



FOURNIERS GANGRENE AND ITS EMERGENCY MANAGEMENT - A STUDY.

General Surgery

**Dr.Md .farhan
Ahmad Neyazi**

senior resident ,dept of surgery,JLNMCH,Bhagalpur,Bihar.

**Dr.Mithilesh
kumar***

3rd yr P.G,(Junior resident)dept of surgery ,JLNMCH,Bhagalpur,Bihar. *Corresponding Author

ABSTRACT

Fournier's gangrene (FG) is a life threatening disease. Although originally thought to be an idiopathic process, FG has been shown to have a predilection for patients with diabetes as well as long term alcohol misuse; however, it can also affect patients with non obvious immune compromise. The nidus is usually located in the genitourinary tract, lower gastrointestinal tract, or in skin. Fournier gangrene is a mixed infection caused by both aerobic and anaerobic bacterial flora. The development and progression of the gangrene is often fulminating and can rapidly cause multiple organ failure and death. Because of potential complications, it is important to diagnose the disease process as early as possible. Although antibiotics and aggressive debridement have been broadly accepted as the standard treatment.

KEYWORDS

INTRODUCTION

Fournier gangrene is a fulminant form of ineffective necrotising fasciitis of the perineal, genital, perianal region which commonly affects men, even though this clinical entity is eponymously credited to the Parisian venerologist Jean Alfred Fournier, who described it as fulminant gangrene of penis and scrotum in young male. Baurienne in 1764 and Avicenna in 1877 had described same disease earlier. Over the years, many terms have been used to describe this clinical condition including idiopathic gangrene of sacrotum. Early surgical debridement of necrotic tissue and antibiotics are fundamental in the treatment of FG. Despite advanced management mortality is average 20-50%.

AETIOLOGY OF F.G – Initially, FG was an idiopathic entity, but source of infection in the majority of cases as either perineal and genital skin infection.

Anorectal or urogenital and perineal trauma or pelvic intervention are other causes of FG. The most common foci include the gastrointestinal tract (20-50%), followed by genitourinary tract (20-40%) and cutaneous injury (20%).

Box 1 lists the commonest causes.

Comorbid systemic disorders are being identified more and more in patients with FG. The commonest being diabetes mellitus and alcohol misuse. Diabetes mellitus is reported to be present in 20-60% of patients with FG and chronic alcoholism in 20-40% of patients (Box 2). The HIV population at risk for developing FG.¹⁰

PATHOGENESIS

In FG, suppurative bacterial infection results in microthrombosis of the small subcutaneous vessels leading to the development of gangrene of the overlying skin.¹¹ Culture from the wounds commonly show polymicrobial infections: aerobes and anaerobes, which include coliforms, *Klebsiella*, *Streptococci*, *Staphylococci*, *Clostridia*, *Bacteroids*, and *Corynebacteria*. On an average, at least three organisms are cultured from each diagnosed patient.¹² Most of the normal commensals in the perineum and genitalia, which, because of the impaired host cellular immunity, become virulent and act synergistically to invade tissue and cause extensive damage.¹³ Even though *E. coli* has been reported to be the commonest organism isolated from the wound, it could be because of the commensal nature of these organisms in the perineal region. Anaerobes are less frequently isolated than expected, which could be because of technical faults.² The impaired defence mechanisms in the host help the infection to proceed unchecked, and at alarming speed, along the fascial planes. The synergistic activity of aerobes and anaerobes leads to the production of various exotoxins and enzymes like collagenase, heparinase, hyaluronidase, streptokinase, and streptodornase, which aid in tissue destruction and spread of infection. The platelet aggregation and complement fixation induced by the aerobes and the heparinase and collagenase produced by the anaerobes lead to microvascular

thrombosis and dermal necrosis.

CLINICAL PRESENTATION

BOX 1 aetiology of F.G

Urogenital	Urethral stricture, prostatic biopsy	Indwelling catheter, urethral calculi etc.
ANORECTAL	Perineal abscess, Rectal biopsy,	Anal dilatation, Haemorrhoidectomy, Appendicitis, Diverticulitis.
Gynaecological	Infected Bartholin's Gland, Episiotomy wound	Septic abortion Coital injury
		Genital mutilation.

FG shows vast heterogeneity in clinical presentation, from insidious onset and slow progression to rapid onset and fulminant course, the latter being the more common presentation. In contrast with Fournier's initial description, the disease tends to present more in elderly men,¹⁶ and also has been reported in women and children.¹⁸ The infection commonly starts as a cellulitis adjacent to the portal of entry, depending on the source of infection, commonly in the perineum or perianal region. The local signs and symptoms are usually dramatic with significant pain and swelling. The patient also has pronounced systemic signs; usually out of proportion to the local extent of the disease. Crepitus of the inflamed tissues is a common feature because of the presence of gas-forming organisms.¹⁸ As the subcutaneous inflammation worsens, necrotic patches start appearing over the overlying skin and progress to extensive necrosis.¹⁹ Unless aggressively treated, the patient can rapidly progress to sepsis with multiple organ failure, the common cause of death in these patients.²⁰ The spread of infection along the fascial planes and is usually limited by the attachment of the Colles' fascia in the perineum. Infection can spread to involve the scrotum, penis and can spread up the anterior abdominal wall, up to the clavicle.²¹ The testes are usually spared as their blood supply originates intra-abdominally. Involvement of the testis suggests retroperitoneal origin or spread of infection.²² Urogenital infections travel posteriorly along the Buck's and Dartos fascia to involve the Colles' fascia, but are limited from the anal margin by the attachment of the Colles' fascia to the perineal body. In contrast, anorectal sources of infection usually start perianally and this variation in initial clinical presentation can serve as a guide to localizing the foci of infection.¹

INVESTIGATIONS

Laor *et al* developed a scoring system (Fournier's gangrene severity index), to quantify the severity of infection, using common vital signs and laboratory data¹⁶ (Box 3). This score helps to prognosticate the illness and helps to predict the mortality. With a score of over 9, they found a 75% probability of death while a score of less than 9 was associated with 78% probability of survival. Chawla *et al* used this scoring system in their series of 19 patients and found that Fournier's

gangrene severity index was useful in predicting survival but not length of hospital stay.²³

Even though the diagnosis of FG is primarily clinical, imaging modalities may be useful in those cases where the presentation is atypical or when there is concern regarding the true extent of the disease. Plain radiography may show air within the tissues. Ultrasonography is useful to differentiate intrascrotal abnormality and can also show thickened and swollen scrotal wall, with gas within.²⁴ Computed tomography and magnetic resonance imaging are useful in select cases to diagnose or rule out retroperitoneal or intraabdominal disease process.²⁵ Box 4 summarises the differential diagnosis for FG.

TREATMENT

FG warrants an aggressive multimodal approach, which includes haemodynamic stabilisation, broad spectrum anti-biotics, and surgical debridement. It must be highlighted however, that early surgical debridement is the primary component of treatment and if delayed will have a negative impact on the prognosis.²⁶ All non-viable and necrotic tissue must be excised, until well perfused viable tissue is reached (figs 1 and 2). The full extent of the disease may not be apparent from the areas of cutaneous involvement, which is usually less than the subcutaneous disease. Care must be taken not to accidentally open up deeper facial planes, which were not initially involved. Urinary or faecal diversion may be necessary depending upon the foci of origin of the disease.¹⁶ Multiple surgical debridement is the rule rather than the exception, with an average of 3.5 procedures required per patient.²⁷ Even though testes are classically spared in the process of FG, orchidectomy, for non-viable testis, is eventually required in upto 21% patients.²⁷



Figure 1A case of Fournier's gangrene in a 45-year old man after live unrelated kidney transplant (transplanted kidney can be seen through the necrotic tissue).

Box 3 Variables in Fournier's gangrene severity index
Temperature
Heart rate
Respiration rate
Serum sodium
Serum potassium
Serum creatinine
Packed cell volume (%)
Whole blood cell count
Serum bicarbonate

Box 4 Differential diagnosis of Fournier's gangrene

Cellulitis
Strangulated hernia
Scrotal abscess
Streptococcal necrotising fascitis
Vascular occlusion syndromes
Herpes simplex
Gonococcal balanitis and oedema
Pyoderma gangrenosum
Allergic vasculitis
Polyarteritis nodosa
Necrolytic migratory erythema
Warfarin necrosis
Ecthyma gangrenosum

Various workers have used different techniques to provide skin cover including transplantation of testes, free skin grafts, axial groin flaps, and myocutaneous flaps. Split thickness skin graft seems to be the

treatment of choice in treating perineal and scrotal skin defects. Parkash *et al* reported their series of treatment of 43 cases in the past 11 years. In three cases the gangrene had spread beyond the scrotum and penis and cover had to be supplemented with split-skin grafts. In all the other cases, cover was provided with scrotal skin remnants at the edge of the lesion and on the penis with the inner layer of the prepuce, which had remained intact.²⁸ On the other hand Black P C *et al* reported their series of Meshed unexpanded split-thickness skin grafting (STSGs) for skin defects. They treated nine patients with penile skin loss between March 2001 and January 2003, with meshed STSGs to the penis.

The underlying condition was FG in four cases, chronic lymphodema in two, skin deficiency from previous surgeries in two, and Crohn's disease in one. Graft thickness was 0.012 or 0.016 inches and meshing was performed in a 1:1 ratio. Meshed slits were oriented transversely without expansion and the graft juncture was located on the ventral surface in zigzag fashion. Graft take, appearance, and sexual and voiding function were assessed postoperatively. All nine patients had 100% graft take. At a mean follow up of six months a satisfactory cosmetic outcome was reported photographically in all except one case involving chronic penile manipulation. Erectile function and ejaculation were preserved in potent patients.²⁹ With the recent advent of the vacuum assisted closure (VAC) system dressing, there seems to be a dramatic improvement with minimizing skin defects and speeding tissue healing. It simply works by exposing a wound to subatmospheric pressure for an extended period to promote debridement and healing.



Figure 2 The same case as in figure 1, after the first debridement: extensive tissue debridement is the rule.

Weinfeld *et al* treated four consecutive cases using negative pressure dressings (VAC) to bolster skin grafts in male genital reconstruction. In this series reconstruction followed one case of tumour ablation and three cases of debridement of abscesses or FG. The VAC was applied circumferentially to secure skin grafts either directly to the penis shaft or to facilitate skin grafting to the sacrotum. Graft area ranged from 75 cm to 250 cm. All cases resulted in successful genital wound coverage, minor complications occurred.

Antibiotic therapy should be broad spectrum to empirically cover all possible organisms. The usual combination includes amoxycillin for the streptococcal species, third generation cephalosporin, with or without an aminoglycoside, for the Gram negative organisms, plus metronidazole for the anaerobes.²⁷ Some topical agents like Dakin's solution (sodium hypochlorite), hydrogen peroxide, or unprocessed honey has been tried to aid in the separation of the slough and accelerate granulation tissue.³² If the initial tissue stain using potassium hydroxide shows the presence of a fungus or if grown in the culture, then addition of amphotericin B is necessary.

Hyperbaric oxygen is widely believed to be an effective adjunctive therapy in the treatment of FG, even though there is no conclusive evidence regarding its effectiveness. Putative benefits of hyperbaric oxygen therapy include neutralisation of anaerobic organisms, improvement in neutrophil function, increased fibroblast proliferation, and angiogenesis.³³

Recent advances in wound healing such as the application of growth hormones, trophic agents, and the use of vacuum dressing (fig 2) to hasten the wound closure has been used successfully.

OUTCOME

Early series reported high mortality rates around 80%,³⁴ but more recent studies show an improvement with lower rates of generally less than 40%.³⁵ Despite better understanding of the aetiopathogenesis of the

disease, the availability of more broader spectrum antimicrobials, and the trend towards early and timely surgical intervention, the continuing high mortality rate reflects the potentially devastating nature of this disease. Factors that have been identified to negatively affect survival include age, primary anaerobic source of infection, delay in treatment, and immunocompromised state.³⁵ There is as yet inconclusive evidence to suggest that diabetes³⁶ or number of surgical debridements adversely affects the prognosis.

Long term complications, for those who survive this life threatening condition are not uncommon. Long term pain is not uncommon after FG. Only 50% of the patients are expected to be free of pain. The sexual function may be impaired by penile deviation or penile torsion as well as by a loss of sensitivity of the penile skin or pain during erection. Some patients may suffer from temporary stool incontinence. However, despite major complaints because of extensive scarring, most patients considered their cosmetic result as well as their quality of life to be satisfactory.³⁷

CONCLUSION

FG is still a life threatening condition with unacceptably high death rates despite insights gained regarding the disease process. Diagnosis should be prompt with early surgical intervention, along with antibiotics and good supportive care. Radiography can be helpful when the clinical picture is not straightforward. Continued medical care in the form of a multidisciplinary approach is necessary as these patients may require reconstructive procedures in the future. Proactive management of the diabetic and immunosuppressed patients with perineal infections is of extreme importance to prevent the development of the condition in the first instance as this condition in the presence of such comorbidities is associated with high mortality.

REFERENCES

- Smith GL, Bunker CB, Dineen MD. Fournier's gangrene. *Br J Urol* 1998;81:347-55.
- Fournier J-A. Gangrene foudroyante de la verge. *Semaine Medicale* 1883;3:345-8.
- Nathan B. Fournier's gangrene: a historical vignette. (Letter). *Can J Surg* 1998;41:72.
- Gray JA. Gangrene of the genitalia as seen in advanced periurethral extravasation with phlegmon. *J Urol* 1960;84:740-5.
- Meleney FL. Hemolytic streptococcus gangrene. *Arch Surg* 1924;9:317-64.
- Pawlowski W, Wronski M, Krasnodebski IW. Fournier's gangrene. *Pol Merkuriusz Lek* 2004;17:85-7.
- Eke N. Fournier's gangrene: a review of 1726 cases. *Br J Surg* 2000;87:718-28.
- Morpurgo E, Galandiuk S. Fournier's gangrene. *Surg Clin N Am* 2002;82:1213-24.
- Clayton MD, Fowler JE Jr, Sharifi R, et al. Causes, presentation and survival of fifty-seven patients with necrotizing fasciitis of the male genitalia. *Surg Gynecol Obstet* 1990;170:49-55.
- Elem B, Ranjan P. Impact of immunodeficiency virus (HIV) on Fournier's gangrene: observations in Zambia. *Ann R Coll Surg Engl* 1995;77:283-6.
- Johnin K, Nakatoh M, Kadowaki T, et al. Fournier's gangrene caused by Candida species as the primary organism. *Urology* 2000;56:153.
- Yaghan RJ, Al-Jaberi TM, Bani-Hanil. Fournier's gangrene: changing face of the disease. *Dis Colon Rectum* 2000;43:1300-8.
- Rotstein OD, Pruett TL, Simmons RL. Mechanisms of microbial synergistic polymicrobial surgical infections. *Rev Infect Dis* 1985;7:151-70.
- Rutichik S, Sanders M. Fungal Fournier gangrene. *Infect Urol* 2003;16:54-6.
- Tleyeh IM, Routh J, Qutub MO, et al. Lactobacillus gassericus causing Fournier's gangrene. *Scand J Infect Dis* 2004;36:501-3.
- Laor E, Palmer TS, Tolia BM, et al. Outcome prediction in patients with Fournier's gangrene. *J Urol* 1995;154:89-92.
- Adams JR Jr, Mata JA, Bocchini JA, et al. Fournier's gangrene in children. *Urology* 1990;35:439-41.
- Patty R, Smith AD. Gangrene and Fournier's gangrene. *Urol Clin North Am* 1992;19:149.
- Laucks LS. Fournier's gangrene. *Surg Clin North Am* 1994;74:1339-52.
- Sutherland ME, Meyer AA. Necrotizing soft-tissue infections. *Surg Clin North Am* 1994;74:591-607.
- Saijo S, Kuramoto Y, Yoshinari M, et al. Extremely extended Fournier's gangrene. *Dermatologica* 1990;181:228-32.
- Gerber GS, Guss SP, Pilet RW. Fournier's gangrene secondary to intra-abdominal processes. *Urology* 1994;44:779-82.
- Chawla SN, Gallop C, Mydlo JH. Fournier's gangrene: an analysis of repeated surgical debridement. *Eur Urol* 2003;43:572-5.
- Kane CJ, Nash P, McAninch JW. Ultrasonographic appearance of necrotizing gangrene: aid in early diagnosis. *Urology* 1996;48:142-4.
- Amendola MA, Casillas J, Joseph R, et al. Fournier's gangrene: CT findings. *Abdom Imaging* 1994;19:471-4.
- Elliot D, Kufera JA, Myers RA. The microbiology of necrotizing soft tissue infections. *Am J Surg* 2000;179:361-6.
- Benizri E, Fabiani P, Migliori G, et al. Gangrene of the perineum. *Urology* 1996;47:935-9.
- Parkash S, Gajendran V. Surgical reconstruction of the sequelae of penile and scrotal gangrene: a plea for simplicity. *Br J Plast Surg* 1984;37:354-7.
- Black PC, Friedrich JB, Engrav LH, et al. Meshed unexpanded split-thickness skin grafting for reconstruction of penile skin loss. *J Urol* 2004;172:976-9.
- Morykwas MJ, Argenta LC, Shelton-Brown EI, et al. Vacuum-assisted closure: a new method for wound control and treatment: animal studies and basic foundation. *Ann Plast Surg* 1997;38:553-62.
- Weinfeld AB, Kelley P, Yuksel E, et al. Circumferential negative-pressure dressing (VAC) to bolster skin grafts in the reconstruction of the penile shaft and scrotum. *Ann Plast Surg* 2005;54:178-83.
- Hejase MJ, Simonin JE, Bihle R, et al. Genital Fournier's gangrene: experience with 38 patients. *Urology* 1996;47:734-9.
- Capelli-Schellpfeffer M, Gerber GS. The use of hyperbaric oxygen in urology. *J Urol* 1999;162:647-54.
- Stephens BJ, Lathrop JC, Rice WT, et al. Fournier's gangrene: historic (1764-

- 1978) versus contemporary (1979-1988) differences in aetiology and clinical importance. *Am Surg* 1993;59:149-54.
- Martinelli G, Alessandrino EP, Bernasconi P, et al. Fournier's gangrene: a clinical presentation of necrotizing fasciitis after bone marrow transplantation. *Bone Marrow Transplant* 1998;22:1023-6.
- Nisbet AA, Thompson IM. Impact of diabetes mellitus on the presentation and outcomes of Fournier's gangrene. *Urology* 2002;60:775-9.
- Theiss M, Hofmockel G, Eckert P, et al. Cosmetic and functional long-term outcome after operation of Fournier gangrene. *Urologe A* 1996;35:338-41.