Necrotising fasciitis of the eyelid with toxic shock due to *Pseudomonas aeruginosa*

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ABSTRACT

Necrotising fasciitis is a rare and rapidly spreading soft tissue infection characterised by widespread necrosis of the superficial fascia and usually occurring in the limbs and the abdominal wall. Periocular necrotising fasciitis is unusual due to the excellent blood supply of the facial region. The usual pathogens are Group A beta-haemolytic *Streptococcus* and *Staphylococcus aureus*. We report a case of *Pseudomonas* necrotising fasciitis of the eyelid with septic shock, initially diagnosed as hordeolum in a young immunocompromised Chinese woman. Early recognition of the condition, followed by timely intervention with surgical debridement and intensive intravenous antibiotic treatment led to a favourable prognosis. It is important for general physicians to recognise the cardinal signs of necrotising fasciitis, as early treatment with timely surgical debridement and supportive medical therapy is the mainstay to successful management.

Keywords: necrotising fasciitis, periocular infection, *Pseudomonas aeruginosa*, septic shock

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INTRODUCTION

Periorbital necrotising fasciitis is an uncommon, life-threatening infection characterised by necrosis of the subcutaneous tissues spreading rapidly along the soft tissue planes. Its mortality rate, usually due to septicaemic shock and organ failure, ranges from 12%–57%. Group A beta-haemolytic *Streptococcus* (GABHS) is the most common isolate, although other pathogens have been implicated.[1] We present the case of a young immunocompromised Chinese woman with left periocular necrotising fasciitis due to *Pseudomonas (P.) aeruginosa*.

CASE REPORT

A 22-year-old Chinese woman with multiple medical problems was admitted in February 2007 to our tertiary care centre with a history of fever, left eye swelling and redness for two days, which became progressively worse. The patient also reported some purulent discharge from the eye. There was neither a history of trauma nor insect bite to the eyelid. The patient had undergone a successful cadaveric renal transplant in August 2006 for end-stage renal failure due to congenital bilateral dysplastic kidneys and was put on long-term oral tacrolimus, mycophenolate and prednisolone after the transplant. She subsequently developed post-transplant diabetes mellitus and was started on subcutaneous insulin.

On the first presentation, the patient's left eye was closed by erythematous and tense swelling of her left upper lid, but her eye movements were normal, and there was no proptosis or relative afferent pupillary defect. The full blood count showed a white blood count of 3.35 $\times$ 10^9/L, which was in the normal range (3.30–9.66 $\times$ 10^9/L), with neutropenia (0.73 $\times$ 10^9/L). A presumptive diagnosis of hordeolum with preseptal cellulitis was made, and the patient was administered with intravenous ampicillin and cloxacillin, 500 mg each, six-hourly.

The patient was followed up closely, and on review four hours after admission, she was noted to be more septic. There was a spike in her body temperature. She also complained of increasing pain over the eyelid. A computed tomography of the orbit performed on the next day revealed diffuse thickening and enhancement of the left preseptal tissues with a loss of fat planes. There was no evidence of intraorbital inflammation or abscess. The following day, an area of blackish discolouration was observed over the patient's left upper eyelid. At the same time, she developed hypotension and tachycardia, and was promptly transferred to the intensive care unit for haemodynamic and cardiovascular support. In view of her rapid deterioration and the new clinical findings, a diagnosis of necrotising fasciitis with toxic shock was made. The intravenous antibiotics were then changed to meropenem 500 mg eight-hourly and clindamycin 300 mg six-hourly. The patient underwent emergency wound debridement. Intraoperatively, she was found to have necrosis of the full thickness of the left upper eyelid

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Fig. 1 Photograph shows the upper eyelid with blackish discolouration and surrounding induration, indicative of necrotising fasciitis.

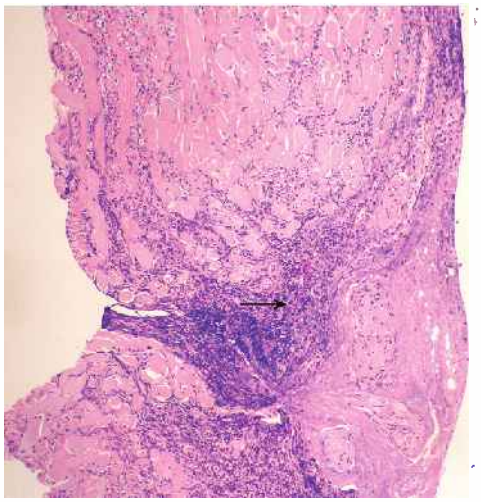

Fig. 2 Photomicrograph of the excised eyelid shows the skeletal muscle (left side) with acute inflammatory cells and adjacent necrotic tissue (black arrow) (Haematoxylin & Eosin, × 100).

margin, measuring 15 mm × 8 mm in the central part (Fig. 1). The necrotic tissue was debrided until healthy viable tissue was present all around the wound. A lateral canthotomy and cantholysis was carried out to enable the wound to be approximated with 6-0 marginal silk suture. The rest of the defect was closed in layers.

The eye swab grew *P. aeruginosa*, which was sensitive to piperacillin, ceftazidime and ciprofloxacin. Intraoperative specimens of the necrotic tissue were sent for histological examination. The sections showed necrotic tissue fragments accompanied by an acute inflammatory infiltrate (Fig. 2) and Gram-negative coccobacillary organisms. The inflammation extended into the underlying viable skeletal muscle tissue. This was consistent with necrotising fasciitis. Interestingly, the patient's blood culture during her admission did not show any bacterial growth.

In view of the clinical improvement in her local and systemic condition, the patient was continued on intravenous meropenem instead of changing to a different culture-sensitive antibiotic. She was also continued on topical ciprofloxacin 0.3% eyedrops and erythromycin 1% ointment to the wound. The patient responded well clinically and her fever subsided. She completed 21 days of intravenous meropenem.

Five days after the first wound debridement, wound breakdown occurred, with a 4.5-mm defect in the left upper eyelid exposing the cornea. Two weeks later, the patient underwent a lid sharing (Cutler Beard) procedure to close the defect. The rest of her hospital stay was uneventful. The Cutler Beard flap was released two months after the procedure. The outcome of the surgery was considered satisfactory, with the patient having a normal functioning upper lid with no lagophthalmos. However, she was left with a small notch in the left upper eyelid margin.

DISCUSSION

Necrotising fasciitis more commonly affects the extremities and trunk, and hardly ever affects the face and the eyelids. Often, the patients are immunocompromised, or there is precipitating trauma to the area which allows a portal of entry for the pathogen. Few cases of periorbital necrotising fasciitis secondary to *P. aeruginosa* have been described.[2-6] Group A and non-Group A *Streptococcus* and *Staphylococcus* species are the most common aetiological agents.[7,8] However, a wide spectrum of other organisms have been recovered, including *Bacteroides*, *Clostridium*, *Peptostreptococcus*, enterobacteriaceae, coliforms, *Proteus*, *Pseudomonas* and *Klebsiella*.[9,10] *Pseudomonas* can cause deranging infections of the eye, including endophthalmitis, dacryocystitis, conjunctivitis, keratitis, corneoscleral ulcers, blepharitis, orbital and periorbital cellulitis. It is one of the most common causes of bacterial keratitis. It can colonise the ocular epithelium by means of a fimbrial attachment to sialic acid receptors. With the compromise of the local defenses, the bacterium can proliferate rapidly, and through the production of enzymes such as elastase, alkaline protease and exotoxin A, cause a rapidly destructive infection that can lead to the loss of the entire eye.[11,12]

Pseudomonas infection can cause necrotising fasciitis.[12] In our case, the patient had neutropenia, which was probably secondary to her immunosuppressive medication and the overwhelming Gram-negative sepsis. In addition, diabetes mellitus may have altered her immune system. In 2002, Dickenson and Yates reported a case of bilateral eyelid necrosis in a 70-year-old man with neutropenia (0.3×10^9/L) from colchicine

therapy for prophylaxis of gout. The colchicine therapy was stopped and the neutrophil count improved to within the reference range in four days, with the patient showing recovery at the same time.[5] In another report of a 16-month-old girl with eyelid necrosis secondary to *P. aeruginosa*, it was noted that the clinical improvement was accompanied by a resolution of leucopenia, which was presumably induced by malnutrition.[2]

Early recognition and prompt aggressive medical and surgical therapy are critical for the management of this potentially fatal disease. Early diagnosis is frequently challenging, as the initial clinical signs and symptoms may be similar to severe cellulitis. Therefore, a high index of suspicion of necrotising fasciitis is necessary, especially in pre-disposed patients. A systemically unwell patient who is not responding to standard antimicrobial therapy, with rapidly progressing skin changes, should raise an alarm. As soon as the diagnosis of necrotising fasciitis is made, urgent surgical debridement is necessary.

In conclusion, widespread and early debridement of devitalised tissue, together with high-dose systemic broad-spectrum antimicrobial therapy, is the mainstay of therapy. In periorbital necrotising fasciitis, there may be marked tissue loss from the eyelids, which needs to be addressed.

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