Giant cell arteritis mimicking spontaneous bilateral vertebral dissections and internal carotid artery fibromuscular dysplasia

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DESCRIPTION

A woman in her 70s presented to the emergency department with a 5-day history of feeling unsteady. Based on CT angiography (CTA) findings, she was diagnosed with left vertebral artery dissection (VAD) involving segments V2 and V3 with underlying fibromuscular dysplasia (FMD). MRI of the brain revealed acute left thalamic ischaemic stroke. Hence, dual antiplatelet therapy (DAPT) with aspirin and clopidogrel was initiated.

Three weeks later, she returned with acute onset left-sided hemiplegia and ataxia. Head CT showed left thalamus subacute infarcts. Head and neck CTA showed interval progression of the left vertebral artery (VA) irregularities, and new multifocal irregularities of the right VA (figure 1), resulting in occlusion at the V2/V3 junction. The anterior circulation redemonstrated mild multifocal beading of the bilateral internal carotid arteries (figure 1). MRI revealed acute infarcts involving the right ventral pons and left middle cerebellar peduncle. DAPT was switched to a heparin drip on admission for evaluation.

The patient also reported an episode of abdominal pain and 15-pound weight loss 3 months prior and a week of bitemporal headaches 2 weeks before presentation. She denied fevers, night sweats, vision changes or arthralgias.

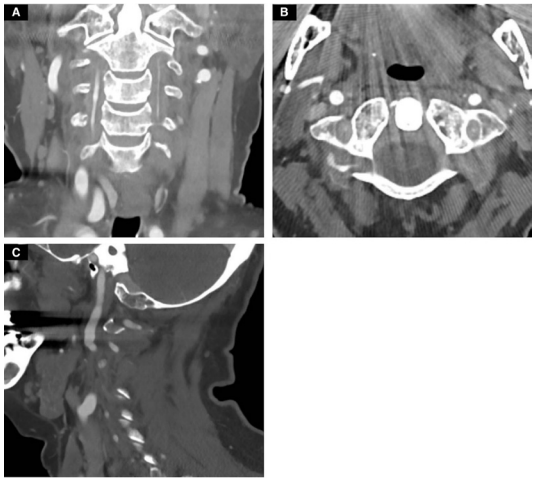

Figure 1 (A) Multifocal irregularity of the bilateral V2 vertebral artery, resulting in occlusion at the right V2/V3 junction (B). Sagittal view of right internal carotid artery showing mild multifocal beading (C).

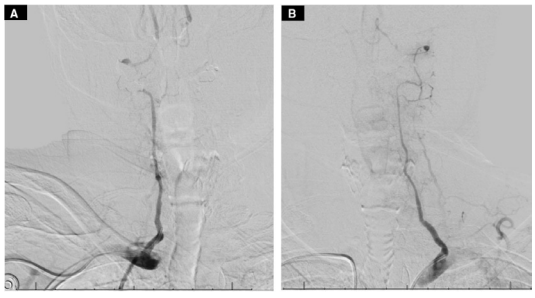

Figure 2 Multifocal areas of narrowing involving (A) the right vertebral artery and (B) the left vertebral artery.

Neurological examination revealed left-sided hemiparesis. Laboratory tests showed normocytic anaemia (haemoglobin 117 g/L), thrombocytosis (486×10^3/μL), elevated C reactive protein (5 mg/dL) and erythrocyte sedimentation rate (ESR) of 56 mm/hour. Digital subtraction angiography revealed multifocal areas of narrowing involving the basilar artery and bilateral VA segments (figure 2). These vessels showed post-contrast circumferential vessel wall enhancement on vessel wall MRI (figure 3). Abdominal and pelvic CTA indicated soft tissue thickening of the distal abdominal aorta and proximal right common iliac artery (figure 4). The patient met three of the five criteria listed by the American College of Rheumatology 1990 (age ≥50 years, new headache and elevated ESR), and was diagnosed with giant cell arteritis (GCA).[1] The patient received intravenous methylprednisolone 1 g for 5 days, followed by tocilizumab and oral prednisone taper starting at 60 mg. Heparin was discontinued and DAPT was re-initiated with the plan to continue clopidogrel for 30 days. The patient was discharged

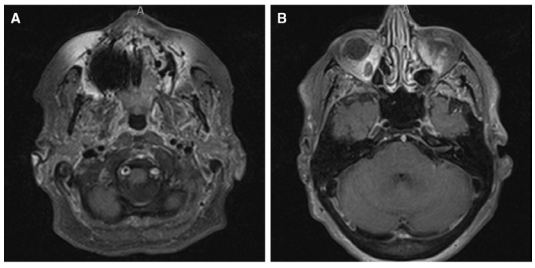

Figure 3 MRI showing post-contrast circumferential vessel wall enhancement (A) in bilateral vertebral arteries and (B) in the basilar artery.

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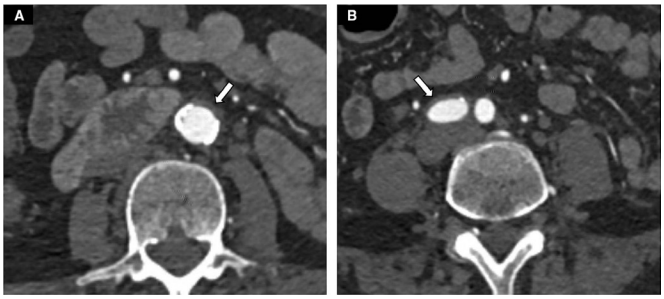

Figure 4 Soft tissue thickening in the distal abdominal aorta (A) and proximal right common iliac artery (B) without adjacent fat stranding or abnormal enhancement suggesting active inflammatory vasculitis.

Patient's perspective

A few months before all of this started, I was having some abdominal pain. I thought it was maybe acid reflux as I have had some episodes of heart burn in the past. I used to have trouble swallowing as well with all of the acid so it made it hard for me to eat. I ended up changing my diet to more fiber and protein and noticed that my heart burn went away! And because of all of that, I've lost about 15-pounds in the last couple of months. In addition to all of this, I started noticing some headaches around my temples, but I thought it could have maybe been related to ear infection.

Learning points

- Giant cell arteritis is a rare cause of stroke, especially in the vertebrobasilar territory.
- Vessel wall MRI can help differentiate vasculitis from other causes of intracranial arterial narrowing.
- Giant cell arteritis should be considered in elderly patients with spontaneous vertebral artery dissection-like presentation.

to a rehabilitation facility in stable condition after 1 week of hospitalisation.

The presence of underlying vasculopathy can be associated with non-traumatic spontaneous VAD, a rare cause of posterior circulation stroke.[2] FMD can present as an underlying cause of VAD, exhibiting the 'string of beads' sign on vessel imaging, usually seen in middle-aged individuals.[3] GCA is typically seen in adults older than 50 years and is a rare cause of vertebrobasilar territory stroke.[4] In this case, the rapid progression of our patient's condition warranted further evaluation. We were able to extract her history of weight loss and temporal headache, and further work-up revealed elevated inflammatory markers and signs of vertebrobasilar artery inflammation. This case of GCA, which initially mimicked the findings of FMD and VAD, highlights the importance of reconsidering the diagnosis when patients develop unexpected clinical courses. GCA should be considered in elderly patients with spontaneous VAD-like presentation.

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

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