An Overview of Necrotizing Fasciitis

Gary Bain

Abstract

Necrotizing fasciitis, a serious infective process, causes extensive tissue damage, resulting in critical illness and potential disfigurement. This article presents a brief review of the pathology, clinical manifestations and treatment of this wound management challenge.

Historical Perspective

The first clear reference to necrotizing fasciitis (NF) dates back to the 5th century BC, with Hippocrates' description of a fatal infection which produced gross discolouration, swelling and eventual gangrene of a man's face (later identified as necrotizing erysipelas) [1]. During the 18th, 19th and early 20th centuries, NF was known variously as 'malignant ulcer', 'hospital gangrene', 'suppurative fasciitis', 'acute infective gangrene' and 'acute dermal gangrene'.

In the late 19th century, outbreaks of NF recorded in England and Wales were thought to be associated with epidemics of scarlet fever. Around the same period, Joseph Jones, a Confederate Army surgeon in the American Civil War, reported NF in 2642 wounded soldiers, with the disease's mortality rate as high as 46 per cent. Then, in 1903, Dr Fournier described the occurrence of NF in the genital area – the condition is now often referred to as Fournier's gangrene. A major advance took place in 1924 when Meleney and Breuer isolated streptococcal infection as the prime cause of lethal NF [1].

From 1987 to 1990, scattered outbreaks of NF were reported in both the USA and Scandinavia, while significant media attention focused on a close cluster of NF cases in West Gloucestershire in 1994 [2]. In 1995 a small number of cases was reported in Canada and California [3]. More recently, Jarrett *et al* provided a detailed report of 15 NF patients in a 6-year period at Waikato Hospital in New Zealand [4].

Nowak [5] suggests there has been an increase in the incidence of severe streptococcal infection throughout the 20th century, resulting in more cases of NF being identified and treated. While most Westernised countries are said to have an incident rate of around one in every 100,000 people [5], accurate Aust-ralian statistics on the disease are currently unavailable.

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Definition

NF, a relatively rare infection, is characterised by rapidly progressing necrosis of the fascia and subcutaneous fat, with subsequent necrosis of overlying skin. Muscle involvement is minimal or non-existent [6].

The Infective Process

Microbiologists generally separate NF into two 'types' [4, 7].

Type I is most commonly associated with sub-acute infection and with the NF occurring as a secondary entity to an existing infection. Usually, at least one anaerobic species – such as *Bacteroides, Clostridium* or *Peptococcus* – is present and may be isolated in combination with one or more aerobes, such as non-Group A *Streptococci, Escherichia coli, Klebsiella* or *Proteus.* The anaerobes and aerobes work in synergy to overwhelm the host's immune defence.

Type II is usually suspected when acute and fulminating infection is identified. Such cases are often idiopathic and there may be no apparent point of entry for the organisms. Group A *Streptococci,* either alone or in combination with *Staphylococcus aureus,* are a common finding. This infection has an extremely rapid course and is most likely to involve the extremities.

Group A *Streptococci* are very virulent and once in the subcutaneous tissue can replicate rapidly, avoiding phagocytosis by neutrophils due to the presence of a surface protein on the

bacterial cell wall. These organisms also produce cytotoxins, streptokinase, haemolysins and pyogenic exotoxins, which have a direct and damaging effect on tissues. The toxins induce a massive release of cytokines, leading ultimately to extravasation of inflammatory fluid, vascular injury to arteries and arterioles, liquefaction of subcutaneous fat and ischaemia.

Predisposing Factors

Numerous factors can increase a susceptible host's risk of contracting NF (see Table 1). Significant among these are diabetes mellitus and intravenous drug use, risk events or conditions the incidence of which has increased rapidly in Western populations over the past hundred or so years [5].

The probability of a correlation between long-term, high-dose use of non-steroidal anti-inflammatory drugs and the oc-currence of NF has also been discussed. It is hypothesised that such drugs both mask the manifestations of inflammation, thereby delaying the cardinal signs of serious infection, and retard the host's natural immune response to bacterial invasion [8, 9].

Portal of Entry for Infection

In the majority of cases there is no obvious mode of entry for the Group A *Streptococci*. Nonetheless, NF has been documented as developing after minor skin trauma, intravenous injections and surgical incisions (such as for blepharoplasty, Caesarean section, appendicectomy and laparoscopy). It has also been reported in patients with perirectal abscesses, diverticulitis, chicken pox, scarlet fever, leg ulcers and bites (animal, insect and human), and following some dental procedures [3, 7].

Table 1. Predisposing or risk factors for necrotizing fasciitis.

- Immunosuppression
- Diabetes mellitus
- Alcoholism
- Malignancy
- Severe malnutrition
- Severe peripheral vascular disease
- Intravenous drug use
- Renal failure
- Radiotherapy
- Obesity

Common Sites of Infection

While any area of the body can succumb to NF, the most common sites are the extremities, the abdominal wall, the perianal and groin area and post-operative wounds.

Typical Patient Presentation

The very early symptoms of NF tend to be 'flu-like' in their characteristics – the patient experiences fever, chills, general aches, diarrhoea and vomiting [10]. Within days or even hours, the affected region becomes oedematous and a localised area of ery-thema develops. Extreme pain – out of all proportion to what would normally be expected – is associated with this reddened area. The skin becomes hot and shiny, superficial blisters may develop and it takes on a dusky hue, advancing to a blue-grey or purple colour. As necrotic tissue begins to appear the com-promised area becomes increasingly demarcated. At this point in the disease process the infection can destroy as much as 4 sq cm of tissue an hour [10].

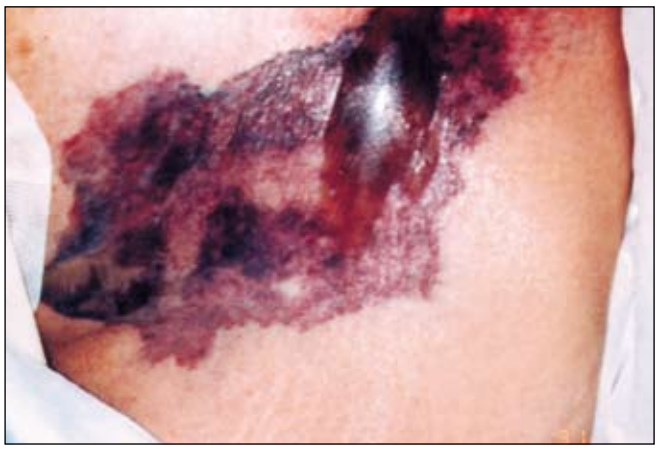
Once necrosis sets in, the amount of pain the patient experiences is reduced, since the skin is anaesthetised with the destruction of nerve endings. Should the blisters burst or the dead skin separate, an offensive, grey, watery discharge is released. Subcutaneous crepitus develops as gas collects under the skin. At this point the patient is grossly unwell, experiencing shock, reduced perfusion, fluid and electrolyte disturbances and an altered mental state. Death from disseminated intravascular coagulation and multi-organ system failure can occur in at least 30 per cent of cases [1, 3, 11, 12].

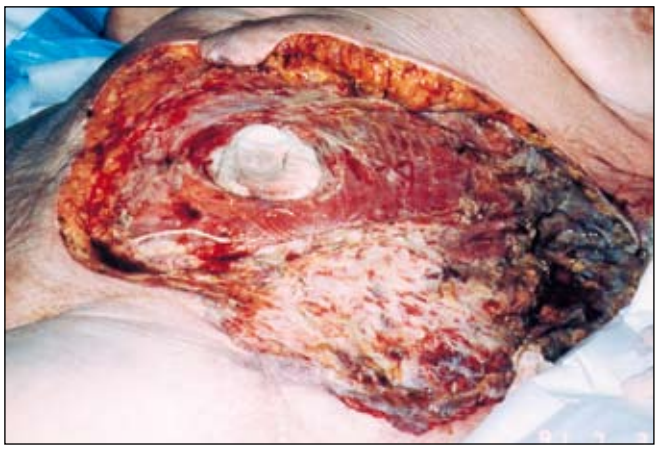
Diagnosis

Diagnosis of NF is largely determined by the nature, speed and severity of clinical events and the patient's failure to respond to routine antibiotic therapy. Bacteria are isolated via microscopy of aspirate or by obtaining a tissue culture. CT scan, MRI or ultrasound will demonstrate the build-up of gas in the subcutaneous tissues [1, 6]. Ultimately, however, surgical exploration most definitively confirms the diagnosis of NF – positive findings are grey and oedematous fatty tissue which strips away readily from the underlying fascia.

Treatment

Primary interventions include extensive surgical excision and broad-ranging antibiotic cover. The most common antibiotics used include combinations of penicillin, erythromycin, clindamycin, cephalosporins and metronidazole [1, 3, 9].





Photos: **Necrotizing fasciitis in a 73-year-old, insulin-dependent diabetic female with a newly created colostomy, showing (a) discolouration and blistering on lower abdomen, and (b) extensive tissue loss post-debridement.**

Supportive measures concentrate on fluid administration, the reversal of metabolic acidosis, correction of electrolyte abnormalities, hyperalimentation, pain relief and wound care. There is some doubt as to the benefit of hyperbaric oxygenation for NF; likewise for the administration of immunoglobulins [6].

There is also some argument regarding the infectiousness of these patients. Group A *Streptococ*i are contagious. Douglas [7] suggests that because NF patients are colonised by this bacteria they should be isolated for the protection of other patients. However, on the basis of the risk factors these individuals possess, their compromised immune status and their significant loss of protective layers of skin, it is more likely that NF patients themselves need defending from the microbial burden of other patients and health-care staff.

Wound Management

Following surgical intervention the wounds of NF patients can be massive in terms of volume and surface area, and this is often psychologically devastating for both them and their relatives. Such wounds are a major challenge for the nursing staff who dress them. There is no best method or choice of wound dress-ing, other than to maintain the principles of moist wound repair.

At the Sydney Adventist Hospital, products used to reduce wound colonisation include hydrogen peroxide short term and diluted povidone-iodine (thyroid and renal function may need to be monitored). In the longer term, wounds have been dress-ed with hydrogel packing and calcium alginates. Other cavity dressings, such as hydrofoams, silastic foam, dextranomer beads, expanding hydropolymer sheets and polyacrylate strands (such as AcryDerm Strands ® from Acrymed), may also be appropriate.

Dressing changes provide a good opportunity to examine the quality of the tissue at the margins of the wound. Any bleeding, change in colour, increase in drainage and odour or further erosion should be reported promptly, since the tissue may not yet be clear of the streptococcal infection. Indeed, it is not unusual for a patient with NF to visit the operating theatre a number of times for further debridement, in an effort to achieve 'healthy' wound margins [11].

Once both patient and wound are stable, the best repair option – whether surgical reconstruction or secondary wound healing – must be considered. Each can be slow and laborious and will certainly involve the institution's entire health-care team, including its community-based resources.

Prognosis

Mortality from NF has remained between 30 and 46 per cent for more than a hundred years. It is much higher for those with a combination of risk factors (see Table 1) – as much as 80 per cent [3, 7, 9]. The site of infection also contributes to the probability of death – the extremities are associated with a low level of fatality, while the abdomen, groin and perineum are linked to high mortality [12]. The most significant prognostic indicator, however, is the time lapse between onset of the infection and instigation of appropriate treatment. Early, aggressive intervention enhances the patient's chances of survival.

Conclusion

While still relatively rare, NF is associated with high mortality and morbidity. The hallmarks of patient care are early diagnosis, aggressive medical and surgical intervention and thoughtful wound management.

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Letters to the Editors

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- the product labelling should include an expiry date for that product and also specify an open shelf-life, and the latter should be supported by the submission of preservative efficacy data;
- the goods should not be promoted for use on third-degree burns or as having an accelerating effect on the rate of wound healing or epithelialisation, and
- the manufacturer must provide evidence that the goods are manufactured in compliance with the principles of good manufacturing practice.

In the interests of patient safety, ***all*** products used to treat wounds ***must*** be subject to the same regulatory controls.

We therefore respectfully suggest that *Primary Intention* publish appropriate warning statements in any article on a product not approved by the TGA for therapeutic use.

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