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Giant cell arteritis presenting with bilateral orbital inflammatory disease and enhancing superficial temporal arteries

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CASE REPORT

A 68-year-old man with metastatic prostate cancer was admitted with a 1-week history of bilateral ‘conjunctivitis’, headaches and horizontal diplopia. On examination, the superficial temporal arteries were bilaterally prominent and indurated,

with conjunctival injection and chemosis. There were bilateral abduction deficits, but visual acuity and fundi were normal.

MR scan of the brain and orbits showed bilateral enhancement of the temporal arteries with intraconal fat stranding but with no extra-ocular muscle enlargement

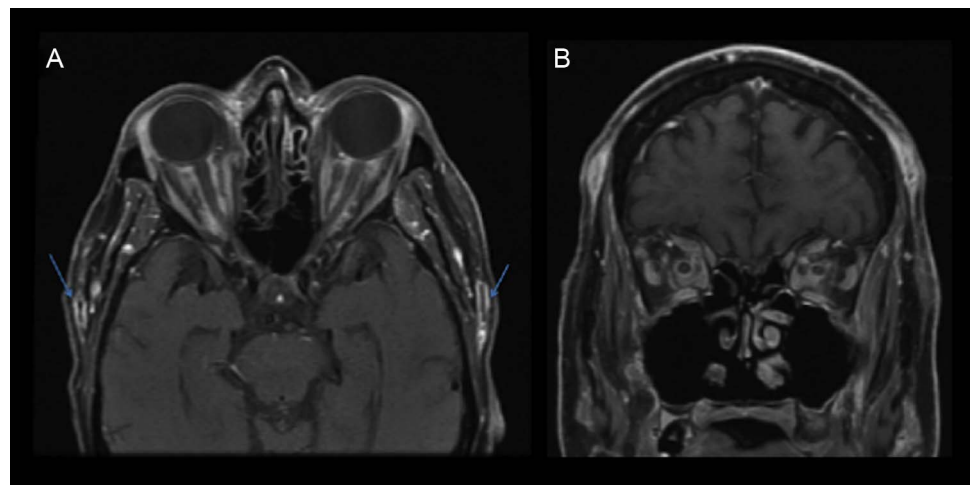


Figure 1 Contrast enhanced MRI orbits. (A) Bilateral enhancement of superficial temporal artery (arrows). (B) Intraconal fat stranding and inflammatory debris.



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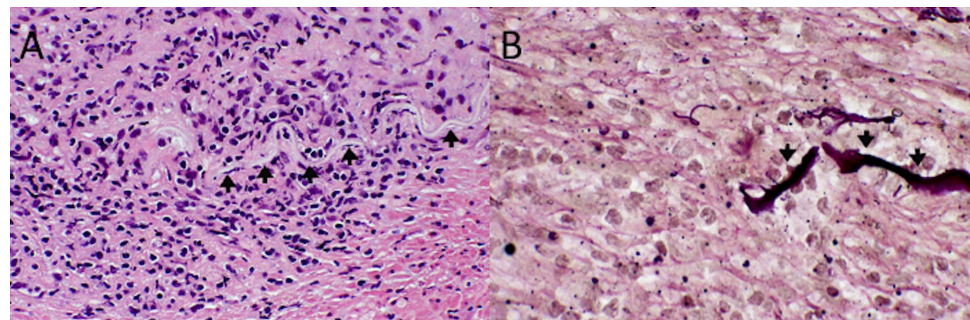


Figure 2 Pathology. (A) Chronic inflammatory infiltrate of vessel and muscle wall, internal elastic lamina (arrows) and intima. Discontinuity of the elastic lamina (left). (B) The inflammatory infiltrate has destroyed the internal elastic lamina (arrows). Superficial temporal artery (A) H&E, x40. (B) Elastic van Gieson, x100.

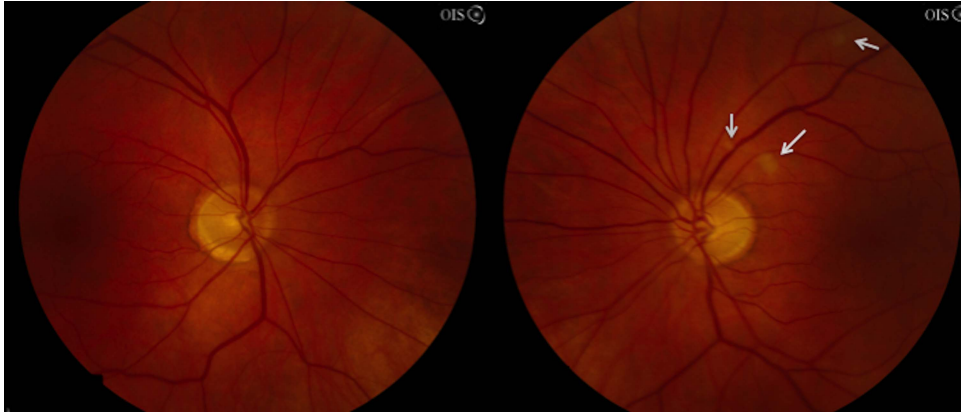


Figure 3 Fundus photographs, Note the three yellow/white lesions ‘cotton wool spots’ representing retinal ischaemia.

or orbital mass (figure 1A,B). His erythrocyte sedimentation rate was elevated at 103 mm/1st h (<20), serum C reactive protein was 14 mg/L (<10) and CSF: glucose 57, protein 31, cell count <1 analysis was normal. We gave him prednisolone 100 mg daily, with significant improvement. A temporal artery biopsy, taken 4 days after starting prednisolone, showed ‘skip’ lesions of the temporal artery wall with chronic inflammatory infiltration of the intima, media and adventitia, damage to the muscle wall, fraying and destruction of the internal elastic lamina, and fibrointimal proliferation (figure 2A,B). The inflammatory infiltrate comprised small lymphocytes and histiocytes, but there were no giant cells.

Re-examination 2 weeks later showed complete resolution of orbital inflammation in both eyes. Visual acuity and visual fields remained normal, but dilated fundoscopy showed three new areas of retinal ischaemia ‘cotton wool spots’ within the superior arcade of the left eye (figure 3). Fluorescein angiogram showed a large vertical area of delayed choroidal perfusion.

COMMENT

Giant cell arteritis is a systemic, inflammatory vasculitis that affects small- to medium-sized arteries. Orbital inflammation is a very unusual clinical presentation of this condition. Our review of the literature found only four previous cases of giant cell arteritis presenting with bilateral orbital inflammation.^{1 2} Our case also showed MR scan findings of dramatic enhancement of the bilateral superficial temporal arteries.

Because early recognition and treatment of giant cell arteritis may prevent severe and irreversible visual loss, clinicians must consider it in elderly patients presenting with idiopathic orbital inflammation, with or without visual loss.

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Competing interests None.

Patient consent Obtained.

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