Periorbital necrotising fasciitis

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Periorbital necrotising fasciitis is an uncommon suppurative condition seen following trauma or in debilitated patients and frequently requires extensive debridement. The basic pathological process is a cellulitis followed by suppuration and necrosis of the subcutaneous tissues and spread along the fascial planes of the submuscular aponeurotic system. The most frequently responsible pathogenic organism is the group A β haemolytic Streptococcus although other pathogens have been implicated. A case in a previously healthy man which resolved on antibiotic and steroid therapy is presented.

Case report

A 78-year-old man presented with a 24 hour history of acute periorbital swelling associated with severe pain. He had had an immediately preceding febrile illness and felt unwell with episodes of rigor and sweating. There was no previous ophthalmic or medical history of note. On examination he had a mild pyrexia, periorbital oedema with discharge from both eyes and was admitted with a provisional diagnosis of bilateral preseptal cellulitis. After conjunctival swabs and blood cultures he was started on intravenous doses of flucloxacillin and ampicillin. Over the next 48 hours the periorbital swelling worsened with ulceration and soft tissue necrosis, a purpuric rash thought to be due to a secondary immune vasculitis developed over the dorsum of both feet and ankles (Figs 1, 2), and he became neutropenic with a white cell count of 2.2 x 10^9/l. Prednisolone was commenced at 60 mg/day.

He was transferred to the eye unit and further tests showed an erythrocyte sedimentation rate of 70 mm/hour, with protein electrophoresis showing a polyclonal increase in gammaglobulins. Other investigations including radiography (and later computed tomography) of the sinuses and orbit, an autoimmune screen, and creatine kinase levels were within normal limits. A subsequent full blood count showed a partial resolution of the neutropenia and his general condition started to improve, his rash settled, and the periorbital swelling and necrosis began to subside. Ampicillin was replaced by penicillin after cultures grew group A β haemolytic Streptococcus and Staphylococcus aureus, and after 1 week his prednisolone was slowly decreased and tapered down over 4 weeks.

His antibiotics were continued and the necrotic crust on the right upper lid sloughed away at 4 weeks (Fig 3) with the left 2 weeks later. Raw areas healed by secondary intention and at 10 weeks he had minimal scarring, good lid closure, no trichiasis, and only slight thickening of the upper epicanthic folds (Fig 4).

Comment

There have been 19 reported cases, including one fatal case, of periorbital necrotising fasciitis and these cases have usually been seen in associa-

![Figure 1](image1.png) Bilateral cellulitis with necrosis 48 hours after admission.

![Figure 2](image2.png) Vasculitic rash before treatment with steroids.
tion with trauma or a concurrent debilitating process such as diabetes, alcoholism, or polymyositis. The role of surgical exploration and debridement with irrigation using a hydrogen peroxide solution together with high dose antibiotics has been emphasised. The pathogenesis of such rapidly spreading necrosis is uncertain. A hypersensitivity component such as an Arthus response has been suggested while an alternative hypothesis of activation of proteolytic enzymes with collagen necrosis has also been postulated. Studies of the Arthus response in experimental animals have shown that it can be suppressed by depleting the animal of complement or neutrophils and the intensity of the response can be reduced in animals and humans by corticosteroids. The transient leucopenia has been reported previously and is due to convergence of granulocytes in affected areas, spleen, and lymphatics.

This patient showed some unusual features of the disease. There was no history of trauma or other underlying pathology, his general condition improved markedly after the introduction of steroids, and he settled without surgical intervention. The use of adjunctive steroids in the reported cases is not well described (except in the case associated with polymyositis) and in cases where the underlying infection is poorly controlled their use might have serious consequences precipitating a potentially fatal bacterial septicemia.

Appropriate high dose antibiotic therapy, often with surgical debridement, is the first line treatment in this disease. It may be, however, that in certain cases where the infection is controlled adjunctive steroids could limit the vasculitis and subsequent tissue damage which occurs. This patient’s excellent recovery on conservative treatment may be due to the absence of any predisposing pathology but may also be a reflection of the effects of steroids on the pathogenesis of this aggressive disease.

I would like to thank Mr J J Kanski for his help and for allowing me to present his patient.


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**Ocular complications associated with bungee jumping**

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Bungee jumping originated in the islands of the South Pacific as a means of initiating young males into the realms of manhood. A length of vine was attached to their legs, but today this is replaced with a bungee rope. The sport is increasing in popularity with a wide range of age groups taking part. It is reported to be dangerous, with lethal accidents caused by miscalculations of the extent to which the rope will stretch, and in one case, a jumper who forgot to attach his
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