vitreoretinopathy.[6,7] Once post-traumatic endophthalmitis develops, over half of patients lose all useful vision despite treatment.[8]

Although the final visual outcome of this case was poor, the post-operative period was complicated by the development of a giant retinal tear, requiring further surgery. The series of post-operative endophthalmitis cases described by Chaudhry *et al.*[2] had a generally favourable visual outcome.

In conclusion, we describe the first case of post-traumatic endophthalmitis caused by *Xanthomonas maltophilia*. Infection with this organism must be suspected and recognised early, as the clinical course can often be turbulent, and the organism exhibits resistance to multiple antimicrobial agents.

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Sir,

Multiple myeloma presenting with optic nerve compression

Neurological complications occur in up to 40% of patients with multiple myeloma as sequelae of hypercalcaemia, hyperviscosity state or nerve compression.[1–5] Significant ocular findings appear during the course of the disease and include pars plana cysts, retinal microaneurysms or haemorrhages, orbital plasmacytomas and ocular motor palsies.[1–5] We describe a man presenting with unilateral visual loss initially mistaken for retrobulbar neuritis. A large intracranial plasmacytoma was found compressing the optic nerve at the optic foramen.

Case report

A 63-year-old man had an acute onset of painless visual loss in the right eye a few days after an upper respiratory tract infection. Two ophthalmologists diagnosed retrobulbar neuritis: best corrected visual acuity of 6/24 in the right eye, intraocular pressures of 15 mmHg bilaterally, decreased right colour perception, prominent disc cupping bilaterally, and large central defects by automated perimetry in the right eye. Two weeks later the patient complained of further visual loss associated with headache and malaise. His internist requested a brain MRI scan. Imaging revealed multiple enhancing bony lesions involving the apex of the right orbit (Fig. 1), the clivus, the base of the skull medial to the mastoid air cells, and the lateral aspect of the right sphenoid wing. There was no increased uptake at the apex of the right orbit or in the skull by bone scan. Brain CT scan showed multiple lytic lesions in the clivus, sphenoid wing, base of the skull and roof of the left orbit, with destruction of the right orbital apex. Bone marrow aspirate confirmed the radiological diagnosis of multiple myeloma. The patient was rehydrated intravenously, treated with intravenous steroids and chemotherapy (adriamycin and vincristine).

Best corrected visual acuity was counting fingers at 3 m in the right eye and 6/6 in the left eye. Afferent pupillary defect could not be elicited because of severe pupillary miosis. Colour vision was depressed in the right eye. Funduscopy failed to reveal pars plana cysts by indentation. Cup-discs ratios were 0.8 in the right eye and 0.7 in the left eye. Intraocular pressure reached a peak of 21 mmHg in the right eye and 19 mmHg in the left eye at 0600 hours. After cycloplegia, intraocular pressures were 23 mmHg in the right eye and 20 mmHg in the left eye. Visual fields of the left eye revealed centrally depressed islands.

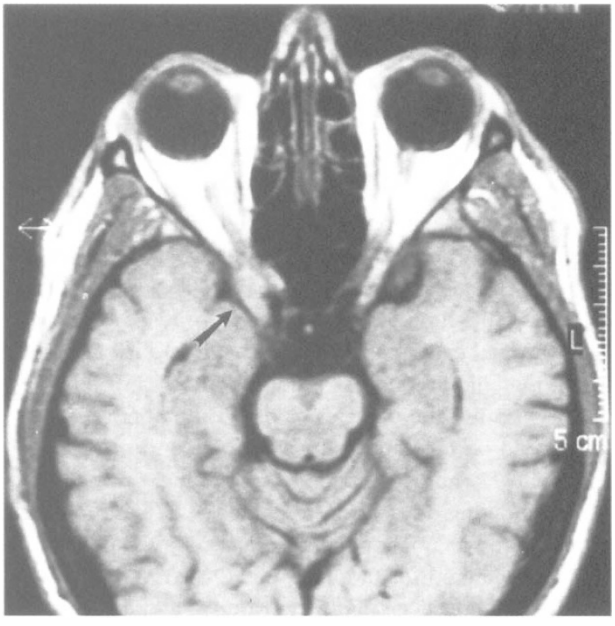

Fig. 1. *Axial T1-weighted view demonstrating a 2 cm lobulated extra-axial mass (arrow) extending to the right optic foramen with compression of the right optic nerve.*

The patient was started on beta-blocker eye drops. Visual acuity improved to 6/21 within 2 weeks of corticosteroid and chemotherapy. Repeat MRI scan failed to reveal any regression of the orbital apex or other skull lesions. Ten days later, visual acuity in the right eye dropped to counting fingers at 2 m with intraocular pressures of 11 mmHg bilaterally. Radiotherapy, chemotherapy and corticosteroid therapy failed to shrink the orbital apex tumour and optic atrophy was evident on the last follow-up 9 months after presentation.

Comment

Multiple myeloma is one of the plasma cell dyscrasias associated with infiltration of malignant cells, immune alterations and metabolic disturbances. It represents 1% of all malignant diseases, and 10% of haematological malignancies.[3] The onset of multiple myeloma is insidious, commonly being heralded by weakness, anorexia and weight loss. More advanced cases present with symptoms related to renal failure, bone involvement, anaemia or infection.[4] It is rare for multiple myeloma to present with visual symptoms.[6–9] Neurological complications in multiple myeloma were first classified into: (a) cranial nerve palsies; (b) intracranial tumour syndromes; and (c) intraorbital syndromes.[1] Intracranial tumour syndromes tend to present with multiple neurological symptoms and signs, occasionally with those of a space-occupying lesion and raised intracranial pressure. Intraorbital involvement commonly presents with optic neuropathy and this is usually secondary to direct infiltration, a retrobulbar tumour or a hyperviscosity state. Optic neuropathy is uncommon secondary to an intracranial lesion and is usually associated with other neurological signs in these latter cases.

Gudas[6] reported on a 42-year-old carpenter who initially presented with periorbital cellulitis. Multiple myeloma was diagnosed 5 months later with findings of myeloma of the optic nerve. In contrast, 3 cases were mistaken for retrobulbar neuritis after presenting with intermittent blurring of vision and good response to corticosteroid therapy.[7–9] The first 2 cases had chiasmal compression[7,8] and the third case had orbital apex compression of the optic nerve.[9] Kamin and Hepler[7] described a 32-year-old woman who had a 10 month history of visual loss in the right eye. Visual acuity in the right eye fluctuated between 6/120 and 6/9 while on corticosteroids. Visual fields demonstrated a central scotoma in the right eye. Subsequently, visual acuity dropped to light perception in the right eye with associated optic atrophy. Craniotomy revealed a plasmacytoma arising from the tuberculum sella and compressing the optic chiasm. Visual acuity was hand movements following cobalt therapy. Kennerdell *et al*.[8] presented a 47-year-old woman who had acute visual loss in the right eye of 12 days' duration associated with pain on ocular movement. She had 6/120 visual acuity, afferent pupillary defect, loss of colour vision and a dense central scotoma in the right eye. The working diagnosis was optic neuritis and visual acuity improved to 6/6 following corticosteroid therapy. She had recurrent episodes of visual loss and worsening visual fields. Thirty-three months later, multiple myeloma was diagnosed following two craniotomies to decompress the mass compressing the optic chiasm and right optic nerve. Visual acuity was hand movements following radiotherapy. Maini and Macewen[9] described a 73-year-old woman who had intermittent blurring of vision of 3 months duration before developing arm and chest pain. Multiple myeloma was diagnosed and ocular examination revealed 6/60 visual acuity in the right eye with a right afferent pupillary defect, and a generalised constriction of the right visual fields. There was radiographic evidence of right optic nerve compression at the orbital apex. Following radiation, visual acuity improved to 6/9.

We report a patient with unilateral visual loss attributed to optic neuritis. Due to the pattern of the unexplained visual loss and headache, brain imaging was performed and this contributed to the finding of optic nerve compression and to the diagnosis of multiple myeloma. Chemotherapy and radiotherapy are useful in reducing the mass of plasmacytomas. The present patient improved transiently on corticosteroid and chemotherapy and did not respond to radiotherapy. It is possible that the very large plasmacytoma decompensated an optic nerve already compromised from chronic glaucoma. The transient improvement from corticosteroid therapy could be attributed to a decrease in oedema around the orbital apex.

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Sir,

'Hyperacute' unilateral anterior uveitis and secondary glaucoma following streptokinase infusion

Streptokinase is a thrombolytic agent that has been used since 1970 in the United Kingdom in acute myocardial infarction. It is used usually only once in an individual patient due to the risk of anergy. Uveitis is a rare complication of its use. We report the following case of non-granulomatous uveitis and secondary glaucoma 12 h after streptokinase infusion.

Case report

A 72-year-old man was admitted with central chest pain associated with nausea and vomiting. His electrocardiogram suggested a myocardial infarction. He therefore received an infusion of 1 500 000 units of streptokinase. Twelve hours later he developed a painful left eye associated with severely reduced vision.

Initial examination was carried out with a direct ophthalmoscope at the bedside. Intraocular pressure was measured with a Tonopen. On examination, his visual acuity with reading spectacles was N5 with the right and counting fingers at 30 cm with the left eye. His right eye was unremarkable and his left eye showed marked conjunctival injection and corneal oedema. Intraocular pressure was 16 mmHg in the right eye and 49 mmHg in the left. There was a vague red reflex but no detailed fundal examination was possible. A presumptive diagnosis of acute angle closure glaucoma was made and he was prescribed oral acetazolamide (250 mg q.d.s.) and prednisolone 0.5% q.d.s. to the left eye.

He was reviewed 24 h later with a slit lamp. The intraocular pressure was 39 mmHg in the left eye. He had a severe non-granulomatous anterior uveitis with a 1 mm hypopyon (Fig. 1). Dilated fundoscopy showed no significant posterior segment pathology. Intensive topical prednisolone 1% and cyclopentolate 1% q.d.s. were commenced. By the next day his vision had improved and the intraocular pressure was 12 mmHg. Due to posterior synechiae formation, a subconjunctival injection of 4 mg of beclomethasone and one dose of Mydricaine No. 2 were administered.

There was no significant past medical history apart from an episode of pneumonia in childhood for which he had been hospitalised. Other possible causes of anterior uveitis were investigated (serum angiotensin converting enzyme, chest radiograph and full blood count were normal). Anti-streptolysin titre was in the normal range at < 200 IU/ml. Three months later, his left visual acuity was 6/9 unaided.

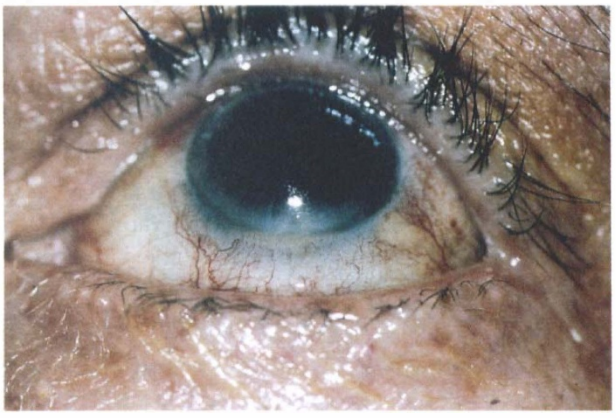

Fig. 1. *Affected eye with hypopyon.*

Comment

Drug-induced acute anterior uveitis is uncommon.[1] The pathogenesis is speculative in most cases.[2] There have been published cases in the literature of bilateral uveitis associated with streptokinase.[3–5] The Committee on Safety of Medicines database revealed 6 reported cases of iritis/uveitis (communication: 1/7/63–10/5/00).

Hypopyon formation is an indicator of severity and is most commonly associated with Behçet's or infectious endophthalmitis. Raised intraocular pressure is usually a late feature in uveitis, the mechanism of which is either related to steroid use or due to damage to the trabecular meshwork.

This case is unusual for several reasons. The patient had unilateral severe uveitis and secondary glaucoma within 12 h of streptokinase administration. The mechanism of anterior uveitis associated with streptokinase has been postulated to be an immune cross-reactivity similar to that described with serum sickness, which typically happens 1–2 weeks after administration of the agent.[5] In contrast to one case report,[5] the patient had no signs of vasculitis and the onset of signs and symptoms was very rapid. Acute anterior uveitis has been described also with trimethoprim–sulphamethoxazole administration, where anterior uveitis recurred within 24 h of reinstitution of therapy.[6] One possible explanation for the rapidity and severity of onset of uveitis in the current case is previous exposure to streptococcal antigen. No other possible cause was uncovered and our patient has no further problems.

We would therefore suggest that, although uncommon, a streptokinase-associated uveitis should be considered by the ophthalmologist in a patient with a recent history of a treated myocardial infarct and a red eye.

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