A contemporary update on Fournier's gangrene

Judith C. Hagedorn and Hunter Wessells

Abstract | Despite advances in the evaluation, treatment, and pathophysiological understanding of necrotizing soft-tissue infections, Fournier's gangrene remains a life-threatening urological emergency. Although the condition can affect patients of any age and gender, it might be more prevalent in some high-risk groups with certain comorbidities. Several prognostic and diagnostic tools have been developed to assist with clinical decision-making once the diagnosis is made — primarily based on the physician's physical exam and potentially supported by laboratory and imaging findings. Expedited treatment with resuscitation, antibiotic administration, and rapid, wide surgical debridement are key elements of the initial management. These procedures must be followed by meticulous wound care and liberal use of planned subsequent surgical debridements. Once the patient has overcome the associated systemic illness, several reconstructive options for the genitalia and perineum can be considered to improve functionality and cosmesis.

Fournier's gangrene is a progressive necrotizing soft-tissue infection (NSTI) of the external genitalia and/or perineum. Although it was first described by Baurienne in 1764, it takes its name from the French venerologist, Jean-Alfred Fournier[1,2]. Fournier's gangrene is a urological emergency requiring prompt diagnosis and treatment — even with administration of parenteral broad-spectrum antibiotics and expedited aggressive surgical debridement, the disease can be fatal. The aetiology of Fournier's gangrene is most commonly polymicrobial and patients with comorbidities, such as diabetes mellitus or renal failure, are at an increased risk of developing the disease, and have a poor prognosis[3]. Prompt diagnosis and immediate treatment is paramount to decrease mortality. The mainstay of treatment for Fournier's gangrene is extensive surgical debridement of the affected tissue[4], but this approach can leave patients with disfiguring wounds that require reconstructive surgery once the patient has recovered from the infection. Several reconstructive surgical techniques have been described that share the goal of minimizing morbidity as well as restoring functionality and cosmesis.

In this Review, we address current concepts of Fournier's gangrene, initially focusing on the findings of the largest epidemiological studies, describing the characteristics of high-risk groups, and introducing the Fournier's gangrene severity index (FGSI) as a prognostic tool. We discuss the pathophysiology and microbiology of the disease and describe clinical tools for diagnosis. We consider the importance of the initial surgical management and definitive management with genital reconstruction.

Department of Urology, University of Washington, 325 9th Avenue, Seattle, Washington 98104, USA.

Correspondence to J.C.H.
judithch@u.washington.edu

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Epidemiology

Incidence and demographics

Fournier initially described his eponymous necrotizing skin infection in 1883 as an idiopathic process found only in young healthy men[1]. However, the epidemiology of Fournier's gangrene has changed considerably from its original description, and the condition is now recognized to occur at any age and in any gender, with several aetiological factors identified.

This sometimes devastating soft-tissue infection has been well documented in case reports and case series, but large epidemiological studies are lacking. To date, only two studies have included a large enough number of patients to assess the epidemiology of the disease with generalizable results. In 2000, Eke reviewed 1,726 patients and in 2009 Sorensen *et al.* studied a total of 1,680 patients[5,6]. Not only did they find that Fournier's gangrene can be observed in children and women and is not exclusively found in men, but they also demonstrated that a large number of cases also occur later in life than initially postulated. The majority of patients affected are between 50–79 years old, with an estimated male:female ratio of 10:1 (REFS 5–7). Sorensen *et al.*[6] reported the overall incidence of this relatively rare condition to be 1.6 in 100,000, comprising 0.02% of hospitalized patients, and identified a total of 1,641 male

Key points

- Fournier's gangrene is a rare but potentially fatal disease
- The Fournier's gangrene severity index (FGSI) has been developed as a prognostic indicator
- The infection is most commonly polymicrobial, but monomicrobial infections with resistant bacteria have also been described
- Clinical tools, including laboratory tests and imaging, exist to assist in the diagnosis of the disease in equivocal cases, but if clinical suspicion of Fournier's gangrene is high, surgical treatment should not be delayed for imaging studies to be carried out
- Fluid resuscitation, broad-spectrum antibiotic therapy, and prompt surgical debridement are key elements of the initial management of the disease
- Genital reconstruction techniques can help with optimal functional and cosmetic outcomes after extensive genital skin loss

patients and 39 female patients with the diagnosis of Fournier's gangrene. In addition 36 of the male patients were children between the ages of 0–9 years. Women with the disease were more acutely ill, frequently requiring mechanical ventilation and dialysis and had a higher fatality rate than the male patients (12.8% versus 7.5%).

Sorensen *et al.*[6] compared regional trends in the USA and demonstrated that the incidence was highest in the southern USA and lowest in the west and midwest states. Eke studied the international geographic distribution of Fournier's gangrene cases, demonstrating that the highest number of reported cases are found in the USA and Canada, followed by Africa and then Europe[5]. He could not identify ethnicity as being a risk factor for the development of the disease. Likewise, an epidemiological review of 171 patients in a national hospital in Nairobi, Kenya, did not identify a difference in susceptibility between ethnic groups[8].

Risk factors and predisposing conditions

Conditions predisposing to Fournier's gangrene include diabetes mellitus, obesity, chronic alcoholism, smoking, renal failure, liver failure, malignancy, and HIV infection[9]. All these conditions have impaired microcirculation and/or immunosuppression in common. In patients who present with Fournier's gangrene the rate of diabetes mellitus is estimated to be between 36–56%[6,9–11]. A study from Germany reported that 51.5% of patients with Fournier's gangrene were overweight with a BMI >25 and 39.4% had a BMI of ≥30 and higher[9]. Alcoholism is present in 25–50% of patients[9,12]. Additionally, Kuo *et al.* showed that liver cirrhosis leads to a high mortality in this patient population[13]. Low socioeconomic status has been postulated to be a possible predisposing factor[5,14], but whether low socioeconomic status is an independent risk factor or whether the association with alcoholism and obesity in this demographic predisposes to Fournier's gangrene is unclear.

As the source of the necrotizing fasciitis is the local skin, the colorectal region, and the urinary tract, Fournier's gangrene is more prevalent in patients with urological conditions and diseases of the perianal area: urethral strictures and indwelling catheters are risk factors, as are genital trauma and perianal abscesses[11,13,15]. Unalp *et al.* identified the anorectal region as a source of infection in 60% and the urogenital area in 40% of patients, with anorectal abscess and primary scrotal abscess being the most common causes[16]. In the largest studies to date by Eke and Sorensen *et al.* no identifiable source or comorbidity was recorded in 26–36% of patients with the disease[5,6].

Mortality and prognostic indicators

Since the nineteenth century, we have made substantial advances in defining necrotizing skin disease, and epidemiological studies have provided a better estimate of the mortality resulting from Fournier's gangrene. Sorensen *et al.* summarized case studies and showed the mortality to be between 20–40% with some studies reporting a case fatality rate as high as 88%[6,17]. The large, population-based studies that are available have demonstrated much lower mortality rates, ranging from 7.5–16%[5,6]. In addition, the mortality was lower in hospitals where a large population of patients with Fournier's gangrene were treated routinely than in those that did not frequently treat such patients. In some studies, the case fatality rate was highest in patients with associated comorbidities, including liver failure, chronic alcoholism, diabetes mellitus, and advanced age[6,12,18–20]. Other investigators did not find an association between these conditions and mortality, but showed that patients with underlying congestive heart failure, renal failure, and coagulopathy were at increased risk of death[21]. Thus, poorer health status of a patient before developing the disease is presumably associated with a poorer prognosis. Laor *et al.* stated that the most important parameter to predict outcome is the degree of deviation from homeostasis, rather than the extent of the disease or surgical debridement[22]. In their study, they assessed physiological profiles of patients and compared survivors to nonsurvivors, reporting that the comorbid medical conditions of renal failure and liver dysfunction were particularly closely associated with death from Fournier's gangrene, as opposed to death from other causes. Another study also showed that the majority of patients who died were on haemodialysis for chronic renal failure[23].

Laor *et al.* have developed a Fournier's gangrene severity index (FGSI)[22] based on nine parameters including body temperature, heart rate, respiratory rate, serum level of Na^+, K^+, creatinine, bicarbonate, haematocrit, and leukocyte count, which acts as a prognostic indicator for outcome of Fournier's gangrene. These parameters are graded on a scale of 0–4 and are summed to produce the FGSI score. In Laor and colleagues' study, a score >9 was associated with a 75% probability of death, whereas a score ≤9 indicated a 78% probability of survival. However, the accuracy of the FGSI to predict the outcomes of the disease remains controversial; several studies have confirmed the prognostic utility of the FGSI, showing that survivors have lower scores than nonsurvivors[11,24,25], but others have not shown a correlation between FGSI score and the death rate from Fournier's gangrene[26,27]. Hence, abnormal metabolic parameters might not accurately predict the severity of the disease, which means that the patient characteristics

Table 1 | **Types of necrotizing fasciitis**

Type	Microbiology	Organisms
I	Polymicrobial	Mixed aerobes and anaerobes
II	Monomicrobial	Group A β-streptococcus, *Staphylococcus aureus*, MRSA, ESBL
III	Marine bacteria	*Vibrio* spp.
IV	Fungal	*Candida* spp.

ESBL, extended-spectrum β-lactamase *Escherichia coli*; MRSA, methicillin-resistant *Staphylococcus aureus*.

and extent of tissue involvement that is not assessed with the FGSI do influence outcome. The FGSI scoring system has since been expanded to include an age score and an extent of disease score, creating the Uludag FGSI[28]. The addition of these variables has made the Uludag FGSI a more powerful tool than the original FGSI score, and has been shown to predict a 94% probability of death in patients with a score >9 and an 81% probability of survival in those with a score of ≤9 (REF. 28). Several other scoring systems, including the Charlson Comorbidity Index (CCI) and Surgical Apgar Score (sAPGAR), which were not developed for patients with Fournier's gangrene in particular, have also been shown to be adequate prognostic tools[29,30]. The CCI predicts the one-year mortality of patients with a variety of comorbidities (such as heart disease, cancer). Each comorbidity is assigned a number from 1–6 depending on the risk of dying associated with the condition[31]. The sAPGAR was developed to predict outcome after major surgery and is based on blood loss, lower heart rate, and lowest mean arterial pressure[32].

Which one of the prognostic tools to use is physician preference and does not depend on the clinical situation. Regardless of the use of a scoring system to predict possible outcomes, Fournier's gangrene is an unpredictable disease process with variable presentation, so no scoring system to predict patient outcome can or should replace clinical suspicion that warrants intervention.

Pathophysiology and microbiology

The necrotizing soft tissue infection (NSTI) of the genital, perineal, and/or perianal regions that constitutes Fournier's gangrene begins in the subcutaneous tissue (the hypodermis); more superficial layers (the dermis and epidermis) are initially not affected[33]. The bacteria within the tissue release toxins that cause tissue breakdown and lead to bacterial spread and tissue necrosis. Importantly, the extent of hypodermic necrosis is greater than can be visualized at the superficial skin level and can progress quite rapidly — within hours the infection can spread along fascial planes, especially when streptococcal infection is present[34]. Tissue hypoxia, and later necrosis and gangrene, are caused not only by tissue breakdown, but also by arterial and venous thrombosis, leading to soft tissue ischaemia that will eventually be visible at the superficial skin layers[35]. The hypoxia and consequent infarction of nerves in the affected area initially causes progressive pain, but later regional hypoaesthesia[33].

Most NSTIs have an anaerobic component that can be gas-forming and can, therefore, present with crepitus and pockets of gas. Aerobic metabolism creates water-soluble CO_2, whereas anaerobic bacteria produce gas that is formed by incomplete oxidation. The resulting hydrogen gas is less water soluble and accumulates in tissues[36]. Another typical tissue characteristic is the 'dishwater' fluid that can be encountered during surgical debridement. This is thought to be created by lysis of polymorphonuclear leukocytes and serous discharge[34].

Four types of necrotizing fasciitis have been described according to their microbiology (TABLE 1). Type I, most common, is a polymicrobial infection with mixed aerobes and anaerobes and is thought to be more indolent than the type II infection, which is monomicrobial and usually caused by group A β-haemolytic *Streptococcus* or *Staphylococcus aureus*[3]. Community-acquired methicillin-resistant *S. aureus* (MRSA) as well as extended-spectrum β-lactamase *Escherichia coli* have been identified as aetiologic agents[15,37]. Type III and IV are rare and are caused by Gram-negative organisms (*Vibrio* species) and fungal infections (*Candida* species), respectively[38–40]. The type III infection is seen in wounds contaminated with seawater and can also be caused by ingestion of seafood[38].

The incidence of type III infection is highest in Asia due to high consumption of seafood[33]. A study from Taiwan by Huang *et al.* analysed wound cultures from patients with necrotizing fasciitis over a 7-year period and found *Vibrio* species in 12% of samples[41]. The type IV infection is related to trauma and is more common in immunocompromised patients. Tang *et al.* reviewed the microbiology of Fournier's gangrene in studies published between 2009 and 2013 and found that the most common causes are polymicrobial (54%), followed by *E. coli* (46.6%) and *Streptococcus* (36.8%). Less common causative organisms included *Bacteroides*, *Enterobacter*, *Staphylococcus*, *Pseudomonas*, *Corynebacterium*, and *Klebsiella pneumoniae*[42].

Presentation and diagnosis

Skin and soft-tissue infections are classified into four categories, according to local and systemic signs and symptoms (TABLE 2). Eron *et al.*[43] categorized class I patients as outwardly healthy and afebrile, but presenting with cellulitis. These patients can be managed with

Table 2 | **Classification of soft-tissue infections**

Class	Patient presentation	Management
I	Healthy appearing, afebrile	Outpatient; oral antibiotics
II	Ill appearing, febrile, no unstable comorbidity*	Outpatient or inpatient observation
III	Toxic and/or critically ill appearing, one unstable comorbidity*	Admission with close monitoring
IV	Septic	Admission with close monitoring

*Such as diabetes mellitus, peripheral vascular disease, chronic venous insufficiency, or morbid obesity.

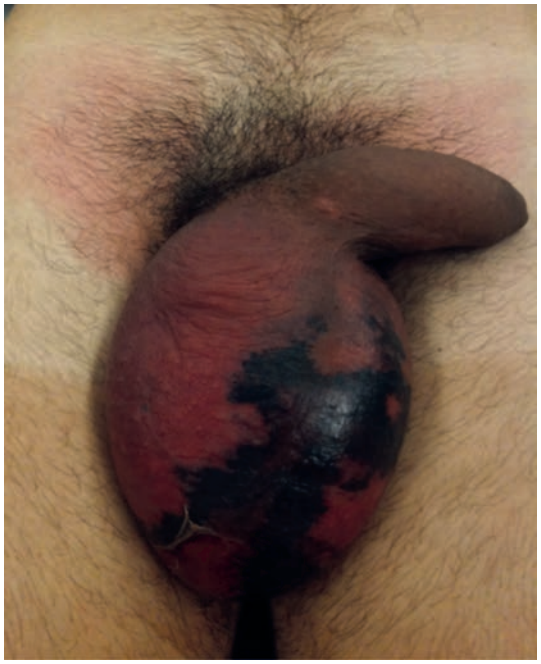

Figure 1 | **Fournier's gangrene of the scrotum.** Necrotic skin changes can be seen along the median raphe of the anterior and inferior scrotum. The erythema extending superiorly over the bilateral inguinal areas is indicative of progressive disease deep to the skin.

oral antibiotics on an outpatient basis. Patients with class II infections are clearly unwell and febrile, but do not have an unstable comorbidity, such as diabetes mellitus, peripheral vascular disease, chronic venous insufficiency, or morbid obesity; they can be managed as an outpatient or observed as an inpatient with antibiotic therapy. Patients presenting with class III infections are toxic and critically ill-appearing at presentation and have at least one unstable comorbidity, and those with class IV infections present with sepsis. These patients benefit from admission and close monitoring on broad-spectrum antibiotics and fluid resuscitation as per sepsis protocols and in severe cases may need intubation for altered mental status and/or respiratory distress[43]. This categorization is being used for NSTI in general, but can also be applied to patients with Fournier's gangrene.

The most common presenting clinical features in patients with Fournier's gangrene are swelling of the external genitalia, fever and pain[44,45]. The mean interval from symptom onset to hospital presentation is ~5.1 ± 3.1 days[44]. Any delay in presentation after symptom onset can result in skin necrosis (FIG. 1). Erythema can rapidly progress along the anatomical fascial planes of dartos, Colles, and Scarpa, often sparing the deeper layers, and, therefore, has the potential of expanding from the perineum along the anterior abdominal wall up to the clavicles[46,47]. Infection of the deeper layers and involvement of the testicles are rare, but can be a sign of a retroperitoneal or intra-abdominal source of infection. It is important to gain control of the source of the infection, which can entail retroperitoneal or intra-abdominal surgery[5,10].

Thorough physical examination and clinical assessment are the cornerstone of the diagnosis of Fournier's gangrene, but laboratory studies and imaging can be useful for risk stratification and to locate a potential source, respectively. The Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) score was proposed by Wong *et al.*[48] to distinguish necrotizing fasciitis from other soft-tissue infections using laboratory markers, including C-reactive protein, total white blood cell count, haemoglobin, Na^+, creatinine, and glucose. A score ≥6 raises suspicion for necrotizing fasciitis, and a score ≥8 is strongly predictive[48]. The LRINEC score has been validated in the literature and is thought to be a useful adjunct to the clinical examination[49].

Radiological studies, including radiography, ultrasonography, and CT, can be of value to assess the extent of disease (FIGS 2–4). CT provides the highest specificity for the diagnosis of Fournier's gangrene[50]. Radiography is the least costly option and can, in some cases, show hyperlucency representing soft-tissue gas before the accompanying clinical crepitus can be detected. A notable weakness of radiography is its inability to detect deep fascial gas[50]. Ultrasonography can be used to examine the scrotal contents, and determine testicular involvement, which is suggestive of an intra-abdominal or retroperitoneal source[50] Overall, CT scan is superior to both ultrasonography and radiography to confirm the diagnosis of Fournier's gangrene and to aid in surgical planning. A review of CT findings in all adult patients undergoing imaging for the evaluation of a soft-tissue infection over a 10-year period at one tertiary medical centre[51] used a scoring system that included presence of fascial air, muscle and/or fascial oedema, fluid tracking, lymphadenopathy, and subcutaneous oedema. More points are assigned to the findings with a higher odds ratio and a score >6 had 86.3% sensitivity and

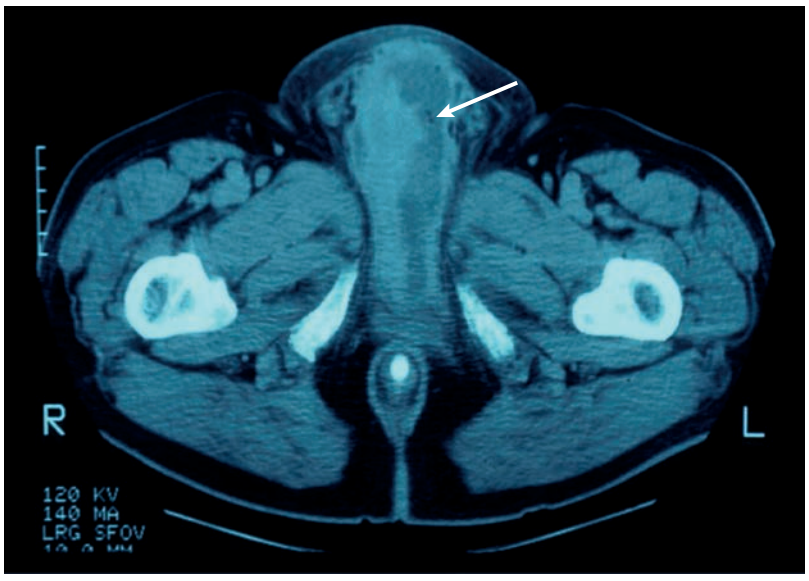

Figure 2 | **Pelvic CT scan of a patient with Fournier's gangrene**. This axial view shows a fluid-filled pocket, and gas in the scrotum and tracking into the perineum, which is indicative of a necrotizing soft tissue infection with a possible anaerobic, gas-forming organisms (white arrow: foci of gas).

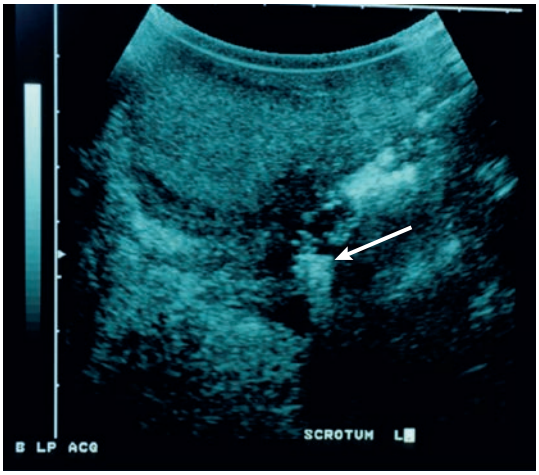

Figure 3 | **Ultrasonography of the scrotum of a patient with Fournier's gangrene.** The pocket of echogenic gas is clearly visible (white arrow) and is indicative of a necrotizing soft-tissue infection with a possible anaerobic, gas-forming organisms.

91.5% specificity for diagnosing NSTIs with a negative predictive value of 85.5%[51].

These tools have been developed to facilitate the diagnosis of Fournier's gangrene, but should never replace a clinical evaluation. A thorough examination of the genital, perineal, and anorectal areas and an assessment of the patient's overall health status remain the cornerstone of accurate diagnosis that should guide expedited management. Most importantly, imaging studies should never delay surgical intervention if the clinical suspicion is high.

Initial management

Antibiotic regimen and fluid resuscitation

Patients with Fournier's gangrene can be critically ill and require immediate resuscitation, much like trauma or burn victims. Fluid resuscitation and correction of electrolyte imbalances are paramount, and broad-spectrum intravenous antibiotics must be administered as soon as the clinical diagnosis is suspected. The antibiotic regimen includes agents that are effective against aerobic, anaerobic, Gram-positive and Gram-negative bacteria[4]. The recommended antibiotics traditionally included a combination of gentamicin, clindamycin, and either ampicillin plus sulbactam or a parenteral third-generation cephalosporin. In cases of clindamycin failure, chloramphenicol can be used. To cover fungal or hospital-acquired infections, agents such as fluconazole, vancomycin, or piperacillin–tazobactam should be chosen[52]. Alternatively, Smaldone *et al.*[53] propose using a fluoroquinolone instead of aminoglycosides and metronidazole instead of clindamycin. The 2014 update by the Infectious Disease Socitey of America agrees with Smaldone and adds the use of a carbapenem as monotherapy (BOX 1)[54].

Wound or tissue samples should be sent for culture and sensitivities, as resistance patterns have been reported and antibiotic treatment must, therefore, be directed toward the drug sensitivities. The antibiotic therapy should be continued until the patient becomes clinically stable and continues to recover. Of note, no correlation has been identified between a specific causative organism and the severity, morbidity, and mortality of the NSTI[29,52].

Surgical debridement

Early surgical intervention is the mainstay of treatment for Fournier's gangrene. However, the visible skin necrosis only represents the 'tip of the iceberg' and a much broader subcutaneous area can be affected and will likely require surgical debridement. Frezza and Atlas[55] proposed a minimal debridement approach, which consists of scrotal skin excision limited to the necrotic tissue with Penrose drains left *in situ* after wound irrigation with betadine and peroxide. The four patients who underwent the minimal debridement in their study recovered from their fasciitis and the authors report primary scrotal closure for all of them[55]. This approach might be successful in some patients, but the majority of the available literature supports wide surgical excision as part of the management policy of NSTI[4,56].

Guidelines are available for early, extensive debridement and planned repeat debridement[57]. The extensive debridement should be performed within the first 12 h of admission (FIG. 5,6); Chao *et al.*[58] reported that patients with NSTI who underwent surgical management within the first 12 h after admission had significantly better outcomes than those in whom surgery was delayed (adjusted hazard ratio (HR), 0.064; 95% confidence interval (CI), 1.6×10^{-7} to 0.25; $P = 0.037$). The Infectious Diseases Society of America practice guidelines for the diagnosis and management of skin and

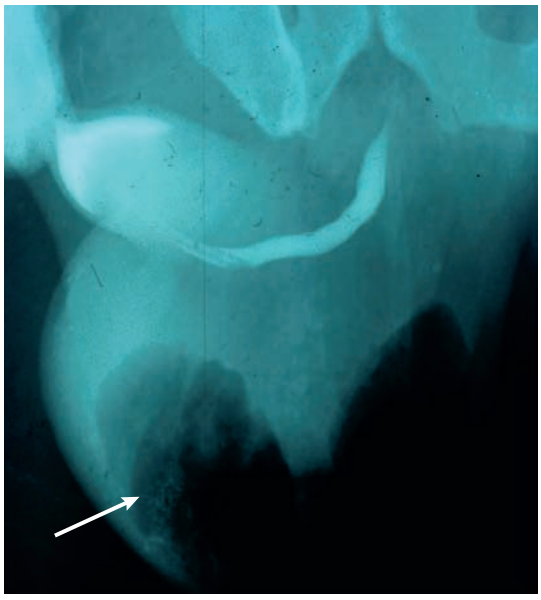

Figure 4 | **Pelvic radiography of patient with Fournier's gangrene.** Black-appearing scrotal gas is clearly visible (white arrow) and is indicative of a necrotizing soft-tissue infection with a possible anaerobic, gas-forming organisms.

Box 1 | **Antibiotic therapy for Fournier's gangrene**

Piperacillin–tazobactam + vancomycin
- 3.37g every 6–8 h intravenously (IV)
- 30 mg/kg/d in two divided doses IV

Cefotaxime + metronidazole or clindamycin
- 2 g every 6 h IV
- 500 mg every 6 h IV
- 600–900 mg every 8 h IV

Imipenem–cilastatin or meropenem or ertapenem
- 1 g every 6–8 h IV
- 1 g every 8 h IV
- 1 g daily IV

soft-tissue infections strongly recommend a scheduled re-debridement within 24 h to assess for local disease spread[54]. Most patients receive multiple surgical interventions with a reported mean of 2–3 debridements per hospital stay[4,21]. At least one re-debridement after the initial operation is recommended, but the role of multiple planned re-debridements is controversial[57]. The patient needs to be monitored closely between surgical procedures for further spread of the infection and potential deterioration of their overall health status, in which case they should definitely be taken back to the operating room for tissue examination and possible further debridement. If the patient is clinically improving further trips to the operating room can be omitted.

Surgical technique. Debridement includes removal of all nonviable tissue and resection until bleeding skin margins are encountered. In our practice, the initial incision is made into the necrotic skin, or into the most oedematous and/or erythematous area of skin if no necrosis is present. Finger dissection is then used to follow fascial planes and to disrupt pockets filled with tissue debris and gas. Throughout the debridement process, nonviable tissue is sharply excised. If the penile skin is involved, the shaft skin must be removed up to the corona without leaving behind any distal skin in order to prevent disfiguring lymphoedema. Once the debridement is completed, the wound is copiously irrigated with saline and haemostasis is achieved using electrocautery for small vessels, or ligation or clipping for larger vessels. We follow the '60-minute rule', which consists of 20 min of wound debridement, followed by 40 min with focus on haemostasis until the entire affected tissue is excised. These time frames are rough estimates and the rule merely reminds us to repeatedly assess the wound bed for any bleeding vessels, as small arteries can vasospasm and, if this process goes unrecognized, can result in substantial blood loss.

For female patients debridement should be carried out similarly as in male patients with expeditious, wide surgical excision of the affected tissue. One case report of a pregnant woman with Fournier's gangrene can be found in the literature and describes an urgent caesarean section with delivery of a 35 week neonate, followed by typical surgical debridement and broad-spectrum antibiotic therapy for the mother as effective managment[45].

In addition, in women the infection does seem to spread more easily to the retroperitoneal space and abdominal cavity than in men, most likely owing to the difference in genital anatomy[59].

Postoperative care. The exposed subcutaneous tissue should be covered with saline-soaked gauze, which should be changed frequently throughout the day. Topical antiseptics such as mafenide or Dakin solution can be used as adjuncts to reduce bacterial contamination of large wounds[60]; the application of honey to the wound bed has also been reported to result in a shorter healing time and hospital stay[61], but is not in widespread use, despite having been shown to possess antimicrobial and healing properties[62]. The use of a vacuum-assisted closure (VAC) system dressing with negative pressure has been proposed by some to help with wound care and closure[63]. The VAC dressing is applied after the acute phase once the patient is medically stable and no further debridements are needed. It is used to achieve a clean wound to facilitate definitive reconstruction (FIG. 7 (REF. 47)). The use of VAC therapy decreases the number of dressing changes from several times daily to every 3–4 days, and also reduces the length of hospital stay and — potentially — cost[64]. In a study from Turkey that reported the treatment of ten patients with Fournier's gangrene, five patients received the conventional gauze dressing and the other five patients had a VAC system placed. The patients in the VAC group had fewer dressing changes, less pain, and greater mobility compared with those who received a conventional dressing[65]. Unfortunately, the location of the wound in the perineal and genital area can make it difficult to keep a good seal and continuous vacuum, often limiting the use of VAC for Fournier's gangrene.

The use of hyperbaric oxygen has been suggested to decrease the number of surgical debridements and

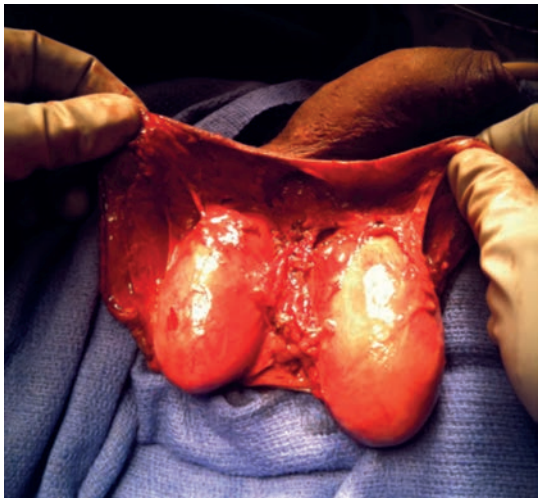

Figure 5 | **The scrotum and testicles immediately after debridement (same patient as shown in** FIG. 2**).** Neither testicle is involved in the infectious process.

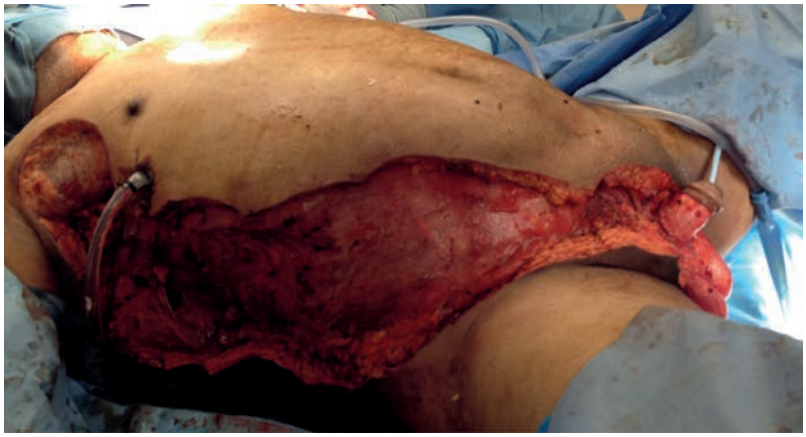

Figure 6 | **Extensive debridement of the entire scrotum, penile shaft, and right flank up to the clavicle, owing to extensive progression of Fournier's gangrene.** The patient has a chest tube and a Foley catheter in place.

possibly the need for reconstruction. Hyperbaric oxygen for 90 min with an average of ten sessions per patient was shown in one study to shorten the healing period when combined with debridement and dressing changes[66]. These data have also been reflected in several other studies that have found hyperbaric oxygen to be a useful adjunct to conventional therapy, keeping in mind that it should never replace nor delay surgical debridement[67–69].

Many patients with Fournier's gangrene need additional procedures, such as suprapubic tube placement, orchiectomy, penectomy, and colostomy, in addition to wound debridement, depending on the extent of their disease. Clear indications for a suprapubic catheter placement cannot be found in the literature. Some authors describe placing a percutaneous suprapubic cystostomy tube in all patients with genital involvement[70]. We recommend a cystostomy for urinary diversion only in patients who present with a urethral stricture, and perhaps in patients in whom a urethral catheter makes the dressing changes challenging. The need for faecal diversion is at the discretion of the general surgery team and remains controversial[71]. In patients with Fournier's gangrene that involves the perianal region, those with rectal injury and/or fistula, or those with rectal sphincter involvement, faecal diversion with either a bowel management catheter or converting colostomy should be considered[71,72]. In such patients the diversion prevents wound contamination and promotes healing without infection.

Approximately 10% of patients require mechanical ventilation and 1.4% need dialysis after surgical intervention[21]. Hence, the admitting hospital should be equipped with intensive care beds, or transfer of a severely ill patient to a centre with more experience or a higher level of care should be considered to reduce mortality[21]. However, even if transfer is being considered, the initial — potentially life-saving — debridement should not be delayed. Analysis of the national State Inpatient Database, established by the Healthcare Cost and Utilization Project showed that the median length of hospital stay in patients with Fournier's gangrene was 8 days and median costs were US$27,646 (REF. 21).

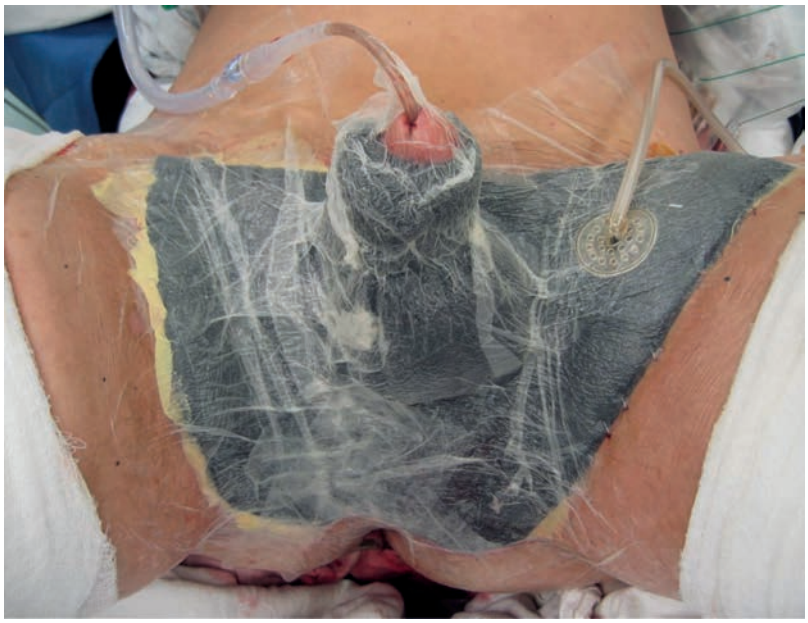

Figure 7 | **Vacuum-assisted closure (VAC) system placement after split-thickness skin graft resurfacing of the penis, testes, and groin.** Reproduced with permission from Hagedorn, J. & Rosenstein, D. in *Advanced Male Urethral and Genital Reconstructive Surgery* (eds Brandes, S. B. & Morey, A. F.) Ch. 39, 543–562 (Springer Science+Business, 2014)[47].

Management of genital skin loss

Once necrotic tissue has been removed, the underlying infectious process has been treated systemically, and the patient is recovering, options for definitive genital reconstruction should be discussed. The quality of the wound bed must be assessed to ensure that healthy granulation tissue has formed without any remaining areas of necrosis. The reconstruction should be delayed for a minimum of 5–7 days after the last debridement until the wound bed is optimal, at which point any reconstructive technique, including primary closure, skin grafting, or flap placement, can be undertaken[73].

Small scrotal wounds with <60% of skin loss can usually be closed primarily[73,74]; a two-layer closure with absorbable suture is preferred. In cases of a larger skin defect or where no scrotal skin remains several reconstructive techniques can be considered. One option is to place the testicles into anteromedial thigh pouches to facilitate wound care of the perineum. For some patients this can offer a permanent solution, especially if fertility and cosmesis is of no concern, and even for patients who desire a reconstructive procedure, thigh pouches can be safely used as a temporary measure. Testicular volume does not seem to be affected even if the testicles remain in the pouches for several months[75].

Split-thickness skin grafting is the most commonly used reconstructive technique for the scrotum. First, the testicles need to be brought to a dependent position (hanging between the thighs) by bluntly dissecting and freeing up the cords to the external inguinal ring. The

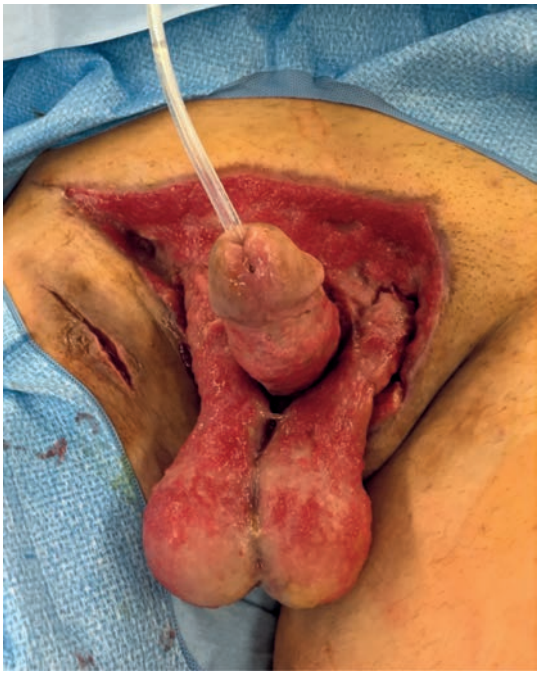

Figure 8 | **Surgical treatment of Fournier's gangrene.** Granulation tissue can be seen in the wound bed and the testicles have been sutured together medially, forming a scrotal unit to facilitate placement of the skin graft.

testes are then prepared for the skin graft by scraping off the granulation tissue and suturing them together in the midline to create one scrotal unit (FIG. 8). The split-thickness skin graft of ~0.012–0.016 inches thick is most often taken from the anterior or lateral thigh and meshed[76]. The graft is then draped over the testicles to cover the wound and fixed into place with absorbable sutures and/or staples (FIG. 9). Around 25 cm^2 of graft is usually needed to cover the scrotum and additional 15 cm^2 to cover the penile shaft. Some surgeons have suggested the use of fibrin tissue glue to help with graft adherence and to minimize the need for suture placement[77]; however, we do not favour this technique because accurate fixation of the graft to the contour of the testes and spermatic cords is easier without the glue. After the graft is fixed into place it is covered with a moist, nonadherent, multilayer, compressive dressing for about 5 days, during which the patient remains bedbound to ensure optimal graft take. The VAC system can be used as a dressing and to help with fixation of the split-thickness skin graft[78].

If the shaft of the penis is involved, split-thickness skin grafting can also be used for resurfacing. The use of an unmeshed graft is favoured for optimal cosmesis and to prevent potential contraction and creation of penile curvature, but Black and colleagues have shown that an unexpanded meshed graft also provides a good appearance and might even have a higher likelihood of graft take, owing to less fluid accumulation and graft bed contamination than that seen with the nonmeshed sheet graft[76]. At a mean follow-up duration of 6 months, Black *et al.* did not note any scar contractures using an unexpanded mesh graft.

Other scrotal reconstruction techniques use various thigh flaps to create a neoscrotum. Rotational fasciocutaneous thigh flaps can be brought together to cover the testicles[79]. Of note, the testicles should be covered with the smallest possible amount of subcutaneous tissue to prevent overinsulation and ensure low temperatures to maintain normal spermatogenesis[80]. Anterolateral thigh flaps using the descending branch of the lateral circumflex femoris artery have been described[79], and show good results at a mean follow-up duration of 8 months[81].

Of note, we usually do not place skin grafts on the perineum since the graft take in our experience is poor due to shearing forces from ambulation. The perineum does in our experience heal well by secondary intention with the use of wet-to-dry dressing wound care.

In female patients the exposed skin of the labia majora can be grafted with split thickness skin, but no data regarding such cases are available in the literature.

Even after successful recovery from the infectious process and genital reconstruction, many patients still need local wound care and help from a caregiver at home, with 30% of patients requiring home health care or a skilled nursing facility after hospital discharge[82].

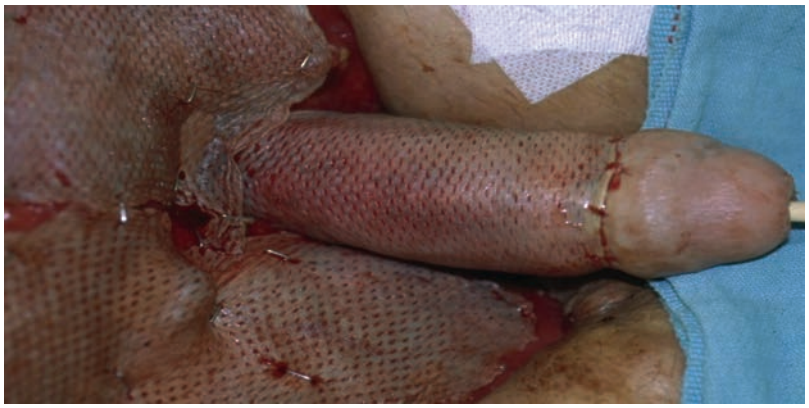

Figure 9 | **Reconstruction after Fournier's gangrene debridement.** A meshed split-thickness skin graft has been fixed with absorbable sutures and staples around the entire penile shaft and suprapubic area.

Conclusions

Fournier's gangrene is a rapidly progressive and potentially fatal necrotizing skin infection of the genital, perineal, and/or perianal regions. A high level of suspicion with prompt resuscitation and surgical intervention are the key for optimizing patient outcomes. For equivocal cases, several diagnostic tools, including laboratory tests and imaging, have been developed to be used in conjunction with physical examination findings. Most infections are polymicrobial, requiring broad-spectrum antibiotics and wide surgical debridement. Wound preparation with dressing changes and further debridements are essential for successful reconstruction once the local necrotic process and systemic infection has been treated. After the wound bed has been optimized, split-thickness skin grafting can be used to reconstruct the external genitalia with good functional and cosmetic outcomes.

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J.C.H. researched data for the article and wrote the manuscript. Both authors contributed to discussions of content and reviewed and edited the manuscript before submission.

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