Necrotizing fasciitis: a plastic surgeon's perspective

Nitisha Narayan
Gavin McCoubrey

Abstract

Necrotizing fasciitis is a serious life- and limb-threatening emergency that warrants early diagnosis and treatment with surgical debridement. The plastic surgeon would be involved once the debridement in complete to reconstruct the resultant defects or sometimes even in the acute stage to help with appropriate debridement. In this article we revisit the pathophysiology, microbiology, clinical features and reconstruction for defects resulting from necrotizing fasciitis and describe the management from a plastic surgeon's perspective.

Keywords Debridement; Fasciitis; Necrotizing; Reconstruction

Background

Necrotizing fasciitis (NF) is a serious surgical emergency. It is a rapidly progressive infection of the soft tissue (skin, subcutaneous fat and fascia) primarily involving superficial fascia that leads to destruction of the blood supply to the skin and causes skin necrosis. NF has been recognized as early as 5th century BC by Hippocrates. However, it was not until 1952 that Wilson et al. described the key features of NF.[1] Necrotizing fasciitis is a rare entity and therefore diagnosis can sometimes be very difficult. The overall incidence of NF has been estimated as 0.24–0.4 per 100,000 adults,[2] which is similar to other Western countries with an estimated 500 new cases every year.[1] The mortality rate is between 15% and 20%.[3] Early diagnosis and aggressive surgical debridement are key elements of treatment, as the condition is associated with high mortality rates if left untreated. The classic presentation consists of pain in the affected area out of proportion to the signs, hypotension, fever, haemorrhagic bullae of the skin, purpura, frank skin necrosis and gas gangrene.[4] At the other end of the spectrum, early presentation may involve erythema of the affected area with temperature in a generally well patient. This can often lead to a delay in diagnosis.

As plastic surgeons, we often deal with referrals where necrotizing fasciitis has to be ruled out. Plastic surgeons would often be involved in the initial debridement of extremity necrotizing fasciitis. NF involving other areas would be managed by general surgeons or ENT/maxillofacial surgeons depending on the anatomical area involved.

Nitisha Narayan ***MBBS MS General Surgery MRCS(Edinburgh)*** *is a Specialist Registrar in Plastic Surgery, Thames Valley and Wessex, UK. Conflicts of interest: none declared.*

Gavin McCoubrey ***BSc MBChB FRCS (Plast)*** *is a Consultant Plastic Surgeon at Salisbury District Hospital, Salisbury, UK. Conflicts of interest: none declared.*

Classification and pathophysiology

The term necrotizing fasciitis was first coined by Wilson in 1952 and was popularized by the media as 'Flesh eating bacteria syndrome'[5] Necrotizing fasciitis has been classified according to the microbiology findings into four categories.[5] It is imperative that good quality microbiology specimens are sent off preoperatively and intraoperatively so that appropriate microbiological diagnoses can be made and appropriate antibiotic treatment can be started after discussion with the microbiology team.

Type I is the most common type, which is also known as polymicrobial caused by anaerobes. This accounts for the majority of the cases and tends to involve two or more pathogens. This is classically seen in patients with Fournier's gangrene or necrotizing fasciitis of the abdominal wall and is associated with significant comorbidities.

Type II is mono-microbial and is caused by beta-hemolytic group A streptococci (*Streptococcus pyogenes*). This is usually associated with necrotizing fasciitis of the extremities. This tends to affect patients who are otherwise fit and healthy and may have a preceding cause, i.e. trauma to the skin. This infection may also be caused by *Staphylococcus aureus*, in which case the risk of developing MRSA (10–30%) and toxic shock syndrome is high.[6]

Type III infection is also caused by mono bacterial infection but usually involving *Clostridium* species or Gram-negative bacteria, e.g. *Vibrio* sp. *Clostridium* infection is associated with crush injuries or surgical wounds and may present with crepitus in the wound and gas under the skin on imaging. *Vibrio* spp. infections are frequently found in Asia especially along the coastal cities. It is found in warm seawater and therefore can affect fishermen. Exposure can occur through ingestion of shellfish or due to trauma to the skin in the marine environment. Other causes include *Aeromonas* infection, which usually resides in fresh water.

Type IV infection is caused by fungal infection usually due to *Candida* spp. This type of infection is rare, aggressive and would usually occur in an immune-compromised patient.

Based on clinical presentation necrotizing fasciitis could be classified into fulminant, acute and subacute. The fulminant variety has very rapid onset and progresses to multi-organ failure. The acute variety progresses over several days, and tends involve large areas of the body. The subacute variety develops over weeks and may be localized in its involvement.[7]

The infection usually begins in the superficial fascia. Destruction of the fascia is a result of direct destruction from enzymes (e.g. hyaluronidase) and toxins released by the bacteria. Invasive bacteria by means of rapid proliferation can then causes thrombosis of the blood vessels. This leads to liquefactive necrosis of the fascia that spreads rapidly along fascial planes causing disruption of the blood supply to the skin, therefore leading to ischaemic necrosis of the overlying skin, which in the early stages can appear normal. Fibrous attachments between subcutaneous tissue and fascia can limit the spread, therefore in larger areas of the body (e.g. trunk) the infection spreads faster as there is a lack of these adhesions.

Risk factors and mortality indicators

The aetiology of necrotizing fasciitis can be multi-factorial. Quite often no direct cause of the disease can be identified. However

majority of the patients would have some comorbidity associated with them.

Diabetes is one of the most common comorbidity associated with necrotizing fasciitis. Other factors that could potentially contribute to developing necrotizing fasciitis include peripheral vascular disease and immunosuppression associated with diabetes.

Immunosuppression is an important predictor of mortality, this includes diabetics, intravenous drug abusers, patients on steroids, HIV infection and malignancy.[8]

Other common comorbidities include liver cirrhosis, chronic heart failure, obesity, alcohol abuse, autoimmune conditions and peripheral vascular disease. The use of NSAIDs as a risk factor is unclear, however, use of NSAIDs or steroids can suppress fever and other inflammatory responses and can therefore delay the diagnosis of necrotizing fasciitis.[6]

Necrotizing fasciitis is known to have a cumulative mortality rate of 34% (6–76%), hence the emphasis on early recognition and aggressive surgical debridement. Patients with higher mortality rate were associated with age >60, serum creatinine >1.6 mg/dl, liver cirrhosis, lower systolic blood pressure (<90), hypoalbuminemia, increased amount of banded leukocytes and thrombocytopenia.[9]

Clinical presentation

Head and neck

Necrotizing fasciitis, although uncommon in the head and neck region, can lead to catastrophic consequences and often difficult to debride and reconstruct. This can arise in the submandibular area or around the eye. Dental pathology is the most common cause of NF in the head and neck region. Other causes include trauma, burns, insect bites or peritonsillar abscesses. Patients usually present with pain, fever, inflammation of the involved skin; however, if not detected early, it could lead to significant complications like airway obstruction. Trauma to the eyelid, i.e. blunt trauma, insect bites, or surgery have been implicated as causative factors for peri-orbital necrotizing fasciitis. The pathogenesis remains the same; however, peri-orbital necrotizing fasciitis can lead to catastrophic complications, e.g. thrombosis of the central retinal artery and deeper involvement of orbital contents requiring exenteration. High index of suspicion, early diagnosis and surgical debridement is the treatment followed by appropriate reconstruction. Joint care by ophthalmology, head and neck teams and plastic surgeons is the key.

Trunk and perineum

Fournier gangrene was described by Jean Alfred Fournier in 1883. It involves the skin and fascia of the scrotum, perineum and lower abdomen (Figure 1).[10] Causative factors include urinary tract infection, peri-anal pathology, local trauma to the area ad associated comorbidities. Treatment entails early debridement, appropriate antibiotics, diverting colostomy (depending on the defect) and appropriate reconstruction. NF of the abdominal wall could present due to surgical site infection from previous abdominal surgery, trauma or intra-abdominal pathology (malignancy or bowel perforation).

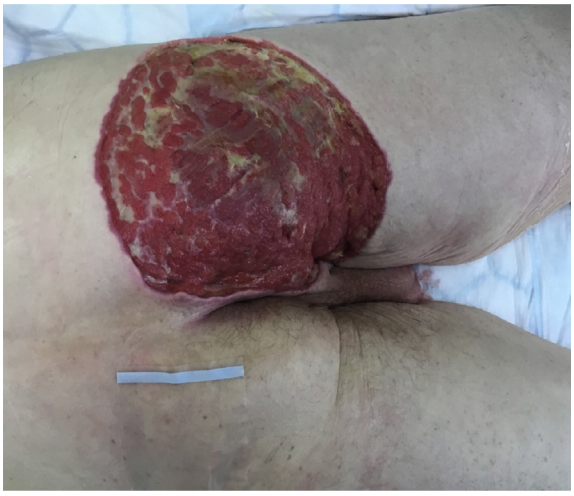

Figure 1 Wound defect following debridement in groin and perineum.

Extremities

NF of the extremities usually presents with pain (out of proportion to signs), erythema of the skin, crepitus, blistering, toxic appearance of the patient, systemic signs of shock. NF of the limbs is a common referral to the plastic surgeons. Often, the diagnosis is delayed to similar presentation of cellulitis. Classically the patient is managed on a medical ward for cellulitis with antibiotics and the diagnosis is made late once the skin necrosis sets in. Like other anatomical sites, management is early, aggressive debridement followed by appropriate reconstruction (Figure 2). Early involvement of physiotherapists is imperative in keeping the joints active and supple.[11]

Multifocal

Multifocal NF is a rare entity but associated with very high mortality rates (70%).[12] NF can affect any part of the body. Once bacteraemia sets in, haematologous metastatic deposition of emboli can occur. This may lead to involvement of more than one area of non-contiguous necrosis. Multifocal NF is usually associated with multiple cop-morbidities and with marine variety of bacteria (type III NF). Management remains the same, however close monitoring of these patients is important in order to detect new areas of involvement early. Clinical diagnosis is key;

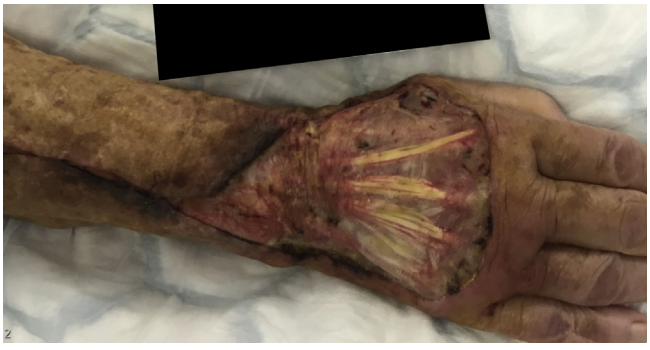

Figure 2 Post debridement appearance of necrotizing fasciitis of the hand.

however, imaging (e.g. gadolinium contrast MRI) can aid in diagnosis.

Necrotizing myositis

Necrotizing myositis is a surgical emergency just like NF. The patient presents with fever, fatigue, intense pain in a muscle compartment, redness and swelling at the site of pain. Classically there is paucity of skin changes, which often leads to a delay in diagnosis. The treatment is immediate resuscitation and surgical debridement just like NF. The mortality rate in literature varies between 6% and 80%. Early debridement is key and it is important to note that planes of debridement are deeper with muscle necrosis.[12]

Investigations

NF is primarily diagnosed clinically; however, adjunctive lab tests can help with diagnosis.

Lab tests are usually not specific. These include leukocytosis, WCC > 20,000, high blood urea nitrogen and creatinine, raised serum creatine kinase and high C-reactive protein. The Laboratory Risk Indicator for Necrotising Fasciitis (LRINEC) is a scoring system proposed for early diagnosis (see Table 1).[13]

Bed side tests (e.g. 'finger sweep' test) can be performed under local anaesthesia. This includes making a 2-cm incision down to deep fascia with gentle probing with a finger. Presence of characteristic 'dishwater pus' with no bleeding is associated with NF.

Similarly an incisional biopsy may be done down to fascia and could be sent for immediate frozen section, culture and Gram stain.

Imaging is not routinely used for diagnosis. Plain radiograph has low sensitivity and specificity for diagnosis. CT and MRI may be used to aid in diagnosis. Gadolinium contrast enhanced MRI is superior to other imaging modalities. T2 hyper-intense signal with thickening of the deep inter-muscular fascia is an important marker for NF on MRI and is useful for early diagnosis of multifocal NF.

Adjunctive treatment

Antibiotics

Once NF is suspected, patients should be empirically started on broad-spectrum antibiotics based on clinical classification of type of NF based on history, examination, Gram stain and cultures if available. When appropriate this should be discussed with on duty microbiologist. Clindamycin is recommended as the empirical antibiotic of choice with its activity against *S. aureus*, streptococci and anaerobes. It acts by inhibiting bacterial protein synthesis at the level of the 50S ribosome. It therefore exerts a prolonged post-antibiotic effect. It may even decrease toxin production and increase microbial phagocytosis at sub-inhibitory levels. A combination of penicillin and clindamycin is recommended for severe NF due to Group A streptococci or *Clostridium perfringens*.[14]

Fluids and monitoring

The patient should have good intravenous access and should receive appropriate fluids for optimization until they go to theatre. In the immediate postoperative period, patients would usually require very close monitoring on a high-dependency unit or intensive care unit. This would include on-going fluids, nutritional support and appropriate physiotherapy.

IV immunoglobulin therapy has been reported with group A streptococcal infections (type II).

The use of hyperbaric oxygen as an adjunct to surgery has been described to improve oxygen tension in the local tissues, thereby reducing the need for further debridements.[15]

Surgical management and reconstruction

As mentioned previously on multiple occasions, NF is a true surgical emergency and early diagnosis and aggressive surgical debridement is the imperative for treatment. Surgical incisions are made along the Langer's lines to achieve better wound healing and scarring. Debridement must continue until healthy bleeding tissue is reached. Debridement is started as a 'melon slice' exploratory excision and the tissue is assessed under vision. The debridement is then carried on in horizontal and vertical axis or in a concentric onion ring fashion (Figure 3). Once debridement is complete, the wound is appropriately dressed. A 'second-look' operation is done within 48 hours to check viability of tissues and debride further tissue if needed.

In the head and neck it is important to prevent contracture from the outset, therefore application of neck collar, physiotherapy and planning reconstruction early is important.

LRINEC scoring system for necrotizing fasciitis

Variable	Score
CRP (mg/L)	
>150	4
WBC (g/L)	
<15	0
15–25	1
>25	2
Haemoglobin (g/dL)	
>13.5	0
11–13.5	1
<11	2

Table 1

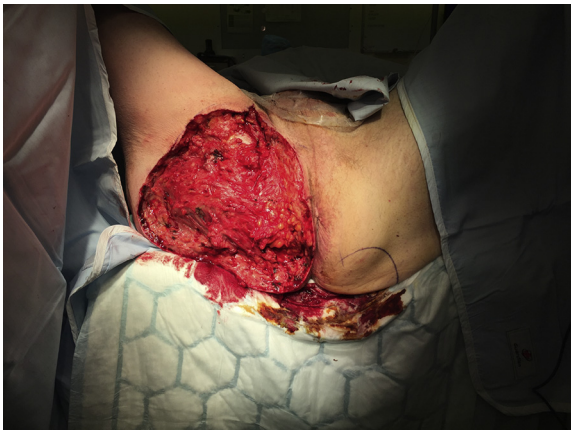

Figure 3 Intraoperative photo of a debrided wound of the thigh.

In abdomen the incisions and made parallel to the muscle fibre. Exploratory laparotomy may be required if there is intra-abdominal spread of infection. A diverting colostomy may be needed with involvement of the perineum.

In the extremities once debridement is complete and wound is dressed, care must be taken to continue appropriate physiotherapy to keep the joints moving and avoid contractures. Where extensive involvement of the deeper tissue is present, amputation may be indicated. Amputation may also be indicated in patients with multi-organ failure and significant comorbidities as it is simpler and leads to less blood loss.

Reconstruction is planned according to the reconstructive ladder and the anatomical site involved. The principle of reconstruction includes coverage of the soft tissue defect, maintaining normal function and aesthetics if possible.

Vaccum-assisted closure

Topical negative pressure dressings can be used following surgical debridement of large areas until healthy granulation tissue is formed (Figure 4). This helps mechanically shrink the size of the wound, reduces bacterial load, and promotes granulation. These areas can then have more definitive reconstruction with skin grafts or flaps.

Skin grafts

Skin grafts are commonly used for reconstruction of defects for NF. Full thickness skin grafts can be used to cover smaller defects and aesthetically sensitive areas like the face and eyelids.

Split skin grafts are more appropriate for larger areas, e.g. abdominal wall and limbs (Figure 5).

Dermal substitutes

These can be used as adjunct to split skin grafting to improve the aesthetic outcome especially in limbs. These include products with (skin graft at the same time) or without silicon layer (skin graft in 2–3 weeks).

Flaps

Flap cover (Figure 6) is required when covering vital structures, i.e. blood vessels, or over-exposed tendons and bone devoid of paratenon and periosteum respectively. The flaps could be pedicled (where the flap remains attached to its original anatomical location) and simply moved to cover the defect. An example of this is using sartorius muscle flap to cover the femoral blood vessels or a gracilis muscle flap to cover defects in the perineum. Free flap cover may be required when pedicled flap cover is not an option, e.g. to cover defects over the popliteal fossa to prevent joint contracture or free flap cover to defects of the neck.

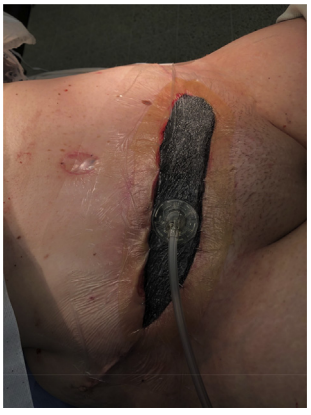

Figure 4 Use of vacuum dressing on a debrided wound.

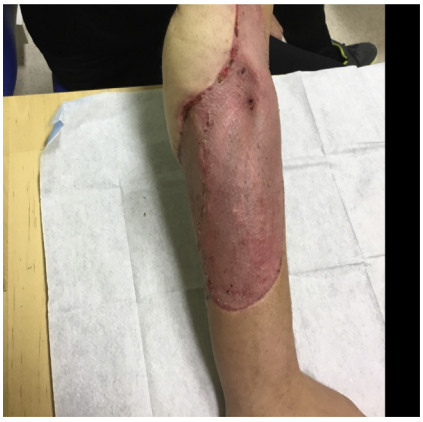

Figure 5 Split skin graft performed for large defect on the forearm.

Figure 6 Anterolateral-thigh flap done to cover defect on the arm and elbow.

Summary

Necrotizing fasciitis is a life-threatening condition that can affect any part of the body and is associated with multiple causative factors and comorbidities. It is a rare entity and usually referred to the plastic surgeons for diagnosis (extremities) and for reconstruction. Laboratory findings and bedside tests are useful but clinical suspicion forms the main stay of diagnosis. NF should be managed by early resuscitation and surgical debridement and a multidisciplinary approach is warranted from the very beginning. This must include intensivists, microbiologists, primary surgical team, nutritionists, therapist and reconstructive surgeons. ◆

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