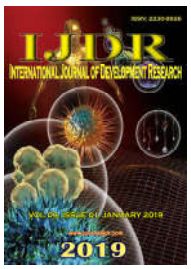
ISSN: 2230-9926

Available online at http://www.journalijdr.com

IJDR

International Journal of Development Research
Vol. 09, Issue, 01, pp.25454-25457, January, 2019



ORIGINAL RESEARCH ARTICLE OPEN ACCESS

FOURNIER GANGRENE – A SURGICAL EMERGENCY

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ARTICLE INFO

Article History:
Received 14th October, 2018
Received in revised form
10th November, 2018
Accepted 25th December, 2018
Published online 30th January, 2019

Key Words:
Gangrene, Necrotizing fasciitis, Sepsis.

ABSTRACT

Fournier Gangrene is a surgical emergency and a potentially life threatening form of necrotizing fasciitis of male genitalia. The infection most commonly arises from skin, urethra or rectal region. Predisposing factors include diabetes mellitus, local trauma, extravasation of urine, perirectal and perianal infections and surgery. Prompt diagnosis is crucial because of the rapidity with which the process can progress with a risk of septicemia and death. We present two cases of Fournier Gangrene recently treated in our hospital.

Citation: Dr. Zahid, M.D., Dr. Raj Kumar and Dr. Santhosh Kumar, K., 2019. "Fournier gangrene – a surgical emergency", *International Journal of Development Research*, 9, (01), 25454-25457.

INTRODUCTION

Fournier gangrene is a synergistic polymicrobial necrotizing fasciitis of the perineum and genitalia. It was named after French venereologist Jean Alfred Fournier. It is also known as idiopathic gangrene of the scrotum, streptococcal scrotal gangrene perineal phlegmon, and spontaneous fulminant gangrene of the scrotum (Fournier, 1883). As originally reported by Fournier in 1883, it was characterized by an abrupt onset of a rapidly fulminating genital gangrene of idiopathic origin in previously healthy young patients that resulted in gangrenous destruction of the genitalia. The disease now differs from these descriptions in that it involves a broader age range, including older patients, follows a more indolent course and has a less abrupt onset; and in approximately 95% of the cases, a source can now be identified (Macrea, 1945). Infection most commonly arises from the skin, urethra or rectal regions.

Predisposing Factors: Predisposing factors include diabetes mellitus, local trauma, paraphimosis, periurethral extravasation of urine, perirectal or perianal infections, and surgery such as circumcision or herniorrhaphy (Eke, 2008). In cases originating in the genitalia, specifically as a result of urethral obstruction, the infecting bacteria probably pass through Buck fascia of the penis and spread along the dartos fascia of the scrotum and penis, Colles fascia of the perineum, and Scarpa fascia of the anterior abdominal wall. In view of the typical foul odour associated with this condition, a major role for anaerobic bacteria is likely.

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Causative Organisms

The bacterial flora includes gram-positive, gram-negative, and anaerobic bacteria. *Escherichia coli, Bacteroides* spp., *S. pyogenes,* and *S. aureus* are common etiologic pathogens (Miller, 1983). Wound cultures generally yield multiple organisms, implicating anaerobic-aerobic synergy (Meleney, 1993). Mixed cultures containing facultative organisms *(E. coli*, *Klebsiella,* enterococci) along with anaerobes (*Bacteroides, Fusobacterium, Clostridium,* microaerophilic streptococci) have been obtained from the lesions. However, group A streptococcal necrotizing fasciitis can occur in healthy immunocompetent individuals.

Fascial Planes

The five fascial planes that can be affected are:

- **Colle's Fascia:** It prevents the spread of infection in a posterior or lateral direction but provides no resistance to spread in an anterosuperior direction towards the abdominal wall. Posterior spread to the anal region is limited by the termination of Colles' fascia in the posterior edge of the perineal membrane.
- **Dartos Fascia:** It is the continuation of Colles' fascia over the scrotum and penis.

- **Buck's Fascia:** It lies deep to the dartos fascia, covering the penile corpora.
- **Scarpa's Fascia and Camper's Fascia:** These are superficial and deep fascial layer of the anterior abdominal wall which continuous as Colles' fascia inferomedially.

Pathogenesis: FG is characterized by polymicrobial infection with subsequent vascular thrombosis and tissue necrosis, aggravated by poor host defense due to one or more underlying systemic disorders (Yaghan, 2000; Rotstein, 1985). Aerobic organisms causes intravascular coagulation by inducing platelet aggregation and complement fixation. Hypoxic tissue leads to the formation of oxygen free radicals which causes membrane disruption, decreased ATP production, and DNA damage, which leads to decreased protein production. Anaerobic organisms secrete various enzymes and toxins such as Lecithinase, collagenase, and hyaluronidase cause digestion of the fascial planes and causes gas formation 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 release of pro-inflammatory cytokines and eventual development of septic shock.

Clinical presentation*:* The hallmark of Fournier Gangrene is a rapid progression from signs and symptoms of cellulitis to blister formation to clinically visible ischemia and ultimately to foul smelling necrotic lesions (Paty, 1992). Patients frequently have a history of recent perineal trauma, instrumentation, urethral stricture associated with sexually transmitted disease, or urethral cutaneous fistula. Pain, rectal bleeding, and a history of anal fissures suggest a rectal source of infection. Dermal sources are suggested by history of acute and chronic infections of the scrotum and spreading recurrent hidradenitis suppurativa or balanitis. The infection commonly starts as cellulitis adjacent to the portal of entry. Initially the involved area is swollen, erythematous, and tender as the infection begins to involve the deep fascia. Pain is prominent, and fever and systemic toxicity are marked. The swelling and crepitus of the scrotum quickly increase and dark purple areas develop and progress to extensive gangrene (Laucks, 1994). If the abdominal wall becomes involved in an obese patient with diabetes, the process can spread very rapidly. Specific genitourinary symptoms associated with the condition include dysuria, urethral discharge, and obstructed voiding. Alterations in mental status, tachypnea, tachycardia, and temperature greater than 38.3°C (101°F) or less than 35.6°C (96°F) suggest gram-negative sepsis (Rajan, 1998).

Laboratory findings and Radiologic findings: Anemia occurs secondary to a decreased functioning erythrocyte mass caused by thrombosis and ecchymosis coupled with decreased production secondary to sepsis. Elevated serum creatinine levels, hyponatremia, and hypocalcemia are common. Hypocalcemia is believed to be secondary to bacterial lipases that destroy triglycerides and release free fatty acids that chelate calcium in its ionized form (Ochiai *et al.,* 2001). Because crepitus is often an early finding, a plain film of the abdomen may be helpful in identifying air. Scrotal ultrasonography is also useful in this regard. Biopsy of the base of an ulcer is characterized by superficially intact epidermis, dermal necrosis, and vascular thrombosis and polymorphonuclear leukocyte invasion with subcutaneous tissue necrosis.

Management: Prompt diagnosis is critical because of the rapidity with which the process can progress. The clinical differentiation of necrotizing fasciitis from cellulitis may be difficult because the initial signs including pain, edema, and erythema are not distinctive. However, the presence of marked systemic toxicity out of proportion to the local finding should alert the clinician. Intravenous hydration and antimicrobial therapy are indicated in preparation for surgical debridement. Antimicrobial regimens include broad-spectrum antibiotics (β-lactam plus β-lactamase inhibitor) such as piperacillin-tazobactam, especially if *Pseudomonas* is suspected, ampicillin plus sulbactam or vancomycin or carbapenems plus clindamycin or metronidazole. Immediate debridement is essential. In the patient in whom diagnosis is clearly suspected on clinical grounds (deep pain with patchy areas of surface hypoesthesia or crepitation, or bullae and skin necrosis), direct operative intervention is indicated (Thwaini *et al.,* 2006). Extensive incision should be made through the skin and subcutaneous tissues, going beyond the areas of involvement until normal fascia is found. Necrotic fat and fascia should be excised, and the wound should be left open. These patients will often require a second look operation after 24 to 48 hours to exclude further disease progression (Gurdal *et al.,* 2003). Orchiectomy is almost never required, because the testes have their own blood supply independent of the compromised fascial and cutaneous circulation to the scrotum. Suprapubic diversion should be performed in cases in which urethral trauma or extravasation is suspected. Colostomy should be performed if there is colonic or rectal perforation. Once wound healing is complete, reconstruction (e.g. using myocutaneous flaps) improves cosmetic results. The indications for adjunctive hyperbaric oxygen therapy in FG remain controversial, although several groups have reported favourable results. There may also be potential benefit to the use of vacuum-assisted closure devices in FG.

Outcome: The mortality rate averages approximately 20%. Infection may spread along fascial planes and hence the exterior skin findings may represent only a small proportion of the underlying infected and necrotic tissue. The progression of infection from genitalia to perineum to abdominal wall may occur extremely rapidly (often within hours). Spread of tissue infection is accompanied by an ever-increasing risk of bacterial septicaemia, usually the eventual cause of death (Dahm *et al.,* 2000). Higher mortality rates are found in diabetics, alcoholics, and those with colorectal sources of infection who often have a less typical presentation, greater delay in diagnosis, and more widespread extension.

Conclusion

Regardless of the presentation, Fournier gangrene is a true surgical emergency that demands early recognition, aggressive treatment with antimicrobial agents, and surgical debridement to reduce morbidity and mortality.

Case 1: A 43 years old male patient, known case of Diabetes mellitus and Alcoholic liver disease with cirrhosis presented with a painful swelling of scrotum and with high grade fever for last 5 days. He was alert, conscious and his vitals were stable. On local examination, the scrotum was swollen with redness of skin and a large ischemic and gangrenous ulcerated area 12x10 cms size was seen with foul smelling discharge (Figure 1). Laboratory findings showed Hb 7.9, TLC 16300 with neutrophilia 85%. His RBS was 268 mg% and serum bilirubin 3.4 mg%. Other investigations were within normal limits. He was immediately taken up for surgery after initial stabilization and broad spectrum antibiotics. Extensive wound excision and wound debridement was done under GA. Almost entire dead skin of scrotum had to be sacrificed. (Figuyre2) The wound was thoroughly cleaned with hydrogen peroxide and betadine solution and antiseptic dressing was done. Figure 3 and 4 showing condition after 2nd and 3rd debridement.

Case 2: A 64 years old male patient with diabetes mellitus and right sided hemiparesis who was admitted in ICU developed a painful scrotal swelling with fever and call to surgery department was sent.

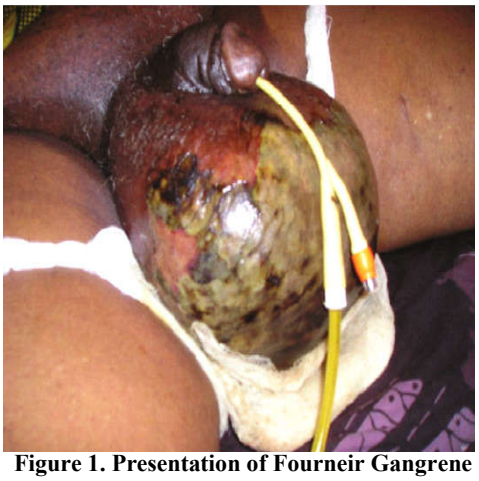

Figure 1. Presentation of Fourneir Gangrene

Figure 2. After extensive debridement

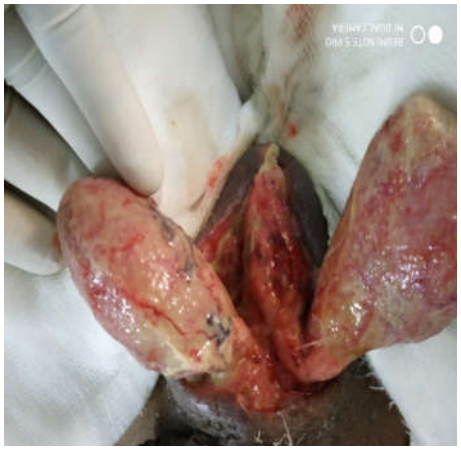

Figure 3. After second debridement

Figure 4. After third debridement

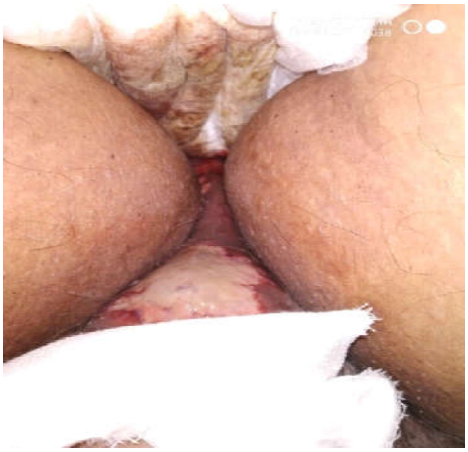

Figure 5. Presentation of FG

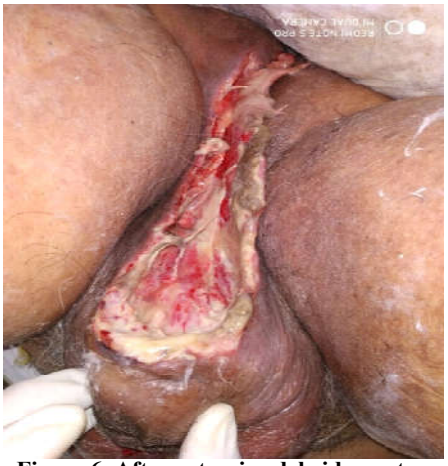

Figure 6. After extensive debridement

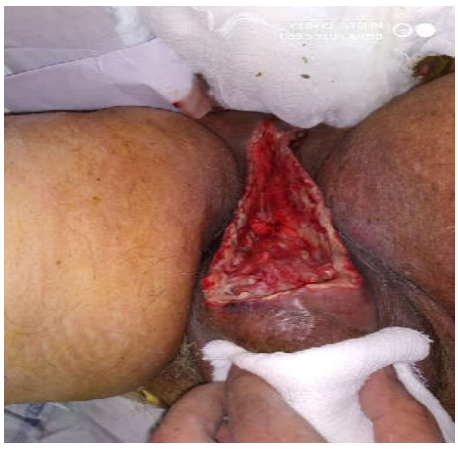

Figure 7. After 2nd debridement)

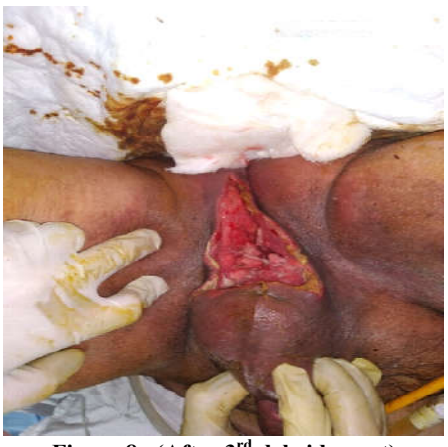

Figure 8. (After 3rd debridement)

On attending the patient and local examination, a painful scrotal swelling with sodden skin over scrotum, penis and perineum was seen with discharge and foul smell (Figure 5) Lab findings-TLC 18800 (Neutrophils-88%), Hb 10.8g/dl, RFT/LFT –N, RBS- 220. The patient was taken up for surgery. Wide local excision of all the dead tissue was done. (Figure 6) About 150 ml of foul smelling pus was drained. Serial wound debridement were done and broad spectrum antibiotics were given. Figure 7 and 8 showing condition after 2nd and 3rd debridement.

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