16 symptomatic aneurysms. All 8 aneurysms (100%) with intramural hemorrhage increased in size on serial examinations while all 8 aneurysms (100%) without intramural hemorrhage did not change in size. In vertebrobasilar aneurysms, T1 hyperintensity consistent with recent hemorrhage is associated with an increased likelihood of growth. Many other aneurysm features associated with growth have been identified, including intramural thrombus, the presence of daughter sacs, and overall size.

These vessel wall features associated with fusiform aneurysm enlargement are important because they demonstrate variable growth over time, generally have a poor prognosis, and are difficult to treat. Cumulatively, the literature has identified many vessel wall features on conventional imaging that can predict such growth, including important aneurysm wall features such as intramural thrombus and peripheral intramural T1 hyperintensity. Improved identification of these features could potentially prompt closer surveillance or treatment.

Acute Dissecting Aneurysms

Acute dissecting fusiform aneurysms result from acute disruption of the internal elastic lamina with subadventitial dissection. These may occur concurrently with subintimal dissection planes and areas of luminal narrowing. This most often affects the vertebrobasilar arteries, but can also occur at other locations including the internal carotid artery (ICA) and middle cerebral artery (MCA). This typically demonstrate an entry-point communication between the pseudolumen and parent vessel lumen. The dissection may end blindly within the pseudolumen or less commonly demonstrate a second exit-point back to the lumen. This distinction is important because dissecting aneurysms that have a second exit point to the lumen have a more stable clinical course.

Initial studies of HR-VWI of intracranial dissection have shown that features of dissection including an intimal flap, double lumen, and intramural hematoma can be identified, including cases with dissecting aneurysms. Both the wall of the lumen and outer margin of the artery can enhance; a small portion of intramural hematomas also reportedly enhance. Wang et al. identified areas suggestive of disruption of the outer margin of the affected artery in 2 (33%) of 6 patients studied with SAH. Park et al. retrospectively studied 41 intracranial vertebral artery dissections with serial HR-VWI to characterize the imaging features over time. This study found a decrease in...
aneurysmal dilation, intramural hematoma signal intensity, and degree of wall enhancement over time. However, aneurysmal dilation and mild wall enhancement persisted in 4 (44%) and 7 (78%) of 9 patients studied in the chronic phase, respectively. Overall, these studies indicate that HR-VWI can identify and help distinguish recent from chronic dissecting aneurysms.

**Blood-Blister Aneurysms**

Blood-blister aneurysms are small, broad-based, hemispherically shaped aneurysms that occur along the dorsal surface of the supraclinoid ICA opposite the origin of the posterior communicating artery. These are very fragile aneurysms and the majority present with SAH. Ishikawa et al. reported such an aneurysm on autopsy at the site of a disrupted internal elastic lamina and tunica media with a thin covering consisting of only fibrous tissue and adventitia. Ishikawa et al. did not identify a dissection, although other authors have postulated that a dissection is the initiating event. Blood-blister aneurysms are sometimes initially angiographically occult and require second-look angiograms for identification.

Horie et al. report a case of HR-VWI used to detect an angiographically occult blood-blister aneurysm. While an abnormality was not identified in the vessel wall itself, a focal T1 hyperintense thrombus was found on the immediate outer surface of the aneurysm. In our experience, vessel wall enhancement can be absent in blood-blister aneurysms on HR-VWI (Fig. 5). Lack of vessel wall enhancement is not surprising with this type of aneurysm given the very thin wall. However, we have also observed wall enhancement in an actively enlarging blood-blister aneurysm (Fig. 6). Because such information is currently based on personal observation and scant data, future HR-VWI investigations assessing the relationship between enhancement and stability are needed to direct use and interpretation in clinical practice.

**Myxomatous and Other Oncotic Aneurysms**

The imaging and clinical features of oncotic aneurysms vary by underlying neoplasm. While the exact etiology is still debated, there is strong evidence that direct implantation of tumor emboli into the vessel wall itself is the initial event. The imaging and clinical features of oncotic aneurysms vary by underlying neoplasm.
Myxomatous aneurysms resulting from embolic tumor particles in patients with cardiac myxoma are the most common subtype of oncotic aneurysm. Histological examination has revealed subintimal growth of tumor and vessel wall disruption. These are typically multiple, fusiform, and involve distal cerebral artery branches and can present concurrently with an atrial myxoma years after resection. These aneurysms are typically slow-growing and difficult to treat, requiring long-term imaging follow-up.

Prior studies on the imaging appearance of myxomatous aneurysms have evaluated the luminal characteristics, but there is little information on the imaging characteristics of the aneurysm walls that harbor the primary pathology. In our experience with HR-VWI, the aneurysm wall appearance demonstrates considerable variation in enhancement patterns and in the presence and appearance of the thrombosed component (Fig. 7). The precise size and morphology of both the lumen and thrombosed components are better depicted on HR-VWI than on conventional-resolution MRI. HR-VWI can also precisely delineate size and mass-effect of thrombosed portions of the aneurysm after treatment.

**Mycotic Aneurysms**

Cerebral mycotic aneurysms may result from various microorganisms including viruses, bacteria, mycobacteria, parasites, and fungi. Immunocompromised status is a risk factor, but these may also develop in immunocompetent patients. In general, these aneurysms have a tendency to be peripheral, multiple, and fusiform, but irregular, dissecting, or saccular aneurysms are reported. The primary pathology includes fibroinflammatory tissue and arterial wall destruction, which can lead to contained pseudoaneurysm or rupture. CNS varicella zoster virus (VZV) vasculopathy can accompany either primary or reactivation zoster infection. VZV vasculopathy may be observed in immunocompromised patients with or without a skin eruption, including those with human immunodeficiency virus (HIV) or those undergoing high-dose steroid treatment. VZV aneurysms are uncommon, but are important to characterize because they have the potential to rupture. HR-VWI has been evaluated in a small number of patients with CNS VZV vasculopathy, but not specifically in patients with VZV aneurysms.

VZV aneurysms may demonstrate complex shapes, develop rapidly, and variably resolve with antiviral and anti-inflammatory medications. In our clinical experience, we have observed peripheral enhancement in multiple aneurysms in an immunocompromised patient with presumed VZV vasculopathy on conventional-resolution MRI (luminal imaging findings reported previously Daugherty et al.; Fig. 8).

Aneurysm formation in the HIV setting is incompletely understood, but may be due to a variety of infections such as VZV or may be attributed to HIV itself.
are more common with low CD4 counts and high viral loads. O’Charoen et al. report peripheral enhancement on conventional-resolution MRI in a cerebral aneurysm attributed directly to HIV vasculopathy. Inflammation may depend on immune status as histopathological evaluation of aneurysms from pediatric patients with HIV have revealed little to no inflammation.

The most common culprits of bacterial mycotic aneurysms are *Staphylococcus* or *Streptococcus* species; response to antibiotics is variable and currently unpredictable. Cerebral bacterial mycotic aneurysms may occasionally mimic a berry aneurysm on conventional imaging methods, and blood cultures are sterile in 18%–50% of patients. By analogy, periaortic enhancement is variably reported in mycotic aortic aneurysms.

Overall, the frequency and clinical significance of aneurysm wall enhancement in the various types of mycotic cerebral aneurysms in either immunocompromised or immunocompetent patients is not fully defined on conventional-resolution MRI and is essentially unexplored on HR-VWI.

**Future Directions and Conclusions**

The biological behavior of the many types of intracranial aneurysms varies widely and is largely dependent on differences in vessel wall pathology including inflammation, neoangiogenesis, intramural hemorrhage, tumor implantation, and physical disruption. HR-VWI offers the potential to identify imaging surrogates of these vessel wall features and to therefore identify imaging biomarkers of aneurysm behavior.

HR-VWI will characterize aneurysm walls in greater in-vivo detail than previously possible and may complement forms of luminal imaging. Although some data on HR-VWI of aneurysms exist, such data are far less abundant than current data grossly characterizing aneurysm walls with conventional MRI or histopathology for many types of aneurysms. The potential exists to build upon this current knowledge with HR-VWI, and future studies of the utility of aneurysm wall findings to direct medical or interventional treatments are needed. It is important to recognize that, while retrospective data evaluating intracranial aneurysms with HR-VWI are emerging, future prospective studies are needed to establish the histopathological basis for imaging findings, assess the ability to predict biological behavior, compare the diagnostic utility with conventional imaging, and determine the impact on patient outcome for the various types of aneurysms discussed herein.
The current understanding of aneurysm wall findings on HR-VWI is evolving; we hope this review of the current knowledge of HR-VWI, conventional-resolution imaging, and key histopathology of aneurysm walls can help guide current clinical practice and future research.

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