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Vessel Wall Imaging in the Management of Subarachnoid Hemorrhage and Multiple Intracranial Aneurysms

Andrew J. Steven, MD,[1] James M. Milburn, MD,[2] Paul Gulotta, MD,[2] Dheeraj Gandhi, MD[1]

[1]Department of Radiology, University of Maryland Medical System, Baltimore, MD [2]Department of Radiology, Ochsner Clinic Foundation, New Orleans, LA

INTRODUCTION

Aneurysmal subarachnoid hemorrhage is a significant cause of morbidity and mortality, with a mortality rate approaching 25% and high rates of permanent neurologic deficits in survivors.[1] A high priority in the management of these critically ill patients is to identify the site of rupture and urgently secure the aneurysm, either through endovascular coiling or microsurgical clipping. The ruptured aneurysm should be treated as early as reasonably possible to prevent a potentially catastrophic rehemorrhage.[1,2]

The presence of multiple intracranial aneurysms in the setting of aneurysmal subarachnoid hemorrhage, which occurs in 15%-35% of patients, can create a significant dilemma in the management of these patients because determining the most likely site of rupture and which aneurysm requires immediate treatment can be difficult.[3] Assessment has traditionally relied on the distribution of subarachnoid hemorrhage and localizing the cranial neuropathy and any focal hematomas in relation to the aneurysms. The size and morphology of the aneurysm may also help determine the culprit lesion, with larger size and irregular shape being more suspicious. While certainly helpful, these methods are indirect and inexact.

Magnetic resonance vessel wall imaging (MR-VWI), an emerging technique for evaluating intracranial vascular disease, may offer additional benefit in the workup of patients with subarachnoid hemorrhage.[4,5] By imaging the wall of the vessel, as opposed to the lumen and intraluminal contents, this technique can identify a site of focal inflammation and offers an alternative method of characterization to traditional angiography techniques.

We present the case of a patient with subarachnoid hemorrhage and multiple intracranial aneurysms followed by a brief discussion of the basic principles and other clinical uses of MR-VWI.

HISTORY

A 49-year-old female with a history of hypertension and hypothyroidism developed a sudden-onset, severe headache while undergoing an ultrasound-guided thyroid biopsy at an outside institution. The patient was diagnosed with an acute hypertensive episode (190 mmHg systolic) and subarachnoid hemorrhage and was subsequently transferred for treatment. During presentation to the neurocritical care unit, the patient was increasingly somnolent yet arousable. Physical examination demonstrated a right-sided ptosis but no other focal neurologic deficit. Her aneurysmal subarachnoid hemorrhage was considered a Hunt and Hess Grade 3, portending a high risk of morbidity and mortality.[2]

RADIOGRAPHIC APPEARANCE AND TREATMENT

Initial imaging included a noncontrast computed tomography (CT) of the brain, a CT angiogram of the head, and a catheter-based digitally subtracted angiogram (Figure 1) that demonstrated a sizable volume of hemorrhage throughout the subarachnoid space, centered in the suprasellar cistern. The hemorrhage extended into the interhemispheric fissure and bilateral Sylvian fissures. The angiograms identified two discrete aneurysms arising from the left anterior cerebral artery, one in the proximal A1 segment and the other at the A1/A2 junction at the level of the anterior communicating artery. Both aneurysms were small, measuring 3-4 mm. Neither exhibited significant irregularity or a pointed apex (Murphy teat) to specifically indicate the site of recent rupture. The distribution of blood suggested the A1/A2 lesion, as we observed a small hematoma in the interhemispheric fissure, but the site of rupture could not be identified with certainty.

The patient subsequently underwent high-resolution MR-VWI using a T1-weighted black-blood vessel wall sequence (turbo spin echo acquisition: field of view 160 $\times$ 160 mm, acquired matrix 512 $\times$ 512, slice thickness 2 mm, repetition time/echo time 550/11 ms) before and after the administration of gadolinium-based intravenous contrast (Figure 2). Our study demonstrated unequivocal T1-hyperintensity suggestive of blood products in the wall of the A1/A2 junction lesion. Prominent enhancement at this site was observed on postcontrast imaging, confirming the A1/A2 junction as the probable site of rupture. No intramural hemorrhage or wall enhancement was found in the proximal aneurysm.

After detailed consultation with neurosurgical and neurointervention staff, the patient and her family elected endovascular repair. The ruptured aneurysm was coiled first, and the A1 aneurysm was treated during the same intervention. The patient spent 16 days in the critical care service and was eventually transferred to a rehabilitation

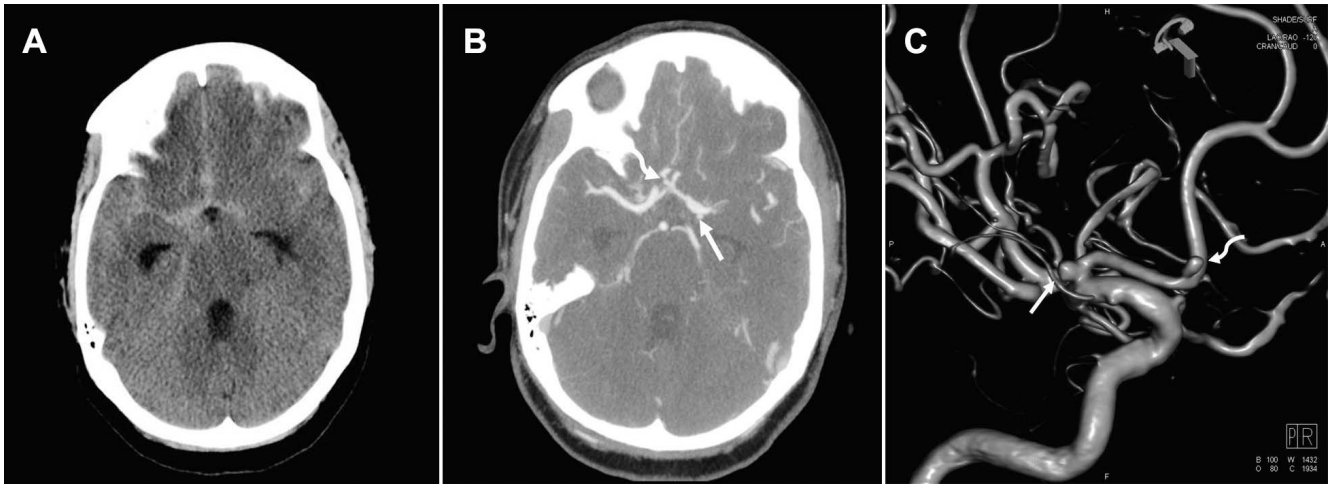

Figure 1. A: Axial noncontrast computed tomography (CT) demonstrates a sizable volume of subarachnoid hemorrhage centered in the suprasellar cistern with extension into the interhemispheric fissure and Sylvian fissures. B: Axial maximum intensity projection reconstruction from a corresponding CT angiogram shows 2 aneurysms arising from the left anterior cerebral artery (straight arrow and curved arrow). C: Three-dimensional reconstruction from a follow-up catheter-based digitally subtracted angiogram shows the proximity of the 2 lesions (straight arrow and curved arrow).

facility with only mild residual deficits. No evidence of rerupture was observed during the hospitalization.

DISCUSSION

Traditional angiography techniques including CT angiography, magnetic resonance angiography, and catheter-based angiography focus on evaluating the lumen and intraluminal contents of blood vessels. While each technique has strengths and limitations, they all rely on evaluating the caliber and contour of the vessels to establish diagnoses, often making inferences on the pathologic process involving extraluminal tissues.

MR-VWI is an emerging technique focused on characterizing the arterial wall.[6,7] The high spatial resolution required to image the arterial wall, measuring only 1-2 mm even in the largest intracranial vessels, has been a significant limitation. However, the technique has evolved with improved magnetic resonance hardware and software. Imaging the vessel wall requires suppressing the intraarterial signal, a so-called *black-blood* technique. The cerebrospinal fluid signal must also be suppressed. Thus, a typical examination will employ a double inversion recovery sequence, acquiring T1-weighted images before and after the administration of intravenous contrast. Both 3-dimensional and 2-dimensional techniques are available.

Early reports demonstrated some utility in evaluating inflammatory conditions such as central nervous system vasculitis, reversible cerebral vasoconstriction syndrome, or active atherosclerotic plaque.[8-11] It is believed that inflammation of the endothelial lining allows gadolinium contrast to leak, causing apparent vessel wall thickening and enhancement on postcontrast images.[4] This enhancement is readily visualized on a black-blood magnetic resonance angiogram sequence where intraluminal blood and contrast are reflected as a low signal flow void.

MR-VWI has notable limitations. As is the case with many magnetic resonance sequences, motion can severely degrade image quality and induce artifact. Obtaining high-quality images in critically ill patients can prove challenging and may require anesthesia. Normal enhancement of adjacent venous structures or slow flow within the periphery of the lumen can simulate arterial wall enhancement, thus producing false-positives.

The literature on MR-VWI is scant and limited to a few case reports. Horie et al described an intraluminal thrombus reflected by a T1-hyperintense signal within the wall of a ruptured aneurysm on precontrast images, similar to our case.[12] The T1 hyperintensity is presumably reflective of subacute blood products, known to cause intrinsic T1 shortening. Matouk et al presented a small case series in which all recently ruptured aneurysms exhibited thick aneurysm wall enhancement thought to reflect gadolinium leakage from disruption of the endothelium.[4] Our case exhibited both of these findings. In our case, we decided to also coil the unruptured aneurysm because of its location in the same arterial distribution and favorable morphology for coil embolization. However, coiling both aneurysms is not always possible in clinical practice. Some aneurysms are extremely difficult to coil because of factors such as small size, large neck, and presence of incorporated branch vessels. In these cases, the treatment of the presumed unruptured aneurysms can be deferred to a later date when dual antiplatelet agents and other adjunctive devices such as neck-bridging stents or flow diverters can be used.

Although much work remains to be done to establish the validity of MR-VWI, the important imaging findings and potential clinical utility of this technique are clearly illustrated in our case.

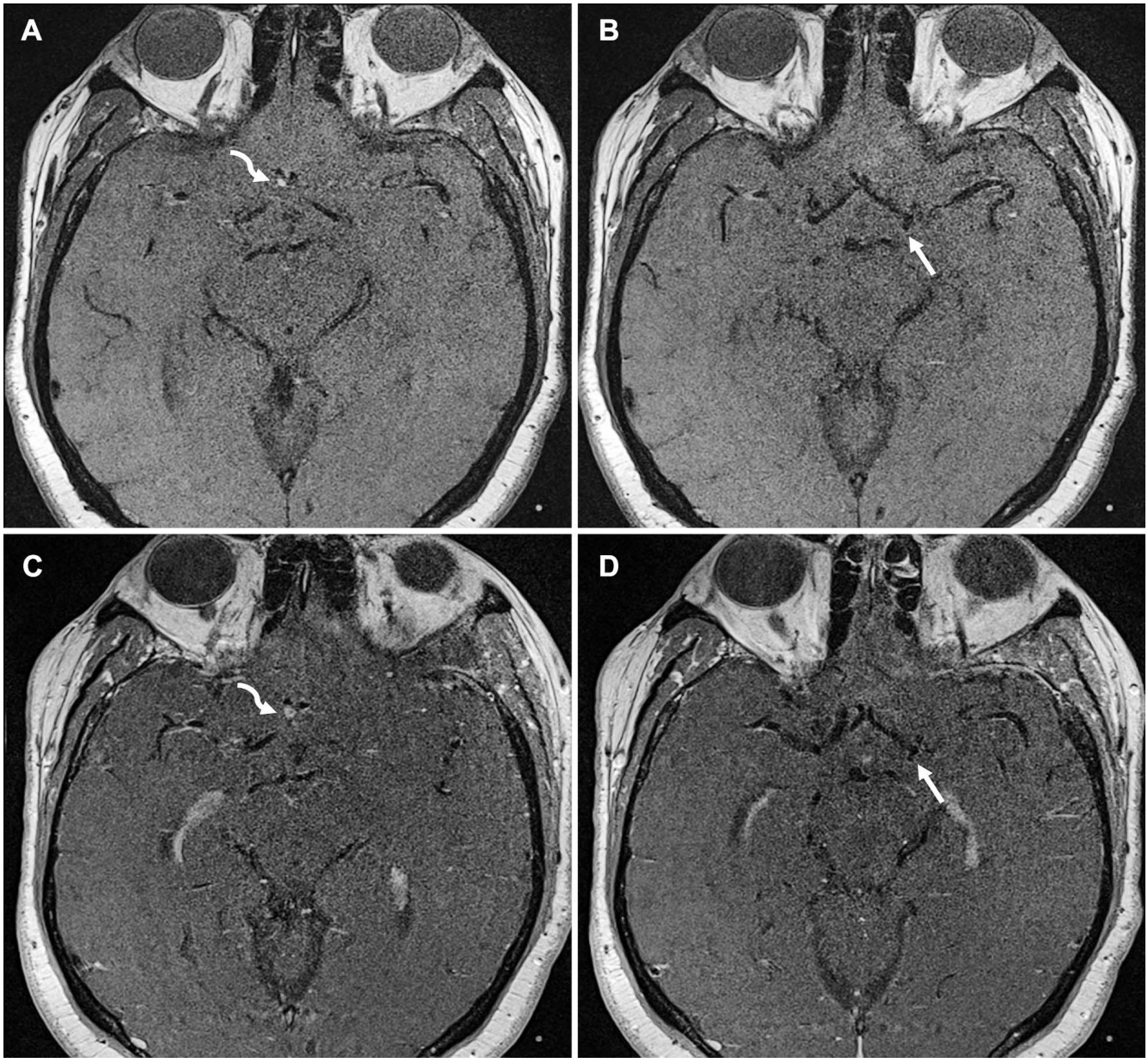

Figure 2. A and B: Precontrast images from a black-blood magnetic resonance angiogram show increased signal along the margin of the aneurysm centered at the A1/A2 junction (curved arrow), with a normal flow void in the proximal aneurysm (straight arrow). C and D: Postcontrast images exhibit focal enhancement of the wall of the A1/A2 junction aneurysm (curved arrow), while the proximal lesion shows none (straight arrow).

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