Fournier Gangrene : Necrotizing Fasciitis

Fournier gangrene is a polymicrobial rapidly progressive necrotizing fasciitis of the perineal, perianal or genital areas of the males.

Gangrene is a local death of soft tissues as a result of disease or injury. The dead tissue is nearly always colonized by bacteria.

Gangrene can be of two types:
1) Noninfected or dry gangrene
2) Infected or wet/gas gangrene.

There are a number of subdivisions including:

- Clostridium cellulitis,
- Clostridium myonecrosis and
- Necrotizing fasciitis.

Types of Fournier gangrene

There are two types of Fournier gangrene

Type I Fournier gangrene. This is due to a mixture of aerobic and anaerobic organisms following abdominal surgery or associated with diabetes mellitus.

Type II Fournier gangrene due to GAS synergistic with a second organism such as Staphylococcus aureus, coliforms, Bacteroides species.

In this condition, there are five fascial planes that can be affected. They include;

1. Colles'fascia is a type of fascia of the anterior triangle of the perineum.
2. Dartos fascia is the continuation of Colles' fascia that is over the scrotum and penis.
3. Buck's fascia is a fascia that lies deep to the dartos fascia. It covers the penile corpora.
4. Scarpa's fascia is continuous with Colles' fascia inferiomedial direction.
5. Lastly, Camper's fascia is a loose areolar fascial layer deep to the abdominal wall skin, but superficial to Scarpa's fascia.

Urogenital causes usually lead to initial involvement of the anterior triangle, while on the other hand anorectal causes involve the posterior triangle.

Pathway for spread

There are two main pathways that this type of gangrene spread.

The first pathway is from the GI tract, from the anorectal region after surgical treatment of some

conditions such as hemorrhoids, or rectal trauma.

The second pathway is from the urogenital tract after prolonged use of urinary catheters or instrumental dilatation of urethra stenosis.

Pathogenesis of Fournier Gangrene

The pathogenesis process of Fournier gangrene is characterized by polymicrobial infection with subsequent vascular thrombosis and tissue necrosis or tissue death. This is aggravated by poor host defense due to one or more underlying systemic disorders.

Aerobic organisms cause intravascular coagulation by inducing the process of platelet aggregation and complement fixation, while anaerobes produce heparinase.

Hypoxic tissue leads to the formation of oxygen free radicals (superoxide anions, hydrogen peroxide, hydroxyl radicals). This lead to cell membrane disruption decreased ATP production, and DNA damage, which leads to decreased protein production

The microorganisms secrete various enzymes and toxins. Lecithinase, collagenase, and hyaluronidase which lead to digestion of the fascial planes. Fascia necrosis and digestion then extend the infection along the fascia planes. They produce insoluble hydrogen and nitrogen, leading to the formation of gas in the subcutaneous tissues, clinically palpable as crepitus.

Endotoxins are released from the cell walls of Gram-negative bacteria. Macrophage activation and subsequent complement activation ensues with the release of pro-inflammatory cytokines and eventual development of septic shock

Thrombosis of these vessels reduces local blood supply and the tissue oxygen tension falls
The resultant tissue hypoxia allows the growth of facultative anaerobes and microphillic organisms

Infection usually advances through the Buck and Dartos, Colles, Scarpa, in all directions.

The infection initiates under normal-looking skin.

Causative Organisms for Fournier Gangrene

The following are common causative microorganisms

- Eschelichia Coli,
- Pseudomonas aeruginosa,
- Streptococcus putridis,
- Staphylococcus, Klebsiella,
- Anaerobic bacteria like Bacteroides, Clostridium perfringens and Bacillus fragilis.
- Enterobacteriaceae
- Fungi

Signs and symptoms of Fournier Gangrene

This disease usually starts with a period of genital discomfort and pruritis that is followed by a sudden onset of perineal pain.
As the gangrene progresses this pain is replaced by anesthesia.

At the start, the skin may appear to be normal with no apparent extent of the subdermal gangrene. This may delay diagnosis.

Skin changes as a result of gangrene result in drainage of the affected area.

Formation of crepitus as a result of anaerobic metabolism. This is composed of hydrogen, hydrogen sulfide, nitrogen, and nitrous oxide, which may be detected on an x-ray. This indicates the presence of dead tissue associated with a foul odor.

There is rapid development of severe toxemia with associated signs, of

- Pyrexia with or without hypothermia,
- Leukocytosis,
- Thrombocytopenia,
- Raised blood urea and nitrogen,
- Tachycardia,
- Hypotension
- Reduced urine output.

Organ failure and, Death.

Causes of Fournier Gangrene

- Poor hygiene,
- Usually, it is due to a less aggressive, more routine infectious process through some point of entry involving the colon, urinary tract, prostate or anorectal area,
- Cellulitis or traumatic injury involving cutaneous structures in the perianal region
- Vaginal deliveries with episiotomies and Caesarean section,
- Cancer of the large intestine,
- Hematological malignancies,
- Severe neutropenia

Diagnosis

Differential diagnosis includes

- cellulitis,
- balanitis,
- orchitis,
- epididymitis,
- torsion,
- strangulated hernia and
- benign scrotal edema.

Diagnosis is usually a clinical diagnosis.

A full blood count will reveal anemia and leukocytosis.

The renal profile is impaired with high urea and creatinine secondary to septic shock.

Coagulopathies secondary to sepsis/septic shock may also be seen.

A mid-stream urine sample is to exclude UTI.

The source of infection should be investigated by whatever means necessary.

An IV pyelogram, barium enema, sigmoidoscopy and/or cystoscopy

Tissue biopsies and pus are sent for culture and sensitivity tests.

A frozen section is useful for visualizing soft tissue necrosis and dense infiltration of the involved area with polymorphonucleocytes.

MRI will show edema but is not necessary for diagnosis

Treatment of Fournier Gangrene

These infections are a surgical emergency

Radical debridement aggressively removing all necrotic tissue and any marginally perfused tissue. Debridement until all remaining tissues are adherent and viable. The penis, testes, bladder, and rectum are spared during debridement.

Intravenous broad-spectrum antibiotics -clindamycin, vancomycin, metronidazole, ampicillin-sulbactam, gentamycin are commonly used.

Clindamycin is useful in the treatment of necrotizing soft-tissue infections due to its activity against gram-positive and anaerobic microorganisms.

Empirical therapy is started and modified on the basis of culture and sensitivity tests.

Daily reviewing of results and creatinine phosphokinase (CPK) levels to monitor myonecrosis.

Enteral nutrition.

The wound is closed later with skin grafting to restore function quickly and provide a good cosmetic outcome.
The resultant scar may predispose patients to squamous cell carcinoma after a long latent.

Tetanus prophylaxis is indicated if a soft-tissue injury is present.

In cases associated with sepsis syndrome, therapy with intravenous immunoglobulin (IVIG), which is thought to neutralize superantigens (eg, streptotoxins A and B) believed to mitigate the exaggerated cytokine response