Clinical review

Necrotising fasciitis

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Necrotising fasciitis is a rare but life threatening condition that requires immediate action, but uncertainties still hamper prompt diagnosis and treatment

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Despite the impression that may have been gained from the British media, necrotising soft tissue infections have been recognised and reported for centuries, the earliest dating back to Hippocrates in the 5th century BC.¹ Such infections represent a large spectrum of clinical entities, ranging from mild pyoderma to life threatening necrotising fasciitis. Although these infections are most commonly caused by streptococcal and staphylococcal species, a multitude of other organisms have also been implicated.²

The term necrotising fasciitis was first used by Wilson³ in 1952 to describe the most consistent feature of the infection, necrosis of the fascia and subcutaneous tissue with relative sparing of the underlying muscle. It can progress rapidly to systemic toxicity and even death if not promptly diagnosed and treated. Once suspected, management should consist of immediate resuscitation, early surgical debridement, and administration of broad spectrum intravenous antibiotics.

As plastic surgeons, we receive several referrals each year from other specialties to diagnose or exclude the possibility of necrotising fasciitis. Since it is part of a spectrum of necrotising soft tissue infections, diagnosis can certainly be difficult for people who are unfamiliar with the condition and treatment may be delayed. This may be compounded by the relative lack of clinical signs and symptoms during the early course of the infection and because surgical consultation is sought only once the diagnosis is obvious and the signs of sepsis are readily apparent. Unfortunately, most adverse outcomes result from this delay in diagnosis.⁴

Fortunately, necrotising fasciitis is rare. The actual incidence in the United Kingdom is estimated at 500 new cases each year but is difficult to record. This in part is due to the confusion that arises by the numerous eponyms given to describe the same condition. A further attempt to classify it according to location (for example, Fournier's gangrene), clinical presentation, or bacteriology only complicates the issue since there is little to distinguish between these entities, and their initial management is the same.^{5,6}

This article aims at eradicating this confusion and looks at the measures that can be taken to increase awareness of necrotising fasciitis among clinicians, with the hope that this will result in earlier referral of patients and improve their overall survival.

Summary points

Necrotising fasciitis is an uncommon but potentially fatal condition and can affect any part of the body

The aetiology is not fully understood but most patients who develop necrotising fasciitis have pre-existing conditions that render them susceptible to infection

Diagnosis is often delayed because of the paucity of symptoms and the unfamiliarity of the condition among clinicians

Laboratory findings and other diagnostic tests may be useful adjuncts, but the diagnosis is still primarily a clinical one and a high index of suspicion is required

Management should consist of immediate resuscitation, early surgical debridement, and administration of broad spectrum intravenous antibiotics

We obtained the information for this article from various sources. References came from medical journal databases such as Medline and PubMed. Clinical photographs came from PRWS and NBH and their vast personal archives.

Aetiology and predisposing factors

The aetiology of necrotising fasciitis is not fully understood, and in many cases no identifiable cause can be found. However, patients often have some prior history of trauma (which may even be trivial), such as an insect bite, scratch, or abrasion.⁷ The disease does not seem to have any predilection for age but occurs slightly more often in male patients.^{8,9} Most patients who develop necrotising fasciitis have pre-existing conditions that render them susceptible to infection. Conditions that result in immunosuppression, such as advanced age, chronic renal failure, peripheral vascular disease, diabetes mellitus, and drug misuse seem to be risk factors (box 1).⁹

Fig 1 Young woman presenting with cellulitis of her lower abdomen after a caesarean section five days earlier. Small areas of skin necrosis are clearly visible

Clinical features

Necrotising fasciitis can affect any part of the body but the extremities, the perineum, and the truncal areas are the most commonly involved. Most patients present with signs of inflammation such as erythema, swelling, and pain at the affected site.⁷⁻⁹ Severe pain disproportionate to local findings and in association with systemic toxicity should raise the suspicion of necrotising fasciitis (box 2). However, these symptoms may be non-specific at first, which often causes the diagnosis to be missed (fig 1). As the infection progresses, the skin becomes increasingly tense and erythematous with indistinct margins. It may change colour sequentially from a red-purple to a dusky blue before progressing to necrosis and formation of bullae and eventually becoming haemorrhagic (box 2, fig 2). Crepitus of the affected area may be palpated and may even be seen as soft tissue air on a plain radiograph. Symptoms may develop over a period of hours to several days, and presentations are varied. Patients presenting at an advanced stage may show signs of systemic shock and sepsis, and it is these patients that often pose diagnostic difficulties since they may be confused, agitated, or even have a reduced level of consciousness.

Box 1: Predisposing factors for necrotising fasciitis

Comorbid conditions

- Immunosuppression
- Diabetes
- Chronic disease
- Drugs—for example, steroids
- Malnutrition
- Age > 60
- Intravenous drug misuse
- Peripheral vascular disease
- Renal failure
- Underlying malignancy
- Obesity

Aetiological factors

- Blunt or penetrating trauma
- Soft tissue infections
- Surgery
- Intravenous drug use
- Childbirth
- Burns
- Muscle injuries

Box 2: Clinical findings in necrotising fasciitis

Early findings

- Pain
- Cellulitis
- Pyrexia
- Tachycardia
- Swelling
- Induration
- Skin anaesthesia

Late findings

- Severe pain
- Skin discoloration (purple or black)
- Blistering
- Haemorrhagic bullae
- Crepitus
- Discharge of “dishwater” fluid
- Severe sepsis or systemic inflammatory response syndrome
- Multiorgan failure

Diagnosis

Certain diagnostic signs that we have come to associate with necrotising fasciitis are not always present. The findings of crepitus and soft tissue air on plain radiograph are seen in only 37% and 57% of patients, respectively.⁹ However, great caution should be taken. Even though they are pathognomonic for the disease, their absence should not exclude the diagnosis. This is an important point to make since many referrals by other specialties are made only once these signs become apparent.

Other findings that are common in necrotising fasciitis include a raised white cell count as well as raised concentrations of glucose, urea, and creatinine. Hypoalbuminaemia, acidosis, and an altered coagulation profile may also be present (box 3).^{7-9,10} Other tests, such as fine needle aspiration and incisional biopsy (which can be performed on the ward), have been used to aid diagnosis since they provide tissue samples for culture and histological analysis, respectively. However, their usefulness is limited by false negative results and the small cohort of patients in completed trials.¹¹ Similarly, computed tomography and magnetic resonance imaging are useful in cases where signs are equivocal or the diagnosis is in doubt.^{12,13} Both have their roles in delineating the extent of the infection and showing soft tissue gas, but the critical condition of patients often precludes their use and may even delay treatment.

Fig 2 Late signs of necrotising fasciitis with extensive cellulitis, induration, skin necrosis, and formation of haemorrhagic bullae

The diagnosis therefore remains a clinical one, and the information drawn from all the investigations should be used in conjunction; clinicians should not rely on individual tests. Clinicians should have a high index of suspicion and a low threshold for surgical referral.

Microbiology

Most studies have shown that necrotising fasciitis is polymicrobial in nature, with most cultures yielding a mixture of aerobic and anaerobic organisms.⁶⁻¹⁰ Those that are isolated reflect their distribution as normal skin commensals that are found adjacent to the site of infection—for example, anaerobic bacteria are usually isolated in necrotising fasciitis affecting the perineum. Infection with a single pathogen is reported in about 15% of cases.⁴ Overall, streptococcus is the most common causative organism (box 4).⁷⁻⁹ Much concern has arisen in recent years with the emergence of toxic shock strains of streptococcus (group A β haemolytic streptococci) leading to fasciitis with organ dysfunction. This virulent organism received much press coverage as the “flesh eating bacterium.” This relates to the fact that group A streptococci can cause necrotising fasciitis and associated toxic shock syndrome with profound discoloration and sloughing of the skin. However, in a recent study, group A streptococci (GAS) were isolated in only 15% of individuals with necrotising fasciitis.⁶ Most cases are caused by M types 1 and 3 and produce exotoxin A and streptolysin O.¹⁴ In a process similar to necrotising fasciitis resulting from a polymicrobial aetiology, the production of these M proteins inhibits phagocytosis and, along with the exotoxins, the proteins act as superantigens, which leads to the liberation of cytokines and a toxic shock-like syndrome.

As most cases of necrotising fasciitis are polymicrobial, empirical broad spectrum antibiotic coverage should be administered. Although subsequent antibiotic management will be guided by sensitivities of cultures taken intraoperatively, initial treatment should provide effective cover against Gram positive cocci, facultative anaerobic Gram negative rods, and anaerobes. In our unit, we use a combination of penicillin, gentamicin (if renal function permits), plus metronidazole or clindamycin to cover against the three principal

Box 4: Organisms identified in necrotising soft tissue infections

Gram positive aerobic bacteria

Group A β haemolytic streptococci
Group B streptococci
Enterococci
Coagulase negative staphylococci
Staphylococcus aureus
Bacillus species

Gram negative aerobic bacteria

Escherichia coli
Pseudomonas aeruginosa
Proteus species
Serratia species

Anaerobic bacteria

Bacteroides species
Clostridium species
Peptostreptococcus species

Fungi

Zygomycetes
Aspergillus
Candida

Other

Vibrio species

groups of organisms, but consultation with the on-call microbiologist should be sought before starting treatment with antibiotics.

Management

Having established the diagnosis, treatment should be instituted immediately. The patient should be resuscitated according to his or her clinical state and may even require intensive care support. Intravenous antibiotics should be started at the same time and be changed according to sensitivities. Patients should be taken to theatre without delay and have aggressive surgical debridement. Surgeons should make incisions to the deep fascia (this may reveal the presence of “murky dishwater fluid” in the wound), and all non-viable tissue including fascia should be excised. Further surgical exploration 24-48 hours later is mandatory to ensure that the infectious process has not extended. Repeated debridements may be necessary (as dictated by the state of the wound) until the infection has been controlled adequately. Haemorrhage is not uncommon after debridement, and in cases of disseminated intravascular coagulation such bleeds can be difficult to control. Having whole blood and clotting products available for the patient before surgery is essential.

Reconstructive surgery should be considered only once the patient has been stabilised and the infection fully eradicated. Sterile dressings may be used to cover the wound in the interim. Wound coverage can be achieved by either split thickness skin grafting or tissue transfer (fig 3). Vacuum assisted closure (VAC) therapy may be employed to promote healing and help close wounds, and this has helped in reducing the size of larger defects that would have been difficult to deal with simply on their own. Other forms of surgery, such as amputation, may be necessary for necrotising infections of the extremities, and a defunctioning colostomy should be considered if wounds are regularly contaminated with faeces.

Box 3: Adjuncts to help diagnosis

Laboratory investigations

Leucocytosis
Acidosis
Altered coagulation profile
Hypoalbuminaemia
Abnormal renal function

Plain radiography

Soft tissue gas

Computed tomography or magnetic resonance imaging

May delineate extent of disease
Soft tissue gas

Incisional exploration or biopsy (can be done at bedside)

Histological confirmation of diagnosis
Tissue culture to identify pathogens and sensitivities

Fig 3 Split thickness skin grafting can be used successfully to cover large defects after surgical debridement

The role of hyperbaric oxygen in the treatment of necrotising fasciitis remains controversial. Authors have advocated its practice in addition to operative debridement in improving overall survival. The principle behind its use is to increase the tissue oxygen tension in zones of infected hypoxic areas. This prevents extension of the disease and the need for further debridements.¹⁵ Conversely, others have shown no survival benefit.⁴ Further studies are therefore required before this treatment can be recommended.

Prognosis

Necrotising fasciitis is an uncommon, but life threatening, condition with a high associated mortality and morbidity. Reported mortality varies from 6% to 76%, but more recent studies report this to be much lower, at around 25%.^{7-9, 10} This is partly the result of early recognition and improved supportive measures that are multidisciplinary in origin.

Summary

Because of the potential fatal course of necrotising fasciitis, prompt diagnosis is the key to a favourable outcome. Laboratory findings and other diagnostic tests may be useful adjuncts, but the diagnosis is still primarily a clinical one, and suspicion alone warrants early surgical referral. The mainstay of treatment is immediate resuscitation of the patient, followed by aggressive surgical debridement. Further explorations may be necessary before soft tissue coverage is undertaken. Intravenous treatment with broad spectrum antibiotics should also be implemented. We have devised an algorithm that we hope will improve the referral service in suspected cases of necrotising fasciitis and avoid unnecessary delays in treatment (fig 4). Initial responses from the different specialties in our hospital have been good, but the algorithm requires formal evaluation to identify any benefit.

The experience of PRWS and NBH, along with help from other specialist professionals (such as N Sohal, consultant microbiologist) allowed SH to set out an article that will hopefully benefit others.

Competing interests: None declared.

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Fig 4 Algorithm for assessment and treatment of suspected necrotising fasciitis

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Endpiece

Not old

A person is not old until regrets take the place of hopes and plans.

Scott Nearing, American writer (1883-1983)

Fred Charatan, retired geriatric physician, Florida