

# Fournier's gangrene

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## Introduction

Fournier's gangrene (FG) is a rare but fulminant form of infective necrotising fasciitis affecting the perineum and external genitalia, which can rapidly progress along fascial planes. It most commonly affects men, but can occur in women and children, with a male to female ratio of 5:1 [1].

### Box 1: Aetiology of Fournier's gangrene

#### Urogenital

- Urethral stricture +/- urine extravasation
- Scrotal abscess
- Epididymo-orchitis
- Indwelling catheter
- Traumatic catheterisation
- Hydrocele surgery / aspiration
- Vasectomy
- Circumcision
- Prostate biopsy
- Insertion of a penile prosthesis
- Insertion of a TVT
- Perineal trauma
- Genital piercing
- Delayed rupture of ileal neobladder

#### Anorectal

- Perianal / ischiorectal abscess
- Rectal biopsy
- Anal dilatation
- Haemorrhoidectomy
- Rectosigmoid malignancy
- Appendicitis
- Diverticulitis
- Strangulated inguinal hernias
- Steroid enemas

#### Gynaecological

- Infected Bartholin's gland
- Vulval abscess
- Septic abortion
- Episiotomy wound
- Coital injury
- Genital mutilation

The French venereologist, Jean-Alfred Fournier gave the first clinical description of an idiopathic and rapidly progressive necrotising gangrene of the penis and scrotum, in an otherwise healthy young man in 1883 [2]. Although the condition is eponymously ascribed to him, Baurienne and Avicenna had described the same disease process earlier, in 1764 and 1877 respectively [3].

Furthermore, contrary to Fournier's original description, subsequent experience has shown that rather than being idiopathic, in most cases FG has an identifiable cause (Box 1) and is commonly associated with several risk factors and / or co-morbidities (Box 2). Despite medical advances, FG continues to be associated with significant morbidity and mortality.

## Aetiology and pathogenesis

FG has an identifiable cause in approximately 95% of cases [4,5]. Commonly, the necrotising process originates from an infection in the anorectum, urogenital tract or genital skin [6]. Trauma, recent surgery, urethral instrumentation, intramuscular

injections, steroid enemas and the presence of foreign bodies have all been reported as causative factors [4]. In children, strangulated inguinal hernias, circumcision, omphalitis, insect bites, and burns have also been linked to the development of FG. Poor perineal hygiene and any condition associated with immunosuppression may also predispose a patient to the development of FG.

Wound and / or tissue cultures from patients with FG typically demonstrate a polymicrobial infection of both aerobic and anaerobic organisms, with an average of four isolates per case [4]. Most of these are normal commensals in the perineum and genital areas, which opportunistically and synergistically become virulent and extremely destructive in patients with compromised immunity.

*E coli* is the predominant causative aerobic, and *Bacteriodes* species, the predominant anaerobe. Other commonly associated organisms include *Proteus*, *Staphylococcus*, *Enterococcus*, *Streptococcus*, *Pseudomonas*, *Klebsiella*, and *Clostridium*. The production of various endotoxins and enzymes including collagenase, heparinase and streptokinase by these organisms plays an important role in the pathological process; with platelet aggregation and complement reaction induced by aerobes, and heparinase and collagenase production by anaerobes, leading to microvascular thrombosis and skin necrosis. In addition, phagocytic activity in the necrotised tissues is impaired which further promotes spread of the infection [8]. Rates of fascial destruction as high as 2-3cm / hour have been reported [5,9,10].

## Relevant anatomy

Understanding the anatomical relationship of the fascial layers, skin and subcutaneous structures in the perineum and abdominal wall, as well as the vascular supply in these regions, is key for any clinician treating the condition, not least because it has important implications for both the initial debridement, and subsequent reconstructive surgery.

The spread of infection is along

### Box 2: Comorbid risk factors for the development of Fournier's gangrene

- Diabetes (present in up to 60% of cases [4])
- Chronic alcoholism (present in 25-50% of patients [7])
- Extremes of age
- Malignancy
- Chronic steroid use
- Cytotoxic chemotherapy
- Radiation therapy
- Lymphoproliferative diseases
- Malnutrition
- Morbid obesity
- HIV infection
- Chronic liver and kidney disease
- Crohn's disease
- Suprapubic catheter

the fascial planes, and is usually limited by the attachment of Colles' fascia to the perineal body caudally and the clavicles cranially. For this reason, infection originating in the perineum or rectum can spread to involve the scrotum, penis and anterior abdominal wall right up to the level of the clavicles. However, whilst the superficial and deep fascia, and the skin (to varying degrees), are destroyed, the deeper muscular structures, corpora, urethra and cord structures are not usually involved unless they are the origin of infection. The testes are also usually spared as their blood supply originates intra-abdominally from the aorta. If the testes are involved it is usually a sign that the infection has originated or spread into the retroperitoneum. Involvement of the anal margin is also a key feature to assess on examination, in order to help determine the origin of infection (if it is not already clear from the history); anorectal sources of infection will usually start perianally and spread outwards, whereas urogenital infections tend to travel caudally along Bucks and Dartos fascia to involve Colles' fascia, but will not normally spread to involve the anal margin because of restrictions posed by the attachment of Colles' fascia to the perineal body.

The blood supply to the anterior abdominal wall and scrotal wall are also relevant; the inferior aspect of the anterior abdominal wall is supplied by branches from the inferior epigastric and deep circumflex iliac arteries, whereas branches of the external and internal pudendal arteries supply the scrotal wall. The internal pudendal artery (which supplies the posterior aspect of the scrotal wall) is the only one of these vessels which doesn't travel within Camper's fascia. Therefore, whilst all of the other vessels may become thrombosed in the pathogenesis of FG, thereby jeopardising the viability of the skin that they supply in the anterior scrotum and perineum, sparing of the internal pudendal artery often means that blood supply to the posterior aspect of the scrotal wall is preserved. This viable skin may then be used later in any reconstructive surgery.

## Presentation

The clinical presentation of FG shows considerable heterogeneity. In some

cases the onset and progression of disease will be insidious, starting with pruritis and discomfort in the external genitalia. In others, disease onset will be rapid with an immediately fulminant course. A high index of suspicion, particularly in those with relevant risk factors and co-morbidities is therefore needed if the condition is to be recognised and treated early.

Commonly, the infection will start as an area of cellulitis adjacent to the portal of entry of the infecting organisms. There may be significant swelling, but often with marked systemic signs and pain out of proportion to the physical findings. Crepitus of the inflamed tissues may be noted because of the presence of gas forming organisms [11]. A distinctive odour exhibited by the infected tissues and bronzing of the tissues are further characteristic features. As the condition and degree of inflammation and microvascular thrombosis within the subcutaneous tissues progresses, necrotic patches start appearing in the overlying skin, leading to extensive necrosis if untreated [12].

## Investigations

FG is a urological emergency, and primarily a clinical diagnosis based on a thorough history and careful examination. Early diagnosis and treatment is key, and should not be delayed in the pursuit of obtaining further investigation results. Failing to treat the patient aggressively can rapidly lead to the development of sepsis with multi-organ failure – the main cause of death in these patients [13]. However, in cases where the presentation is atypical or where there is concern regarding the true extent of the disease, imaging modalities may have a role. Plain x-ray may show gas within the tissues whilst ultrasound may be useful to differentiate intrascrotal abnormality and might show a thickened gas-containing scrotal wall [14]. CT and MRI are useful in select cases to diagnose or exclude retroperitoneal or intra-abdominal pathology [15].

Other investigations are essential to identify co-morbid as well as causative factors. These include urinalysis, blood sugar measurements, appropriate bacterial evaluation of pus and / or tissue samples, full blood count, and renal and liver function

blood tests.

The eventual need for further investigations such as cystoscopy, colonoscopy and biopsies may also arise in order to identify the source of sepsis.

## Treatment and management

FG warrants an early, aggressive, multi-modal approach, which includes resuscitation and supportive care, broad-spectrum antibiotics, and early surgical debridement. One can argue, that of all these treatments, it is the latter which is most important; as if surgery is delayed there will be a negative impact on prognosis [16]. All non-viable, suspicious and necrotic tissue must be excised down to well-perfused bleeding tissue (see Figure 1 and 2). The areas of apparent cutaneous involvement data may be misleading in terms of the true extent of the subcutaneous disease below. Care must be taken not to inadvertently open up the deeper fascial planes, which can allow the infection to spread into fascial layers that were initially spared.

Initial antibiotic therapy should be broad spectrum to empirically cover all possible organisms but may then be rationalised once culture results and sensitivities are available. The usual combination includes a penicillin for the streptococcal species, a third generation cephalosporin, with or without an aminoglycoside for the gram-negative organisms, plus metronidazole for the anaerobes.

Urinary and / or faecal diversion may also be required depending on the original focus of the disease [11]. Re-look surgery is critical, and on average, 3.5 procedures are required per patient [17].

There is evidence to suggest the use of vacuum assisted closure (VAC) dressings may dramatically improve the speed of tissue healing and can minimise the size of residual skin defects [19]. Hyperbaric oxygen is also widely believed to be an effective adjunctive therapy in the treatment of FG. It is thought to neutralise anaerobic organisms and promote neutrophil function, fibroblast proliferation and angiogenesis [19]. However, there is little conclusive evidence regarding its effectiveness [20].

Depending on the extent of the skin defect after debridement and



Figure 1



Figure 2

**Table 1: Summary of the major studies reporting on Fournier's Gangrene**

Study (period studied)	Country	No. of cases	Mortality rate (%)
Proud et al. (2001-2010) [22]	Australia	219	15.98
Medina et al. (1975-2008) [29]	Spain	90	34.4
Lin et al. (1989-2011) [30]	Taiwan	85	18.8
Carvalho et al. (1996-2006) [31]	Brazil	80	13.5
Ghnnam et al. (2002-2007) [23]	Egypt	74	21.6
Corcoran et al. (1996-2006) [32]	USA	68	10
Garcia et al. (1998-2012) [33]	Spain	59	26
Benjelloun et al. (2003-2009) [24]	Morocco	50	24
Morua et al. (2004-2009) [25]	Mexico	50	12
Vargas et al. (2003-2008) [26]	Columbia	42	17
Altarac et al. (1995-2010) [34]	Croatia	41	36.6
Chen et al. (2000-2008) [27]	Taiwan	41	12
Sallami et al. (1995-2010) [35]	Tunisia	40	17.5
Vyas et al. (2011-2012) [28]	India	30	20
Yeniyol et al. (1995-2000) [36]	Turkey	25	24
Spirnak et al. (1973-1983) [5]	USA	20	45

subsequent healing, further surgical reconstruction may be required to provide skin cover once the infection has resolved. Several techniques can be employed, including split skin grafts and myocutaneous flaps. The testes may also require auto-transplantation (often into the inner thigh compartment).

### Prognosis

Early series reported high mortality rates with FG at around 80% [21]. More recent studies show an improvement with mortality rates generally less than 25% [22-28] (Table 1).

Factors that have been identified as negatively affecting survival include advancing age, primary anorectal source of infection, delay in treatment, extension of infection to the anterior

abdominal wall, renal failure on admission, occurrence of septic shock, the need for postoperative mechanical ventilation, and immunocompromised states [24,37].

Laor et al. [38] have developed a scoring system; the Fournier's Gangrene Severity Index (FGSI), which may be used to quantify the severity of infection and help prognosticate the illness and predict mortality, using commonly measured vital signs and laboratory data. In this scoring system (Box 3), a score >9 was associated with a 75% probability of death, while a score <9 was associated with a 78% probability of survival [38].

In patients who do survive FG, morbidity rates can be high; long-term complications of the disease are not

### Fournier's Gangrene Severity Index Score

The FGSI is based on deviation from reference ranges of the following clinical parameters:

- Temperature
- Heart rate
- Respiratory rate
- White blood cell count
- Haematocrit
- Serum sodium
- Serum potassium
- Serum creatinine
- Serum bicarbonate

Each parameter is assigned a score between 0 to 4, with increasing values indicating greater deviation from normal. The sum of all the parameter values is then used as the final FGSI score.

### TAKE HOME MESSAGE

- FG remains a life-threatening condition with high mortality rates.
- Early diagnosis and multimodal treatment with surgical debridement, broad-spectrum antibiotics and good supportive care remain fundamental in the management of affected patients.
- Proactive management of diabetic and immunocompromised patients with perineal infections is important to try and prevent development of FG.

uncommon and may include chronic pain, chronic cellulitis and oedema, and impaired sexual function.

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