

A case of giant cell arteritis mimicking vertebral dissection on contrast-enhanced magnetic resonance angiography

Sirs,

A 78-year-old woman presented with a 2-week history of temporal headache and anaemia (haemoglobin, 8.5 g/dL). She had blood vessel swelling, tenderness, and pulsatile pain in the temporal region and jaw claudication. We suspected giant cell arteritis (GCA) and she was referred to our hospital in July 2016. The initial laboratory findings revealed an inflammatory response (C-reactive protein, 8.28 mg/dL and erythrocyte sedimentation rate, 135 mm/h). She met the American College of Rheumatology's criteria for temporal arteritis based on her age, presence of a new headache, temporal artery abnormality, and elevated inflammatory markers (1). Magnetic resonance imaging (MRI) performed on day 5 after admission incidentally revealed an acute infarction in the left cerebellar hemisphere, so we started antiplatelet therapy with aspirin. Contrast-enhanced (CE) three-dimensional (3D) time-of-flight (TOF) angiography source images showed a crescent-shaped high-signal area along the left vertebral artery to the basilar artery, which suggested acute arterial dissection with a mural haematoma (Fig. 1A). However, the findings had low intensity, and the lumen in this area was not clearly visible on plain 3D-TOF angiography images during the follow-up MRI performed on day 12 (Fig. 1B). These findings suggested a thickened arterial wall with enhancement, compatible with a diagnosis of vasculitis. Retrospectively, we observed mild vessel wall thickening and enhancement around the right occipital artery on a previous MRI (Fig. 1C). Abdominal computed tomography angiography on day 12 showed vessel wall thickening in both femoral arteries, the abdominal aorta, superior mesenteric artery, and both common iliac arteries. We confirmed the GCA diagnosis based on findings and the slightly elevated pentraxin 3 levels (8.42 ng/mL) (2). We began treatment with prednisolone 30 mg/day, and her symptoms and inflammatory response improved immediately. The vessel wall thickening around the vertebral and occipital arteries was improved on follow-up MRI 3 weeks after treatment initiation (Fig. 1D). MRI is a non-invasive diagnostic tool that can assess the vascular involvement in GCA. On MRI, mural thickening and contrast enhancement of the vessel wall indicate typical signs of inflammatory vessel processes (3). CE 3D-TOF angiography source images have recently been demonstrated to be useful for assessing cervical arterial dissection. It can analyse the abnormalities of the vessel lumen (occlusion,

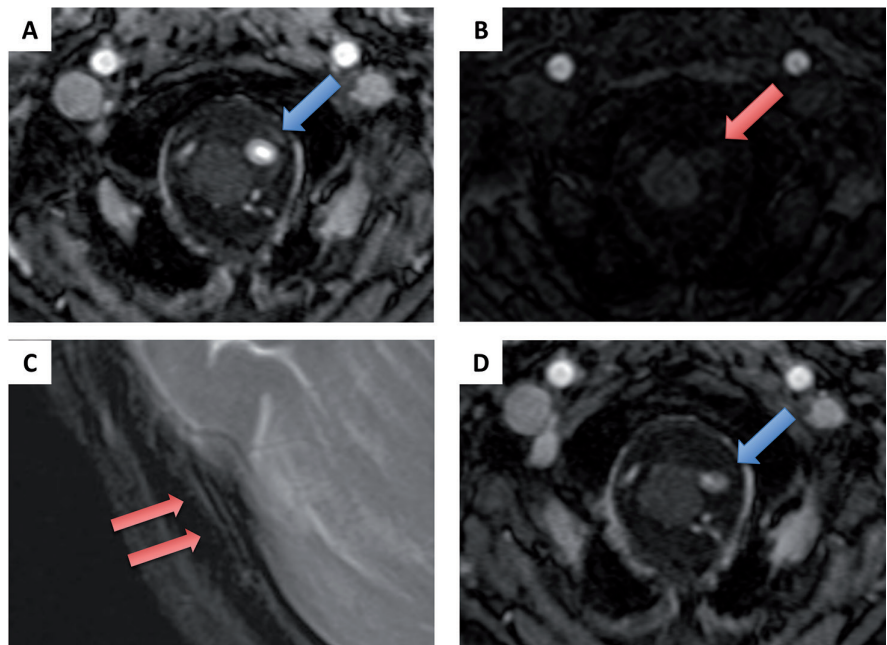


Fig. 1. Brain magnetic resonance imaging and three-dimensional time-of-flight magnetic resonance angiography (3D-TOF MRA) source images.

A. Contrast-enhanced (CE) 3D-TOF MRA source image on day 5 shows a crescent-shaped high-signal area along the left vertebral artery (blue arrow), suggesting a mural haematoma.
B. Plain 3D-TOF MRA source image on day 12. The lumen of the left vertebral artery is not clear (red arrow).
C. Fat-suppressed T2-weighted image on day 5. The faint high-signal area around the right occipital artery (red arrow) suggests vessel wall thickening.
D. CE 3D-TOF MRA source image on day 40. The mural thickening and enhancement are improved (blue arrow).

stenosis, and luminal thrombus) and vessel wall (crescent mural haematoma, pseudoaneurysm, double lumen, and intimal tear) of the dissected artery. Mural haematomas show high intensity with all pulse sequences at the acute phase (4). CE 3D-TOF angiography source images can also show both vessel wall thickening accompanied by enhancement and vessel lumen contraction of the inflamed artery (5). Therefore, this tool is also useful for evaluating inflammatory changes in systemic vasculitis, such as GCA.

In our case, the crescent-shaped high-signal area along the left vertebral artery was originally thought to be a mural haematoma. However, since it was not hyperintense on plain 3D-TOF angiography source images on day 12, we concluded that it was not a mural haematoma, but mural thickening with enhancement. The improvement in these findings after the initiation of corticosteroid also indicated their compatibility with vasculitic changes. Moreover, a comparison of CE and plain 3D-TOF angiography source images was useful for differentiating between vasculitis and arterial dissection. Both mural haematomas and mural thickening are hyperintense on CE MRA, while haematomas have high intensity compared to the low-intensity vascular wall on plain MRA.

We experienced a case of GCA in which mural thickening of the vertebral artery showed mural haematoma-like findings on

MRA. We cautiously suggest that the vasculitic findings of GCA can mimic dissection on CE MRA. Thus, comparing CE and plain 3D-TOF angiography source images might be useful for differentiating between arterial dissection and vasculitis.

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