Necrotizing fasciitis

Necrotizing fasciitis is an extremely severe soft tissue infection that spreads along the fascia planes and causes necrosis. It is often caused by mixed aerobes and anaerobes.

It is also known as haemolytic streptococcal gangrene, hospital gangrene and gangrenous erysipelas .

When the male genitalia are involved, called Fournier’s disease Or Fournier's gangrene.

This condition is an extremely severe, life-threatening infection.

It starts as a cellulitis that dissects into the fascial planes of the skin.

Causes of necrotizing fascitis

Streptococcus and **Clostridium perfringens** are the most common organisms because they are able to produce a toxin that further worsens the damage to the fascia.

In general, this infection is caused by haemolytic streptococci and, less commonly, haemolytic staphylococci.

Other organisms that have been identified in some of these infections, include coliforms, Bacteroides, diphtheroids and Pseudomonas.

Most commonly, the condition arises following surgery or trauma.

Spontaneous cases have also been described.

The most commonly affected sites are the extremities, followed by the lower trunk, including the external genitalia and the perineum.

Risk factors

- Diabetes mellitus,
- Intravenous drug use,
- Chronic liver or renal disease, and
- malignancy are associated risk factors

Pathophysiology

The exact mechanism for the subcutaneous necrosis is unknown but appears to be related to the binding of the mucopeptide fraction of the bacterial cell wall with dermal collagen.

The necrosis does not involve the muscle layer and skin involvement is secondary to thrombosis of the perforating vessels coursing through the infected necrotic area.

Necrotizing fasciitis is always serious and carries a definite mortality if not managed early and promptly.

The affected part is initially very painful but then becomes numb owing to the involvement of sensory nerve fibres. Then the process spreads rapidly through the subcutaneous fatty/ fascial plane with reddish discoloration, inflammatory oedema, necrosis and eventual sloughing of the overlying skin.

Clinical Presentation.

The features that distinguish necrotizing fasciitis from simple cellulitis are;

1. A very high fever,
2. A portal of entry into the skin,
3. Pain out of proportion to the superficial appearance,
4. Presence of bullae, and
5. Palpable crepitus.

Systemic manifestations are always present and the toxaemia may be severe with pyrexia, tachycardia and shock.

In untreated infection, the overlying skin develops blue-gray patches after 36 h, and cutaneous bullae and necrosis develop after 3–5 days.

Necrotizing fasciitis due to a mixed flora, but not that due to group A streptococci, can be associated with gas production.

Without treatment, pain decreases because of thrombosis of the small blood vessels and destruction of the peripheral nerves which is an ominous sign.

In patients to whom the causative organism is clostridium perfringens, these patients are extremely toxic and the mortality rate is high.

Within 48 hours, there is a rapid tissue invasion and systemic toxicity that is associated with hemolysis and death ensues.

The distinction between this entity and clostridial myonecrosis is made by muscle biopsy.

Diagnosis.

Blood cultures should always be taken in these kind of patients.

Laboratory evidence of necrotizing fasciitis is an elevated creatine phosphokinas.

These patients are prone to development of hypocalcemia.

An x-ray, CT scan, or MRI demonstrates presence of air in the tissue or necrosis.

All of these laboratory methods of establishing a diagnosis lack both sensitivity and specificity.

Surgical debridement is the best way to confirm the diagnosis and is also the mainstay of therapy.

Treatment.

Treatment includes resuscitation with crystalloids and blood, antibiotics and surgery is the mainstay of therapy with an early wide surgical excision and drainage with delayed skin cover being recommended.

The best empiric antibiotics are the beta-lactam/beta-lactamase combination medications, such as ampicillin/sulbactam, ticarcillin/ clavulanate, or piperacillin/tazobactam.

Vancomycin at 15mg/kg every 12 hours plus clindamycin 600mg every 8 hours together with gentamycin 5mg/kg per day. These treatmet regimen is adjusted with culture results in that; If there is a definite diagnosis of group A Streptococcus (pyogenes), then treat with clindamycin and penicillin.

Without adequate therapy, necrotizing fasciitis has an 80% mortality rate.