A case report on necrotising fasciitis

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This case report discusses the management of an elderly woman patient who presented with a provisional diagnosis of cellulitis of the right lower limb to a district hospital. Subsequent rapid deterioration of the limb and the development of clinical features suggestive of necrotising fasciitis led to immediate surgical intervention.

Wilkes A. Discovering an unusual wound. A case report on necrotising fasciitis. Primary Intentions 2005; 13(2): 83-88.

Case history

A 71 year old woman was transferred from a peripheral hospital to District Hospital, New South Wales, for management of what was initially thought to be cellulitis of the right lower limb. This patient's medical history included chronic obstructive airways disease (she was a current smoker), transient ischaemic attacks, and a reported past history of chronic venous insufficiency and venous ulceration of the lower limb. There was no known history of diabetes and all tests for diabetes were negative. Current medications at the time of admission included salbutamol and tiotropium inhalers. Allergies to both penicillin and pethidine were reported.

One week prior to transfer, this patient had presented to the peripheral hospital with a painful and erythematous right lower leg, fever to 38.5 degrees Celsius, tachycardia and mild hypotension. There was an unclear history of onset; specifically the patient denied any recent trauma to the leg. Investigations at that time revealed a total white cell count of $19.7 * 10^{9}$/L and a haemoglobin of 129g/L. Given her history of allergies, intravenous ceftriaxone and oral roxithromycin were commenced.

Throughout the course of the next six days the patient remained intermittently febrile and her leg failed to show any significant improvement. A venous Doppler ultrasound of the right leg excluded a deep venous thrombosis. The ultrasound, however, confirmed the presence of extensive oedema of the subcutaneous tissues.

After surgical consultation it was felt that transfer to a facility with readily available anaesthetic services would be appropriate in the event that debridement may be required. Upon arrival to our hospital, her leg was noted to be erythematous and oedematous with the suggestion of ulcerative changes around the medial malleolus. Investigations at this time revealed a total white cell count of $25.3 * 10^{9}$/L. Intravenous erythromycin was commenced and a simple dressing was applied to the affected area. Over the subsequent twenty-four to thirty-six hours, there were some small signs of improvement. The wound care nursing consultant for our hospital also reviewed this wound and concurred that the wound did not present as a typical venous or arterial leg ulcer.

Shortly after this consultation, the leg began to show some rapid changes. Over the course of a few hours the lower leg began to develop multiple pustules, which began to spread rapidly over the dorsum of the foot and proximally up the lower leg. These were associated with a purulent exudate and areas of visible fat necrosis (see Figures 1 & 2). Upon recognition of the necrotising nature of this wound, urgent surgical debridement was planned. At this time, the patient remained haemodynamically stable. Pre-operative investigations revealed an escalating white cell count of $39.8 * 10^{9}$/L, a haemoglobin of 93g/L, a coagulopathy with a prothrombin time of 17 seconds, and an activated partial thromboplastin time of 35 seconds. The C-reactive protein, sodium, and creatinine levels were CRP 389 mg/L; sodium 138 mmol/L; creatinine 93 respectively. Liver function tests showed marked elevation of her transaminases, felt likely to be a combination of underlying infection and recent erythromycin therapy. Renal function remained within normal limits.

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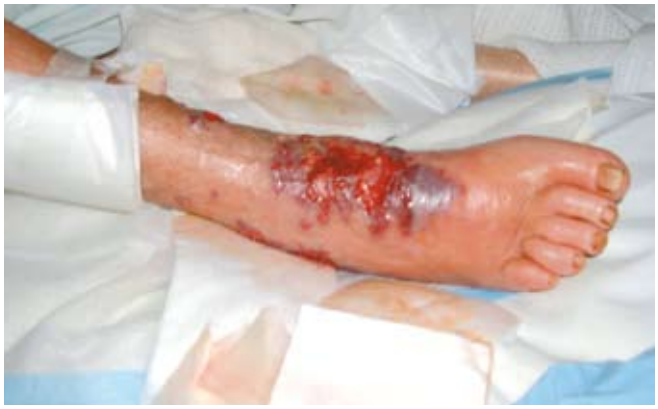
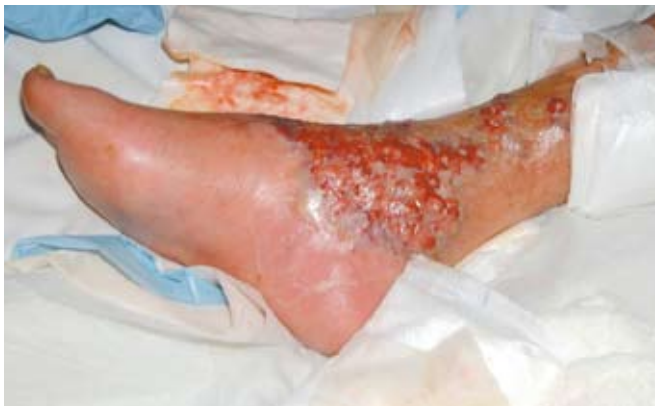
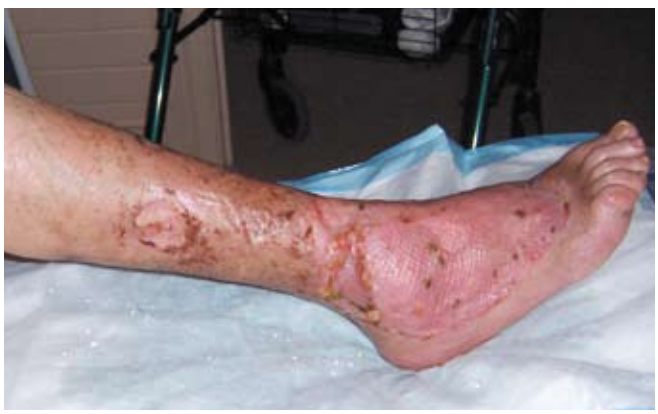
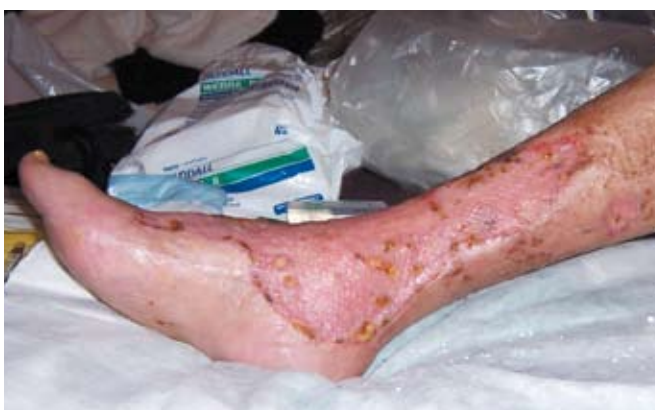




Figures 1 and 2. Patients right foot prior to debridement.





Figures 3 and 4. Wound post skin grafting

Surgical and post operative management

Extensive debridement including the skin, subcutaneous tissue and deep fascia of the dorsal aspect of the right foot, medial malleolus and the distal anterior surface of the right leg was undertaken. The fascia was noted to be dull and grey, consistent with necrotising fasciitis, and the right posterior tibial artery thrombosed. The speed of spread of necrosis was such that areas of necrosis developed beyond that just excised. Initially it was thought that amputation may have been the best way to contain this process, however with persistence, over the course of a few hours the oedema and erythema reduced and no further necrosis was visualised. Intra-operatively the patient became haemodynamically unstable and required the commencement of inotropes.

After discussion with the hyperbaric unit of the Prince of Wales Hospital, Sydney, the patient was transferred to the unit for hyperbaric therapy. The wound itself required only a small amount of further debridement under local anaesthetic, and, after just a fortnight from the initial debridement, a split skin graft was successfully applied (Figures 3 and 4). The patient underwent a short period of rehabilitation prior to discharge home.

Histopathology of fascial biopsies taken intra-operatively showed prominent necrosis and an infiltrate comprised predominantly of neutrophils consistent with NF, thus confirming the diagnosis of necrotising fasciitis. At no stage, despite multiple cultures, was an organism cultured from this wound.

Discussion

Necrotising fasciitis, which is also known by the more sensational term – 'flesh eating bacteria syndrome' is uncommon. It is characterised by rapidly spreading inflammation and necrosis of the skin, subcutaneous fat and fascia [1]. In the early stages of its development, it can be easily mistaken for cellulitis.

The most common sites for the development of necrotising fasciitis are the abdomen, perineum and the extremities. Factors which predispose to the development of necrotising fasciitis include immunocompromised states such as diabetes mellitus, HIV/AIDS and malignancy. People with chronic illnesses such as cardiorespiratory, hepatic or peripheral vascular disease are also at risk [1, 2, 3].

Types of Necrotising Fasciitis

It is likely that this case was a Type II NF; although cultures can not support this. There are two main types of necrotising fasciitis depending upon which organism is responsible. Type I necrotising fasciitis is typically associated with multiple bacteria such as Clostridia and Bacteroides.

Type II is typically associated with group A (beta haemolytic) Streptococcus with or without a co-existing Staphylococcal infection [1, 2].

Typically, the organism will enter the subcutaneous layer in one of two ways – from external trauma involving breaks in the skin, or from internal trauma resulting from intestinal fistulae. Once infection is established, it can spread at a rate of 2cm/hr with death occurring rapidly in undiagnosed cases – in some cases as quickly as 24 hours, secondary to sepsis and subsequent multi-organ failure. Death rates range from 20-60% of affected patients [1, 2, 4, 5, 6].

Clinical presentation

Early in the course of the disease, patients present in a similar fashion to cellulitis. Typically, erythema and oedema are seen at the site with a surrounding diffuse inflammatory response that blends into the surrounding skin without any clear line of demarcation [1,2,5]. Severe pain, out of proportion to the nature of the lesion, is characteristic of necrotising fasciitis [2].

The speed of spread of the disease is variable. In fulminant cases, patients present with rapid disease progression, often only having symptoms for several hours. In acute or subacute cases, infection progresses for two to three days prior to the skin erythema changing to purple/purple-black as tissue necrosis develops. Numbness may occur over the affected skin. Bullae develop – the fluid contained within them turns into grey malodorous fluid referred to as 'dishwater pus'. As bullae rupture, a dry black eschar may develop. Over time, cutaneous gangrene may extend beyond the skin involving subcutaneous fat and fascial planes below [1]. Separation of the necrotic tissue along the fascial planes may occur.

In addition to these skin changes, which are readily visible, most patients will also manifest systemic symptoms such as a high fever, rigours, tachycardia, hypotension, confusion and often multi-organ failure – especially respiratory and renal failure [1, 2, 3, 5].

Pathophysiology

Why is it that the bacteria associated with necrotising fasciitis cause such a dramatic spectrum of changes in patients who develop necrotising fasciitis? There are a number of factors related to the patient and the bacteria.

Firstly there is often a breakdown in tissue resistance, often secondary to immunosuppression, which allows bacteria to enter through the skin. The bacteria associated with necrotising fasciitis produce enzymes and toxins, which facilitate the spread of bacteria through the skin, especially the subcutaneous tissue and fascial planes [1]. The toxins produced cause inflammation and damage to the lining of blood vessels, which allows fluid to escape from the vessels into the tissue surrounding the blood vessels. This leads to oedema as well as reducing the blood flow to the wound itself. This, in turn, leads to tissue hypoxia, that is reduction in oxygen perfusion, and, subsequently, tissue death occurs [7]. Necrosis of subcutaneous tissue and fat is thought to occur as the result of bacterial enzymes such as hyaluronidase and lipase [1, 2].

In cases caused by Streptococci, recent research has also revealed that Streptococci is associated with the production of super antigens which activate T helper lymphocytes. These are cells of the immune system which normally act to help fight infection. The activation of these lymphocytes results in the production of further inflammatory mediators which stimulate production of free radicals and nitrous oxide which, together, lead to shock, immunosuppression, depression of myocardial function, and multi-organ failure [1, 8, 9, 10, 11].

In essence, as bacteria multiply and release toxins, the local protective tissue responses are inhibited, ultimately leading to tissue death [4].

Diagnosis

Essentially, the diagnosis of necrotising fasciitis is based on the clinical presentation, that is the appearance of the skin, extreme pain, fever and systemic features. Laboratory, radiological, and histological investigations can aid in the diagnosis. Typically, the haematological profile will reveal an elevated white cell count, anaemia, and an elevated erythrocyte sedimentation rate and C-reactive protein.

More recently, Wong *et al*, developed a scoring system referred to as the Laboratory Risk Indicator for Necrotising Fasciitis. This scoring system is based on readily available blood results such as total white cell count, haemoglobin, C-reactive protein, sodium, creatinine and glucose. It is proposed that this scoring system provides a system for detecting early cases of necrotising fasciitis amongst patients with severe soft tissue infections. Each of these factors has been found, by these researchers, to be independently predictive of necrotising fasciitis. The maximum score obtainable is 13. According to researchers, a score of 6 or more should raise suspicion of necrotising fasciitis and a score of 8 or more is strongly predictive of necrotising fasciitis [12]. Interestingly, the subject of this case report would have obtained a score of 8.

X-ray can detect soft tissue gas, however, the extent of

damage is more clearly visualised on CT scan or MRI [1]. These modalities, therefore, are often not used in diagnosis as a consequence of their limited availability and the fact that they may delay appropriate treatment.

Fascial biopsies and surgical exploration are helpful. Characteristic for necrotising fasciitis is the ability to introduce an artery forcep into the wound and slide it easily along the fascial planes [1].

Treatment

The main features of treatment are:

1. Firstly to recognise or suspect necrotising fasciitis in the patient.
2. Administer intra-venous antibiotics – in this regard a combination of broad spectrum anti-biotics is required to cover all potential bacteria, until culture and sensitivities are available to guide further anti-biotic choices. Typically, penicillin or a third generation cephalosporin; an aminoglycoside such as gentamicin; clindamycin or metronidazole are used [1,2,5].
3. Surgical debridement – aggressive surgical debridement is the cornerstone of managing this disease. Debridement should include extensive incision of the skin and subcutaneous tissue wide into healthy tissue, followed by excision of all necrotic fascia, and non-viable skin and subcutaneous tissue. Subsequent debridements may be required and, when infection cannot be controlled, amputation may be required to prevent the patient's death [1,3,5].
4. Intensive care unit support – this includes fluid resuscitation, inotropic support, if required, and nutritional support. These patients are in a catabolic state and require extra calories delivered either orally or parenterally [1,2].
5. Hyperbaric oxygen – the role of hyperbaric oxygen in treating necrotising fasciitis remains controversial. Some studies have shown that hyperbaric oxygen therapy lowers the mortality rate associated with necrotising fasciitis by up to 40% [13] whilst other studies have failed to demonstrate this [14].

   The proposed mechanisms by which hyperbaric oxygen aids the treatment of necrotising fasciitis are multiple, and include:

   - increasing the pressure of oxygen in the tissues, that is improving the oxygenation of tissues, which in turn increases the killing ability of leucocytes.
   - increasing the killing of anaerobes, one of the common bacterial species responsible for necrotising fasciitis.
   - a reduction in tissue oedema.
   - promotion of wound healing by stimulating fibroblast growth, increasing collagen formation, stimulating angiogenesis and promotion of granulation tissue [1, 2, 3].
6. Intravenous immunoglobulin - immunoglobulins bind to bacterial toxins and bacteria, reducing the proliferation of T cells and, subsequently, reducing the production of tumour necrosis factor, which, in turn, will reduce the inflammatory response [1, 2, 5]. Further research into the role of intravenous immunoglobulin in the management of necrotising fasciitis is required to establish its benefit.
7. Wound management – in the interim between surgical debridement and application of the split skin graft, skilled wound management is required.

Conclusion

Necrotising fasciitis is not common but dramatic. Given the severity of its nature and the rapidity with which it develops, health care professionals need to be aware of its existence and presentation and institute rapid and aggressive therapy if therapy is to be successful

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