



**HIGH RESOLUTION VESSEL WALL MAGNETIC RESONANCE IMAGING:
TECHNICAL IMPLEMENTATION, CLINICAL USES, DIFFERENTIAL
DIAGNOSIS AND PITFALLS**

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ABSTRACT

Vessel wall magnetic resonance imaging (VW-MRI) executes an increasing aspect in diagnosing vascular disease, as it contributes direct visualization and characterization of wall of the vessel. Traditionally, diagnosis of vascular diseases was based on conventional luminal imaging techniques which provide limited information, since they only assess vessel lumen. With the widespread use of this imaging technique, clinical doctors and radiologists should be familiar with the imaging specifications, clinical uses, methods of evaluation and restriction in the analysis of these images. This review article provides concise overview of technical implementation and various clinical uses of VW-MRI with common imaging findings of various vascular diseases (such as atherosclerotic disease, arterial dissection, moyamoya, vasculitis, reversible cerebral vasoconstriction syndrome, intracranial aneurysm) and carotid web. The review also provides some potential pitfalls of VW-MRI.

Keywords: Vessel Wall- MRI; Atherosclerotic disease; Arterial dissection; Carotid Web

INTRODUCTION

Early diagnosis of vascular diseases (such as, atherosclerotic disease, arterial dissection, vasculitis and reversible cerebral vasoconstriction syndrome) is important, as delay or inappropriate treatment may contribute to worse clinical outcomes [1,2]. Traditional diagnosis of vascular diseases was primarily based on conventional luminal imaging such as computed tomography (CT) or magnetic resonance angiography (MRA) techniques and invasive technique such as digital subtraction angiography (DSA) which are useful in evaluating the vessel lumen [3,4,5,6]. However, these techniques have restricted strength to differentiate vascular diseases, as some of the diseases can display the similar defect in vessel lumen [3,7,8]. Direct visualization of vessel wall allows the possibility to differentiate between these diseases. Currently, vessel wall magnetic resonance imaging (VW-MRI) is building up significant place in analysis of CNS vascular diseases [2,3,5,9,10]. VW-MRI contributes direct visualization and characterization of wall of the vessel and also provides superior soft tissue contrast and spatial resolution, which can provide diagnostic clue which are not feasible with traditional imaging techniques, that can help clinicians plan early appropriate treatment [1,5,6,8].

The aim of this review is to describe concisely some of the technological necessities of VW-MRI, accompanied with review of the rising clinical uses, differential diagnosis of various vascular diseases and some pitfalls of VW-MRI. Since, most of the clinical VW-MRI is carried out using 3T MRI machines, most of the following reviews on imaging features and techniques consideration will be based on 3T MRI.

VW-MRI Technical Implementation:

Selection of scan parameters and sequences for VW-MRI are mostly dependent on the particular scanning software and hardware available at the center. The American Society of Neuroradiology Vessel Wall Imaging Study Group [3] has stated some key technical requirements for intracranial VW-MRI. Here are some key technical requirements described below:

Spatial Resolution:

In MRI scanners, field strength most frequently used are 3 tesla(T) or 1.5T. 3T MRI is far appropriate for vessel wall imaging as it provides higher signal-to-noise ratio (SNR) and contrast-to-noise ratio (CNR) with high field strength [3,11]. 7T MRI with ultra-high field strength may provide visualization of smaller vascular lesions and detailed vessel wall imaging [12]. As, the availability of 7T MRI is limited. Besides this shortcoming, it generates nonuniform B0 and B1 fields, that may lead to deteriorate quality of scan and restrict the evaluation of the cerebellar and temporal areas [8,11]. Another concern in vessel wall imaging is head channel coil. With the high-resolution maintenance at the periphery of field of view, a 32- or 64-channel coil are better, compared to 8-, 12- or 16-channel coil for vessel wall imaging [13].

2D or 3D acquisitions:

Detailed analysis of VW-MRI needs visualization of wall of the vessel in both planes (long- axis and short-axis). Options are to use 2D (high in-plane spatial anisotropic resolution) or 3D (high spatial isotropic resolution) sequences [11]. The advantages of 2D acquisition are: takes lesser time and targets on particular

vessels of interest, but are highly dependent on correct placement of field-of-view and need for multiple acquisitions [3,8]. 3D sequence provides advantage as reconstruction of images in every direction is possible and is appropriate for torturous intracranial vessels [3,8,11,14]. 3D sequence takes more time to acquire compared to 2D sequence, but the newly introduced vendor-specific developments in 3D VW-MRI have the ability to shorten the acquisition time [8].

Multiple Tissue Weightings:

T1-weighted sequence (T1WI), plain and contrast enhanced is required in almost all vessel wall examinations. Proton density (PD) weighted sequence is also acquired instead of T1-weighted, but there are some disadvantages of PD weighted sequence. The signal intensity of CSF can appeal signal intensity of wall of the vessel and contrast enhancement are less apparent in PD weighted sequence [3,15]. Fat suppressed VW-MRI is sometimes useful and necessary while assessing branches of external carotid artery (such as in suspected temporal arteritis). T2-weighted VW-MRI (T2WI) sequence is often acquired and is beneficial in differentiating vascular diseases. Time-of-flight magnetic resonance angiography (TOF-MRA) describes lumen abnormality and are usually acquired [3]. Other sequences such as FLAIR, DWI, SWI, perfusion and cervical MRA are often required for the conclusive analysis corresponding to the clinical events [15].

Signal Suppression in Blood and CSF:

VW-MRI requires signal suppression emerging from CSF and blood [3,15]. Black blood (BB) VW-MRI was established in 1990s and broadly been used in imaging of cardiovascular diseases. BB-MRI allows finer visualization of vessel wall and helps differentiating similar vasculopathies, as it suppresses signal intensity of blood flow in the lumen. The frequently used methods for blood-signal suppression are spin echo and BB-double inversion recovery (BB-DIR) technique [14]. The most commonly used 3D VW-MRI techniques is variable flip angle refocusing pulse, fast spin echo sequences with different brand names such as CUBE (GE Healthcare, Milwaukee, WI, USA), VISTA (volume isotropic turbo spin-echo acquisition; Philips healthcare, Best, Netherlands) or SPACE (sampling perfection with application optimised contrasts by using different flip-angle evolutions; Siemens, Erlangen, Germany) [3,8,14].

Clinical Uses:

The clinical uses of VW-MRI, the typical imaging features of some of the CNS vasculopathies on VW-MRI and their related clinical significance will be review in the following parts.

Atherosclerotic Disease:

Intracranial atherosclerotic disease (ICAD) is a major cause of stroke causing long-lasting impairment and death worldwide. The cause of stroke in 50-60% of Asians, 29% of African-Americans and 15% of Caucasians populations is due to ICAD [4,13,16]. The core of describing atherosclerotic plaque has been changed from the grade of stenosis to assessment of pattern of plaque, location and composition [17]. Histopathology studies of plaque composition have provided evidence that inflammation and intraplaque hemorrhage (IPH) are important risk factors for occurrence of stroke, despite of the severity of stenosis [18].

VW-MRI enables to evaluate plaque morphology, distribution, vessel wall remodeling, hemorrhage and enhancement [1,8]. The component of plaque, such as surface irregularity, large lipid necrotic core, IPH, neovascularization and inflammation, signifies vulnerable plaque, that can be related to occurrence of ischemic symptoms [14,19].

On VW-MRI, intracranial atherosclerotic plaque generally illustrates eccentric thickening of arterial wall. On T2WI, the plaque close to the lumen often has high signal intensity, whereas the adjoining component to it often has low signal intensity. In some cases, in the periphery of the plaque, a thin enhancing layer is seen. This layered appearance of atherosclerotic plaque on VW-MRI has been correlated with carotid specimens after endarterectomy, which have shown that the enhancing layer adjacent to the lumen, the non-enhancing layer and the enhancing thin layer in peripheral represents fibrous cap, lipid core and increased vasa vasorum in the adventitia respectively [3,8,15].

Many authors have described the relation between plaque instability and enhancement of plaque [6-8,11,16,18,19] but has not been well established. Many studies reported that enhancement of plaque is associated with recent stroke and are less commonly reported in asymptomatic atherosclerotic plaques [6,8,11,19,20]. Baseline no enhancement or decrease enhancement of plaque at follow up, are likely to be stable whereas, persistence or increase in degree of enhancement at follow up, are likely to be identify as vulnerable plaque [16]. The mechanism of plaque enhancement may be multifactorial, but mostly related with inflammation and neovascularization [6,13,16]. The temporal change may consider the process of plaque activity. Furthermore, longitudinal studies are needed to illustrate the relation of plaque enhancement with plaque instability in ICAD.

Intraplaque hemorrhage (IPH) is considered as one of the significant risk factors determining plaque instability with yearly recurrence rate of stroke ranging from 15% to 45% [1,13]. Pathological studies shown that IPH can lead to progression and plaque rupture [14]. The IPH presence leads to higher rates of silent ischemia to occurs compared to those without IPH [21,22]. IPH illustrates as T1 hyperintensity, signal intensity greater than 150% of internal muscle tissue reference [1,6,13,23]. IPH detection and differentiation can be made by using various sequences such as T2WI or SWI [4].

Vessel wall might respond to evolvement of plaque either by positive or negative remodeling [6]. Positive remodeling can be beneficial by maintaining the lumen but the wall itself hysterically thickens, which may lead to instability and rupture of plaque [1,4,6,13]. On VW-MRI wall remodeling is illustrated before angiographically detectable stenosis occurs.

Arterial Dissection:

Cranio-cervical artery dissection is an important cause of stroke in young and middle age patients, accompanying 20% of all ischemic strokes and 27% of cerebellar and brainstem strokes [1,4,10,24]. Previously, isolated intracranial arterial dissections were poorly characterized but with the widespread use of VW-MRI, differentiation of dissection from other intracranial vasculopathies has been established [4,19,25]. Sub-adventitial dissections often maintain lumen shape, making it challenging to detect on conventional imaging

techniques [23]. With the use of VW-MRI, detection of intracranial dissection improved from 11 to 22%, compared to TOF-MRA [19]. VW-MRI allow detection of arterial dissection by characteristics features such as double lumen, intimal flap, intramural hematoma (IMH), stenosis and dilatation [1,3,4,19].

Intimal flap which separates true lumen from false lumen illustrates curvilinear shape hyperintensity on T2WI [3,15]. Visualization of intimal flap in T1-weighted images are better with gadolinium enhancement [14]. One of the advantages of VW-MRI is detection of IMH in patient with arterial dissection. IMH is detected as eccentric arterial wall thickening, often hyperintense on T1WI, but like hemorrhage, the signal intensity evolves with time [3,15]. Arterial dissection without IMH are less likely to be progressive than dissection with IMH [14]. Intraluminal contrast enhancement represents intraluminal thrombus formation which is more likely to be associated with occurrence of ischemia in corresponding territories of the vessel [26]. Vessel wall enhancement are also sometimes related to arterial dissection. Although the mechanism for wall enhancement in dissection is not clear, but has been considered as result of stagnant blood present in the false lumen, inflammation or vasa vasorum enhancement. Extensive enhancement might anticipate for recurrent and multiple dissections [4,10]. Furthermore, longitudinal studies are needed to illustrate whether vessel wall enhancement is the cause or the result of dissection.

Moyamoya:

Moyamoya disease is rare progressive vascular disease caused by occlusion or severe stenosis of distal internal carotid artery (ICA) leading to evolution of compensatory collateral circulation at the base of the brain [4,6,10]. Moyamoya disease usually affects children and young population and common in Asian populations [27]. Moyamoya syndrome is characterized as occlusion or stenosis of distal ICA due to atherosclerotic disease, vasculitis, sickle cell disease, neurofibromatosis type 1, radiation therapy or other disease processes [1,28]. Typical changes in moyamoya disease can be recognized via CT angiography (CTA) or MRA [14]. But, early moyamoya disease usually does not illustrate the typical basal “puff of smoke” compensatory collaterals and cannot be differentiated from other steno-occlusive vasculopathies on conventional imaging techniques. Identification of moyamoya disease from moyamoya syndrome and other vasculopathies is important, because it is mostly surgically treated rather than medical management [17,23].

On VW-MRI, the most common pattern identified in moyamoya disease is concentric wall thickening and enhancement of distal ICAs and middle cerebral artery (MCA) diminution [1,3,18]. The concentric thickening and enhancement suggest hyperplasia or inflammation and diminution or wall thinning represent media shrinkage [6,17]. Moyamoya syndrome caused by atherosclerotic disease illustrates imaging features described as above, mostly with eccentric plaques with positive remodeling and enhancement [23], whereas moyamoya syndrome caused by radiation therapy illustrates concentric wall thickening and enhancement similar to moyamoya disease, but may persist for long duration [29].

Vasculitis:

CNS vasculitis is a rare heterogeneous vascular disease that is caused by diverse inflammatory disease processes [4,8,10]. The Chapel Hill Consensus Conference in 2012, categorized vasculitis in relation to size of

artery involved and accompanied pathologic lesions such as primary angiitis (PACNS) or accompanied with systemic disorder such as giant cell arteritis (GCA) and polyarteritis nodosa, or vasculitis secondary to collagen vascular diseases, tumors, infections and substance abuse [10,18]. The clinical diagnosis of CNS vasculitis is challenging [17]. Conventional angiograms and biopsies are constantly needed to determine the diagnosis; still, the specificity and sensitivity are limited to as low as 30% and 27% respectively and even have risk related to procedure [8,17]. Recently, VW-MRI has been used widely to diagnose CNS vasculitis.

VW-MRI often illustrates homogeneous, smooth, concentric wall thickening and enhancement [3,4,17]. Mostly concentric enhancement is seen, but eccentric enhancement has also been seen in minority of patients [14,17]. The wall enhancement may either be thin or thick, which usually extends margin of the vessel wall. In spite of lumen not clearly visible, VW-MRI illustrates a linear enhancement in periadventitial layer which go along with region of small vessels [30]. Leptomeningeal or parenchymal enhancement can also be seen [31]. The likely mechanism of vessel wall enhancement in vasculitis are due to increase endothelial permeability or vasa vasorum related leakage of contrast from lumen to vessel wall [3,14]. The appearance or degree of enhancement cannot differentiate among diverse vasculitis etiologies, but the location of vessel involve and clinical symptoms may help to narrow down the cause in individual cases [32].

With the widespread use of VW-MRI, need for biopsy to diagnose vasculitis may be limited, but in case where biopsy is needed, VW-MRI may help in localization of active disease and select an appropriate biopsy target [8,23]. Another potential application of VW-MRI is evaluating response to treatment as degree of enhancement can signify the disease activity [4,17].

Reversible Cerebral Vasoconstriction Syndrome (RCVS):

RCVS is a group of various conditions with common clinical presentation marked by thunderclap headache [14]. RCVS have overlapping clinical finding with subarachnoid hemorrhage and imaging findings with CNS vasculitis. Therefore, early differentiation is necessary to provide appropriate treatment and avoid unnecessary examination such as brain biopsies and lumbar puncture [14,18].

VW-MRI illustrates minimal, smooth, concentric thickening of vessel wall with no enhancement. Mild enhancement has been seen in minority of patients [1,18,19]. No enhancement of vessel wall is consistent with histopathologic examination in RCVS, lack of vessel wall inflammation [3]. If enhancement present, it is mild compared to vivid vessel wall enhancement present in CNS vasculitis. Reversibility of wall thickening, enhancement and luminal stenosis is specific for RCVS, so follow up vessel wall imaging could signify the disease activity and would be more beneficial [14].

Intracranial Aneurysm:

Intracranial aneurysms are common in 3–5% of the population [33] and subarachnoid hemorrhage due to aneurysm leads to higher rate of disability and mortality [19]. CTA and MRA have accuracy upto 90% to detect intracranial aneurysms [8]. Recently, VW-MRI is not used for diagnosing intracranial aneurysm but has been used to evaluate the relationship between aneurysm wall features and its stability [14].

Overall aneurysmal wall enhancement more than 1 mm detected on VW-MRI has been interpreted in vulnerable aneurysms [8,14,33,34]. The mechanism for aneurysm wall enhancement are likely due to neo-vascularization, macrophage infiltration and degeneration of elastin, which, may increase the risk of rupture. Other theories for aneurysm wall enhancement can be due to slow blood flow along the aneurysm wall and endothelial dysfunction [8,33]. The lack of aneurysm wall enhancement may seem to have strong prediction of stable aneurysm than enhancing lesions in predicting risk of rupture or instability [17,33].

Another potential use of VW-MRI is to determine the likely responsible aneurysm causing subarachnoid hemorrhage in case of multiple aneurysms present [17,34]. On VW-MRI, the aneurysm responsible for subarachnoid hemorrhage typically illustrates circumferential enhancement or focal enhancement at the site of rupture [17,23]. The mechanism regarding aneurysm wall enhancement in rupture aneurysm is likely endothelial injury related with mural breakage or due to healing process of inflammation [35].

Carotid Web:

Carotid web is nonatherosclerotic disease, radiologically described as abnormal shelf-like protrusion or filling defect in the posterior aspect of ICA bulb [36,37]. Carotid web has been one of the under-recognized etiology for cryptogenic stroke and recurrent stroke, particularly in younger patients. Till now, DSA is the “gold standard” imaging technique for detection of carotid web [38].

At present, there are only few studies that have demonstrated the role of VW-MRI in diagnosing carotid web. VW-MRI presents visual clue of the carotid web morphology and location [37]. VW-MRI illustrates thin, linear membrane or shelf-like intraluminal filling defect with slightly low signal intensity to normal vessel wall with peripheral enhancement and central hypo-enhancement in T1WI [39]. More studies are needed to determine more about imaging features and diagnostic performance of VW-MRI in diagnosing carotid web.

Differential Diagnosis of Vasculopathies on VW-MRI:

VW-MRI have been promoted for differential diagnosis of various vascular diseases. Imaging features such as vessel wall pattern (concentric vs eccentric), contrast enhancement, remodeling patterns and characteristics signal intensity in T2WI helps in differentiating various vascular diseases.

The VW-MRI features to differentiate between atherosclerosis, RCVS and vasculitis includes T1WI (plain and contrast enhanced) and T2WI [1]. In most the cases with atherosclerosis, plaque illustrates eccentric vessel wall thickening and enhancement. Concentric wall enhancement was also found in some of the patients, mimicking RCVS and vasculitis. In this case, additional T2WI would be beneficial for differential. Atherosclerotic plaque shows heterogenous hyperintense T2 vessel wall signal as compared to vasculitis shows homogeneous isointense signal and minimal smooth wall thickening in RCVS [1,6]. Additionally, atherosclerotic plaque usually shows outward remodeling, which is not a feature associated with vasculitis and RCVS [1]. No or mild wall enhancement with multiple vascular stenosis favors RCVS over vasculitis and atherosclerosis [8]. The VW-MRI characteristics to differentiate between RCVS and CNS vasculitis includes RCVS shows no or mild vessel wall enhancement which resolves on follow-up vessel wall imaging, while CNS vasculitis shows

multifocal intense enhancement with persistence of luminal narrowing and enhancement on follow-up imaging [1,14].

The differentiation between intramural hematoma (without double lumen and intimal flap) and intraplaque hemorrhage on VW-MRI may be challenging occasionally and differentiation may be dependent on clinical data such as age of the patient, risk factors, presenting symptoms and location of vasculopathy [4].

When a patient presents with indistinct clinical and luminal imaging features, similar to atherosclerotic disease and moyamoya, differentiation may be difficult especially in Asian patients. In this setting, on VW-MRI moyamoya disease shows concentric enhancement, whereas atherosclerotic plaque shows focal eccentric enhancement on the symptomatic segment. In addition, moyamoya disease has features such as MCA shrinkage that can help in differentiating it from intracranial atherosclerosis [4,6].

Carotid web and atherosclerotic plaque generally locate at carotid bifurcation and present as focal endoluminal projection, so can mimic each other. VW-MRI helps differentiating between atherosclerotic plaques and carotid web. On VW-MRI, carotid web illustrates smooth and well-defined border, whereas, atherosclerotic plaque illustrates irregular border and increase vessel wall thickness is more likely to be present in atherosclerosis [36].

There are some important cautions noting for use of VW-MRI. In children, vascular enhancement can be normal imaging findings. Such type of enhancement is commonly thin and linear, often symmetrical to contralateral vessels and usually does not course through CSF. Due to wall injury after mechanical thrombectomy, vessel wall enhancement can be seen, which can mimic vasculitis [11,19].

Pitfalls:

In spite of the advancement in imaging techniques and significant clinical uses of VW-MRI, there are some limitations and pitfalls of this imaging technique. Mostly are associated with the analytical experience and imaging acquisition, which may lead to normal findings being misread as abnormality (earlier mentioned that normal perivascular enhancement or post thrombectomy enhancement might be misread as CNS vasculitis). In addition, venous enhancement, incomplete suppression of signal in the edge of lumen or slow blood flow can also imitate enhancement of vessel wall. Likewise, vasa vasorum can also cause circumferential wall thickening and enhancement that may be misinterpreted as vasculitis [3,8]. Another limitation is the long duration for acquisition which may lead to patient discomfort and motion artifact that could not be repaired afterward [14].

CONCLUSION

For the past few years, VW-MRI has been progressively used as diagnostic tool for evaluating CNS vasculopathies. Since, limited information are provided through conventional luminal imaging techniques for evaluation of vasculopathies, VW-MRI allows direct visualization and characterization of the vessel wall and provide accurate diagnosis and potentially impact in patient outcomes. In addition to diagnostic evaluation, VW-MRI can also be used to evaluate response to treatment. With continuous advances in technique and analysis, VW-MRI carries much potential and likely to play an important role in further clinical practice.

Competing Interests:

None declared.

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