Necrotising fasciitis caused by *Vibrio vulnificus*

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SUMMARY A case of necrotising fasciitis caused by *Vibrio vulnificus* is described. The need for early recognition and aggressive surgical treatment are highlighted, and the necrotising infections due to *V vulnificus* described in the published work are reviewed.

Necrotising fasciitis is a rapidly progressing necrotising process involving subcutaneous tissue and fascia but sparing muscles, and is accompanied by severe systemic toxicity.^{1,2} The syndrome is often caused by group A streptococci,^{1,3} but it is now recognised that other bacteria may be involved, usually in mixed culture. Organisms that have been implicated include a variety of anaerobes, enterobacteria, non-group A streptococci, pseudomonas, and staphylococcus.^{4,5} *Vibrio vulnificus* is a recently described halophilic vibrio which causes necrotic skin lesions following wound infection.⁶ We have recently seen a child who presented with classic necrotising fasciitis caused by *V vulnificus* alone.

Case report

An 8 year old schoolboy was admitted with a high fever (40.2°C), chills and rigors, and a peripheral white cell count of $19 \times 10^9/l$. On the day before admission he had suffered a small abrasion over his left thigh, which he rubbed with dirty water from a basin. On admission, he was toxic and lethargic. His left thigh was swollen and extremely tender and showed several black blisters (about 1-2 cm in diameter) delimited by necrotic purple-blue skin. His condition deteriorated rapidly despite antibiotic treatment. On the next day he went into septicæmic shock, but he was resuscitated successfully. The skin over the left thigh and upper leg became dusky with bullous formation. The margin of the lesion progressed rapidly, spreading up to the left groin and down to the lower calf (Figs. 1 and 2). Discoloration of the left lower quadrant of the abdominal wall was also noticed on the third day.

Sixty two hours after admission necrotising fasciitis was diagnosed and an emergency radical

debridement was performed under general anaesthesia. Necrotising fasciitis was confirmed at operation. There was extensive necrosis of the skin, subcutaneous tissue, and the deep fascia, but muscle was spared. Vessels in the subcutaneous tissue were thrombosed, and tissue necrosis was more widespread than the skin lesions had suggested. The patient improved almost immediately after the operation. The wound required skin grafting, but was completely healed after two months.

MICROBIOLOGY

Cultures of blood and debrided tissue yielded a pure growth of an aerobic, halophilic, oxidase positive, Gram negative bacterium with the characteristics of *V vulnificus*. This strain was sensitive to ampicillin, chloramphenicol, co-trimoxazole, gentamicin, and amikacin, but resistant to cephalothin. The identification was confirmed by Dr JV Lee of the Public Health Laboratory Service, Centre for Applied Microbiology and Research, Porton Down, Salisbury, UK.

HISTOPATHOLOGY

Histology of debrided tissue showed focal necrosis in the skin and subcutaneous tissue with subepidermal bulla formation. There was diffuse infiltration with polymorphonuclear and mononuclear inflammatory cells. The subcutaneous vasculature was congested. Numerous Gram negative vibrios were seen in necrotic areas and spreading along the deep fascia. Careful search failed to find any other organism.

This strain of *V vulnificus* was used in the animal model of Poole and Oliver.⁷ The LD₅₀ was about 7×10^3 colony forming units. This dose produced necrotic skin lesions in survivors when given to ICR adult mice by intradermal and subcutaneous injection, while control injections of the same dose of

Figs. 1 and 2 Skin lesions of the left leg.

V parahaemolyticus did not. Tissue sections of the experimental lesions showed typical appearances of necrotising fasciitis, with extensive necrosis of dermal tissues and numerous polymorphonuclear leucocytes and vibrios spreading along fascial planes. Muscle tissue was much less affected than the dermis, but it too showed some necrotic changes.

Discussion

There is no doubt that in this patient necrotising fasciitis was caused by *V vulnificus* alone. The patient had all the classic clinical, anatomical, and histological features of the disease: the organism was isolated in pure culture from both blood and tissue taken at different times; the isolated strain produced a similar syndrome when injected into mice; and the organism with its characteristic

appearance was seen in tissue sections of lesions from both the patient and experimentally infected animals.

Giuliano *et al*⁵ suggested that streptococci are the most common single bacterial cause of necrotising fasciitis, and non-group A streptococci with *Bacteroides fragilis* is the most common combination. Numerous other bacteria have also been implicated, including peptostreptococci, peptococci, clostridia, other bacteroides species, fusobacteria, *Staphylococcus aureus*, *Escherichia coli*, citrobacter, klebsiella, enterobacter, serratia, *Proteus mirabilis* and *Pseudomonas aeruginosa*.⁵ *V vulnificus* has not previously been given as a cause of necrotising fasciitis, but there are a large number of reports noting necrotic skin lesions associated with *Vibrio vulnificus* (or "lactose-positive halophilic vibrio"^{8,9}) wound infection and septicaemia.

Fig. 2

Roland¹⁰ reported a case of gangrene of the leg with massive necrosis of adipose and muscular tissue after sea bathing and "clamming." "*Vibrio parahaemolyticus*" was isolated from the exudate of the leg¹⁰ but was later identified as a lactose fermenting halophilic vibrio.⁸ A case of erythema multiforme with fatal fulminating septicaemia was described by Zide.¹¹ Blood culture yielded "*Vibrio parahaemolyticus*," which was later identified as a lactose fermenting halophilic vibrio.⁸ Microscopical examination of the skin lesions showed focal acute fat necrosis in subcutaneous tissues, and the dermis was infiltrated by Gram negative bacteria. Thorsteinson *et al*¹² reported three cases of halophilic non-cholera vibrio infection. One of the patients had a

non-purulent thumb infection that required fasciotomy and another had gangrene of the leg and died despite amputation. Fernandez and Pankey¹³ described three cases of severe necrotising cellulitis, two of which yielded a lactose positive, halophilic vibrio. Five patients with septicaemia and wound infections caused by a similar organism were described by Hollis *et al*.¹⁴ All five died shortly after admission, but details of the skin lesions were not given. Matsuo *et al*¹⁵ described a fatal case of septicaemia caused by a lactose positive halophilic vibrio, presenting with bullae on the legs and eruptions over the arms. Histological examination of the skin lesions showed areas of fibroadipose necrosis in the dermis and subcutaneous tissue. Massive bacterial

infiltration in the skin was also noted.

Blake *et al.*,¹⁶ Mertens *et al.*,¹⁷ and Tacket *et al.*¹⁸ reported altogether 18 patients with *V vulnificus* wound infections, the lesions being variously described as bullous, cellulitic, or necrotic. Castillo¹⁹ described two patients with *V vulnificus* septicaemia who developed haemorrhagic and necrotic skin lesions of the hands that required urgent surgical debridement. Finally, Kelly *et al.*²⁰ reported a fatal case of *V vulnificus* septicaemia with acute necrotising myositis of the leg. Postmortem dissection showed focal necrosis and haemorrhage of the calf muscles.¹⁷

In all these cases of serious wound infection, spreading necrotic skin lesions appear to have been regarded as complications of an often fatal *V vulnificus* septicaemia. Detailed clinical information was not always available, however, and some of these patients may have had necrotising fasciitis. *V vulnificus* necrotising fasciitis may be part of a range of conditions, varying from localised focal necrosis of the dermis to widespread skin and muscle necrosis.

Wound infections due to *V vulnificus* are usually caused by contamination with sea water or sea food.⁶ In our case there was no history of such exposure. The domestic water supply was cultured but no pathogen was isolated, and the basin of dirty water which the patient used to rub the abrasion had been poured away.

Although caused by an unusual organism, this case of necrotising fasciitis illustrates the general principles of management of this condition, including early recognition and immediate aggressive surgical treatment.²

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