Correspondence

Necrotising fasciitis of the eyelids

Sir. A fatal case of this infection, involving a 35-year-old man in Southampton was described recently in your journal by Walters. He has referred to four previously described cases, including two in Southampton, but Carruthers et al., Bahna M, Holliman and Catford have described nine others.

The provisional diagnosis of preseptal cellulitis, with erythema and oedema of the right forehead, eyelid, cheek, and neck following a poke in the eye by the patient's daughter, together with purulent discharge from the eye containing Gram-positive cocci (? streptococci) should have prompted the diagnosis of β-haemolytic streptococcal (BHS) or staphylococcal sepsis. Coliform bacteria, as suggested by Walters, are not associated with causing this infection at this site, but can cause necrotising fasciitis of the abdominal wall in association with Bacteroides spp following abdominal injury or surgery. The initial drugs of choice are benzylpenicillin in very large doses (20 to 30 megaunits per day intravenously) together with flucloxacillin 4 G intravenously per day until results are known. Third generation cephalosporins, namely, cefotaxime, together with gentamicin and chloramphenicol, are not indicated, nor is metronidazole, as anaerobic bacteria such as Bacteroides spp, fusobacteria, and anaerobic streptococci, which rarely cause this infection originating from an oral focus, are sensitive to benzylpenicillin. If the patient is allergic to penicillin, then cephalosporin or erythromycin should be used instead.

It is known that sudden death in this infection can occur from disseminated intravascular coagulation (DIC) due to BHS septicemia, from which the patient probably suffered. Anticoagulation with heparin should be considered in the early stages of management, supported by haematological parameters, and is life saving. The infection spreads rapidly through tissue causing thrombosis of vessels above the fascial layer, for which surgical debridement is preferred as antibiotics penetrate into the necrotic tissue with difficulty, reflecting the need for very large doses. Bahna M, Canalis RF. Necrotising fasciitis. Arch Otolaryngol 1999; 106: 648–651. Holliman RE, Catford GV. Periorbital necrotising fasciitis with loss of vision. J Infect 1986; 13: 35–6.

Early recognition of the clinical features of these infections can be life saving, but fortunately they occur rarely nowadays and the actiology is often missed. The infection advances rapidly, giving the skin a patchy blue appearance; if blistering occurs it is pathognomonic. Patients also develop toxaemia with a characteristic euphoria and sloughing of the affected part, especially when it becomes anaesthetic. Some patients develop early fatal septicemia and DIC, often occurring over as little as 12 hours, while in others the infection remains localised to necrotic tissue, probably reflecting the host response for both opsonic phagocytosis as well as the propensity for small vessel thrombosis. This type of infection is more common in diabetics and the elderly.

BHS group A (Streptococcus pyogenes) M type 73 was isolated in pure culture from the blood and lid of Walter's patient. We and others have similarly isolated a pure growth of BHS growth A from four patients with this type of lid infection. but, in their review, Bahna and Canalis identified three out of eight patients in whom both BHS group A and Staphylococcus aureus were isolated; Carruthers et al. also isolated this combination. In a rabbit model of this spreading infection we have found synergy between Staph. aureus or its α-lysin and BHS group A for initiating spread. Such initial synergy may occur in humans as well, though Staph. aureus has only been isolated from 17% of 36 cases. This synergy is missed if the spreading edge of the infection, or the necrotic slough, is cultured rather than the initial site of invasion; ideally, both sites should be investigated. The cause of the spreading thrombosis is probably toxin mediated and not due to the inflammatory effect of streptococcal cell wall.

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References


Sir. Dr David Seal's letter is interesting and comprehensive and I am grateful for the information regarding other cases of necrotising fasciitis involving the eyelids.

I would certainly concur with Dr Seal that the β-haemolytic streptococci is the principal causative organism of necrotising fasciitis and that high dose benzylpenicillin is the treatment of choice (with the support from flucloxacillin as cover against Staphylococcus aureus). The choice of anti-
bacteria. This diagnosis has been established and organisms isolated, but I feel that when the patient is first seen and admitted a high dose broad spectrum antibiotic is justified but agree that it should be accompanied by flucloxacillin. The diagnosis of necrotising fasciitis is first made on clinical grounds, and it can take 24 to 48 hours for the pathognomonic clinical features to appear. I suspect that many cases are missed. I would strongly recommend that, as in this case, the microbiologists are involved from the start and that an opinion should also be sought from one’s dermatological colleagues.

Disseminated intravascular coagulation (DIC) can indeed occur and cause death but was not found in the patient I described.

One should always watch out for orbital cellulitis when confronted with lid infections. However, my understanding is that necrotising fasciitis is confined to the skin and should not directly affect deeper tissues or penetrate the orbital septum to cause an orbital cellulitis.

I am aware that some cases of necrotising fasciitis of the eyelid have been treated conservatively and that the patient has survived, though requiring reconstructive lid surgery. However, I still feel that early surgical débridement (together with intensive antibiotic treatment) is the treatment of choice provided the diagnosis is established early enough.

I have recently heard of a case in which the affected area of skin was surgically reflected and the deep surface cleaned of inflamed and necrotic tissue. The underlying fascia was also cleaned. The skin was then laid back into position (as per a skin graft with a pedicle) and recovered well. This is an interesting approach and may be a way of avoiding the serious cosmetic consequences of excision of a large area of affected facial skin.

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Optic nerve involvement in methanol poisoning

Sir, I was very much interested to read the paper entitled ‘Optic nerve involvement in a case of methanol poisoning’ by Peter Naeser. This paper contains some serious errors of omission as well as commission. To set the record straight, I would like to point these out. They are as follows:

1. We produced experimental methyl alcohol poisoning in rhesus monkeys and investigated exhaustively the subject of ocular and optic nerve toxicity by conducting detailed clinical, morphological, and biochemical studies on the subject.5 The author makes no mention of our studies at all. Our studies proved that the principal ocular lesion seen in methanol poisoning is the development of toxic optic neuropathy; this contradicts the statement by the author that ‘the optic nerve has only infrequently been investigated.’ We investigated the optic nerve in detail by light and electron microscopy and found swelling of the oligodendroglial cytoplasm in contact with the axons and of the astrocytes in the retrolaminar optic nerve and the intra-orbital optic nerve. We found no vascular lesions in the

2. The author states that ‘The perfusion of the central parts of the nerve from a central optic nerve vessel with different extension may be of importance.’ The author cites a 25-year-old paper by Francois and Neetens in support of the existence of a central artery of the optic nerve; a few months after that paper was published I pointed out the fallacies in this paper by Francois and Neetens and disproved conclusively the existence of any such central artery of the optic nerve, based on my studies.6,7,8 My detailed studies on the ophthalmic artery and blood supply of the optic nerve9 (to which the author makes no reference in the paper) showed that no such artery exists in man.

This finding was later confirmed in studies by several other authors, including Francois’ own group subsequently,9,10,11 when they stated that in their series of 40 optic nerves they did not find any central artery of the optic nerve.11,12 I thought the mythical central artery of the optic nerve expired long, long ago. The concept of a watershed zone in the retrolaminar part of the optic nerve, postulated by Roodman and Butler,13 totally lacks any scientific proof. Thus the basic assumptions regarding the blood supply of the optic nerve in this paper are invalid.

It is a tragedy that the subject of the blood supply of the optic nerve has been plagued for decades by a very serious problem. To explain ischaemic disorders of the optic nerve according to their preconceived theories many authors from time to time have resorted to misstating and distorting the observed facts about the blood supply of the optic nerve: to suit their own convenience they have ignored well established anatomical and physiological facts. To create anatomy to suit a pet theory instead of vice versa is the exact opposite of scientific inquiry. No matter how often they are disproved, such pet theories once published, persist like skeletons which may tumble out of the closet years later, like the mythical central artery of the optic nerve, so miraculously resurrected in this paper.

The author, based on one case, implies that optic nerve damage in methanol poisoning is due to vascular disturbance in the optic nerve. Our studies, and those by many other authors, based on much more extensive material, lend no support to this view. Thus this misleading paper, in an effort to explain the optic nerve lesions in one case, ignores all the weight of evidence available on the subject of changes in the optic nerve in methanol poisoning and on the blood supply of the optic nerve. This seriously undermines its scientific credibility.

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References