Intracranial Black-Blood Vessel Wall Magnetic Resonance Imaging: Initial Clinical Experience in a Single-Center Study

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ABSTRACT

Objective: To assess the adjunct value of high-resolution black-blood vessel wall MR (VW-MR) imaging in the context of ischemic stroke and other vascular diseases in the clinical practice.

Methods: All patients with vascular abnormalities underwent MR on a 3T system. In addition to the conventional clinical scans, VW-MR imaging was performed by the volume isotropic turbo spin-echo acquisition (VISTA). Typical imaging parameters were: isotropic voxel size 0.35 × 0.35 × 0.35 mm^3. VW-MR findings in adjunction to MR angiography were reviewed by two neuroradiologists and presented as case series to compare with the final diagnosis based on the clinical data.

Results: 14 patients were recruited within 12 months, among which 12 completed MR scans. The final diagnoses included 6 cases of atherosclerotic plaque, 4 cases of aneurysm, 1 case of Takayasu vasculitis, and 1 case dissection. In general, VW-MR imaging before and after gadolinium allowed successful detection of vasculitis with circumferential smooth and thick enhancement, as well as active plaque with eccentric enhancement. Reasonable interpretation could be made for clot and thrombus with careful inspection of the clinical context. Signal void artifact could be observed in post coiling and stent placement arteries. Dark signal was also demonstrated in heavily calcified aneurysmal wall.

Conclusion: Despite relatively small sample size, our preliminary study has demonstrated the useful adjunct information provided by VW-MR imaging for diagnosis and characterization of intracranial vascular diseases in the clinical practice. Pitfalls should be aware in certain cases and large cohort clinical studies are warranted to further evaluate its clinical value.

Keywords: Vessel wall; intracranial artery; magnetic resonance imaging; black blood (Siriraj Med J 2018;70: 327-334)

INTRODUCTION

Imaging of intracranial vessels, especially arteries has been used in clinical practice for a long time. Conventional angiography with intraluminal catheterization is generally accepted as the standard imaging modality. With the development of computed tomography and magnetic resonance imaging, non-invasive techniques such as CTA or MRA have been proposed as alternative ways to demonstrate abnormality of the vessels especially intracranial arteries or veins. However, these luminal imaging cannot demonstrate the pathology at the vascular wall. For extracranial vessel wall imaging, ultrasonography and MRI have been tried for evaluation of carotid bifurcation disease such as vulnerable plaque in order to predict high risk of future stroke. The conventional MRI with suppression of flowing proton or so called black-blood technique has been proved histologically for tissue component of the abnormal signal and findings on multiple pulse sequences.[1] However, different scenarios for intracranial arteries are slower flow and smaller

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vascular size/wall. Also ethical issue makes it difficult to be conclusive from the imaging findings. Most recent reports were finalized with clinical context rather than direct comparison with pathologic result.[2]

With the new technology of MRI recently developed, it is possible for intracranial vessel wall imaging (VWI). The commercially available pulse sequence has been launched with improvement in image quality. At our hospital, we have evaluated the technique in clinical practice to consider the usefulness of the vessel wall imaging for intracranial arteries.

MATERIALS AND METHODS

This retrospective study was performed after approval from the Institute Ethical Committee (Si 396/2017). The subjects were recruited from the department PAC system for availability of vessel wall imaging and MRA. The MRI finding was retrospectively reviewed in consensus by two neuroradiologists without clinical data. Final conclusion of the diagnosis was summarized from clinical data. The result was presented as case series for the MRI findings and possible diagnosis.

MR imaging: The conventional brain imaging was performed as routine protocol of the institute. MRA was performed with 3D-TOF and contrasted enhanced MRA techniques for luminal imaging. Vessel wall imaging was performed with volume isotropic turbo spin echo acquisition (VISTA) technique (Philips, Best, the Netherlands). Since this study was a retrospective review, the pulse sequence used was varied according to the judgment of the radiologists on duty. All except case number 3, were performed with T2W-VISTA and gadolinium enhanced T1W-VISTA. Some cases were performed with PD-VISTA and some with T1W-VISTA. The parameters were shown in Table 1.

RESULTS

In total 14 cases underwent VW-MR imaging during 12 months. While 12 cases were obtained with complete clinical data, two cases did not have conclusive clinical data and were excluded for further assessment.

Among 12 cases, 8 cases of stroke were finally diagnosed as atherosclerotic plaque in 6 cases, Takayasu vasculitis in 1 case, and arterial dissection in 1 case. The other four cases were finally diagnosed as ruptured aneurysm in 1 case and unruptured in 3 cases. The demographic data of each case was presented in Table 2.

DISCUSSION

VW-MR imagings were successful in all cases.

Atherosclerosis

Case 1: Multiple small cortical infarction was demonstrated on cMRI. The VW-MRI showed enhancing eccentric enhancing plaque at the luminal narrowing M1 segment of right middle cerebral artery (MCA). Old pontine infarction was noted with irregular nonenhancing plaque at the adjacent basilar artery (BA).

Case 2 (Fig 1)**:** MRI brain showed no parenchymal lesion. VWI showed small focal enhancing plaque at M1 segment of right MCA with stenosis on MRA, compatible with active plaque.

Case 3: MRA showed focal stenosis of left posterior cerebral artery (PCA) with suspected ulceration. VWI showed small eccentric plaque with no ulceration. No contrasted VISTA was available due to low GFR.

Case 4 (Fig 2)**:** MRA showed mild stenosis of the distal basilar artery (BA). The rest of the BA was smooth normal

TABLE 1. MRI vessel wall imaging parameters.

	PD-VISTA (VIRTA)	T1-VISTA	T2-VISTA
Field of View (FOV) [mm^3]	200x165	200x251	150x150
Matrix size	304x252	268x336	228x156
Voxel size (acquired) [mm^3]	0.66x0.65x0.7	0.75x0.75x0.75	0.66x0.96x0.7
Voxel size (reconstructed) [mm^3]	0.35x0.35x0.35	0.37x0.37x0.38	0.35x0.35
Repetition time TR / echo time TE [msec.]	1450/33	700/33	2000/120
SENSE factor	2	1.7	2
Scan time [sec.]	5.30	6.45	5.26

Abbreviations: TR= repetition time; TE= echo time; PD= proton density; VISTA= volume isotropic turbo spin echo acquisition

TABLE 2. Demographic data of the cases.

Number	Age	Sex	History	Conclusive diagnosis
1	43	M	Left sided weakness for 1 day, previous history of repeated neurological deficit on left side more than 10 times in 2 weeks.	- Active plaque at right MCA with stenosis and Multiple cortical lacunar infarction – Non-active plaque at Basilar artery and old pontine infarct
2	37	F	Left hemiparesis for 5 minutes with fully recovery (TIA)	Right MCA plaque with stenosis.
3	80	F	Underlying hypertension presenting with gait ataxia with no other neurological deficit.	Left PCA plaque with mild narrowing
4	53	F	Previous pontine hemorrhage with right hemiplegia.	Short segment of mild distal basilar stenosis with Atherosclerotic plaque along the basilar artery.
5	38	M	Sudden onset of left hemiparesis. NCCT showed old lacunar infarction at right basal ganglion	Right MCA thrombosis with occlusion and old basal ganglion infarct.
6	51	M	Underlying hypertension presenting with left hemiparesis	Dolicoectasis with plaque along the left VA.
7	52	M	Incidental finding of abnormal intracranial arteries.	Right VA dissection.
8	18	F	Left hemiparesis and motor aphasia at age of 12 years old. Clinically diagnosed as Takayasu vasculitis.	Vasculitis of left ICA.
9	70	F	Subarachnoid hemorrhage	Right superior hypophysial aneurysm post coiling and residual neck
10	82	F	Left homonymous hemianopia and bilateral PCA infarction. Incidental finding of ACom aneurysm on CTA.	Post coiling AComA aneurysm with artifact.
11	73	F	Memory impairment with incidental finding of aneurysm at tip of BA on MRI brain.	Post coiling aneurysm at BA tip with stent placement in BA.
12	70	F	Fever with sinusitis with incidental finding of calcified aneurysm on CT.	Calcified aneurysm right M2 MCA

Abbreviations: M= male, F= female, AComA= anterior communicating artery, ICA= internal carotid artery, MCA= middle cerebral artery, PCA= posterior cerebral artery, VA= vertebral artery, BA= basilar artery, NCCT= non-contrast CT, CTA= CT angiography, TIA= transient ischemic attack

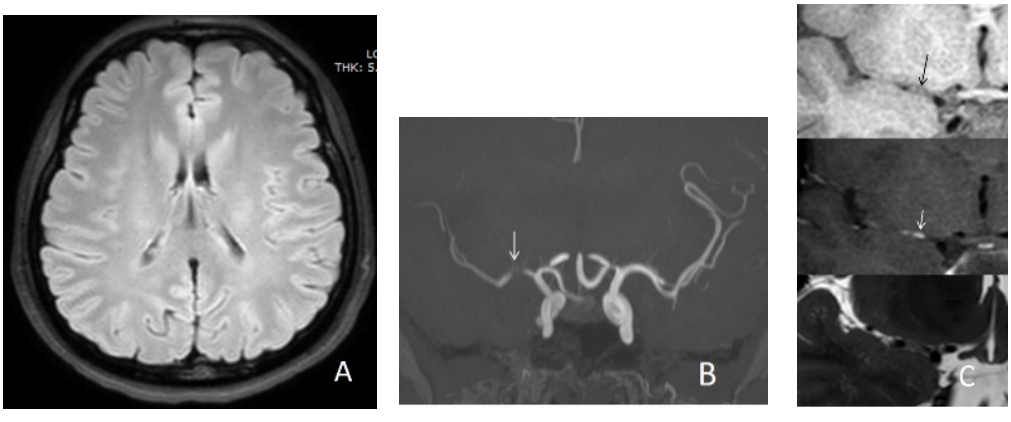

Fig 1. Case 2 patient with TIA. (A) no parenchymal lesion, (B) focal narrowing right M1 MCA (arrow), (C) VWI T1W(upper), Gd-T1W(middle) and T2W(lower) show enhancing plaque at the stenotic MCA.

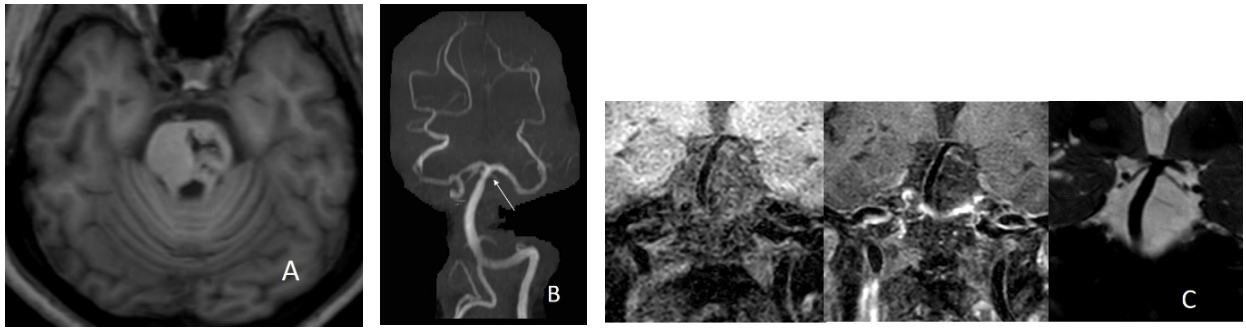

Fig 2. Case 4 patient with pontine hemorrhage (A) T1W shows old hemorrhagic region, (B) MRA shows short segment of stenosis at basilar tip, (C) VWI T1W(left), Gd-T1W(middle), T2W(right) show irregular enhancing plaque along the basilar wall.

flow signal. VISTA showed multiple irregular eccentric plaque along the entire BA wall with mild enhancement.

Comments (Case 1-4): Intracranial atherosclerosis has been reported more in Asian stroke than western one. About 10% presented with TIA and 30-50% with ischemic stroke.[3] MCA is the most common site of atherosclerotic stenosis in Asian stroke.[4] Active plaque is generally defined as enhancement of the plaque.[5] However, no definite evidence has been reported about how long the culprit plaque has been enhanced.[6] We noticed enhancement of the plaque in most of our cases corresponding with the symptomatic brain supplied by the arterial lesions. Even more, case 2 (Fig 1) showed no detectable parenchymal lesion with repeated TIA, but abnormal vessel wall. This may be useful information for confirming ischemic process rather than local convulsive process. Intensive medical treatment has been reported regression of the stenosis and plaque enhancement at the symptomatic side.[5,7] VWI may be useful for decision in medical treatment and follow up of the treatment outcome. Case 4 (Fig 2) also showed additional information of VWI on MRA. Luminal imaging may not explain the etiology of the stroke like hemorrhagic pons in case 4 in which more pathology was seen to be more extensive on VWI.

On VWI, the most common pattern of small intracranial atherosclerotic plaque is eccentric.[8] MCA is the most common artery with intracranial atherosclerotic plaque. Common location of MCA culprit plaque is opposite to the perforator orifice or just at the origin of the branches.[9]

Case 5 (Fig 3)**:** A 38-year-old male with acute left hemiparesis and old basal ganglion infarction on CT underwent mechanical thrombectomy. The endovascular procedure was performed at the occluded right MCA with yielding of small amount of white clot. Recanalization of the right MCA was achieved with irregular narrowing of the lumen. Followed up MRI showed occlusion of the right MCA. VWI demonstrated high signal clot in the lumen with irregular enhancement, possible mixed organized and non-organized thrombus, as well as active plaque.

Summary of signal appearance on VW-MR images of cases 1-5 was shown on Table 3.

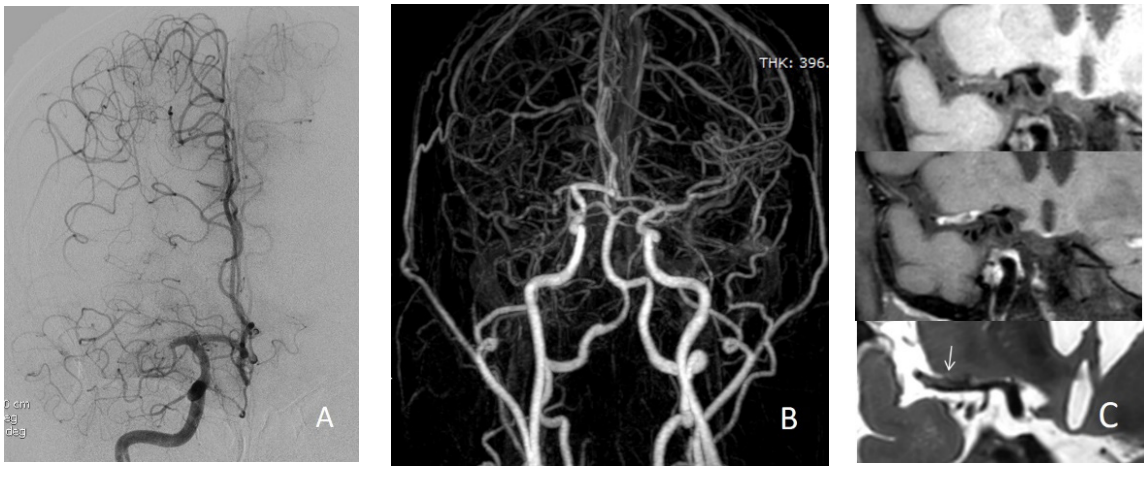

Fig 3. Case 5 patient with acute stroke. MRI showed old infarction at right basal ganglion (not shown). (A) DSA shows occlusion of right MCA with scanty clot from mechanical thrombectomy. (B) MRA still shows occlusion right MCA. (C) VWI T1W (upper), Gd-T1W(middle) and T2W(lower) show thrombus with irregular enhancement.

TABLE 3. Signal intensity of lesions in case 1-5 on vessel wall imaging.

Case number/location of lesions	VISTA-T1W	VISTA-T2W	Gd-VISTA-T1W (enhancement)
Right MCA	Iso-SI	Low-SI	Positive
Basilar artery	Iso-SI		Negative
Right MCA	Iso-SI	Iso to increased SI	Positive
FU 4 months	Low-SI	Iso-SI	Positive
Left PCA	Iso-SI	NA	NA
Basilar artery	Iso-SI	Low-SI	Positive
Right MCA	High and iso-SI	Iso-SI	Positive

Abbreviations: SI= signal intensity; NA= no information/not done

Comment (Case 5): The component of plaque in intracranial atherosclerotic stenosis (ICAS) cannot be separated as in neck arteries due to small lesion out of MRI resolution.[10] Variable signal intensity has been noted even in cervical carotid artery.[10-14] Lipid core may have low or iso-SI onT1W and T2W due to component of cholesterol and rich-water proton.[15,16] Signal intensity of blood component on VISTA is not so familiar for radiologists, unlike conventional T1W or T2W. By solely using high signal T1W as the symptomatic plaque is not reliable by visual assessment.[17] Low signal on T2W and SWI was also reported helpful for identifying hemorrhagic plaque.[10] Generally, enhancing plaque is accepted as the most reliable marker for unstable and symptomatic stroke.[5] The careful point is misinterpretation of periarterial enhancement from vessel of the adjacent meninges.[18,2,19]

However, case 5 demonstrated mixed high and iso-signal of the blood which was believed to be subacute or chronic stage of the inflammatory thrombus with organization. The enhancement of the clot was difficult to separate from plaque at the wall of the small size artery. With history of relapsed stroke and old infarct in this case, atherosclerotic plaque was believed to be the cause of severe stenosis and explained yielding of scant clot in thrombectomy.

Arterial Dissection

Case 6: DSA showed dilatation of left intracranial vertebral artery. No definite intraluminal flap was detected. VWI showed thick wall with mixed high and iso signal nearly circumferentially. The wall was also enhanced suggestive of active process. No definite intimal flap was detected.

Case 7: DSA showed multiple areas of intracranial arterial stenosis. The most severe was noted at right VA with post-stenotic dilatation. VWI was requested for possible dissection. MRA showed sharp linear filling defect inside the irregular lumen, not artifact. VISTA confirmed the enhancing intimal flap, compatible with dissection.

Comments (Cases 6-7) (Fig 4): Intracranial arterial dissection has been found more in young adults and more in Asian population than Europeans.[20-22] Definite diagnosis of arterial dissection is present intimal flap and two lumens or change of characters after follow up.[23] The criteria may not be true for intracranial dissection. Many cases may have no symptoms for immediate diagnosis at time of event.[24] Most reported imaging findings included multistages of the disease. Thrombosed false lumen may be seen as high signal clot at the wall, either eccentric or circumferential location, and compromise the true lumen.[25] After healing, the lumen may be back to original size or dilated.[26] To separate from atherosclerosis, dilatation was used to be believed the clue. However, more evidence of remodeling of intracranial atherosclerotic arteries has shown this concept might not be true.[5,27] We demonstrated two cases with diagnostic problem of dissection. Case 7 clearly showed the intimal flap. For case 6, the circumferential high signal wall may represent vulnerable atherosclerotic plaque or thrombosed false lumen. Dilatation of the vertebral artery may be the process of remodeling which has been found more in posterior circulation.[27] Surprisingly, both cases had no symptom from the involved arteries. Positive remodeling is suggestive of high risk, whereas negative one is more compatible with fibrotic healing process.[27]

It should be emphasized that when dilated artery or intramural hematoma is found, diagnosis of arterial dissection should be cautioned. Given the differential diagnosis for both dissection and atherosclerosis it may be more proper to suggest follow up imaging to observe if any change of the lesion feature, is reported.

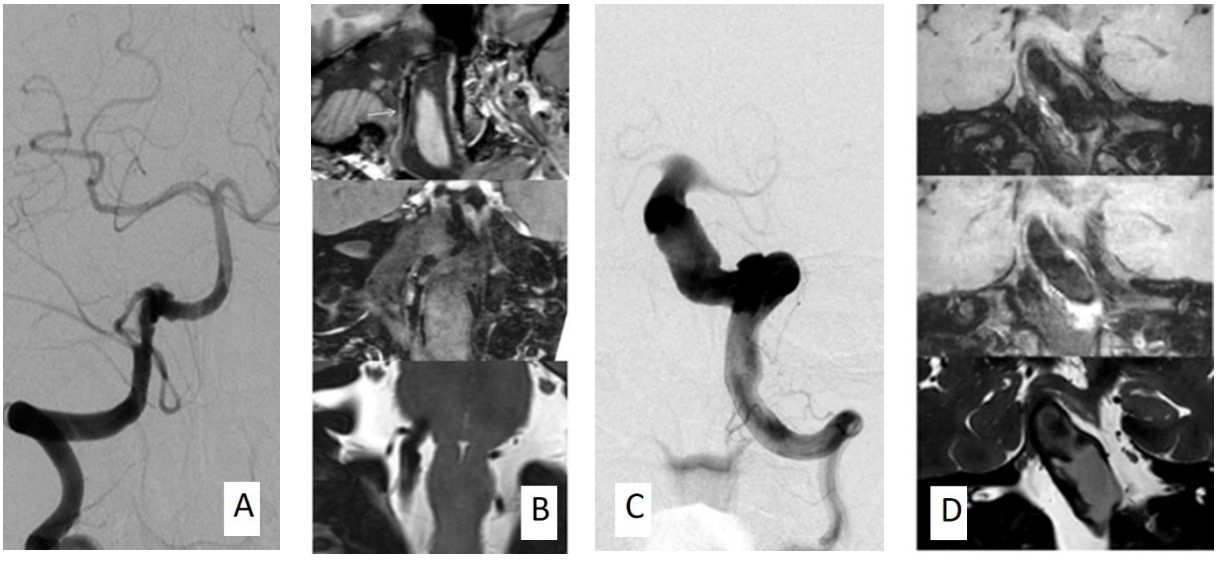

Fig 4. Case 6 (A, B): left atherosclerotic plaque and positive remodeling left vertebral artery. (A) DSA, (B) VWI shows irregular plaque with hemorrhage component and enhancement along the VBA wall. ; Case 7 (C, D) dissection of right VA. (A) DSA, (B) VWI T1Wshows enhancing intimal flap (arrow) of right VA.

Vasculitis

Case 8 (Fig 5)**:** Known case of Takayasu vasculitis showed left MCA infarction and occlusion left supraclinoid ICA causing developed Moya-like pattern The VWI was performed 6 years after the event and showed small left ICA with thick enhancing wall at the petrous and cavernous part, suggestive of active process.

Comment: Usually, CNS involvement in Takayasu vasculitis is in large extracranial arteries. Involvement of intracranial artery was reported in 15.2% and 7.6% was vasculitis.[28] Moya-like pattern is rarely reported.[28,29] Arterial wall edema was reported in Takayasu vasculitis in 94% of clinically active disease and 56% in clinically inactive disease.[30]

Vasculitis has been well demonstrated with VWI. Circumferential smooth and thick wall enhancement is the clue for diagnosis.[31] Dilated neovasculature has been demonstrated in arterial wall of Takayasu arteritis.[32, 33] Evidence of thinning media of arterial wall and finally luminal stenosis (vanishing MCA) was reported in idiopathic Moyamoya disease.[34] In Takayasu arteritis, involvement starts at adventitia and progresses to intima. We demonstrated rare Moya-like Takayasu arteries with VWI. The stenotic ICA was totally occluded with no enhancement, suggestive of inactive process. However, the rest of more proximal ICA was still enhanced, and so probably active disease. Small arterial size of the involved ICA implied the same process as reported in iMoyamoya disease, or less functional artery. With the history of the patient, the diagnosis is usually in no doubt if arterial stenosis is seen. VWI might be useful for areas with no luminal stenosis to demonstrate active process like this case.

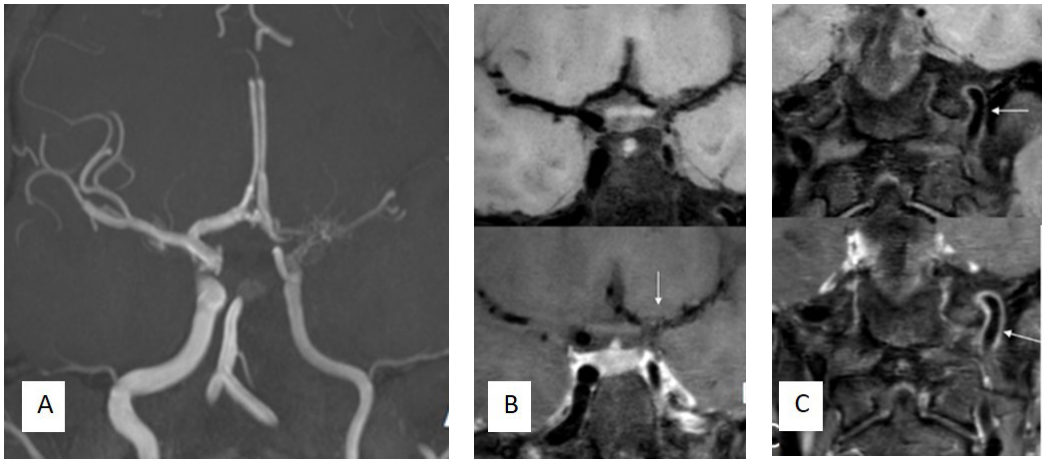

Fig 5. case 8 patient with Takayasu arteritis (A) MRA shows severe stenosis of left distal ICA with moya-like pattern, (B) VWI shows no enhancement of the arteries around the stenotic left ICA (arrow), but enhancement of wall along the more proximal left ICA is noted (arrow in C) and small size of the artery.

Aneurysm and instrument placement

Case 9: DSA showed right superior hypophyseal aneurysm before and after coiling. VWI showed small focal signal void at the aneurysm site with wall enhancement. CE-MRA confirmed small residual neck of aneurysm at the lesion.

Case 10: DSA showed before and post coiling aneurysm at left anterior communicating artery. VWI showed signal void of the aneurysm, suggestive of patent lumen. The aneurysmal wall showed no definite enhancement. However, CE-MRA showed no residual lumen of the aneurysm. This might be artifact from coil on VISTA.

Case 11 (Fig 6)**:** DSA showed post coiling basilar tip aneurysm with stent placement in BA. VWI showed signal void of the BA lumen which appeared larger than demonstrated on CE-MRA. This may be blooming effect from stent in the basilar lumen. The BA wall was enhanced suggestive of inflammatory process. The coiling aneurysm was heterogeneous signal on both VISTA and CE-MRA. Aneurysmal wall enhancement was clearly seen.

Case 12 (Fig 7)**:** Incidental finding of calcified aneurysm at right M2 MCA on CTA. VWI showed patent lumen of the aneurysm. The calcification appeared dark on VISTA. No enhancement of the aneurysm wall implied an inactive one.

Comment (Cases 9-12): VWI has demonstrated thick peripheral wall enhancement in ruptured aneurysm and predicted high risk ruptured one.[35,36] We demonstrated one large calcified aneurysm at right MCA with no enhancement of the wall (case 12). This might suggest low risk rupture. We have been following up this patient up to now. For the other cases, the study was performed after coiling. Enhancing aneurysmal wall might be from foreign material or manipulation during intervention.

Patent vascular lumen is dark signal on VISTA. Calcification is also dark signal on VWI.[1] However, metallic material could cause signal void too which was demonstrated in cases 10 and 11. Using VWI for follow up of aneurysmal lumen in post coiling or stenting should be considered. Up to our current knowledge, this finding has not been reported yet.

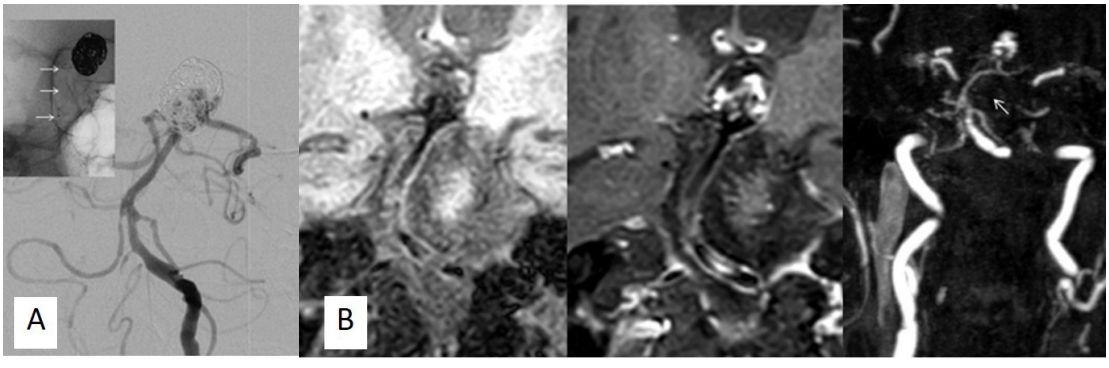

Fig 6. Post coiling aneurysm (A) DSA of case 11 shows coiling and stent in the aneurysm and basilar artery, (B) VWI T1W(left), Gd-T1W (middle) demonstrate patency of the BA not corresponding with stenotic lumen on CE-MRA(right).

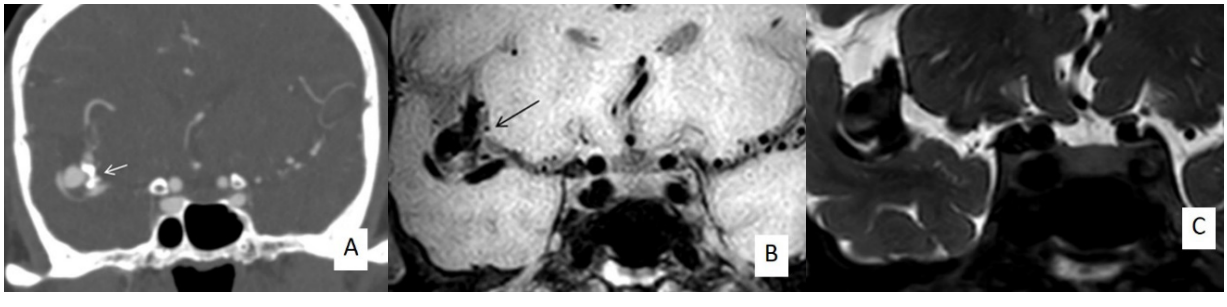

Fig 7. Case 12 patient with calcified aneurysm at right MCA (A) CT shows calcification at the wall of the aneurysm, (B, C) VWI show low signal of the calcification which may be missed as patent lumen.

CONCLUSION

Vessel wall imaging with MRI is a useful method for demonstrating the pathophysiology of the intracranial vascular disease. The findings add on the information in clinical management of the patients. With advancement of the MR technology, better high resolution of the vascular wall would be achieved. Important pitfalls should be aware in selected cases. Radiologists should get familiar with the signal of the findings and inform the clinicians that the technique is not validated yet. Clinical context is still highly needed for final conclusion.

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