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The Utility of Vessel Wall Imaging in the Postulation of Acute Ischemic Stroke With Spontaneous Recanalization Pathophysiology

Mohamad Syafeeq Faeez Md Noh, MD, MMed (Radiology),*† Anna Misyail Abdul Rashid, MD, MMed (Internal Medicine),‡§ Fan Kee Hoo, MD, MRCP (UK),†¶ and Norafida Bahari, MD, MRad†||

Abstract: Recent advances in technology, particularly in the field of magnetic resonance imaging, have brought forth new sequences, including vessel wall imaging (VWI). Traditionally, the workup for intracranial vascular pathology has always turned to luminal imaging using computed tomography angiography, magnetic resonance angiography, or digital subtraction angiography. Since its introduction, VWI has enabled researchers and practicing clinicians to better understand disease processes and manage patients to the best standard of care possible. Spontaneous recanalization in acute ischemic stroke (AIS) is a known but understudied phenomenon. Available literature has looked at this phenomenon and postulated the occurrence based on conventional cross-sectional imaging and angiography; however, objective evidence pointing to the occurrence of this phenomenon is scarce. We would like to share our experience using VWI in a patient who was clinically suspected to have a middle cerebral artery syndrome at onset, with resolution of the symptoms 3 hours after initial presentation. VWI showed vessel wall enhancement at the suspected vessel involved, with evidence of acute infarcts at the vascular territory supplied. A presumptive diagnosis of AIS with spontaneous recanalization was made. Our experience could potentially aid in the understanding of spontaneous recanalization in patients with AIS, particularly in the postulation of the pathophysiology.

Key Words: acute ischemic stroke, magnetic resonance imaging, spontaneous recanalization, vessel wall imaging

(Top Magn Reson Imaging 2022;31:40-42)

From the *Department of Radiology, Faculty of Medicine and Health Sciences, Universiti Putra Malaysia, Serdang, Selangor, Malaysia; †Department of Radiology, Universiti Putra Malaysia (UPM) Teaching Hospital, Persiaran MARDI-UPM, Serdang, Selangor, Malaysia; ‡Department of Neurology, Faculty of Medicine and Health Sciences, Universiti Putra Malaysia, Serdang, Selangor, Malaysia; \$Department of Neurology, Universiti Putra Malaysia (UPM) Teaching Hospital, Persiaran MARDI-UPM, Serdang, Selangor, Malaysia; ¶Department of Neurology, Faculty of Medicine and Health Sciences, Universiti Putra Malaysia, Serdang, Selangor, Malaysia; and ∥Department of Radiology, Faculty of Medicine and Health Sciences, Universiti Putra Malaysia, Serdang, Selangor, Malaysia.

Received for publication June 29, 2022; revision received July 30, 2022; accepted August 2, 2022.

Address correspondence to Mohamad Syafeeq Faeez Md Noh, MD, MMed (Radiology), Department of Radiology, Faculty of Medicine and Health Sciences, Universiti Putra Malaysia, 43400 Serdang, Selangor, Malaysia (e-mail: msf.mdnoh@gmail.com).

The authors declare no conflicts of interest.

- Written informed consent was obtained for the publication of this case report and clinical details from the patient.
- Our institution does not require ethical approval for the publication of single case studies.
- No funding was received for this work.
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DOI: 10.1097/RMR.000000000000298

Spontaneous recanalization in acute ischemic stroke (AIS) is a known but understudied phenomenon. The mechanisms bringing about spontaneous recanalization are unclear to this day. Although some studies have suggested possible involvement of the autologous fibrinolysis system, development of collateral vessels favoring spontaneous recanalization, and the contributory role of hypertension, all these remain hypothetical. Although previous studies using conventional imaging methods have attempted to document evidence of its occurrence, studies using advanced imaging methods, such as vessel wall imaging (VWI), are lacking. We would like to share our experience using VWI in a patient with AIS with spontaneous recanalization, particularly in the postulation of the pathophysiology.

CASE REPORT

A 66-year-old man was referred to us from an outside hospital after an acute onset of syncope. He was found by family members on the floor, being confused. He had no similar episodes before. On examination at the emergency department, 3 hours after symptom onset, his Glasgow Coma Scale score was 10 (E3 V1 M6). His blood pressure was 160/90 mm Hg, and he was not hypoglycemic. There was right-sided facial asymmetry, and the patient was aphasic. Mild right-sided limb weakness was noted. His National Institutes of Health Stroke Scale (NIHSS) score was noted to be 5. A possible middle cerebral artery (MCA) syndrome was suspected. No imaging at the outside center was performed, and no intravenous (IV) thrombolysis was administered. He was referred to us for further management and potential administration of intravenous thrombolysis with a view to mechanical thrombectomy. However, during transfer to our center, his symptoms resolved. Reassessment of his neurological symptoms gave a NIHSS score of 0. Magnetic resonance imaging was performed at approximately 3.5 hours after onset of symptoms as per our institution's protocol, which showed acute infarcts at the left head of caudate nucleus and smaller infarcts at the left parietal lobe (Fig. 1). VWI showed noneccentric vessel wall thickening and enhancement involving the M1 segment of the left MCA, with no evidence of an atherosclerotic plaque. No large or medium vessel occlusion was noted on magnetic resonance angiography (Fig. 2). In view of the resolution of his clinical symptoms and the imaging findings, no acute stroke intervention with either IV thrombolysis or mechanical thrombectomy was pursued. He was admitted to our center for observation and optimization of his medical illness and was eventually discharged well.

DISCUSSION

AIS is a common disease affecting people worldwide, which is usually attributed to an arterial occlusion. Persons afflicted with AIS tend to have a high rate of disability and mortality. Modern medicine



FIGURE 1. (A) DWI shows hyperintensity at the left head of caudate nucleus (white arrow). (B) DWI shows hyperintense foci involving the left parietal region (white circles). (C) ADC shows corresponding hypointensity at the left head of caudate nucleus, in keeping with acute infarct. (D) ADC shows corresponding hypointense foci involving the left parietal region, in keeping with acute infarcts. ADC, apparent diffusion coefficient; DWI, diffusion-weighted imaging.

has enabled physicians to offer hyperacute stroke treatment, with intravenous thrombolysis and/or mechanical thrombectomy. The idea behind this is to relieve the vascular occlusion as urgently as possible so as to save as much viable brain tissue as possible, limiting disability and/or complications.

However, there have been instances where the vascular occlusion seems to self-recanalize, a phenomenon termed spontaneous recanalization. This phenomenon, seen in patients with AIS, is rather understudied and poorly understood, despite advances in modern medicine. Previous studies have attempted to prove the occurrence of this phenomenon, albeit being limited by a variety of factors. One such study looked at spontaneous recanalization in patients who have undergone cerebral angiography and noted that the actual rate occurs in approximately 17% of patients, with much more cases being confounded by nonocclusion rates.¹ In other studies, the presence of intracranial atherosclerotic plaques enables a clearer diagnosis to be made, with the plaque still being present, and the stenosis or occlusion improved from baseline after clinical improvement and imaging evidence of spontaneous recanalization.²



FIGURE 2. (A) Postgadolinium VWI sequence shows noneccentric vessel wall thickening and enhancement involving the M1 segment of the left MCA vessel (white circle). Comparing that with the normal, contralateral MCA vessel, this shows no vessel wall enhancement. (B) TOF MRA shows no evidence of large or medium vessel occlusion on the left side (white circle). MCA, middle cerebral artery; MRA, magnetic resonance angiography; TOF, time of flight; VWI, vessel wall imaging.

		FOV	Slice	Gap	TR	ТЕ		FA			NSA/	Total Time
MRI sequence	Image Plane	(mm)	(mm)	(mm)	(ms)	(ms)	SNR	(°)	Mode	Matrix	NEX	(min:s)
DWI	Axial 2D	230×230	5	1	3213	86	1	90	SE	192	1	1:46
FLAIR	Axial 2D	230×230	4	1	9000	144	1	90	TSE	320	1	2:42
MRA	Axial	200×200	180	-0.6	22	3.5	1	18	FFE	512	1	4:36
	TOF (3D)											
SWI	Axial 3D	230×189	145	-1	31	7.2	1	17	FFE	672	1	4:59
REACT	Coronal 3D	350×488	3	-1.5	3.7	0	1	12	TFE	448	2	3:56
EPI-dynamic	Axial 2D	224×224	4	1	9000	144	1	75	FFE	128	1	2:27
(postgadolinium perfusion)												
VWI/black blood	Axial 3D	200×251	475	-0.4	700	35	1	8	SE	432	2	5:30
(postgadolinium)												

2D indicates two-dimensional; 3D, three-dimensional; DWI, diffusion-weighted imaging; EPI, echo planar imaging; FA, fractional anisotropy; FLAIR, fluidattenuated inversion recovery; FOV, field of view; MRA, magnetic resonance angiography; NSA/NEX, number of signal averages/number of excitations; SNR, signal-to-noise ratio; TE, time to echo; TOF, time of flight; TR, repetition time; VWI, vessel wall imaging.

In cases where there is no imaging evidence of a plaque or stenosis, it is understandably difficult for clinicians to diagnose spontaneous recanalization when patients initially presenting with AIS suspected to have vascular occlusion improve dramatically, but no imaging evidence is present to verify the site of occlusion. This task is likely not achievable using conventional imaging methods. A potential method to provide a solution to this dilemma noninvasively, based on our experience, is VWI.

VWI is a relatively new MRI technique that acts as an adjunct to conventional angiographic imaging using computed tomography angiography, magnetic resonance angiography, or digital subtraction angiography. Apart from luminal imaging that is achieved by these conventional angiographic imaging techniques, it is also important to be able to image abnormalities occurring in the vessel wall. This is made possible by VWI, with the following principle technical requirements, to be clinically useful—high special resolution, multiplanar 2D or 3D acquisitions, multiple tissue weightings, and suppression of signal in luminal blood and cerebrospinal fluid.³

VWI and enhancement on MRI have been used in the workup and prognostication of clinical conditions such as vasculitis, dissections, Moyamoya disease, and intracranial aneurysms.⁴ In the field of AIS, a huge interest in intracranial atherosclerotic disease has meant a wider usage of VWI.⁵ Our institution's stroke protocol⁶ routinely incorporates VWI for the imaging of patients clinically presenting with acute stroke, with the scan parameters presented in Table 1.

The VWI finding in our case was noneccentric thickening and enhancement of the M1 segment of the left MCA vessel compared with the contralateral side. Based on the pattern of acute infarcts on DWI, it is possible that the initial insult was multiple thromboembolism affecting the anterior and the media (distal M1/proximal M2) segment) territories. Typically, intracranial atherosclerotic disease presents with VWI features of eccentric wall thickening because of the presence of the atherosclerotic plaque, while patients with vasculitis tend to have a more uniform, concentric vessel wall thickening and enhancement when imaged with VWI. However, these features are not true in all cases. Mandell et al,⁷ in their study which looked at the VWI features of recanalized vessels either using mechanical thrombectomy or medical therapy, showed that patients had noneccentric vessel wall enhancement and thickening of the involved, recanalized vessels, when imaged with VWI. The VWI features were compared with the contralateral, nonaffected vessel, which was what we observed in this case.

Taking into consideration the patient's initial symptoms, the NIHSS score at onset (n = 5), and the revised score at our center (n = 0), we postulated that there was a high likelihood of a transient large vessel occlusion involving the M1 segment of the left MCA vessel, with spontaneous recanalization while being transferred to our center. This was evident by the noneccentric wall enhancement and thickening seen with no plaque present and the acute infarcts on the DWI/ADC sequence. We do acknowledge that this is a single case study, which would need to be corroborated by larger studies in the future. It is with exactly that in mind that we hope that our limited experience opens up more areas of study and that VWI is routinely incorporated in the practice of stroke medicine in many more centers throughout the world.

In conclusion, VWI could potentially serve as an imaging method to presumptively diagnose spontaneous recanalization in patients with AIS. In cases where there is no clear imaging evidence of stenosis or plaque using conventional methods, evidence of wall enhancement at the suspected vessel, with acute infarcts involving the vascular territory supplied, combined with the clinical signs and symptoms may suggest the site of initial occlusion. More studies are needed to validate this theory.

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