Case report

A 60-year-old otherwise healthy woman has been treated for epilepsy for 10 years. The first drug she was prescribed was carbamazepine, and 2 years ago vigabatrin was added at a dose of 500 mg q.i.d. A year later sodium valproate was also added. Six months after she started vigabatrin she developed photophobia, flickering lights and saw 'shadows at both sides'. On ocular examination her visual acuity was 20/20 both eyes. She could easily identify Ishihara plates. There was no afferent pupillary defect. Anterior segments and fundi were within normal limits. Intravenous fluorescein angiography did not reveal any abnormality. Automated field testing using the 30-2 program disclosed bilateral crescent-shaped nasal field defects (Fig. 1). A 60-4 full threshold test showed marked annular peripheral field defects; 60-4 blue-on-yellow perimetry demonstrated denser annular scotomas (Fig. 2). Scotopic and photopic electroretinography (ERG) recordings were normal. On electro-oculography (EOG) the Arden ratios were clearly reduced (42% and 30% for the right and left eye respectively). Vigabatrin was tapered to 500 mg t.i.d. Photophobia and flashing lights have resolved. However, the visual field defects persisted without progression. The patient is currently checked every 3 months.

Comment

Visual field defects reportedly develop in 30% of adult patients and in up to 65% of children taking vigabatrin.[2,3] These defects are usually symmetric, absolute and binasal, sparing the temporal field.[1] The concentric contraction of visual fields does not change or improve when the medication is discontinued.[4] Currently, the visual field changes are explained by the accumulation of GABA in the Mueller cells and the GABA transaminase inhibition of the rod bipolar cells in the peripheral retina.[5] This is further supported by the fact that most observed ERG abnormalities involved scotopic b-waves, produced by Mueller cells.[5] As in our patient, there is often a reduced Arden ratio on EOG that is yet to be explained.[1,5]

Some authors believe that the effect is cumulative over a prolonged period of treatment and found a strong correlation between the percentage of visual field changes and total dose of the drug.[2,3] It was suggested that a daily dose of 1500 mg or more would be likely to cause visual field defects.[2] In children, field defects were observed from 1 year up to 6.5 years after the start of vigabatrin.[3] Others did not find any relationship between the dose and duration of therapy and the risk of developing visual field losses.[5] The emergence of this side-effect as early as 6 weeks after the institution of vigabatrin could suggest an idiosyncratic response as well.[6] Our case favours the latter point of view.

The majority of studies in the current literature were conducted on patients who had been taking vigabatrin for many years, and in most of these patients it was not possible to determine exactly when the visual field changes first started. The patient reported here demonstrates that visual field loss may occur soon after the institution of vigabatrin therapy.

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Sir,

Primary tuberculosis of the conjunctiva

Conjunctival tuberculosis is now a very rare condition in the developed world, although it was not uncommon in the nineteenth and the early part of the twentieth century. We present a case of primary conjunctival tuberculosis in a 34-year-old man.

Case report

A 34-year-old male Caucasian General Practitioner with no past medical history presented with a papillomatous lesion in the right upper tarsal conjunctiva. There had been a mucoid discharge for the previous 3 months. The lesion was excised and the patient was treated with topical chloramphenicol. Histopathological examination revealed the inflamed conjunctival tissue with occasional giant cells in the underlying fibrous tissue associated with lipoid material. The appearances were consistent with a diagnosis of chalazion.

The lesion recurred 3 months later, was excised, and the base cauterised. Histopathological examination revealed the presence of mixed acute and chronic inflammatory cells. There were areas of granuloma

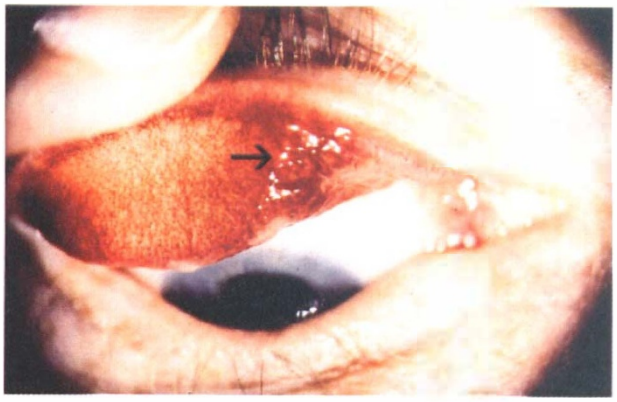

Fig. 1. *Clinical photograph showing a papillomatous lesion of the right upper tarsal conjunctiva (arrow). There is some conjunctival scarring medial to the papillomatous lesion following excision of a previous similar lesion.*

formation and occasional giant cell were seen. No acid-alcohol fast bacilli were seen. This appearance was again thought to be consistent with a diagnosis of chalazion.

Four months later the lesion recurred once again at the original site (Fig. 1) and at this time preauricular lymphadenopathy was noted. After excision of the lesion a sample was sent for histopathological examination and microbiological study. Histopathological examination revealed a granulomatous inflammation (Fig. 2) and *Mycobacterium tuberculosis* was cultured from the tissue. A further nodular lesion had also appeared in the inferior conjunctival fornix. There was no evidence of previous pulmonary tuberculosis. A diagnosis of primary conjunctival tuberculosis was made and the patient was started on a triple therapy with isoniazid, rifampicin and pyrazinamide. The lesion resolved in the next 3 months leaving behind some scarring of the upper palpebral conjunctiva. The treatment was continued for 18 months with no side-effects or recurrence over 3 years follow-up.

Comment

The early history of this condition was reviewed by John Eyre. He reviewed 206 cases which had been reported over a 30 year period from 1882 to 1912.[1] Lundsgaard reported 48 cases in 1915 and Igeisheimer another 22 cases in 1922. There have been only isolated case reports since then.[2–8]

Eyre classified conjunctival tuberculosis into four types: ulcerative, nodular, hypertrophic granulomatous and pedunculated. He found that it commonly occurred in children, adolescents and young adults. It is uncommon for both eyes to be involved. The palpebral conjunctiva was more often involved than the bulbar, and the commonest site was the upper lid. There is usually enlargement of the preauricular lymph nodes. Later, if the infection is untreated, it may spread to the cervical lymph nodes.

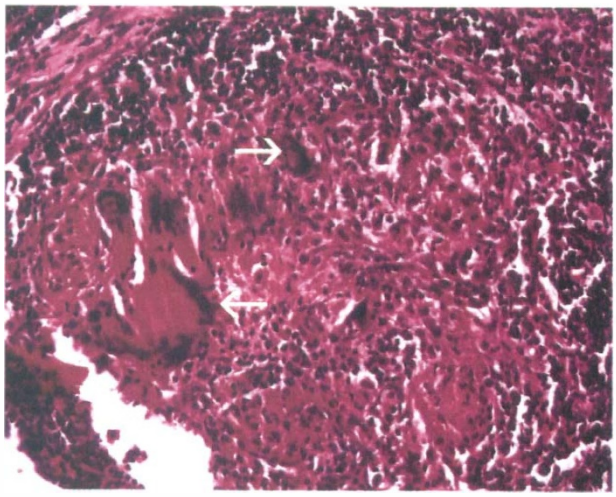

Fig. 2. *Histological section showing granulomatous inflammation with lymphocytes, epitheloid cells and occasional Langhan's giant cells (arrows).*

It is thought that droplets of sputum from patients with active respiratory tuberculosis expelled during coughing can contaminate the conjunctiva of a contact. However, in some patients the infection is autogenous and derived from the patient's own respiratory tract.

The histological picture is characteristic, showing typical tubercles with epitheloid cells, Langhan's giant cells, plasma cells and lymphocytes. The diagnosis is confirmed by identification of the bacilli in the tissue. However, it is important to remember that failure to demonstrate the bacilli in sections is not evidence against a tuberculous aetiology and such a failure might be due to technical difficulties resulting from the small number and size of the bacilli and the difficulty of acid fast tissue staining.[9]

Treatment is with systemic anti-tuberculous therapy, and there is usually rapid resolution of the conjunctivitis with this therapy.

In the last 30 years there has been no case report of conjunctival tuberculosis in the UK. However, it is important to note that although the developed world has seen a dramatic decline in the incidence of tuberculosis, there has been an increase in the annual notification rates of tuberculosis in Europe and the USA since the mid-1980s.[10] This is partly due to the development of the AIDS pandemic, although other factors such as increased mobility across the international borders, a declining public health infrastructure and poverty are also partly to blame. It is quite possible, therefore, that cases of conjunctival tuberculosis might occur more frequently in the future.

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Sir,

Necrotising fasciitis of the periorbital region

Necrotising fasciitis is a life-threatening bacterial infection characterised by necrosis of subcutaneous tissue and underlying fascia, with rapid spread of the infection and inflammation along fascial planes. It rarely involves the face and periorbital region.[1] Early diagnosis and prompt treatment are essential to prevent the high morbidity and mortality associated with this condition. Although *Streptococcus pyogenes* is the most frequent isolate, a mixture of facultative and anaerobic bacteria can also lead to this condition.[2,3] We present a case where prompt diagnosis and instigation of appropriate high-dose antibiotics limited the need for extensive debridement.

Case report

A 37-year-old Caucasian man presented at 4 a.m. to the Eye Casualty. He had allegedly been assaulted and hit with a baseball bat on the right side of his face at 11 a.m.

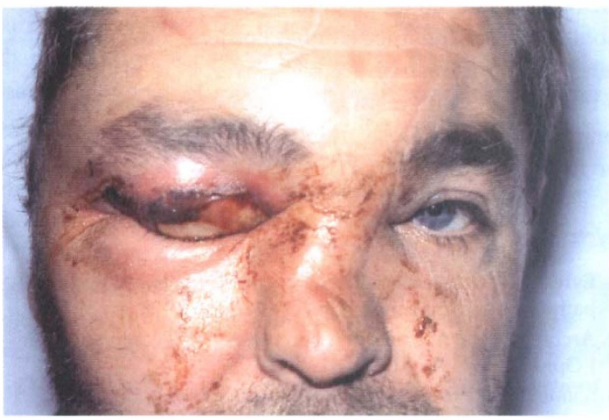

Fig. 1. *Photograph showing sloughed necrotic skin of the upper lid and woody swelling on the right side of the face.*

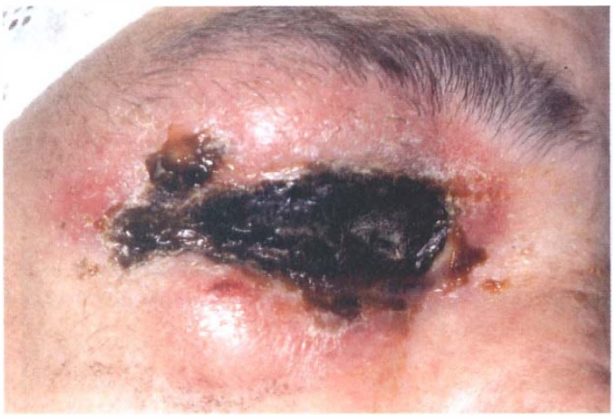

Fig. 2. *Photograph showing necrotic eschar formation of the upper lid.*

the previous day. He gave a history of brief loss of consciousness. He was a known alcoholic with consumption of 60 units of alcohol per week.

At the time of presentation his temperature was 38.5 °C. He felt unwell and complained of considerable pain on the right side of his face. On examination there was gross swelling of the eyelids and periorbital region on the right side. He also had a superficial laceration on the upper eyelid above the lateral canthus. Eye examination revealed a normal anterior segment. There was no proptosis and ocular movements were normal. The pupillary reactions were equal with no relative afferent pupillary defect. Skull and facial radiographs showed no fractures. His white blood cell count was $20.15 \times 10^9/mm^3$ which was suggestive of significant bacterial infection. The plasma urea and electrolytes, liver function tests and clotting factors were normal.

A provisional diagnosis of traumatic haematoma with preseptal cellulitis was made and he was started on intravenous augmentin 1.2 g t.i.d. After 5 h he was still unwell and remained pyrexial. He also developed woody swelling of the periorbital region, which extended from the forehead down to the side of his face to the mandibular angle. There was a darker purple area over the upper lid with some flaking of the skin (Fig. 1). At this point a clinical diagnosis of necrotising fasciitis was made. He was started on intravenous benzylpenicillin 1.2 g 4-hourly, intravenous cefuroxime 1.5 g t.i.d. and intravenous metronidazole 400 mg t.i.d. A wound and eye swab was taken and microscopy showed Gram-positive cocci suggestive of streptococci. An urgent microbiology and dermatology opinion was sought. Twenty-four hours later his temperature had lysed and he felt much better but the dark area in the upper lid had worsened and looked necrotic and crusted (Fig. 2).

The blood culture and wound swab grew Gram-positive Group A streptococci after 3 days. He continued to improve clinically. After 7 days of intravenous antibiotics the woody swelling had subsided but the area of necrosis was larger. He therefore underwent debridement of the upper lid on the eighth day. Three days later he had upper lid reconstruction with axillary skin graft.