

Why a standard contrast-enhanced MRI might be useful in intracranial internal carotid artery stenosis

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Abstract

In patients with ischemic stroke of unknown cause cerebral vasculitis is a rare but relevant differential diagnosis, especially when signs of intracranial artery stenosis are found and laboratory findings show systemic inflammation. In such cases, high-resolution T1w vessel wall magnetic resonance imaging (MRI; 'black blood' technique) at 3 T is preferentially performed, but may not be available in every hospital. We report a case of an 84-year-old man with right hemispheric transient ischemic attack and signs of distal occlusion in the right internal carotid artery (ICA) in duplex sonography. Standard MRI with contrast agent pointed the way to the correct diagnosis since it showed an intramural contrast uptake in the right ICA and both vertebral arteries. Temporal artery biopsy confirmed the suspected diagnosis of a giant cell arteritis and dedicated vessel wall MRI performed later supported the suspected intracranial large artery inflammation. Our case also shows that early diagnosis and immunosuppressive therapy may not always prevent disease progression, as our patient suffered several infarcts in the left middle cerebral artery (MCA) territory with consecutive high-grade hemiparesis of the right side within the following four months.

Keywords

Intracranial internal carotid artery stenosis, cranial magnetic resonance imaging, giant cell arteritis, cerebral vasculitis, ischemic stroke

Introduction

Symptomatic intracranial stenosis accounts for up to 10% of ischemic strokes in white patients.¹ It is mostly caused by atherosclerosis and diagnosed by duplex ultrasound and magnetic resonance or computed tomography (CT) angiography.²

About 5% of patients with giant cell arteritis (GCA) face cerebrovascular ischemic events.^{3,4} Until recently, intracranial vessel involvement in GCA was believed to occur only in rare cases.^{2,5} For diagnostic imaging in GCA, the implementation of a dedicated high-resolution 3 T vessel wall magnetic resonance imaging (MRI) protocol using pre and post contrast-enhanced T1w sequences with nulling of the signal of flowing blood ('black blood' technique) in addition to the color-coded duplex sonography is commonly recommended,^{6,7} but not routinely available in standard stroke centers.

This case illustrates that also a standard contrast-enhanced MRI can point the way to the correct diagnosis of intracranial internal carotid artery (ICA) stenosis due to active GCA.

Case report

An 84-year-old patient (arterial hypertension, hyperlipidemia, diabetes mellitus type II, curatively treated colon carcinoma) presented with a transient sensorimotor hemisyndrome on the left. For two months he had noticed a headache, especially when chewing and elevating the arms.

Laboratory tests showed an increase in the erythrocyte sedimentation rate to 37 mm/h (reference range <20 mm/h); C-reactive protein was elevated to 5.4 mg/dL (reference range <0.5 mg/dL). Ultrasonography

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revealed indirect signs for a distal occlusion of the right ICA and atherosclerotic plaques at the origin of the left ICA. The initial cranial CT was unremarkable. A contrast-enhanced MRI was performed because of the cancer history. It showed significant stenotic wall

changes with intramural contrast uptake in the intracranial right ICA and vertebral arteries and a small infarction in the right middle cerebral artery (MCA) territory (Figures 1–2). Temporal artery ultrasound revealed an inflammatory wall thickening (halo); the

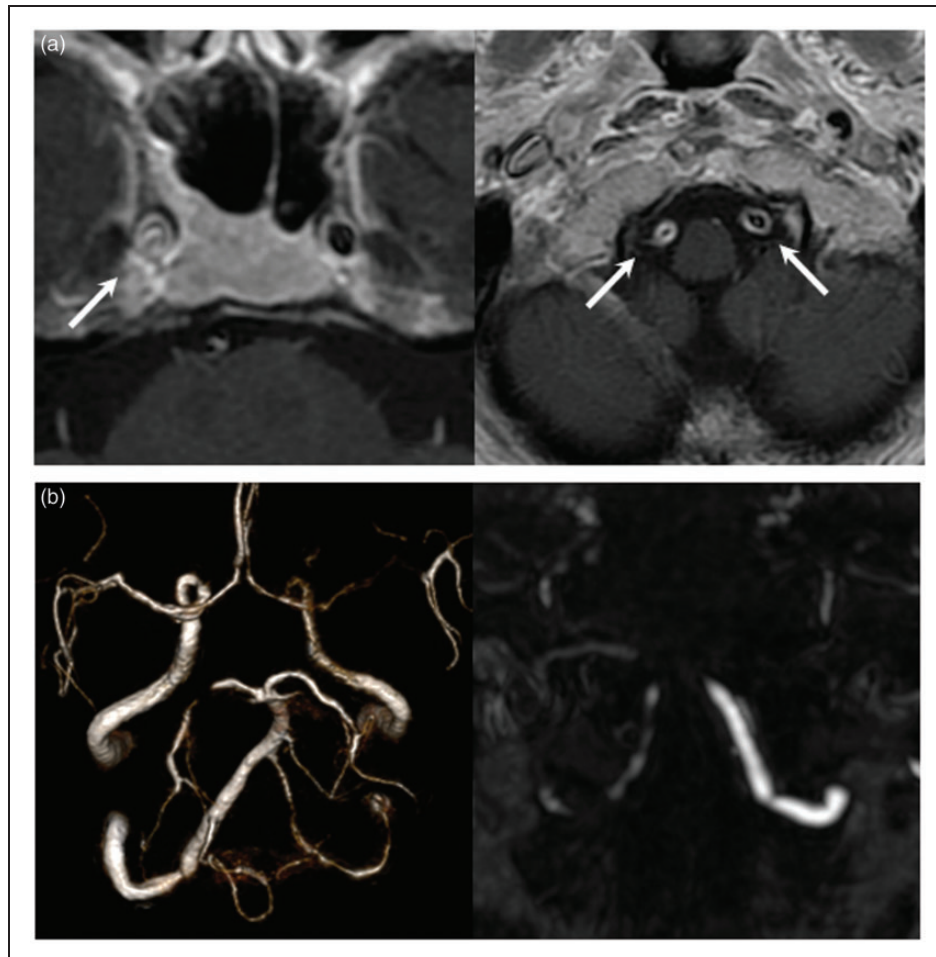


Figure 1. (a) Axial T1w-MRI sequences showing concentric intramural contrast enhancement of the right cavernous ICA with target sign (left image) and both vertebral arteries (right image) $191 \times 93 \text{ mm}^2$ (72 \times 72 DPI). (b) Left image: three-dimensional reconstruction of cerebral arteries based on a TOF-MRI sequence showing distal right ICA stenosis. Right image: MRI angiography showing stenosis in both V4 segments of the VA, in particular on the right side. $191 \times 99 \text{ mm}^2$ (72 \times 72 DPI).

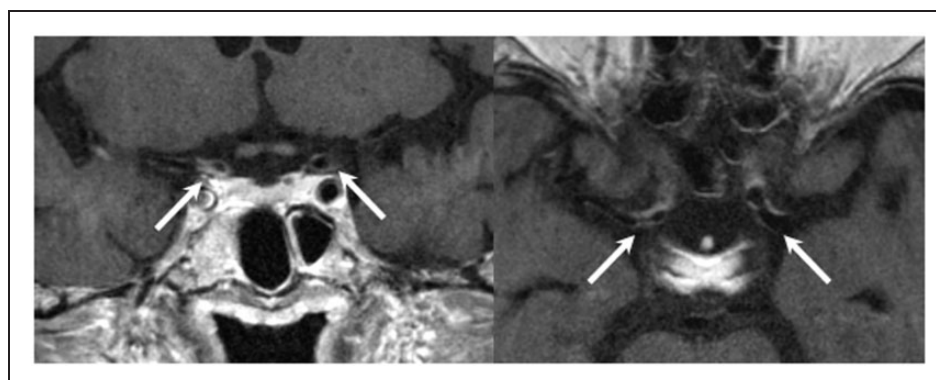


Figure 2. Post-contrast high-resolution T1w FLAIR black blood imaging sequences showing intramural contrast enhancement in both terminal ICAs, especially on the right side. $200 \times 73 \text{ mm}^2$ (72 \times 72 DPI).

vertebral arteries showed similar wall alterations, especially on the right side (Figure 5). A biopsy of the left temporal artery confirmed the suspected diagnosis of GCA (Figure 6).

The patient was started with aspirin in combination with dipyridamole, immediate high-dose glucocorticoid (intravenous prednisolone for five days 250 mg/d, afterwards 100 mg/d orally), and methotrexate. The wall changes did not decline, and there were again hemodynamic and embolic infarctions in the right MCA territory 19 days later. A high-resolution dedicated vasculitis MRI then revealed inflammatory wall changes in both ICAs and MCAs. Digital subtraction

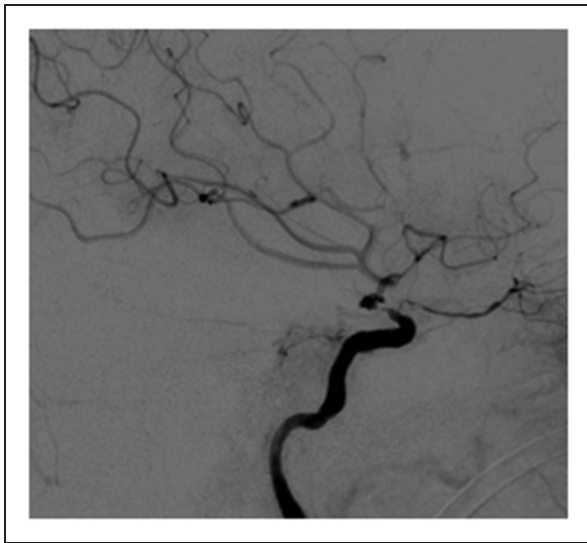


Figure 3. DSA image of right ICA injection irregular high-grade stenosis of the ICA terminus with a beaded appearance located distal the origin of the ophthalmic artery. $102 \times 93 \text{ mm}^2$ (72×72 DPI).

angiography (DSA) was performed because of a recurring blood-pressure dependent hemiparesis on the left side and showed beaded wall alterations of the distal ICA with high-grade stenosis (Figure 3). The therapeutic option of balloon angioplasty was discussed interdisciplinary but discarded because of the increased risk of a vessel rupture. The patient did not benefit from a therapy escalation to intravenous cyclophosphamide either.

During the four-month follow-up, further right-sided MCA infarcts occurred (Figure 4) and the patient finally suffered from a high-grade hemiparesis on the left. The arterial stenosis did not diminish despite the intensified immunosuppressive therapy.

Although he received a rehabilitation treatment, the patient remained severely disabled. He finally moved into a nursing home and died about one year after the first ischemic stroke.

Discussion

This case illustrates that highly-active GCA of the intradural arteries could be solely demonstrated by a standard MRI with contrast-enhanced T1 imaging.

Our patient showed inflammatory involvement both of the intracranial ICA and vertebral arteries. In fact, previous reports showed that GCA may affect the vertebral arteries rather than the vessels of the anterior circulation.² However, in a recent study evaluating 3T MRI with an high-end optimized protocol for the detection of extra- and intracranial involvement in GCA,⁸ 10 out of 20 patients with GCA showed vessel wall enhancement of the intradural ICA (as compared with 9 out of 20 patients for the vertebral arteries). Still, vessel stenosis or occlusion was more frequent in patients with vertebral artery involvement. This fact possibly explains the clinical impression of vertebral

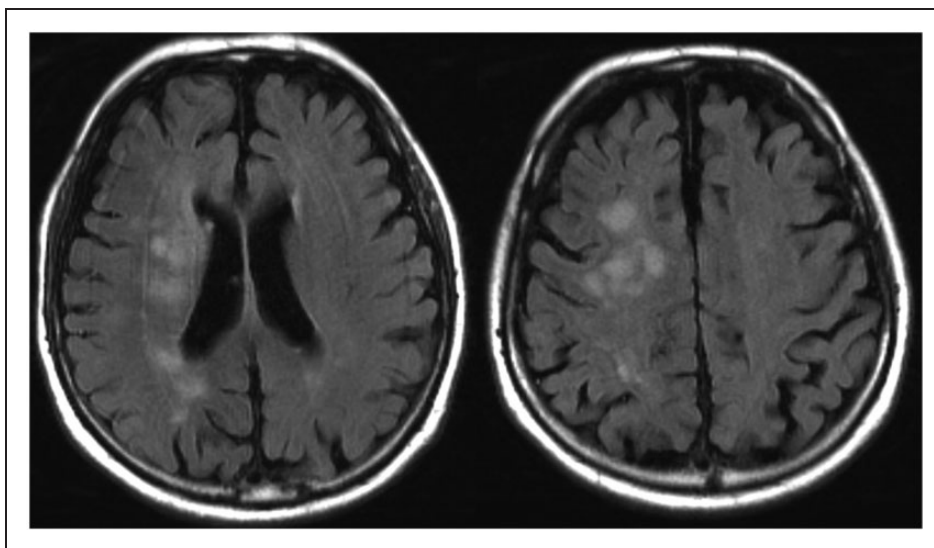


Figure 4. T2w FLAIR dark-fluid imaging sequences four months after diagnosis of GCA showing ischemic lesions in the right MCA territory. $187 \times 104 \text{ mm}^2$ (150×150 DPI).

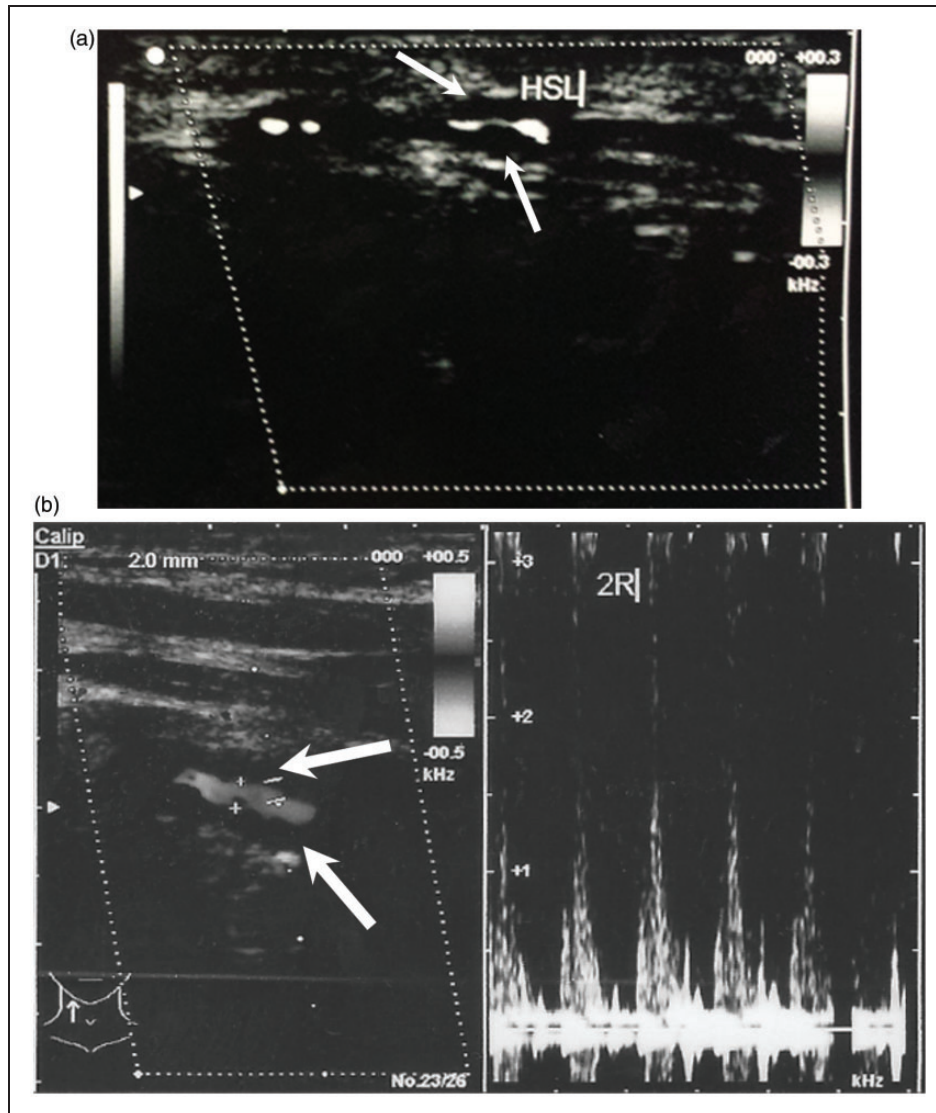


Figure 5. (a) Duplex sonography reveals a concentric inflammatory wall thickening (marked by arrows) of the left temporal artery. $289 \times 181 \text{ mm}^2$ ($150 \times 150 \text{ DPI}$). (b) Duplex sonography of the right vertebral artery in segment V2. The arrows indicate the inflammatory wall alterations. $211 \times 135 \text{ mm}^2$ ($150 \times 150 \text{ DPI}$).

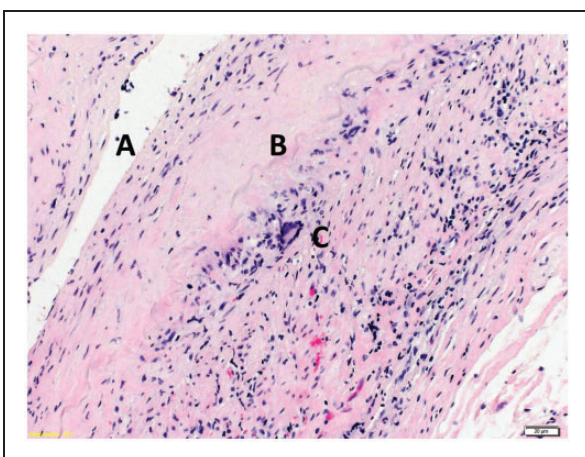


Figure 6. Histopathologic image of the biopsy of the left temporal artery. Markers: A - Artery lumen and adjacent tunica intima. B - Lamina elastica interna. C - Tunica media with giant cell. $254 \times 190 \text{ mm}^2$ ($150 \times 150 \text{ DPI}$).

arteries being more frequently affected by GCA. Also, ischemic stroke in GCA patients has been reported more frequently for the posterior circulation territory.⁹

While the diagnostic accuracy of specific MRI for extracranial temporal artery involvement has been found to be high,^{7,10} there are no data on the diagnostic accuracy for intracranial involvement because of the lacking gold standard. As in other forms of intracranial large artery vasculitis, the findings of high-resolution vessel wall MRI for intracranial GCA have only been published in selected case reports or smaller series.^{2,11-15} However, as found in a recent study by Siemonsen et al.,⁸ involvement of the intradural ICA was detected in 50% of GCA patients using a dedicated 3T high-resolution vessel wall MRI protocol (small series only). Likewise, it may be assumed, as in our case, that extensive acute intracranial artery involvement of GCA may not be seen so infrequently on routine contrast-enhanced MRI, although this lacks

prospective evaluation. As a further limitation, vessel wall contrast enhancement of the intracranial arteries can also occur in atherosclerotic intracranial stenosis or occlusion probably because of an inflammatory process within the acute plaque or thrombus. In atherosclerosis, however, the vessel wall enhancement is usually rather focal eccentric affecting only the plaque area, decreases with time following an ischemic event, and does not reveal a concentric circular involvement characteristic for large artery vasculitis or Moyamoya disease.^{15–17}

The optimal treatment strategy for GCA with intracranial involvement is still not clear. In general, an early immunosuppressive treatment is recommended, but, as also found in our experience,¹⁸ regression of arterial stenosis does rarely occur.⁹ In the present case clinical deterioration could not be prevented even under intensified immunosuppression including intravenous cyclophosphamide. Future strategies in such non-responding patients might involve newer agents such as the anti-IL6 receptor monoclonal antibody tocilizumab.¹⁹

In highly-active cases GCA of the intradural ICA may already be detected by standard contrast-enhanced MRI. In elderly patients with intracranial ICA or vertebral artery stenosis and corresponding signs of inflammation in the routine laboratory, intracranial GCA may be a cause and, in addition to temporal artery ultrasound, high-resolution vessel wall MRI or if not available contrast-enhanced T1w MRI of the brain should be considered as a diagnostic step.

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Conflict of interest

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