Temporal artery enhancement on cranial magnetic resonance imaging

A 68-year-old white female presented to her outside ophthalmologist with acute vision loss in the left eye (OS) described as a "green line across the top half of her vision" and dull pain OS. She then experienced progressive worsening of vision OS, pain with chewing, temple pain, and scalp tenderness. The visual acuity was 20/20 in the right eye (OD) and counting fingers at 1/2-foot distance OS. There was a left relative afferent pupillary defect and optic disc edema OS. The right optic nerve was normal, but the cup-to-disc ratio was 0.4 OD. The remainder of the eye examination was normal OU. An erythrocyte sedimentation rate (ESR) was 25 mm/h, a C-reactive protein (CRP) level was <0.30 mg/dL, and platelet count was $349 \times 10^3/mm^3$. She was started on oral prednisone 60 mg/day. Medical history was significant for hypertension and Raynaud phenomenon. The remainder of the social, family, allergy, and medication history was noncontributory. She denied the use of any immunosuppressive drugs. The patient was then referred to the neuro-ophthalmology service at Houston Methodist Hospital. Cranial magnetic resonance imaging (MRI) with and without contrast for atypical headache and amaurosis fugax at the outside facility showed on the T1 precontrast axial MRI some mural thickening of the left superficial temporal artery. T1-weighted, fat-suppressed, postcontrast axial MRI showed mural enhancement of the artery and a partial flow void on all sequences consistent with partial arterial flow in the vessel more consistent with an artery rather than a vein (Fig. 1). Axial T2 MRI demonstrated the course of the superficial temporal artery all the way down to the internal maxillary artery (Fig. 2).

A left-sided temporal artery biopsy (TAB) taken at the same anatomical site as the enhancing artery seen radiographically on the MRI was positive for giant cell arteritis (GCA). Additional special staining with Movat pentachrome stain showed disruption of the internal elastic lamina, and additional immunohistochemistry studies using CD68 were positive for macrophages consistent with the diagnosis of GCA. Three months after the positive TAB, the patient reported recurrence of temple headache and scalp tenderness. ESR and CRP results were normal. She underwent repeat cranial MRI at the outside facility. Comparison of the pre- and post-TAB cranial MRI with gadolinium showed postoperative reduction in the size of the previously enhancing temporal artery, and there was some residual surrounding soft-tissue edema caused by postoperative changes at the site of the left TAB (Figs. 3 and 4).

GCA is a chronic vasculitis of elderly adults that manifests in large- and medium-sized arteries.[1,2] The diagnosis is a clinical one supported by elevated acute-phase reactants (e.g., ESR and CRP) and confirmed histologically by a TAB.[2] Cranial and orbital MRI studies are generally not performed and are usually not necessary for the diagnosis of GCA, but a few prior MRI studies have demonstrated similar radiographic findings in the temporal artery in GCA.[3] We suspect that these MRI findings may be underrecognized in GCA. Bley et al.[4,5] reported the use of high-resolution MRI in revealing mural inflammatory changes of the temporal artery in GCA. Similar findings have been seen with variable sensitivity and specificity by ultrasound of the temporal artery but have not superseded the use of the presumed gold standard of the TAB.[6] Nevertheless, the presence of these MRI findings might prompt a clinician evaluating an elderly patient to more aggressively evaluate and treat for GCA.

Although clinical suspicion, elevated serum acute-phase reactants, and a positive TAB are critical for the diagnosis of GCA, we believe that these MRI findings suggestive of GCA occasionally might be useful to clinicians, especially

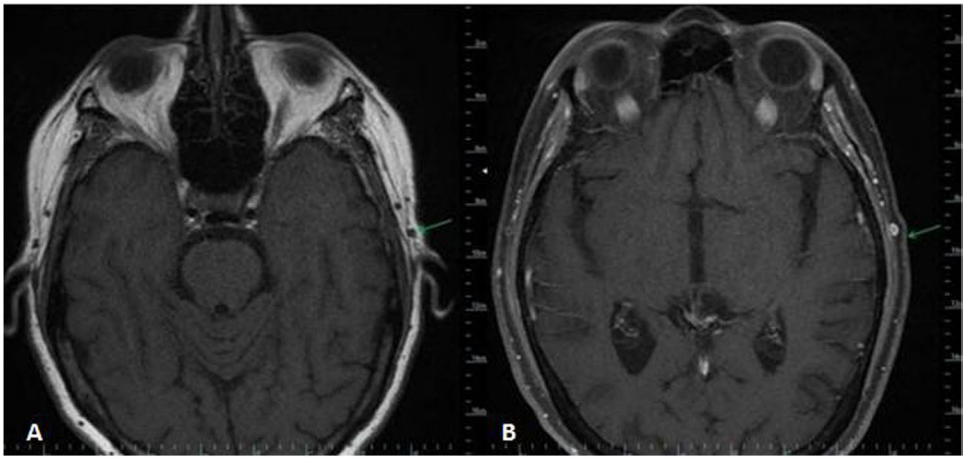

Fig. 1—A, Axial T1 precontrast magnetic resonance imaging (MRI) demonstrating thickening of the left superficial temporal artery with central hypodensity demonstrating the flow void in signal in comparison with contralateral normal-sized superficial temporal artery. B, Axial T1 postcontrast MRI demonstrating enhancement of the left superficial temporal artery.

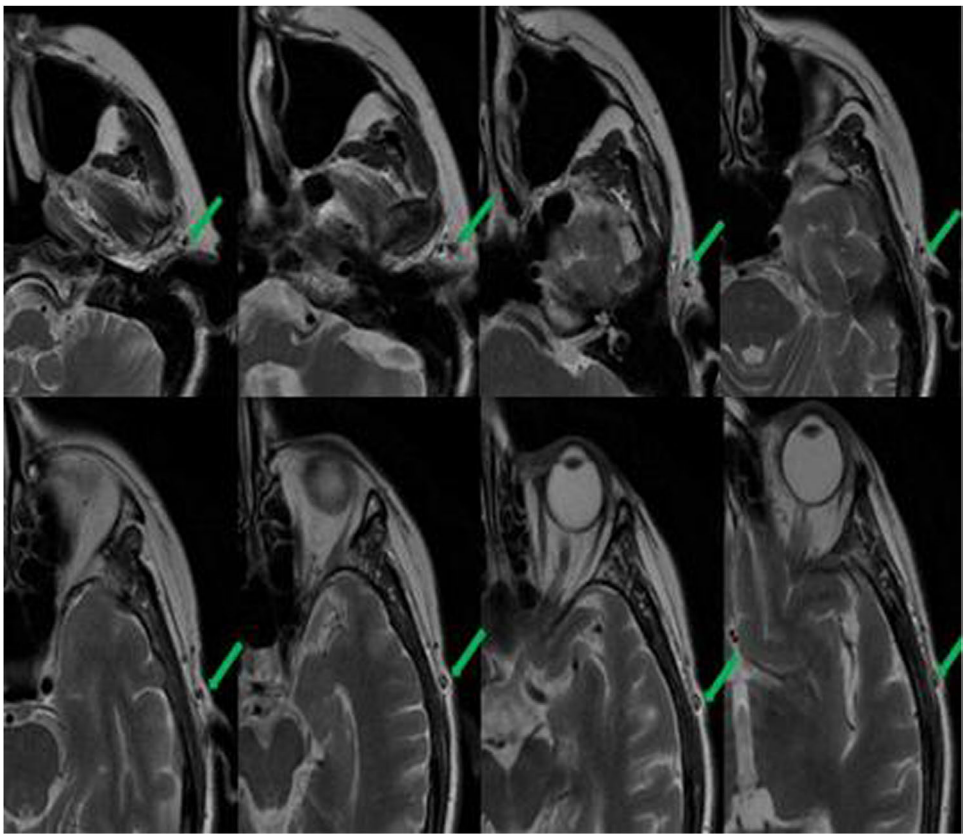

Fig. 2—Axial T2 magnetic resonance imaging demonstrating the course of the superficial temporal artery all the way down to the internal maxillary artery (*green arrows*).

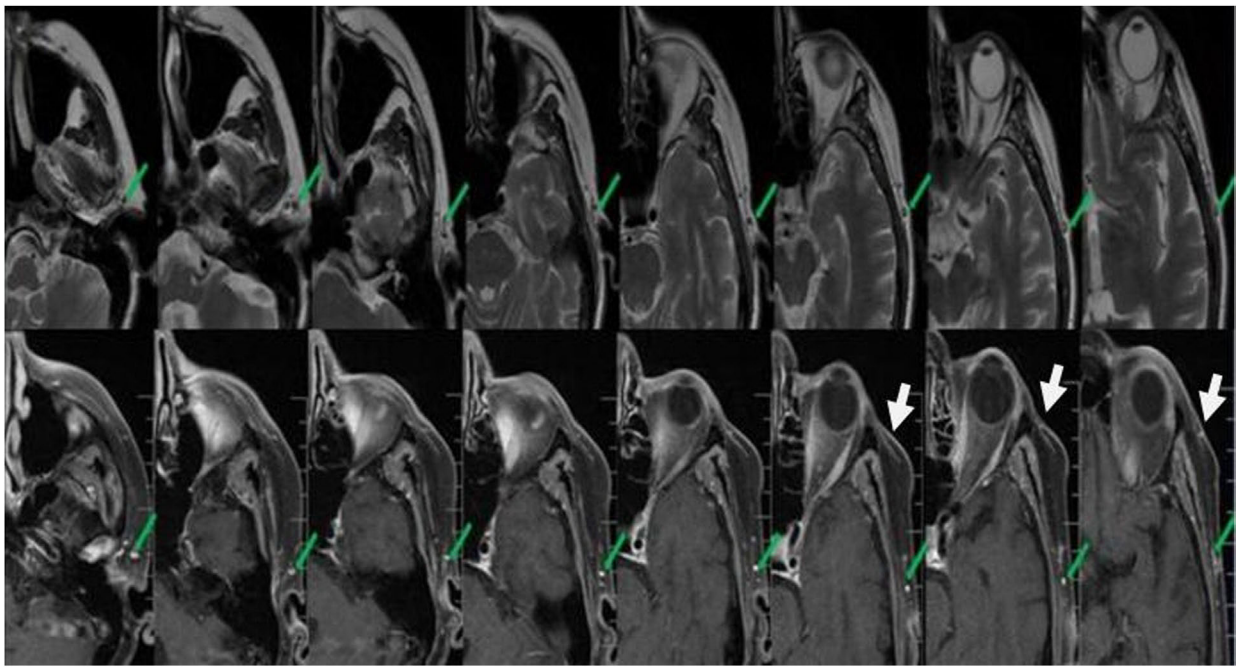

Fig. 3—The preoperative axial T2 magnetic resonance imaging (MRI) (top row) tracing the origin of the vessel up to the area of the superficial temporal artery biopsy (TAB) site and postoperative T1 postcontrast axial MRI show that at the site of the left TAB, there is a reduction in the thickening of the previously enhancing temporal artery wall (*green arrow*) and some residual surrounding soft-tissue edema caused by postoperative changes from the left-sided TAB (*white arrow*).

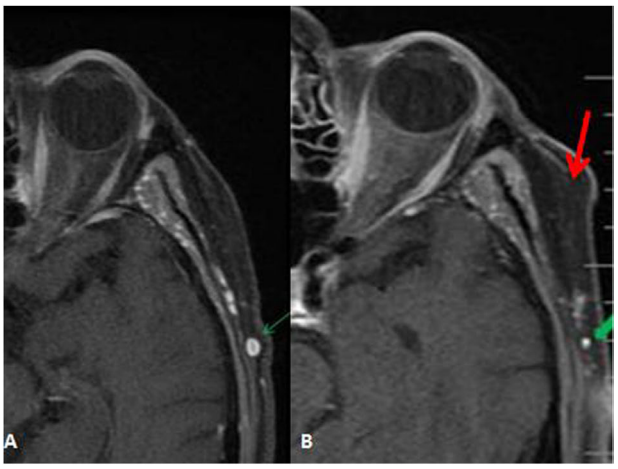

Fig. 4—A direct comparison between the T1 precontrast (pre–temporal artery biopsy [pre-TAB]) (A, *green arrow*) and T1 postcontrast (post-TAB) (B) axial MRI shows the reduction in the lumen of the temporal artery, and there is postoperative change in the surrounding soft tissue (*red arrow*) around the left TAB site (*green arrows*). Note the postoperative TAB changes with soft-tissue edema and the resolution of the previously seen mural enhancement differentiating the vessel from a vein.

for elderly patients who have already undergone neuroimaging for headache or other nonspecific complaints of GCA.

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Acute eosinophilic meningitis and orbital inflammation from presumed angiostrongyliasis

Angiostrongyliasis, the most common cause of eosinophilic meningitis, results from infection by the nematode *Angiostrongylus cantonensis.* The most common clinical form is eosinophilic meningitis, and orbital inflammation has not been previously reported as a manifestation of angiostrongyliasis. We present a unique case of presumed angiostrongyliasis manifesting as orbital inflammation.

A 66-year-old previously healthy Asian male presented to the emergency department with a 3-day history of decreased mental status accompanied by right-sided periorbital swelling and pain (Fig. 1A). Vision was reported to be 20/200 in both eyes without afferent pupillary defect. No ophthalmic consultation was initiated at that time. The patient was admitted for intravenous antibiotics for presumed meningitis and possible orbital cellulitis. Magnetic resonance imaging (MRI) showed right-sided proptosis, severe right periorbital and orbital soft-tissue swelling, and enlargement and abnormal enhancement of the right superior and lateral rectus muscles. Thickening and enhancement of meninges on the right side was evident (Fig. 1B).

The ophthalmology service was consulted on the third day of admission because of increasing periocular swelling. The patient showed right orbital compartment syndrome with finger counting vision, intraocular pressure of mid-fifties mm Hg, marked periorbital swelling, and severe conjunctival chemosis. Lateral canthotomy with cantholysis was performed with reduction of the intraocular pressure to 20 mm Hg (Fig. 1C). A tentative diagnosis of idiopathic orbital inflammation was made and intravenous methylprednisolone 100 mg daily was started. Biopsy of orbital fat and lateral rectus muscle was performed and pathology showed diffuse eosinophilic infiltrate with rare lymphocytes (Fig. 1D).

Review of diagnostic work-up revealed eosinophilia in the serum (eosinophils 22%, normally $<7\%$) and cerebrospinal fluid (CSF; only 1–3 white blood cells/high powered field but $>50\%$ of white blood cells present were noted to be eosinophils, normally not present). Otherwise, the rest of the complete blood cell count; thyroid function tests; CSF Gram, acid-fast, and India ink staining; CSF cryptococcal antigen; blood; and CSF bacterial and fungal