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Necrotizing fasciitis: Lethal soft tissue infection

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Abstract
Necrotizing fasciitis (NF) is a rapidly progressive, life-threatening soft tissue infection that spreads along the connective tissue planes. It not only involves superficial tissues but may also extend into deeper layers, including the muscles. Due to the rapid spread of infection and systemic toxicity, the morbidity and mortality associated with NF is extremely high. Understanding the etiopathogenesis, diagnostic and therapeutic approaches to this serious condition, is essential for early diagnosis and prompt treatment. Aggressive management of shock, appropriate antibiotic therapy and early extensive surgical debridement can significantly reduce morbidity and mortality in affected patients. This article reviews the pathophysiology, diagnostic workup, and therapeutic approach to necrotizing fasciitis.

Keywords: Necrotizing fasciitis, soft tissue infection, pathophysiology, diagnosis, surgical debridement, antibiotic therapy

Introduction
Necrotizing fasciitis is an aggressive skin and soft tissue infection that causes severe necrosis of the fascia and subcutaneous tissues. The infection spreads along fascial planes, which typically have poor blood supply and may extend to involve the muscles. Severe, widespread tissue necrosis, systemic toxicity, and multi-organ dysfunction are commonly associated with NF, contributing to high morbidity and mortality. The causative organisms are usually polymicrobial, although gas production may occur in some cases.

Classification
Necrotizing fasciitis (NF) based on the causative organisms can be classified as polymicrobial or mono microbial [1].

Type 1-Polymicrobial
This type involves a mixture of aerobic and anaerobic bacteria, such as entero-bacteriaceae, bacteroides, enterococcus, and anaerobic Streptococcus species. It is commonly seen in perineal and abdominal wall infections, postoperative wounds following colonic surgery and in immunocompromised patients.

Type 2-Monomicrobial
This type is typically caused by Group A Streptococcus (streptococcus pyogenes) and staphylococcus aureus, including methicillin-resistant Staphylococcus aureus (MRSA). These infections usually occur in otherwise healthy individuals following minor trauma.
Other causative organisms include:
- *Vibrio vulnificus* (associated with marine exposure)
- *Aeromonas hydrophila* (associated with freshwater exposure)
- *Clostridial* species

Alternate Classification-Based on type of bulla [1, 2]
This classification is based on the presence and type of bulla seen in NF:
- **Group N:** No bullae present; typically associated with Staphylococcus species.
- **Group S:** Serous-filled bullae; commonly caused by β-haemolytic Strepto-coccus.
- **Group H:** Haemorrhagic bullae; associated with Vibrio species.

Group H has the worst prognosis, often requiring intensive care admission and carrying a higher risk of limb amputation.

Pathology

Tissues obtained from the operating room following debridement typically show extensive superficial fascial necrosis. The majority of small and medium-sized blood vessels are usually thrombosed. Aggregates of neutrophils are commonly observed. Small vessel vasculitis and extensive fat necrosis are also evident. All glands in the dermis and subcutaneous tissues are usually necrotic.

Risk Factors

The following conditions increase the risk of developing necrotizing fasciitis:

1. Diabetes mellitus
2. Peripheral vascular disease (PVD)
3. Immunosuppression (e.g., steroid use, malignancy, HIV infection)
4. Recent surgery, trauma, or intramuscular injections
5. Chronic liver and kidney diseases
6. Intravenous drug abuse
7. Obesity and malnutrition

Clinical features

Early signs of necrotizing fasciitis may be subtle and non-specific. However, rapid progression of infection is the hallmark of this condition.

- **Prodrome:** Severe pain disproportionate to the local findings usually fol-lowing a breach in the skin.
- **Local signs:** These include erythema, swelling, warmth, tenderness, and rapidly spreading erythema with indistinct margins.
- **Skin changes:** Patients may develop tense oedema, blistering, bullae, ecchymoses, dusky discoloration, and crepitus if the infection is caused by gas-forming organisms. (Figure 1)
- **Systemic symptoms:** Fever, tachycardia, hypotension, mental obtundation, and features of septic shock may be present.
- **Late features:** Anaesthesia over the affected skin due to nerve destruction, frank necrosis, and foul-smelling discharge are common.

Diagnosis

A strong clinical suspicion remains the mainstay of diagnosis. Early detection is crucial to prevent serious complications.

Laboratory investigations: The LRINEC score (Laboratory Risk Indicator for Necrotizing Fasciitis) is useful in evaluating patients [1, 2, 3]. (Table 1)

- A score of 6 has a positive predictive value of 92% and a negative predictive value of 96%.
- A score of 8 or higher indicates a 75% risk of necrotizing infection.

Imaging

- **Plain X-ray:** Plain x ray will reveal subcutaneous gas, indicative of gas-forming infections.
- **Ultrasound:** Can demonstrate thickened fascial planes and fluid collections.
- **CT scan:** Useful for detecting fascial thickening, presence of gas, and flu-id tracking along tissue planes.
- **Vascular Doppler:** Assesses the blood supply status to the affected extremity.

Surgical Exploration

Surgical exploration remains the gold standard for diagnosing necrotizing fasciitis. Confirmatory signs observed during surgery include:

- Tissues that separate easily (often described as “dishwater fluid”)
- Grey, necrotic fascial tissue
- Lack of bleeding from the affected tissues
- Absence of normal tissue resistance during dissection

Management

The management of necrotizing fasciitis (NF) necessitates urgent, multidisciplinary intervention. Early and aggressive resuscitation combined with prompt surgical debridement constitutes the mainstay of effective treatment.

Resuscitation and Supportive Care:

Initial management mandates aggressive intravenous fluid resuscitation alongside continuous hemodynamic monitoring to maintain adequate tissue perfusion [4]. In cases refractory to fluid therapy, ionotropic agents should be initiated to support circulatory function. Patients presenting with advanced disease frequently require organ support modalities, including mechanical ventilation and renal replacement therapy. Optimal glycemic control is imperative, particularly in diabetic patients, to mitigate metabolic derangements. Additionally, correction of coagulopathies is essential to prevent progression to disseminated intravascular coagulation (DIC), which is associated with increased morbidity and mortality.

Surgical Management

Immediate and extensive surgical debridement is critical in the management of necrotizing fasciitis [5, 6]. Debridement should proceed until viable, bleeding tis-sue margins are encountered, thus ensuring complete removal of necrotic skin, fascia and subcutaneous tissue. All fluid collections and abscesses must be thoroughly evacuated to reduce bacterial load.

Serial debridement is frequently required within the subsequent 24 to 48 hours to achieve adequate infection control and to assess for viable tissue. (Figure 2) Failure to control infection despite repeated interventions may necessitate more radical procedures.

In cases of severe limb involvement accompanied by septic shock and hemodynamic instability, amputation of the affected extremity may be indicated to prevent systemic deterioration.

Post-debridement wound management involves regular dressing changes using appropriate antimicrobial agents. Negative pressure wound therapy (NPWT) can be beneficial in patients with significant exudation from exposed tissues, promoting wound contraction and granulation. Hyperbaric oxygen therapy serves as an adjunctive treatment by enhancing tissue oxygenation and improving vascular perfusion, potentially reducing morbidity. (Figure 3) Once the wound shows healthy granulation tissue, reconstruction in the form of skin grafting or flaps can be performed. (Figure 4)

Antibiotic Therapy

Empiric broad-spectrum intravenous antibiotic therapy should be initiated promptly to cover Gram-positive organisms (including methicillin-resistant Staphylococcus aureus [MRSA]), Gram-negative bacteria, and anaerobes. Carbapenems, in combination with clindamycin or vancomycin,

are considered the antibiotics of choice [5, 6].
Clindamycin is specifically recommended for its ability to inhibit toxin production in Streptococcus and Staphylococcus infections, thereby reducing systemic toxicity.
Metronidazole may be added to enhance coverage against anaerobic organisms.
Intravenous immunoglobulin (IVIG) therapy is reserved for patients with severe staphylococcal toxic shock syndrome, where it may help neutralize circulating toxins and modulate the immune response [7].

Prognosis
Mortality rates for necrotizing fasciitis vary widely, ranging from 10% to 40%, depending on factors such as the causative organisms, presence of comorbidities, development of septic shock, and the timeliness of surgical intervention.
Common complications include limb loss, renal failure, multi-organ dysfunction, and prolonged rehabilitation.
Poor prognostic indicators include advanced age (>50 years), delayed surgical debridement, hypertension, renal failure, uncontrolled comorbid conditions, and extensive tissue involvement.

Table 1: LRINEC score (Laboratory Risk Indicator for Necrotizing Fasciitis)

Criteria	Interpretation
CRP (mg/L)	<150 (0) >150 (4)
Total WBC Count (cells/mm)	<15 (0) 15-25 (1) >25 (2)
Haemoglobin (gm/dl)	>13.5 (0) 11-13.5 (1) <11 (2)
Sodium (mmol/L)	>135 (0) <135 (2)
Serum Creatinine (mg/dl)	<1.6 (0) >1.6 (2)
Glucose (mg/dl)	<180 (0) >180 (1)

Fig 1: Necrotizing fasciitis causing extensive necrosis of the skin and soft tissues.

Fig 2: After three sessions of debridement

Fig 3: After regular dressings and 10 sessions of hyperbaric oxygen therapy.

Fig 4: After split thickness skin grafting

Conclusion
High index of suspicion in superficial soft tissue infections is essential for early diagnosis.
Aggressive resuscitation followed by extensive debridement in the mainstay of treatment.
Optimization of co-morbidities such as control of diabetes significantly contributory for a successful outcome.
Structured wound care followed by reconstruction is the final treatment.

Conflict of Interest
None

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