Most reports of self-inflicted eye injury record attempted self-enucleation,[4] which is commonly associated with schizophrenia but also with drug use,[5] obsessive-compulsive neuroses and organic states such as epilepsy and encephalitis. Other forms of ocular self-mutilation are described such as eye-banging,[6] alkali burns[3,7,8] and cellulitis due to self-injection.[3] This case emphasises the addition of cicatricial conjunctivitis to this list even in cases with no apparently significant psychiatric history.

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Sir,

Periorbital necrotising fasciitis in a child

Necrotising fasciitis (NF) is a soft tissue infection caused by toxin-producing virulent bacteria. NF of the face is extremely rare in children; only 3 cases have been described in the literature. We describe a case of periorbital NF in a child.

Case report

A 5 years and 6 months old boy presented to the eye casualty with swelling of the right uppr and lower lids 12 h after being kicked in the right eye. He had a temperature of 39.1°C, pulse was 110/min, he vomited once and on central nervous system examination he was fully conscious and orientated but lethargic. He had marked periorbital redness and swelling making it difficult to assess his vision, but he could see 6/5 with his left eye. A plain radiograph of the orbits did not reveal any fracture.

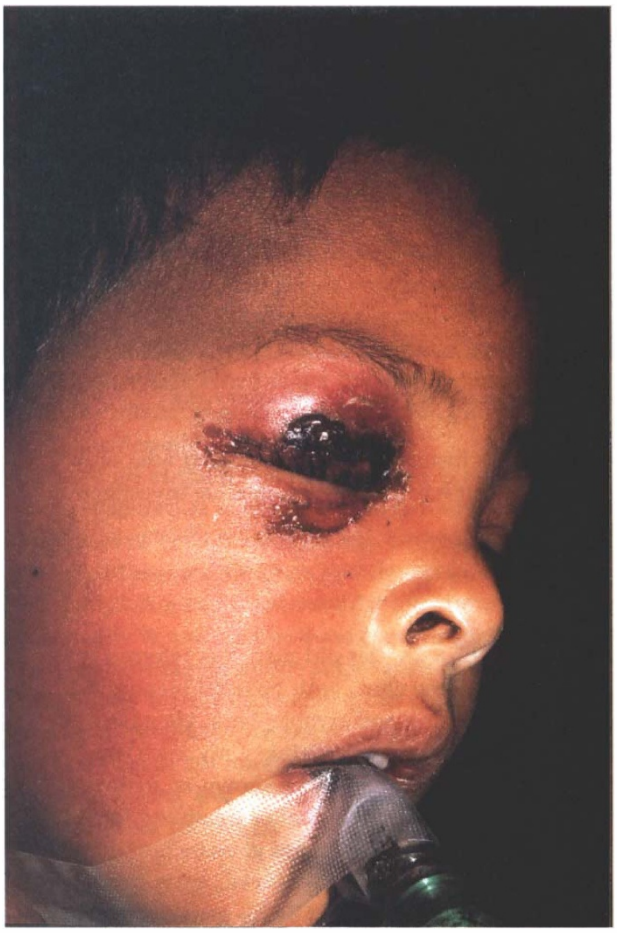

Fig. 1. *Oedema and necrosis involving the whole upper lid.*

A working diagnosis of orbital cellulitis was made and the child was admitted under the paediatricians. FBC, CRP, U+Es were all normal (WCC 15.7); also an eye swab and blood culture were taken. The child was then started on intravenous flucloxacillin (100 mg/kg per day), cefotaxime (200 mg/kg per day) and metronidazole (22.5 mg/kg per day).

On the second day a CT scan showed evidence of large soft tissue mass in the front of the right orbit extending medially to the right side of the nose and laterally to the lateral orbital margins without involvement of the optic nerve or the extraocular muscles. There was no evidence of gas collecting within orbital tissues.

On the third day the child was apyrexial but with increased pain, tenderness and erythema of the right cheek. There was no obvious proptosis and ocular movements were normal.

An eye swab grew group A *Streptococcus pyogenes* and *Staphylococcus aureus*, while blood culture grew *Streptococcus pyogenes* (group A). Antibiotics were changed to intravenous benzyl penicillin (1 g) and chloramphenicol (500 mg q.d.s.), according to sensitivity results. Blood chloramphenicol levels were checked daily.

On the fourth day black necrotic spots started to develop on the skin of the upper lid and the suspicion of necrotising fasciitis was raised.

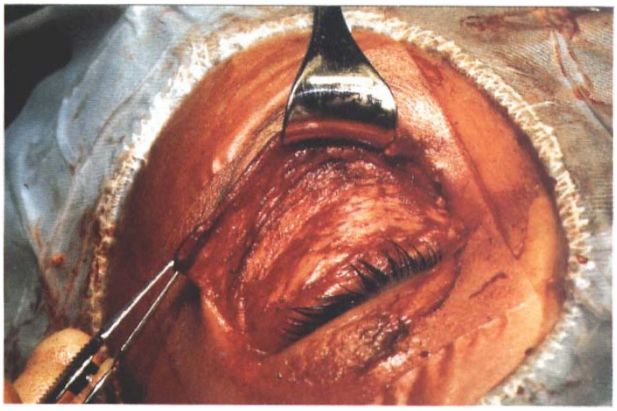

Fig. 2. *Extent of the defect following debridement.*

On examination under anaesthesia, gross upper lid tissue necrosis was seen which was extending beyond the right eyebrow but with no evidence of gas collection. All necrotic tissue was excised. Fundus examination showed a normal optic disc and vessels. Over the next few days the child continued to be apyrexial, there was no spread of lid necrosis and further blood cultures were negative. He continued the course of antibiotics for 7 days and then was discharged with 6/6 vision in his right eye and full ocular motility. In clinic follow-up visits most of the skin of the upper lid was seen to be replaced by granulation tissue, with incomplete lid closure and signs of corneal exposure.

Three months later he underwent excision of scar tissue with upper lid skin graft (from the post-auricular area). Post-operatively good lid closure was achieved with good corneal protection.

Comment

NF is characterised by widespread necrosis of superficial and deep fasciae sparing the skin and underlying muscle in the early course of the disease, but later the infection spreads to involve the surrounding tissue and can lead to systemic toxicity and septicaemia. It was first described by Meleney in 1924, who gave the name 'haemolytic streptococcus gangrene' to the disease.[1] Hippocrates described NF as a complication of erysipelas.[2]

NF most commonly affects the abdominal wall, perineum and the extremities[3,4] and can be predisposed to by diabetes, alcohol abuse, immunosuppression, intravenous drug use, metastatic cancer, peripheral vascular disease, chronic renal failure, malnutrition and myxoedema.

The head and neck area is not a common site for NF but in the face it usually affects the periorbital region secondary to trauma, eyelid infection or eyelid surgery (one case was reported after laser blepharoplasty).[5] Only 3 cases of facial NF have been described in children, two of whom had head and neck disease;[6] only one child had periorbital NF.[7]

The skin of the affected area is erythematous, swollen and tender. Later bullae, containing serous fluid, appear. Black discoloration of the skin and liquefactive necrosis follow, secondary to the release of hyaluronidase and lipase from bacteria. It is important to note that skin necrosis is a relatively late sign and that the extent of fascial necrosis is more widespread than the extent of the overlying skin lesions.

NF of the face, in comparison with periorbital cellulitis and erysipelas, has more rapid progression and skin necrosis can develop within hours. Myonecrosis (clostridial or non-clostridial) and bacterial gas gangrene (skin necrosis as opposed to fascial necrosis) can also give clinical picture similar to NF.

Diagnosis of NF is based on the clinical picture, laboratory tests and imaging. Laboratory tests show a high white blood cell count with positive tissue and blood cultures. Rapid streptococcal screen of serosanguinous fluid from bullae can be helpful and is performed in some emergency departments.[8] The most common bacterium responsible for NF is GAS (group A, beta haemolytic streptococcus). Other organisms involved are *Staphylococcus aureus*, *Bacteroides* species and *Haemophilus influenzae* type B.[3,4,9] There has been one report of NF due to *Clostridium septicum* without myonecrosis[10] and one case due to *Serratia marcescens*[11] in a diabetic patient.

According to the organisms identified in cultures, NF has been classified into: polymicrobial type 1 caused by non-group-A streptococci, anaerobes and/or facultative anaerobes; type 2 caused by GAS alone or in combination with *Straphylococcus aureus;* and type 3 caused by *Vibrio* species after exposure to sea water.[4] The preferred site for obtaining culture material is the centre of the necrotic lesion, in distinction to cellulitis where the leading edge of the lesion is aspirated.

CT and MRI are useful diagnostic tools to delineate the extent of the infection[12] and to reveal gas dissecting along fascial planes. MRI is superior to CT in delineating abnormal soft tissue, but in our case CT was chosen as it was readily available.

Treatment of NF includes antibiotics, surgery and hyperbaric oxygen.

The initial choice of antibiotic should cover Gram-positive and Gram-negative aerobes and anaerobes, which is adequately achieved by a combination of cephalosporin or penicillin, aminoglycoside and either metronidazole or clindamycin. The choice of antibiotics should be modified according to culture and sensitivity

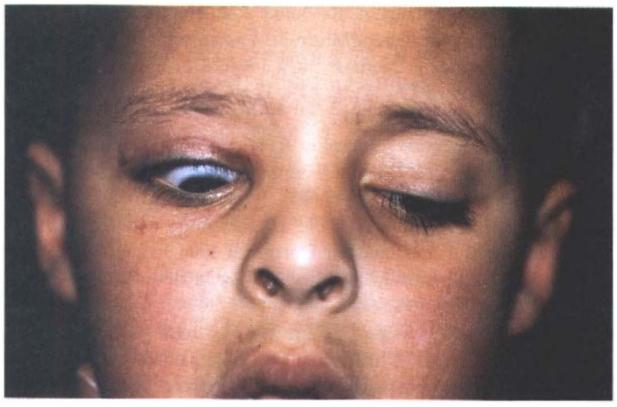

Fig. 3. *Lagophthalmos 3 months after debridement.*

results. It is often noted that patients do not do very well on B-lactam antibiotics despite bacteria being sensitive to them in cultures. The classic treatment of NF would be a combination of benzyl penicillin and clindamycin. In our case the isolated *Streptococcus pyogenes* was sensitive to penicillin. The *Staphylococcus aureus* was sensitive to chloramphenicol, but resistant to both penicillin and erythromycin, so clindamycin was not a first choice antibiotic, due to the phenomenon of dissociated resistance with erythromycin, and chloramphenicol was used instead. Chloramphenicol also gives good cover for anaerobes; hence metronidazole was stopped.

Antibiotics alone are unable to penetrate necrotic tissues and have limited effect until extensive fasciotomy and excision of all necrotic tissue is done. After surgery the involved areas should be watched carefully for expansion of necrosis and repeated debridement should be performed if necessary. The skin defect is initially allowed to granulate and secondary reconstruction with skin grafts should be delayed for 3–6 months unless there is corneal exposure; then reconstruction should be performed as soon as the infection has resolved.

Good nutritional support, aggressive fluid resuscitation and analgesia are important measures of treatment. The use of hyperbaric oxygen has been suggested but its efficacy is not known.

NF, if not treated adequately, can lead to ophthalmic artery occlusion, severe skin loss with lagophthalmos, ectropion, epicanthus and significant cosmetic disfigurement. NF involving the neck area carries a higher mortality rate as it tends to spread caudally to the chest and mediastinum, often resulting in pulmonary complications and death. This stresses the importance of early recognition and proper treatment of the disease.

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Sir,

Traumatic prolapse of the globe into the maxillary sinus diagnosed as traumatic enucleation of the globe

A pure blow-out fracture is a fracture of one or more of the orbital walls without involvement of the orbital margin.[1] To our knowledge there have been only a few documented cases of blow-out fracture with prolapse of the entire eyeball into the maxillary sinus.[2–4]

CT scan is diagnostic and is helpful for subsequent management.[5] In our patient the condition was initially misdiagnosed as traumatic enucleation of the globe. Two months later a CT scan of the orbits and paranasal sinuses showed the prolapsed eyeball in the maxillary sinus.

Case report

A 29-year-old man presented to the eye casualty department with severe pain in the left orbit. He had been the victim of an assult in which he had suffered blunt trauma to the left orbit. The patient could not recall the exact circumstances of the accident because he was under the influence of alcohol. Following the trauma he had immediately lost all sight in the left eye and experienced neurosensory loss in the distribution of the infraorbital nerve. On examination he had no light perception on the left side and there was marked left periorbital oedema.

The eyelids were opened with a speculum and the globe appeared to be absent. On examination under general anaesthesia the globe, extraocular muscles and optic nerve were not found. A diagnosis of traumatic enucleation of the globe was made. A central conjunctival defect was sutured. The patient was subsequently followed up in the eye clinic and 2 months later was seen in the oculoplastic clinic for consideration of secondary orbital reconstructive surgery. A pre-operative CT scan showed a huge pure blow-out fracture of the orbital floor with prolapse of the entire globe into the maxillary antrum (Fig. 1).

The patient was subsequently referred to the oculoplastic service at Manchester Royal Eye Hospital. The patient had developed a marked socket contracture (Fig. 2). A surgical exploration of the socket and of the blow-out fracture was undertaken. The globe was firmly adherent to the walls of the maxillary antrum and could not be reposited into the contracted socket (Fig. 3). The globe was enucleated. Of the extraocular muscles only the horizontal recti could be identified. A scleral wrapped 18 mm hydroxyapatite implant was placed