CASE REPORT

A case of necrotising fasciitis caused by Pseudomonas aeruginosa

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Necrotising fasciitis is a rare but severe infection of soft-tissue associated with rapid progression, systemic toxicity and high mortality. Monomicrobial necrotising fasciitis caused by Pseudomonas aeruginosa is exceptionally uncommon with only 12 cases reported in the literature. We describe a fatal case with an atypical presentation in a patient following spinal decompression for a metastasis from prostate cancer.

In 1952, Wilson used the term necrotising fasciitis to describe a rapidly progressive and destructive infection of the subcutaneous tissue, associated with high mortality and long-term morbidity. Early clinical recognition is often challenging, and aggressive surgical debridement together with appropriate antimicrobial therapy is critical. Group A Streptococcus is the most common organism associated with monomicrobial necrotising fasciitis, but mixed growths can also occur. The risk factors for infection include penetrating trauma, recent surgery, burns and intravenous drug use. Individuals with other systemic illnesses, immunosuppression, diabetes mellitus or cancer are more susceptible. Monomicrobial necrotising fasciitis caused by Pseudomonas aeruginosa, is exceptionally rare with only 12 reported cases in the literature.

We describe a case of necrotising fasciitis with an atypical presentation secondary to Pseudomonas aeruginosa.

Case report

A 62-year-old man presented acutely with fever and severe pain in his left knee. Three weeks earlier he had undergone spinal decompression for a metastatic tumour in the spinal cord which had failed to improve following four courses of radiotherapy (Fig. 1). He had made an unremarkable recovery from this surgery. He had normal sensation in his lower limbs, but some residual motor weakness bilaterally.

On admission he was pyrexial (38.0°C) with a mild tachycardia, and hypotensive. Examination revealed an erythematous, a mildly tender area over the medial aspect of his left knee and a non-tender pigmented area over his left shin. His surgical wound was slightly swollen, but there was no surrounding erythema. His white cell count, renal and liver function tests and blood gas analysis were all normal. His CRP was elevated at 67 mg/L. Blood cultures and aspirate from the left knee joint were sent for microbiological analysis. Intravenous benzylpenicillin (1.2 g) and flucloxacillin (2 g) were given four times a day for 24 hours for presumed systemic sepsis secondary to infection of his spinal wound.

Over the next 12 hours his clinical condition improved, although the appearance of his left leg was unchanged. He then deteriorated rapidly. Gentamicin was given intravenously. An ultrasound scan of his left leg failed unexpectedly to identify any collection of fluid or air. However, an MR scan of his spine revealed a collection of fluid extending to the dura at the level of the operation. The wound was therefore explored and debrided. The pigmented region on his shin had turned dusky in appearance and was therefore also explored. There was clear fluid in the soft tissues. The subcutaneous fat was brown in colour and separated easily from the fascia. During the procedure the patient became increasingly unwell, and the decision was made not to debride this site. He died four hours post-surgery.

The pre-operative blood cultures were negative. The specimens taken at operation from the spinal wound and from the left leg all yielded isolated growths of Pseudomonas aeruginosa. Histological examination confirmed a diagnosis of necrotising fasciitis.

Discussion

Necrotising fasciitis is a rare but severe infection. Early clinical recognition is a considerable
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challenge. Differentiation from cellulitis may be particularly difficult but is critical, as necrotising fasciitis requires prompt surgical intervention.

The initial signs and symptoms include swelling, pain and erythema. As the infection progresses, typical features include tense oedema outside the area of compromised skin, disproportionate pain, discoloration of the skin, bullae and crepitus. Systemic findings include fever, tachycardia and hypotension. Although these findings are typical and fairly specific for necrotising fasciitis, they are only present in 10% to 40% of cases.3,4

Plain radiography and ultrasonography can aid the diagnosis by identifying subcutaneous gas, but although this is a very specific finding it is rarely seen. CT has the advantage of identifying other causes of infection, particularly deep abscesses. However, a hyperintense signal along the deep fascial plane on a T2-weighted spin echo MR scan is considered to be pathognomonic.5-7

The mainstay of treatment is early surgical debridement together with appropriate broad-spectrum antibiotics ensuring cover for Gram-positive, Gram-negative and anaerobic organisms. At operation the macroscopic findings include grey necrotic tissue, a lack of bleeding, thrombosed vessels, foul-smelling grey oedematous fluid (dishwater pus) and non-contraction muscle. There is a characteristic lack of resistance to finger dissection in normally adherent tissue. These findings should prompt additional debridement.

Necrotising fasciitis commonly presents in one of two ways.8,9 The first includes patients with antecedent surgery, immunocompromise, diabetes mellitus or vascular insufficiency, in whom the infection is usually polymicrobial with aerobic and anaerobic bacteria such as Clostridia and Bacteroides species. Treatment with corticosteroids is known to increase susceptibility by reducing the patient’s immune response.10 The second consists of patients with no underlying diseases in whom necrotising fasciitis is induced by a single pathogen, usually group A Streptococcus, popularised as the ‘flesh-eating bacterium’.9

The sole organism responsible in this case was Pseudomonas aeruginosa, which is unusual. The infection probably arose from the spinal wound, associated with radiotherapy and oral steroids, with haematogenous spread to his left leg by means of a transient bacteraemia.

The absence of pain or tenderness in the left leg on clinical assessment and the initial clinical improvement may have been secondary to his infection disrupting the nerve supply locally.

This case highlights the variability of the clinical presentation of necrotising fasciitis and the need to maintain a high index of suspicion, particularly in a septic patient with cellulitis, where the possibility must be actively excluded.

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References