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High-resolution intracranial vessel wall imaging: imaging beyond the lumen

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Abstract

Accurate and timely diagnosis of intracranial vasculopathies is important due to significant risk of morbidity with delayed and/or incorrect diagnosis both from the disease process as well as inappropriate therapies. Conventional vascular imaging techniques for analysis of intracranial vascular disease provide limited information since they only identify changes to the vessel lumen. New advanced MR intracranial vessel wall imaging (IVW) techniques can allow direct characterisation of the vessel wall. These techniques can advance diagnostic accuracy and may potentially improve patient outcomes by better guided treatment decisions in comparison to previously available invasive and noninvasive techniques. While neuroradiological expertise is invaluable in accurate examination interpretation, clinician familiarity with the application and findings of the various vasculopathies on IVW can help guide diagnostic and therapeutic decision-making. This review article provides a brief overview of the technical aspects of IVW and discusses the IVW findings of various intracranial vasculopathies, differentiating characteristics and indications for when this technique can be beneficial in patient management.

INTRODUCTION

Traditionally, intracranial vascular diseases have been evaluated with luminal imaging techniques, whether traditional catheter angiography or noninvasive luminal imaging techniques (MR angiography, MRA or CT angiography CTA). However, these angiographic techniques can only identify abnormalities affecting the vessel lumen, and many cerebral

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vasculopathies can have similar luminal imaging appearances.[1–4] In recent years IVW techniques have been developed to directly characterise disease processes affecting the walls of arteries.[4–7] Intracranial vasculopathies can require dramatically different treatments and a delay in treatment or inappropriate therapy can result in significant morbidity and even mortality. MR intracranial vessel wall imaging (IVW) can provide diagnostic information not available with traditional angiographic techniques that can assist clinicians planning appropriate treatment. A previous review article focused on the technical aspects and implementation of IVW with a broad discussion of imaging findings as they relate to stroke subtype and plaque imaging characteristics.[8] This review article focuses on a comprehensive overview of the IVW imaging findings of various intracranial vasculopathies, differentiating characteristics that may aid clinicians in diagnosis and indications for when this technique can be beneficial in patient management.

INTRACRANIAL MR VESSEL WALL IMAGING PROTOCOL DESIGN

IVW, as compared to extracranial vessel wall imaging, is more challenging due to the small caliber and tortuous course of the intracranial arteries. For example, the middle cerebral artery (MCA) diameter can range from 3 to 5 mm, with a vessel wall thickness from 0.5 to 0.7 mm.[9] The smallest practical voxel is necessary to be able to depict the normal vessel wall and differentiate pathological states, and with larger voxels there is increased likelihood of inaccurate measurements.[7] While some protocols have used 1.5 T scanners, because of the need for very high resolution and thus higher signal, higher field strengths are optimal, preferably at least 3 T, with reports demonstrating improved results with 7 T with utilisation of a magnetisation prepared inversion recovery sequence with PD, T1 and.[10–12] While a majority of the studies in the literature have used two-dimensional (2D) black blood imaging techniques, there has been recent application of three-dimensional (3D) IVW techniques.[26713–21] 3D acquisitions allow for improved through plane resolution, with increased brain coverage, and can allow for isotropic resolution which can be reformatted in multiple planes. This is advantageous for interrogating the intracranial arteries due to their inherent small size and tortuous course. Many of the current 3D IVW protocols, however, utilise non-isotropic resolution, which can also be reformatted, but may result in suboptimal image quality due to blurring. 3D variable refocusing flip angle (VRFA) sequences (VISTA, Philips Healthcare, Best, the Netherlands; SPACE, Siemens Healthcare, Erlangen, Germany; CUBE, GE Healthcare, Milwaukee, Wisconsin, USA) have been the most extensively used and studied 3D techniques to date, as these sequences provide improved image quality, coverage and blood flow suppression in a shorter scan time relative to conventional 3D and 2D imaging techniques.[7132223] VRFA techniques have been used with T1 and proton density (PD) weightings, before and after gadolinium contrast administration, as the pattern and degree of contrast enhancement can be helpful in differentiating and characterising vasculopathies.[27] High-resolution T2-weighted imaging has also shown some promise for IVW. While a majority of studies have investigated enhancement characteristics of vasculopathy, Mossa-Basha *et al*[24] showed that a multicontrast protocol including T1 pre and postcontrast and T2 IVW improves accuracy of vasculopathy differentiation. In addition, there was very strong to substantial inter-reader agreement for assessment of IVW characteristics, including the assessment of lesional T2 signal (κ=0.8), pattern of wall

thickening (κ=0.87), presence (κ=0.9), pattern (κ=0.73) and intensity (κ=0.77) of enhancement.

2D imaging techniques that have been employed for IVW include T1, T2 and PD-weighted sequences. There are multiple publications that have primarily performed 2D imaging in a plane perpendicular to plane of the lumen, which is likely the most important plane of imaging, however, multiplanar imaging is beneficial for optimal lesion assessment, performing axial imaging and imaging in a plane perpendicular to the axis of the interrogated artery. This allows for more complete visualisation of the lesion, assessment of its effects on the lumen and lesion morphology. Obtaining imaging in a plane perpendicular to the lumen is important to avoid volume averaging effects and to obtain a more accurate estimate of lesion and wall thickness. Vessel obliquity, slice thickness and in-plane resolution are all factors that will affect wall measurements and the sharpness of the vessel wall borders.[25] Cardiac gating may be performed for 2D IVW, though it is not frequently used.[7]

Intracranial vessel wall imaging protocols can be performed in a time efficient manner. 3D PD-weighted VRFA sequence (0.4–0.5 mm^3 isotropic voxels) with coverage of the major intracranial arteries can be performed on a 3 T system in 7–8 min.[7] Thus a full protocol, including time-of-flight MR angiography (TOF MRA) for localisation and PD VISTA pre and post contrast can be performed in under 30 min. As MRI scanners improve in efficiency and field strength, coil elements improve and compressed sensing techniques become more efficient, sequences will become shortened and allow for improved coverage[26] (refer to table 1 for the IVW MR protocol used at our institution).

INTRACRANIAL ATHEROSCLEROTIC DISEASE

Intracranial atherosclerotic disease (ICAD) is the root cause of a significant portion of ischaemic strokes. In the USA, it has been shown to account for up to 9–15% of ischaemic strokes, although this percentage approaches 50% for people of Asian or African descent.[27–30] ICAD is presumed to represent the most common cause of ischaemic strokes worldwide.[31] ICAD is one of the most commonly investigated intracranial vascular pathologies in studies utilising IVW and demonstrates the utility of such techniques.[2111432–41] An example of an ICAD lesion on IVW can be seen in figure 1.

While utilising vessel wall imaging to investigate extracranial carotid disease is a relatively mature field, the extent of research on ICAD is smaller. Whereas previous approaches to ICAD assessment considered the degree of plaque-related stenosis, IVW directly evaluates plaque characteristics, starting with the degree and pattern of wall thickening at the site of disease.[42] The histological components of ICAD are similar to those seen in extracranial carotid disease, and preliminary IVW studies have also assumed similar imaging characteristics of the various atherosclerotic features.[43] Preliminary studies correlating in vivo or ex vivo IVW to histology have shown good agreement between plaque MR signal characteristics and lesion components on histology, including intraplaque haemorrhage, lipid rich necrotic core, fibrous cap and fibrous tissue.[44–47] ICAD, similar to atherosclerosis in other vascular beds, frequently initially grows outward, and can reach prominent size before

resulting in detectable stenosis.[48] For this reason, luminal imaging frequently underestimates the burden of intracranial atherosclerosis, and may not detect non-stenotic lesions, which can be symptomatic.[334041] A French autopsy study showed that 62% of patients who died from ischaemic stroke had ICAD. Of the 200 patients with ICAD, only 126 were found to have obvious stenosis (>30%) with conventional luminal imaging, suggesting 37% of all ICAD plaques cannot be easily assessed by angiographic imaging.[49] Some studies have indicated that plaques that show positive remodelling (outer wall remodelling) are more frequently symptomatic than negative remodelling plaques and have a higher association with microemboli detected on transcranial Doppler.[343842] The degree of stenosis and ratio of wall thickening to lumen were also found to be significantly associated with symptomatic plaques when compared to asymptomatic lesions.[42]

Wall thickening alone is not a good predictor of symptoms; wall signal characteristics must be examined.[23738] Atherosclerotic lesions frequently have a juxtaluminal T2 hyperintense band, which represents the fibrous cap.[3848] This juxtaluminal band will also appear isointense to hyperintense on PD sequences.[48] Histological comparison of ICAD to in vitro 7 T MR IVW has confirmed the presence of the fibrous cap which was found to have hyperintense signal on 3D VRFA MR sequences.[45] Underlying the juxtaluminal band is a lipid-rich necrotic core, which is isointense on T1WI and hypointense on PD and T2WI.[48] ICAD lesions will frequently have heterogeneous T2 IVW signal (figure 1C).[24] Increased size of the lipid core has been associated with higher rates of rupture.[3842] In atherosclerosis of the extracranial carotid arteries, intraplaque hemorrhage (IPH) has proven to be one of the most important risk factors for plaque vulnerability.[50] With ICAD, IPH has also been shown to occur in preliminary studies and to be associated with symptoms, though more investigation is necessary.[363743] IPH will appear as intraplaque T1 hyperintensity, which is greater than 150% the signal intensity of the internal reference muscle tissue.[3637] Presence of ICAD IPH is associated with higher rates of plaque complications and stroke, similar to what is seen in extracranial carotid artery plaques.[3743] Intracranial atherosclerotic plaques are frequently eccentric in morphology and often will have incomplete postcontrast enhancement (figure 1D, E).[424] ICAD can, however, have a circumferential pattern of vessel wall involvement, mimicking vasculitis. In this setting, evaluating the T2-weighted IVW can be helpful for differentiation.[24] Enhancement has been shown in plaques upstream to ischaemic infarcts within the first 4 weeks after the stroke, and with the passage of time, the degree of enhancement progressively diminishes.[35] In patients with multifocal disease, culprit lesions consistently enhance, and they do so more avidly than non-culprit lesions, which enhance inconsistently.[14] Plaque enhancement may relate to inflammation and/or ingrowth of vasa vasorum. It is important to note the role for vasa vasorum in the initiation of an inflammatory cascade that contributes to atherogenesis development and propagation.[51] The radial location of plaques along the MCA has been shown to be important as plaques along the superior wall of the MCA are associated with deep infarcts, presumably due to ostial stenosis or occlusion at the origins of lenticulostriate perforators.[39]

IVW has not only shed light on ICAD features associated with ischaemic symptoms, it is providing new insights that are changing our understanding of the natural history of this disease. Traditional schema that determined treatment algorithms for ICAD has depended on degree of stenosis. The Warfarin-Aspirin Symptomatic Intracranial Disease (WASID) trial

demonstrated a roughly linear relationship between degree of stenosis and rates of ischaemic stroke, with an apparent asymptote at approximately 90% of stenosis.[52] Stenosis of 70–99% then became a cut-off for endovascular treatment of refractory ICAD lesions and was used as an inclusion criterion in the only randomised clinical trial evaluating, but failing to show the effectiveness, of intracranial stenting.[53] However, current IVW techniques show that stenosis may not be appreciated with atherosclerotic lesions. Intracranial IVW has confirmed coronary artery findings that initial vessel wall remodelling can maintain the lumen while the wall itself dramatically thickens.[345455] Such expansive remodelling is associated with vulnerable plaque characteristics, higher rates of plaque rupture and acute coronary syndrome and similar early ischaemic pathology is now being seen in ICAD.[3438425455] There are multiple possible pathophysiological routes from ICAD to stroke. These include hypoperfusion, plaque rupture and thrombus formation leading to artery-to-artery embolisation, and occlusion of a vessel by a plaque or plaque-associated thrombosis.[56] Lesions, however, may be asymptomatic despite high-grade stenosis, most likely due to long-term luminal narrowing resulting in protection via collateralisation. With this new understanding of ICAD, it now appears possible that prior investigations of ICAD therapies have not been adequately targeting the lesions most likely to cause symptoms, particularly when determining which lesions should be targeted for endovascular therapy. Considerable future investigation is needed to examine the roles of plaque components during the mildly stenotic or non-stenotic positive remodelling phase of ICAD. IVW can provide a better imaging marker for ICAD for future therapeutic trials than luminal imaging techniques that have been used.[57] This could lead to improved treatment algorithms that identify which patients should undergo endovascular interventions in addition to aggressive medical management. Finally, in candidates for endovascular therapy, specific features of individual lesions can be identified that may impact planning for such interventions, such as proximity to ostia of larger branch vessels and perforators.[394058]

VASCULITIS

IVW can differentiate intracranial vasculopathies that look nearly identical on catheter and cross-sectional angiography, including vasculitis, RCVS, ICAD, vasospasm, infection and radiation-related vasculopathy.[5960] Nearly all causes of vasculitis, whether autoimmune or infectious, can affect the cerebral arteries.[61] Typically vasculitides are imaged with angiographic methods, but findings of multifocal luminal narrowing are nonspecific. In addition, catheter angiography has limited sensitivity to central nervous system (CNS) vasculitis especially when considering small artery vasculitis such as lymphocytic or granulomatous subtypes.[62] The sensitivity of catheter angiography for changes related to CNS vasculitis ranges from 27% to 90%,[6364] with specificity that can be as low as 30%.[60]

The morbid natural history of these vasculitides necessitates prompt, accurate diagnosis. While systemic vasculitides can be diagnosed by biopsy of peripheral arteries or tissues, biopsy of cerebral vasculitis is a riskier undertaking. In addition, the sensitivity of biopsy for the diagnosis of CNS vasculitis may be lower than 50%.[65] IVW directly visualises vessel wall inflammation and oedema. Küker *et al*[66] evaluated 27 patients with a diagnosis of CNS vasculitis using IVW, and found that 25/27 showed vessel wall thickening and 23/27 showed postcontrast vessel wall enhancement. Vasculitis typically shows thickening and multifocal

or diffuse homogeneous, smooth, concentric enhancement of the vessel wall (figure 2A–D).[366–68] A majority of cases of vasculitis showed stable degrees and extent of vessel wall enhancement on follow-up.[68] Vasculitic enhancement seen on postcontrast MRI may also extend beyond the vessel wall into adjacent periadventitial tissues, increasing conspicuity of the affected segments. Parenchymal enhancement that may be seen could represent microvascular involvement that is angiographically occult with inflammatory spillover into the surrounding tissues.[69] On T2-weighted IVW, vasculitis typically shows homogeneous vessel wall signal that is isointense to grey matter.[24] With advances in IVW, findings diagnostic for vasculitis may help guide or potentially limit the need for biopsy.[7071] However, IVW cannot differentiate between the types of vasculitis, and thus the necessity for fluid or tissue sampling may remain for definitive diagnosis. With treatment, the degree and extent of vascular enhancement will diminish; however, the extent of luminal stenosis may not appreciably change.[72]

Important caveats bear noting for this application of IVW. Perivascular enhancement can be found normally at baseline in children. Such normal enhancement is typically linear and thin in appearance, often symmetric with the contralateral vessels and typically not present in vessels coursing through cerebrospinal fluid.[73] Enhancement patterns mimicking those seen in vasculitis can be seen shortly after mechanical thrombectomy for the treatment of acute ischaemic stroke.[74] In addition, steroid therapy can affect vasculitis-related vessel wall enhancement, diminishing the degree of abnormality.[72]

REVERSIBLE CEREBRAL VASOCONSTRICTION SYNDROME

Patients with reversible cerebral vasoconstriction syndrome (RCVS) present with a sudden, severe, intermittent headache, at times with concomitant neurological deficits.[7576] Clinical history is often instructive, with young to middle-aged women most commonly afflicted in the setting of one or more of many known inciting factors.[7576] Known risk factors are often present, such as ecclampsia, postpartum status, strenuous activity, bathing or showering, recent use of tobacco, amphetamine, stimulants, cannabis, ethanol or selective serotonin reuptake inhibitors.[76] Possible complications of RCVS include infarction, subarachnoid haemorrhage, parenchymal oedema or intraparenchymal haemorrhage. Clinical evaluation and diagnosis can be performed difficult due to patients presenting in critical condition and unable to relay an accurate history due to the aforementioned disease complications. In this setting, differentiation of RCVS from other vasculopathies based on clinical and luminal imaging can be difficult. IVW studies show minimal smooth thickening of vessel wall that is concentric with mild to no enhancement (figure 2E–I).[24676877] This is consistent with the pathophysiological process leading to RCVS, specifically non-inflammatory vasospasm. There is near uniform resolution of these findings within 3 months of symptom onset.[6876]

MOYAMOYA

Moyamoya disease (MMD) is an idiopathic disease causing progressive narrowing of the bilateral carotid termini, leading to development of abnormal collateral branches at the base of the brain. In Japanese patients, MMD affects patients in a bimodal distribution, with peaks at 5 years and mid-40 years of age. MMD will affect both carotid termini, however,

the severity of disease between each side may differ. In addition, disease manifestation may not be simultaneous. In Moyamoya syndrome (MMS) there may be unilateral or bilateral supraclinoid internal carotid artery stenosis from one of several disease states, including ICAD, inflammatory vasculopathy, radiation therapy, sicklecell disease, neurofibromatosis type 1, trisomy 21 or other genetic syndromes.[78] Distinguishing between these two processes is important, because symptomatic MMD can be treated with external carotid to MCA bypass procedures, whereas some of the underlying processes causing MMS are potentially reversible with initial medical management.[78]

On IVW, Ryoo *et al*[15] showed concentric vessel wall thickening and enhancement of the distal internal carotid arteries in the setting of MMD, while others have indicated a lack of wall enhancement in the setting of MMD (figure 3).[14] MMD has been described as showing wall shrinkage,

DISSECTION

Intracranial arterial dissections (IAD), while still considered relatively rare, are an important cause of ischaemic stroke and subarachnoid haemorrhage, especially in younger patient populations.[79] The aetiology of IAD is unknown in most cases, but predisposing factors include hypertension, history of migraine headaches, oral contraceptives, genetic factors, trauma, as well as inflammatory/infectious states.

Dissections can be subintimal, subadventitial or both. When blood collects between the media and internal elastic lamina via an intimal tear, this is termed a subintimal dissection; a subadventitial dissection occurs when the haematoma extends through the media.[79] Subintimal dissections frequently lead to hypoperfusion or ostial occlusion and thromboemboli, resulting in ischaemia.[8081] Intracranial subadventitial dissections frequently will result in subarachnoid haemorrhage, and may maintain luminal contours, making them difficult to detect on luminal imaging. Subadventitial dissections can also result in pseudoaneurysm or giant partially thrombosed aneurysm formation, which can result in compression of the brainstem or cranial nerves.[79] The most common locations for intracranial dissections include the supraclinoid ICA, the mainstem M1 MCA and the intracranial vertebral arteries.

Luminal imaging can be performed for the evaluation of intimal dissections, however, these techniques are not sensitive for the detection of subadventitial dissections that may maintain luminal contours.[82] T1-weighted imaging with fat saturation often can demonstrate thrombus within the false lumen of a dissection, but IVW techniques with improved resolution and dark blood imaging can improve characterisation and detection.[21783–85] Additionally, dissections can be further evaluated with postcontrast imaging that may show eccentric enhancement.[6] 3D IVWhas been shown to better characterise and detect vertebrobasilar dissections than both MRA and 2D spin echoMRI techniques, while suffering only minimal flow artefacts that did not affect diagnostic assessment.[1617] 3D IVW techniques afford a number of advantages for the detection of dissections relative to 2D fat-saturated T1 sequences, specifically, improved isotropic resolution for detection of small

dissections and multiplanar reformations, improved suppression of flow artefacts that can obscure or mimic dissections, and no need for cardiac gating for flow suppression.[84]

CEREBRAL ANEURYSMS

IVW is adding to our understanding of the factors that cause aneurysm growth. Vasa vasorum is not normally present in the cerebral vasculature until the development of pathology like ICAD or aneurysm (saccular aneurysms >4 mm in diameter, giant and fusiform aneurysms).[6] One difficulty with histological vasa vasorum investigation, however, is adventitial stripping with autopsy sampling, especially in smaller intracranial arteries, may result in underestimation of the presence of vasa vasorum.[51] The vasa vasorum may leak and cause haemorrhage into the wall of an aneurysm, inciting an inflammatory cascade that includes the release of growth factors, leading to aneurysmal enlargement and wall thinning, which may lead to rupture. IVW can identify the vasa vasorum, the aneurysm wall itself, and any mural haematoma.[6] IVW can also accurately delineate the relationship between aneurysms and adjacent anatomic structures.[86] High-resolution techniques can accurately measure wall thickness as long as the wall thickness is above the imaging resolution threshold.[86–89] MR measurements showed progressive overestimation relative to histological measurements with decreasing arterial wall thickness.[8889] Sherif *et al*[89] defined the imaging threshold to be the in-plane voxel size (0.4 mm) and indicated significant differences between true wall thickness and MR wall thickness in measurements smaller than this imaging threshold (0.24±0.06 mm for true aneurysm dome wall thickness vs 0.30±0.068 mm for MR wall thickness (p=0.0078)). The imaging resolution threshold for accurate wall measurement depends on imaging parameters, including in-plane and through-plane resolution and orientation of the vessel wall and lumen being evaluated.[25] The evaluation by Sherif *et al* was performed with 2D IVW, and has yet to be investigated using 3D isotropic techniques that would result in more accurate wall measurements from reduced volume averaging and wall distortion effects. In the setting of multiple aneurysms with acute subarachnoid haemorrhage, thick aneurysm wall enhancement (figure 4) helps identify the culprit lesion.[90] IVW may help in identifying an aneurysm as the source of subarachnoid haemorrhage that is angiographically occult.[91] T1, T2-weighted and steady state free precession MRI techniques can also identify and characterise aneurysmal intraluminal thrombus.[92] Such early demonstrations of the feasibility of IVW for aneurysms may now lead to further investigation to identify which characteristics are predictive of aneurysm rupture, allowing neurointerventionalists and microvascular neurosurgeons offering open surgical techniques more information to determine when and what type of treatment is appropriate.[56]

IVW FOR THE DIFFERENTIATION OF VASCULOPATHIES

The differentiation of intracranial vasculopathies using luminal imaging techniques can be difficult, and this is an area of promise for IVW imaging. Table 2 provides a summary of vessel wall imaging characteristics of the various intracranial vasculopathies. Multiple vessel wall characteristics have been utilised for disease differentiation, including presence of contrast enhancement, pattern of wall involvement (eccentric vs circumferential), pattern of enhancement, presence of remodelling and T2 signal characteristics. Mossa-Basha *et al*[24]

evaluated the ability of these IVW characteristics to differentiate intracranial atherosclerosis, vasculitis and RCVS using a multicontrast protocol including T1-weighted pre and postcontrast and T2-weighted high-resolution techniques. A majority of atherosclerosis cases showed eccentric vessel wall involvement, varying degrees and patterns of enhancement and mixed T2-weighted lesion signal intensity. Vasculitis on the other hand typically showed a circumferential lesion with diffuse grade 1 or 2 enhancement and isointense T2 lesion signal. RCVS lesions typically showed circumferential vessel wall involvement with no to mild enhancement and mild isointense wall thickening on T2-weighted IVW.

ICAD can, however, have a circumferential pattern of vessel wall involvement, mimicking vasculitis and RCVS.[2477] In this setting, evaluating the T2-weighted IVW can be helpful for differentiation.[24] On T2-weighted IVW, vasculitis will differ in appearance from ICAD and typically shows homogeneous vessel wall signal that is isointense to grey matter, as compared to the heterogeneous T2 wall signal seen in ICAD and the mild wall thickening in RCVS. In addition, ICAD frequently will show outward remodelling, a characteristic not described in RCVS and vasculitis.

While differentiation of RCVS from CNS vasculitis using luminal imaging is nearly impossible, IVWcan readily differentiate between the two disease states.[6768] Mandell *et al*[67] found that RCVS showed no vessel wall enhancement and resolved on follow-up luminal imaging, while cerebral vasculitis showed multifocal enhancement associated with the regions of stenosis with persistence of luminal narrowing on follow-up luminal imaging. Obusez *et al*[68] found that most cases of RCVS showed negligible to mild enhancement and circumferential wall thickening that frequently resolved, whereas circumferential thick wall enhancement was seen in most cases of vasculitis. Dieleman *et al*[77] described a case of RCVS which showed circumferential enhancement, however, the degree and extent of enhancement was not described. The presence and degree of enhancement are the best criteria to differentiate RCVS from ICAD and vasculitis, however, the T2-weighted vessel wall appearance of RCVS typically shows subtle mild T2 isointense wall thickening, which also differs from other intracranial vasculopathies.[24]

Angiography does not distinguish between MMD and MMS well, with both processes demonstrating high-grade stenosis of the carotid termini with robust collateralisation. Ryoo *et al*[15] showed concentric vessel wall thickening and enhancement of the distal internal carotid arteries in the setting of MMD, while others have indicated a lack of wall enhancement in the setting of MMD (figure 3).[14] In addition, MMD will not show outer wall remodelling and expansion, in distinction from atherosclerosis. MMS caused by ICAD will show the findings described in the atherosclerosis section, most notably eccentric plaques often showing positive remodelling and enhancement.[14] MMS caused by vasculitis will have concentric mural enhancement.[36667]

IVW can help differentiate disease states when luminal imaging and clinical markers are inconclusive. This can be especially helpful in differentiating RCVS, ICAD and vasculitis or in differentiating MMD from MMS. There can exist overlap in imaging appearances in terms of pattern of wall involvement and presence of enhancement, but other characteristics

including outer wall remodeling and T2-weighted IVW signal characteristics can provide additional differentiating data.

LIMITATIONS OF IVW IMAGING

While much promise has been shown with IVW for the evaluation and differentiation of intracranial vasculopathies, further investigation is necessary. There are only a small number of studies with histological validation of ex vivo IVW imaging findings of ICAD,[4547] and a handful of studies with correlation of IVW findings with histological or cerebrospinal fluid analysis of inflammatory vasculopathies,[66–68709394] surgical confirmation of aneurysm rupture[90] or aneurysm wall thickness.[8689] This is primarily due to limited availability of histological specimens for most intracranial vasculopathies. Further prospective, longitudinal investigation of outcomes relating to specific vessel wall imaging lesion characteristics and particular diseases as they correlate to vulnerability and risk of stroke is necessary to promote this field of clinical and investigation considering the aforementioned limitations with histological confirmation. Considering the need for high-resolution and adequate signal-to-noise for the evaluation of small, tortuous arteries, IVW scans can be time-consuming, which can be a limiting factor for wide use due to its impact on patient throughput, limited quality due to patient motion[95] and patient discomfort. With software and hardware improvements going forward, improved resolution should be attainable within shorter scan times, making these examinations more clinically feasible for all imaging environments. The benefit of using higher field MRI systems, typically with at least 3 T field may limit the ability to perform these examinations for certain institutions.

FUTURE CONSIDERATIONS

As refinement and validation of IVW techniques continue, these tools can serve a complementary and additive role to luminal imaging techniques and become more routine in the evaluation and differentiation of the above-described cerebrovascular pathologies. In certain disease processes, with further investigation, IVW may be able to identify lesions with a poorer prognosis that may require earlier or more aggressive treatment. IVW may allow for detection of disease prior to changes seen on luminal imaging. Newer techniques can be implemented for improved evaluation of intracranial atherosclerotic plaque characteristics including intraplaque haemorrhage, outer wall boundary detection, and simultaneous acquisition of luminal and vessel wall imaging.[181996]With improved CSF suppression using preparation modules such as delay alternating with nutation for tailored excitation (DANTE), improved assessment of normal and diseased arterial segments can be achieved on multiple contrast weightings, allowing for increased acceptance of multicontrast protocols that will permit improved disease characterisation.[1820] In addition to CSF suppression, DANTE suppresses flowing blood without any effect on tissue contrast, thus limiting flow artefacts that may be confused with vessel wall pathology or intraluminal thrombus. Considering the requirement of high-resolution imaging for IVW, in order to attain adequate signal constrictively long sequences may be employed. With the utilisation of advanced parallel imaging and compressed sensing techniques that can further accelerate imaging without loss of image quality through more efficient k-space undersampling, shorter scan times that are clinically feasible may be attainable.[97–99] The development of improved

postprocessing techniques that can allow for more efficient interpretation of examinations by evaluating the degree of remodelling, plaque composition, the degree of eccentricity and degree of enhancement automatically. These tools will allow for increased acceptance of IVW techniques into clinical practice by simplifying the evaluation and interpretation processes. Beyond diagnostic evaluation, IVW can also be used to assess response to treatments. Evolution of plaque volume and vulnerable plaque characteristics, as well as evolution of changes related to inflammatory vasculopathy in the setting of therapy can guide the clinical team as to when aggressive therapy can be stopped or if the disease is refractory to the current therapy. Such techniques hold much promise and will likely play a prominent role in the future everyday practice of clinicians diagnosing and caring for patients with intracranial vasculopathies.

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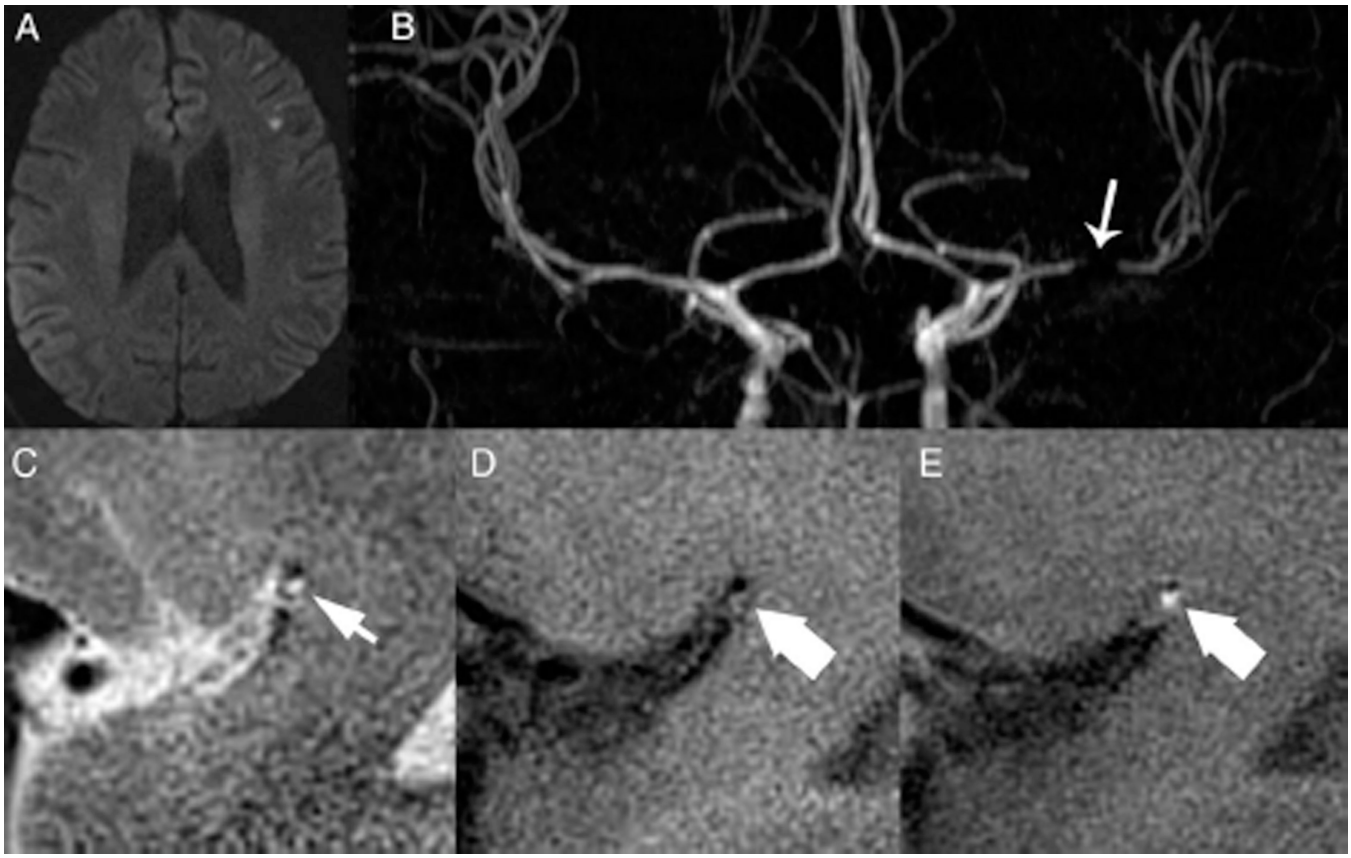

Figure 1.

Sixty-two-year-old male with history of hypertension, diabetes mellitus and coronary artery disease presents with right-sided weakness and dysphasia. On axial DWI (A), there were multiple foci of diffusion abnormality corresponding to acute ischemic infarcts. On Time of Flight MRA (B), there is focal high-grade stenosis of the left M1 MCA (white arrow). On sagittal T2-weighted IVW (C), there is eccentric wall thickening with heterogeneous T2 signal and outer wall remodelling (short white arrow). There is mild T1 hyperintensity within the lesion (thick arrow) on T1 pre-contrast IVW (D). The lesion shows incomplete enhancement (thick white arrow) on T1 postcontrast IVW (E). Clinical and imaging findings are compatible with intracranial atherosclerotic disease. DWI, diffusion weighted imaging; IVW, intracranial vessel wall; MCA, middle cerebral artery; MRA, MR angiography.

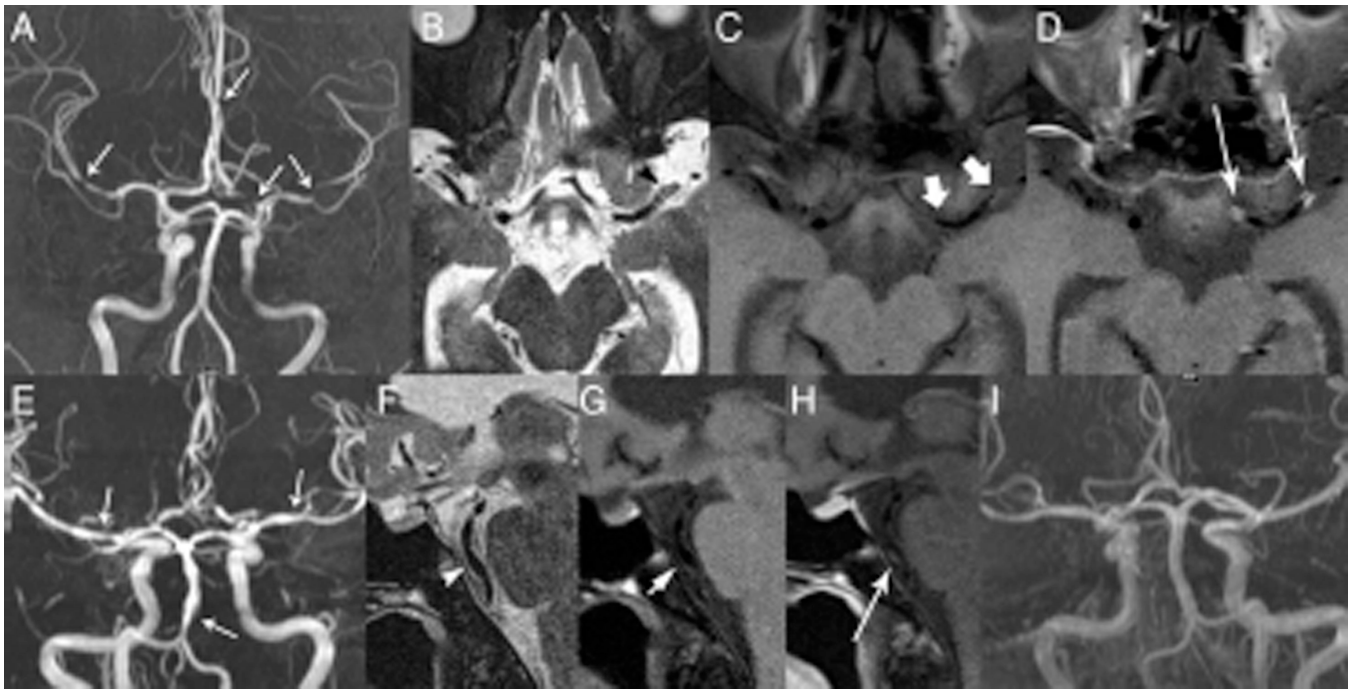

Figure 2.

Twenty-nine-year-old female with varicella vasculitis (A–D) and 36-year-old female with RCVS. 3D TOF MRA coronal volume rendered reformat (A) shows multifocal luminal stenosis and irregularity (white arrows), corresponding to areas of circumferential wall thickening (black arrowhead) on T2 IVW (B). There is corresponding wall thickening (thick white arrows) on T1 pre-contrast (C) with circumferential enhancement (long arrows) on postcontrast IVW (D). On 3D TOF MRA coronal volume rendered reformat (E), there is multifocal luminal stenosis and irregularity (white arrows) in a beaded pattern involving multiple arterial segments. On sagittal T2 (F) and T1 pre-contrast IVW (G), there is minimal vessel wall thickening (arrowhead on F and short arrow on G). There is no evidence of appreciable enhancement (long white arrow) on sagittal T1 postcontrast IVW (H). On 3D TOF MRA coronal volume rendered reformat performed 3 weeks later (I), there is marked improvement in luminal stenoses. 3D TOF MRA, three-dimensional time-of-flight MR angiography; RCVS, reversible cerebral vasoconstriction syndrome; T2 IVW, T2-weighted intracranial vessel wall.

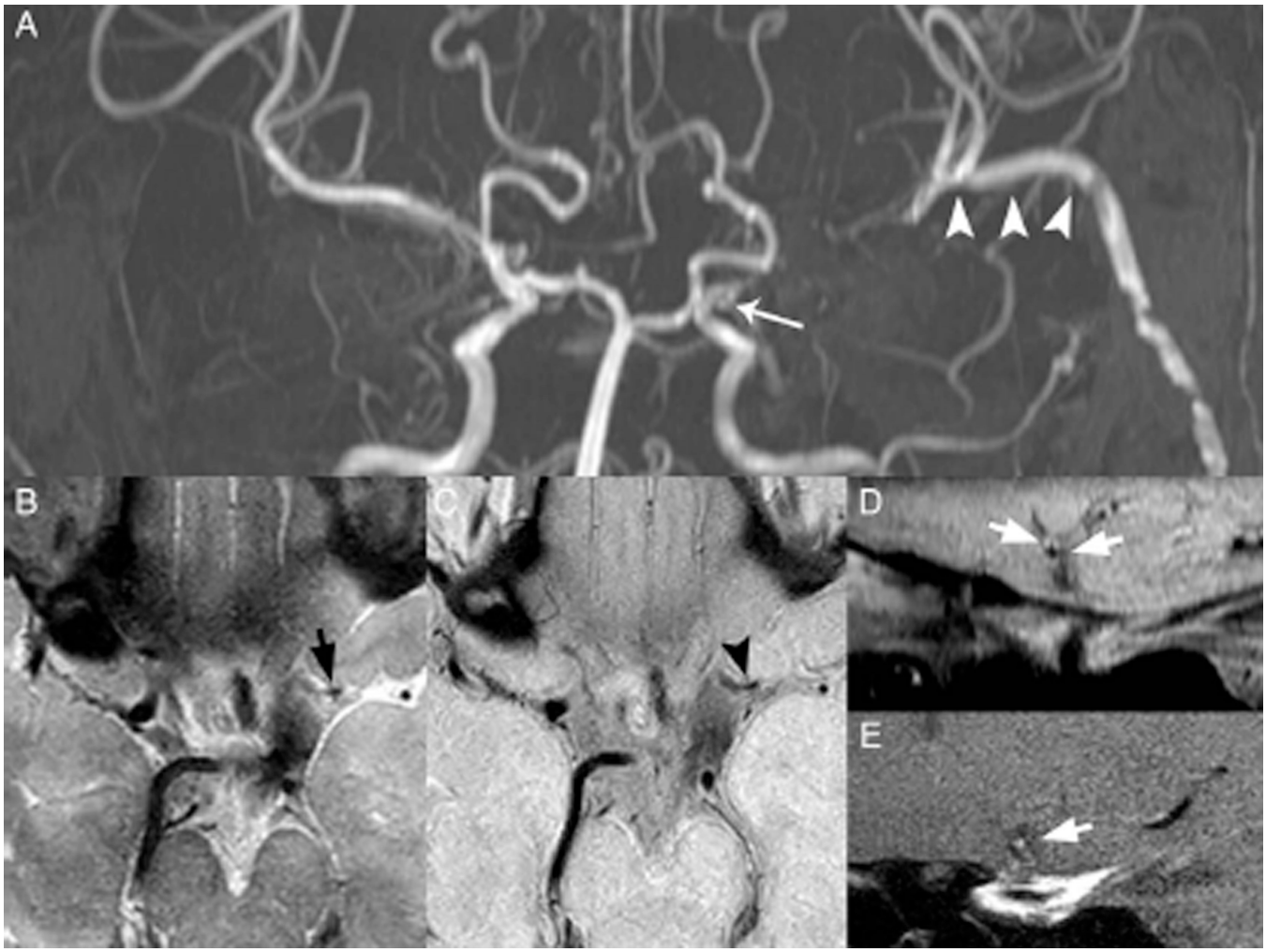

Figure 3.

Twenty-one-year-old female with history of Moyamoya disease on the left who had undergone previous direct left external carotid artery to middle cerebral artery bypass. On 3D TOF MRA coronal volume rendered reformat (A), there is occlusion of the left carotid terminus (white arrow). Bypass can be seen (arrowheads) with filling of left M3 and M4 branches. On axial 3D T2 VISTA (B), there is diminution of the left carotid terminus (short arrow) without evidence of outer wall remodeling. This is also seen on axial PD VISTA pre-contrast sequence (arrowhead) (C). On sagittal reformatted postcontrast PD VISTA (D), there is circumferential vessel wall enhancement (short white arrows) confirmed (short white arrow) on sagittal T1 postcontrast IVW (E). 3D TOF MRA, three-dimensional time-of-flight MR angiography.

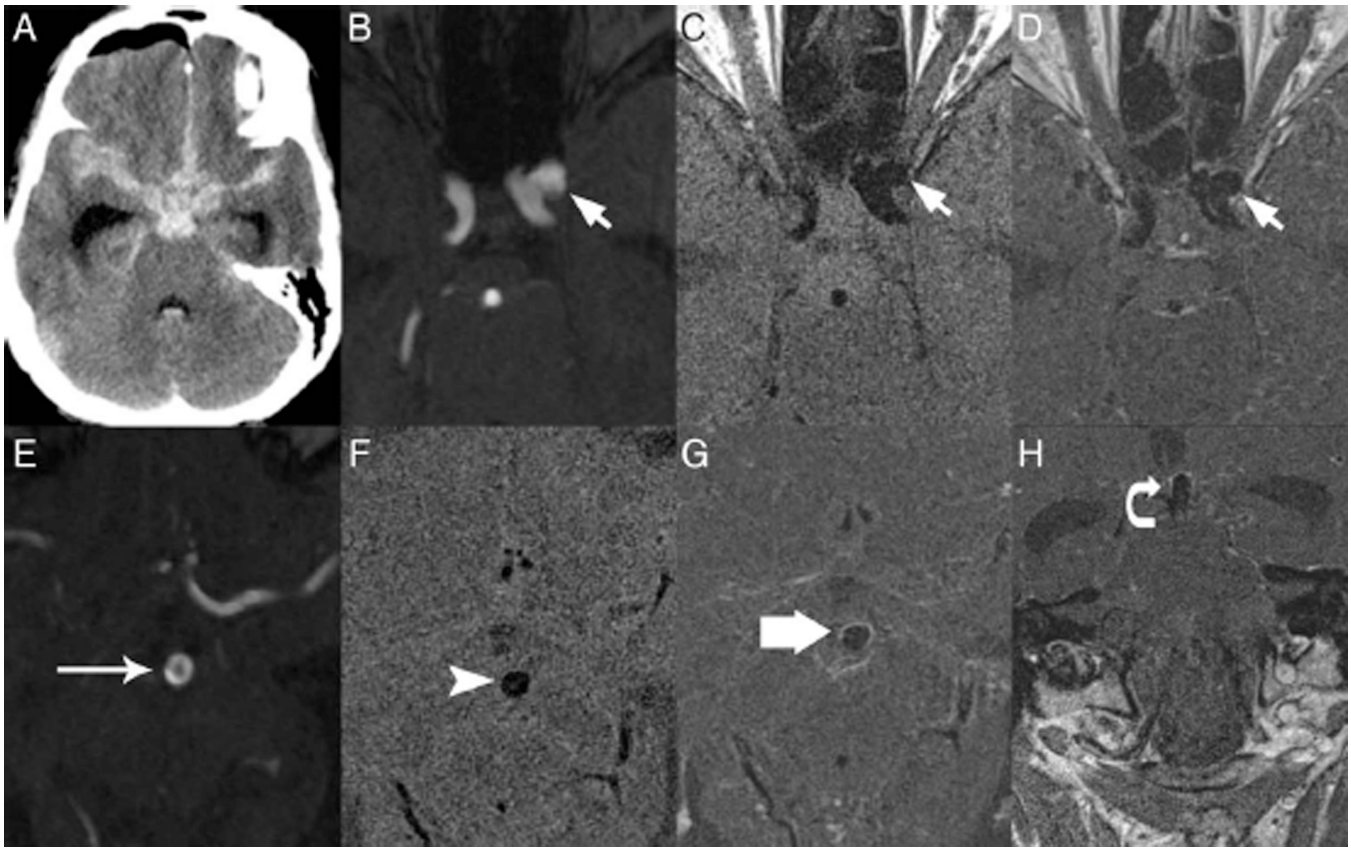

Figure 4.

Fifty-six -year-old female presenting with thunderclap headache. Axial non-contrast CT head (A) shows diffuse basal cistern subarachnoid hemorrhage. On 3D TOF MRA (B and E), there is a left supraclinoid ICA aneurysm (B short white arrow) and basilar tip aneurysm (E long white arrow). There is no corresponding enhancement of the left supraclinoid aneurysm (short white arrows) on T1 pre (C) and postcontrast (D) IVW, while the basilar tip aneurysm (arrowhead on F and thick arrow on G) shows circumferential wall enhancement when comparing T1 pre (F) and postcontrast (G) IVW. The basilar tip aneurysm was emergently treated endovascularly (curved arrow) as seen on coronal T1 postcontrast IVW (H). 3D TOF MRA, three-dimensional time-of-flight MR angiography; IVW, intracranial vessel wall.

Table 1

IVW MRI sequences and parameters at 3.0 T

Parameters	2D T2W	2D T1W	3D PD VISTA	3D T2 VISTA
Echo time (ms)	72	10	38	90
Repetition time (ms)	3550	1000	2000	3000
Field of view (cm)	18×18	18×15.8	18×16.5	25×19
Matrix	448×448	448×448	448×413	500×380
Slice thickness (mm)	1	2	0.4	0.5
In-plane resolution (mm)	0.4×0.4	0.4×0.35	0.4×0.4	0.5×0.5
Number of averages	3	4	1	2
TSE factor	22	18	60	60
Startup echoes	–	–	4	4
Acquisition time per slice (seconds)	10.4	45	–	–
Oversampling factor	–	–	1.2	1.2
# of slices	26	4–10	90	70
Bandwidth	223	207	361.7	164.5
Time	–	–	8:30	8:51

2D T1W, two-dimensional T1-weighted; 2D T2W, two-dimensional T2-weighted; TSE, turbo spin-echo.

Table 2

Vessel wall imaging characteristics of vasculopathies

	Vasculopathy				
Characteristics	**RCVS**	**Vasculitis**	**Atherosclerosis**	**Moyamoya Disease**	**Dissection**
Pattern of disease	Circumferential, Rarely Eccentric	Circumferential, Rarely Eccentric	Eccentric, Rarely Circumferential	Circumferential	Eccentric, Rarely Circumferential
Contrast Enh	±	++	++	±	±
T1 Hyperintensity	−	−	±	−	+
T2 Signal Characteristics	Isointense, Homogeneous	Isointense, Homogeneous	Heterogeneous, Juxtaluminal Hyperintensity	N/A	Variable
Outer Wall Remodeling	−	−	++	−	+
Potential Overlap in Findings	Vasculitis	RCVS, Atheroslerosis	Vasculitis	Vasculitis	Atherosclerosis

−, No abnormality; +, Mild abnormality; ++, More pronounced abnormality.

NA, not applicable; RCVS, reversible cerebral vasoconstriction syndrome.

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