rence.^{4,5} Because the results reported by several authors cannot be equally compared because of differences in their detailed records of courses of diseases, it is hard to select a unique factor affecting the ophthalmic findings in these patients.

Recently, massive corticosteroid treatment of VKH was clinically reinvestigated.⁶ Yamamoto *et al*⁶ found a similarity in the prognosis of patients treated with non-steroidal anti-inflammatory agents and those treated with massive doses of systemic corticosteroids. Therefore, it was difficult to determine if the remission of the disease during pregnancy in our patient was spontaneous or due to the pregnancy.

Although a good clinical result has been

obtained without the systemic administration of high doses of corticosteroids or topical corticosteroids on the basis of careful examinations of a patient with VKH in early pregnancy, further discussions will be required about its treatment during pregnancy.

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Fatal bilateral necrotising fasciitis of the eyelids

Department of
Ophthalmology, King's
College Hospital,
Denmark Hill, London
SE5 9RS

D Kent
P L Atkinson
E W G Davies

Department of
Medical Microbiology,
King's College School
of Medicine and
Dentistry, Bessemer
Road, London SE5 9PJ
B Patel

Correspondence to:
Dr D Kent, Department of
Ophthalmology, Link 8Z,
Royal Liverpool University
Hospital, Prescott Street,
Liverpool L7 8XP.

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D Kent, P L Atkinson, B Patel, E W G Davies

Necrotising fasciitis is an uncommon soft tissue infection usually affecting the trunk, perineum, or legs after surgery or trauma. Infection spreads rapidly along subcutaneous fascial planes and produces overlying skin necrosis. Head and neck involvement is rare, with scalp and neck fasciitis almost always after trauma or dental sepsis. Midfacial and peri-orbital infection is notable in that a history of trauma may be absent or such trauma may be minor. We report a fatal case of bilateral necrotising fasciitis of the eyelids with no history of preceding trauma in which *Streptococcus pyogenes* (β haemolytic streptococcus

Lancefield group A) was cultured from the eyelids, blood, and throat.

Case report

A 40-year-old man with a history of alcoholic liver disease presented with a 24 hour history of increasing bilateral painful periocular and facial swelling. On examination he was afebrile, mildly jaundiced with ascites and hepatomegaly. The lid skin was markedly swollen with a small patch of dusky skin on the right lower lid. Intravenous benzylpenicillin and flucloxacillin were commenced and the facial swelling began to subside over the next 24 hours so the eyes could be opened revealing normal globes. However, within 48 hours the lid skin became dusky, and progressed to frank lid gangrene (Fig 1). Investigations revealed anaemia (haemoglobin 9.7 g/dl), a white blood count of $8.2 \times 10^9/l$, thrombocytopenia ($82 \times 10^9/l$) and an abnormal clotting screen (INR 2.4). *Streptococcus pyogenes*, sensitive to penicillin and erythromycin, was isolated from the cultures of the eyelids, blood, and throat. A diagnosis of necrotising fasciitis was made. Clinically the patient began to deteriorate and surgical debridement was delayed until 7 days after presentation. Under local anaesthesia all necrotic periorbital skin and subcutaneous tissue were debrided revealing healthy orbicularis muscle beneath (Fig 2). However, renal and circulatory failure developed and, despite intensive supportive measures, he died 15 days after his admission.

Figure 1 Severe bilateral gangrene of the periorbital skin and subcutaneous tissue.

Figure 2 Appearance following debridement of all necrotic tissue.

Figure 3 Intense acute inflammatory response with polymorphonuclear leucocytes infiltrating between striated muscle fibres (haematoxylin and eosin, $\times 83$).

Histology of the debrided tissue showed full thickness necrosis of the skin and underlying fat with a diffuse heavy infiltrate of neutrophils with sparse Gram positive cocci (Fig 3). Subsequent typing of the organism showed it to be a *Streptococcus pyogenes* type M1, T1.

Comment

Necrotising fasciitis is a soft tissue infection caused by group A β haemolytic *Streptococcus* either alone or in combination with other organisms, most commonly with *Staphylococcus aureus*.¹ Progression is rapid, with initial erythema and tenderness becoming frank gangrene in as little as 24 hours. Spread along

subcutaneous planes results in necrosis of fascia, fat, and overlying skin but usually with preservation of underlying muscle. Systemic upset is characteristically severe with fever, neutropenia, and septicaemia which may progress to multiple organ failure and death.

Necrotising fasciitis without preceding trauma or surgery is rare, but has been reported in three cases of periorbital infection.^{2,3} In this case infection could have occurred via unnoticed skin trauma or sepsis, or possibly haematogenous spread from the pharynx.

Invasive disease due to group A *Streptococcus* has been reported more frequently since the late 1980s and this may be due to a change in serotype distribution, with the more invasive types M1, 3, and 18 being more prevalent (M protein is an organism cell wall constituent⁴). The rapid progression and spread seen in necrotising fasciitis may well be related to the exotoxins produced by the *Streptococcus* such as pyrogenic exotoxin, streptokinase, and hyaluronidase.

Successful treatment depends on early recognition of the condition, early surgical debridement, and the currently recommended antimicrobial treatment of high parenteral doses of benzylpenicillin with the addition of clindamycin in severe cases.⁵ Furthermore, it must be remembered that the subcutaneous spread is often more extensive than the involvement of the overlying skin while the preservation of the orbicularis and the eyelid margins will simplify reconstructive surgery.

Although rare, particularly without a history of trauma, prompt recognition of this potentially fatal infection is necessary for successful treatment. The apparent change in the spectrum of the disease caused by *Streptococcus pyogenes* may be related to the prevalent M serotypes and the ability of the organisms to produce pyrogenic exotoxins. If these serotypes become more prominent then invasive *Streptococcus pyogenes* may become more common.

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