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Christina J. Flaxel[1,3]
Michael J. Wheeler[2]
Bridget Mulholland[1]
Zdenek J. Gregor[1]

[1]Moorfields Eye Hospital London, UK
[2]Department of Chemical Pathology
St Thomas' Hospital
London, UK

[3]Current address:
University of Southern California
Doheny Eye Institute
Los Angeles
California, USA
e-mail: flaxelcj@aol.com

Zdenek J. Gregor, FRCS, FRCOphth ✉
Moorfields Eye Hospital
City Road
London EC1V 2PD, UK

Tel: +44 (0)171 253 3411
Fax: +44 (0)171 253 4696

Sir,

Occult giant cell (temporal) arteritis presenting with bilateral sixth and unilateral fourth nerve palsies

Giant cell arteritis is regarded as the 'prime medical emergency in ophthalmology, there being no other disease in which the prevention of blindness depends so much on prompt recognition and early treatment'.[1] We describe one occult manifestation, namely bilateral sixth and unilateral fourth nerve palsies with no systemic malaise.

Case report

An 80-year-old man presented with a 2 week history of intermittent pain below the right eye. He had had a trabeculectomy performed previously on the left eye for chronic open angle glaucoma. Topical medication was timolol and dorzolamide b.d. to both eyes. He had noticed diplopia for the past 3 days. He had no systemic malaise, headache, or jaw or lingual claudication.

Best corrected visual acuity was 6/9 bilaterally. Ocular movements were limited laterally in either eye. A Hess recording suggested bilateral sixth nerve palsies (Fig. 1a). Pupil reactions were normal. Intraocular pressure was 24 mmHg in each eye. A left trabeculectomy bleb and iridectomy and glaucomatous optic discs were noted. There was no evidence of anterior ischaemic optic neuropathy. Erythrocyte sedimentation rate (ESR) performed on presentation was 132 mm/h.

The next day a right temporal artery biopsy was performed. This showed a mixed inflammatory infiltrate affecting all sections of the artery wall but was most marked in the media of the vessel (Fig. 2). Although no giant cells were seen, the pathologist felt the appearance was suggestive of temporal arteritis. The patient was commenced on oral prednisolone 80 mg/day. The following day his eye pain had gone. After 3 days a repeat Hess chart demonstrated that a right fourth nerve palsy had developed in addition to his bilateral sixth nerve palsies (Fig. 1b). His steroid regime was slowly reduced and 2 months following admission he was on 10 mg/day of oral prednisolone, his ocular nerve palsies had resolved and his ESR was 35 mm/h.

Other investigations performed before oral steroid treatment was commenced included a full blood picture, urea and electrolytes, liver function tests, autoimmune screen, anti-neutrophil cytoplasmic antibodies, C-reactive protein, serum immunoglobulin assay and serum electrophoresis, chest radiograph and a CAT scan of the brain. Abnormal results included the C-reactive protein, which was markedly elevated at 7.4 mg/dl (normal range < 1 mg/dl); the leucocytes, marginally elevated at 11.4 $\times 10^3/\mu l$ (normal range 4–11 $\times 10^3/\mu l$); and the serum electrophoresis, which showed an elevation of the alpha-1 and alpha-2 globulins consistent with an increase in acute phase reactants. Results of other investigations were normal.

Comment

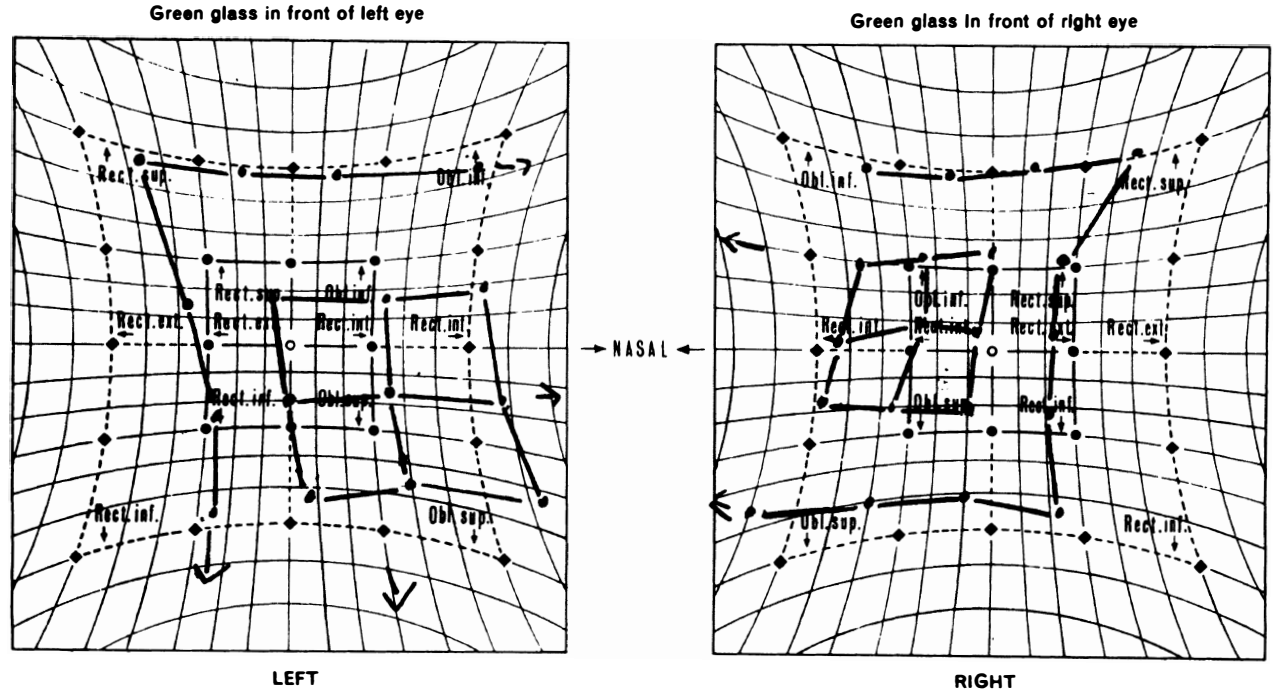
Patients with giant cell arteritis may present with no systemic complaints, as in this case. The incidence of occult giant cell arteritis has been reported as between 8% and 38% of cases.[2,3]

The commonest ocular presentation is anterior ischaemic optic neuropathy,[4] but many other ocular complications may be produced including retinal artery occlusion, choroidal ischaemia, anterior segment ischaemia and even orbital infarction resembling orbital cellulitis.[5,6]

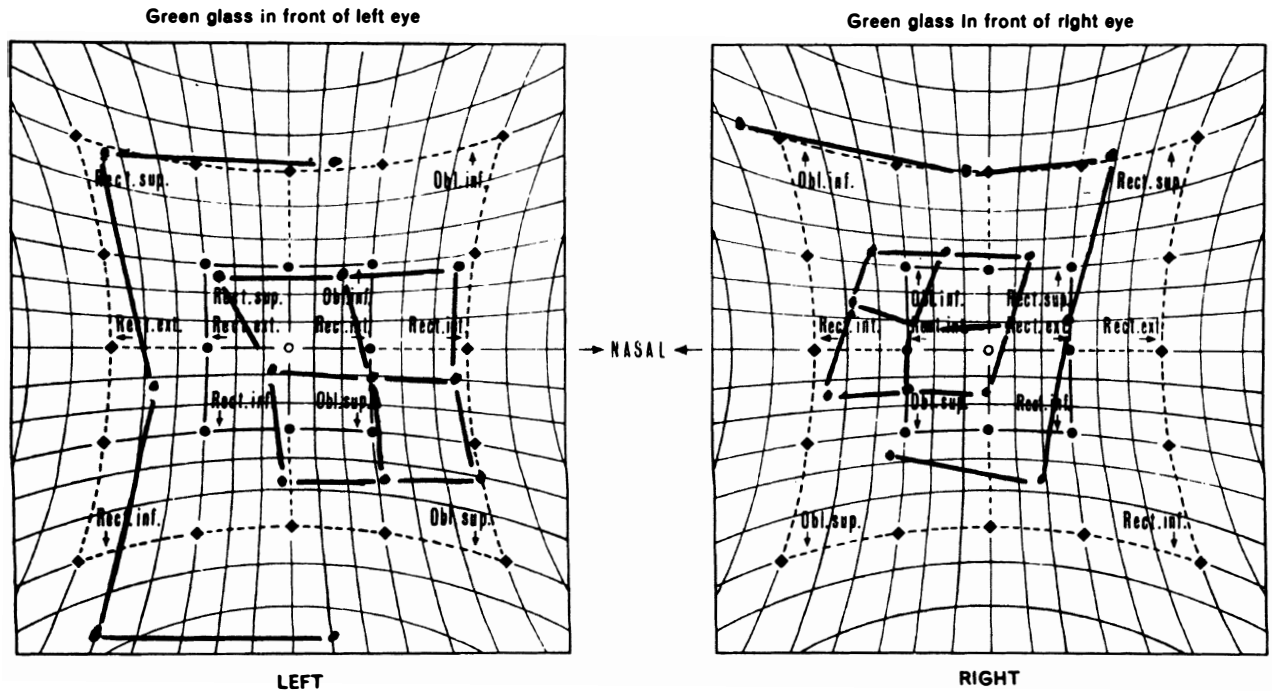
Ocular motor palsies are recognised in giant cell arteritis[5] with the oculomotor (III) nerve most commonly involved.[7] Abducens (VI) or trochlea (IV) nerve palsies are less frequently described.[7]

The occurrence of bilateral abducens (VI), and subsequent trochlea (IV), nerve palsies with no associated systemic malaise is a very rare presentation of giant cell arteritis. It was the initial ESR of 132 mm/h that suggested to us giant cell arteritis might be the underlying diagnosis. This was confirmed by checking C-reactive protein levels and performing a temporal artery biopsy. A space-occupying lesion and a systemic vasculitis were excluded by CAT scan and vasculitic screen respectively.

An elevated ESR in combination with a C-reactive protein level of > 2.45 mg/dl, as in this case, has been shown to have a 97% specificity for the diagnosis of giant cell arteritis.[8] This high specificity depends on a consistent clinical picture for giant cell arteritis, as



(a)



(b)

Fig. 1. *(a) Hess chart illustrating initial bilateral sixth nerve palsies and (b) subsequent development of a unilateral right fourth nerve palsy.*

conditions such as other vasculitides, connective tissue diseases or infection will give similar elevations of both the C-reactive protein and the ESR. There was no evidence of any such confounding conditions in our patient.

We further confirmed our diagnosis with a positive temporal artery biopsy. No giant cells were seen on our biopsy. However, these are not required for diagnosis when other histological features of inflammation are present.[8,9]

In summary, ocular nerve palsies are relatively common in elderly patients. They may represent an occult presentation of giant cell arteritis. In these cases an ESR is a useful first-line investigation. If the ESR is elevated it makes exclusion of giant cell arteritis mandatory.

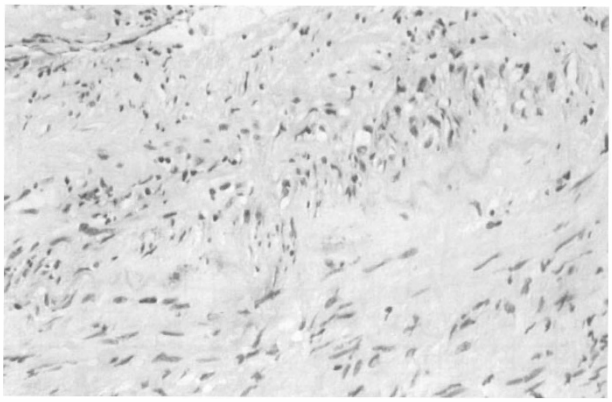


Fig. 2. *Temporal artery biopsy demonstrating a mixed inflammatory infiltrate of the vessel wall.*

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Andrew Lotery
Jane Best ✉
Stephen Houston
Department of Ophthalmology
Royal Victoria Hospital
Belfast BT12 6BA
Northern Ireland
Tel: +44 (0)1232 240503, ext 3152
Fax: +44 (0)1232 330744

Sir,

Management of orbital emphysema

Orbital emphysema is a well-known clinical and radiological finding in cases of orbital trauma with fracture.[1] The condition occurs when a fracture of the orbital wall is associated with a laceration of the adjacent sinus mucosa that allows air to enter the orbit. The vast majority of these patients are asymptomatic and the air spontaneously resolves without adverse sequelae.[2] Rarely severe visual loss from optic nerve or retinal ischaemia can occur due to the intraorbital air mass causing a compressive orbital compartment syndrome.[3,4] Transient reduction of the ocular circulation is not necessarily followed by permanent visual loss[5] and therefore prompt diagnosis and management of these cases is essential.

Case report

A 76-year-old man presented with a 1 day history of a swollen, red, painful left eye with reduced vision. He had fallen 4 days previously suffering blunt facial trauma, and a fractured nasal bone had been diagnosed clinically; radiological investigations were not deemed necessary. There was no significant past ophthalmic history. Regular medications included nifedipine for hypertension and aspirin following a transient ischaemic attack.

On examination he was apyrexial with a pulse rate of 80 beats/min and blood pressure 150/90 mmHg. Visual acuity was 6/5 right eye and count fingers left eye, with a left afferent pupillary defect. There was a 4 mm left-sided ptosis with a haemorrhagic, chemosed conjunctiva (no lid crepitus was palpable). The left eye was proptosed 3 mm, displaced downwards 5 mm and ductions were restricted in all directions of gaze. The intraocular pressure was 20 mmHg left (16 mmHg right) and fundoscopy, including the retinal vasculature, was normal in both eyes.

A clinical diagnosis of optic nerve compression secondary to orbital emphysema was made. An urgent computed tomogram (CT scan) demonstrated a fracture in the lateral wall of the left maxillary antrum with involvement of the orbital floor. There was a marked left-sided proptosis with a large air collection situated laterally in the orbit and retrobulbar space (Fig. 1). With the patient supine a 25 G needle attached to a syringe was passed into the lateral orbit under topical anaesthesia and approximately 5 ml of air aspirated. The proptosis reduced and the patient's discomfort improved immediately. Oral and topical antibiotics were commenced.

On review 12 h later, the left visual acuity was 6/18 with normal pupillary responses. There was a substantial improvement in the ptosis, hypoglobus and proptosis with less restriction of the ocular movements. A repeat CT scan (Fig. 2) demonstrated a small residual amount of intraorbital air. One week later the patient was asymptomatic and the ocular examination, including visual acuity, colour vision and visual fields, was normal.

Comment

The development of orbital emphysema is thought to involve the forceful passage of air through a sino-orbital communication. The pressure gradient required is most likely a consequence of sneezing or nose-blowing. Positive orbital pressures are probably maintained by either a hinged fragment of bone acting as a one-way valve, or orbital fat functioning as a ball-valve.[3] Confinement by the osseous walls, periorbita and septum restricts spontaneous decompression of the