The diagnosis and management of Fournier’s gangrene

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Fournier’s gangrene is a fulminating, polymicrobial, necrotising fascitis of the anogenital region, which spreads rapidly along the deep fascial planes. This eponymous syndrome was first mentioned by venereologist Jean Alfred Fournier in 1883 when he described an idiopathic, rapidly spreading genital gangrene occurring in five healthy young males.¹

Fournier’s gangrene is rare, accounting for <0.02% of hospital admissions² and is now understood to be the result of opportunistic infection, most commonly arising from the ano-rectum (30–50%), uro-genitalia (20–40%) or genital skin (20%).³ A resultant oblitative endarteritis ultimately leads to tissue ischaemia and necrosis.

Immunocompromised populations are most at risk, with males over 50 years old being the most commonly affected. The disease has a significant associated morbidity, and mortality rates lie between 20–40%.² Early identification, resuscitation, and administration of broad-spectrum antibiotics and timely surgical debridement are the basic principles of successful management.

Clinical presentation
Fournier’s gangrene should be considered in anyone with painful swelling of the scrotum or perineum with features of sepsis. The most common presenting feature, in over 75% of patients, is perianal/scrotal pain and swelling.⁴ Systemic features such as pyrexia and tachycardia are frequently present and may be associated with end organ dysfunction and increased mortality.⁵ The onset of Fournier’s appears to be insidious in nature, with the average time from initial symptoms to presentation being five to seven days - hence patients may present with late features such as skin hyperaemia or necrosis⁶ (Figure 1). Purulent, foul smelling discharge and crepitus have been described in the late stages of the disease process.⁴

Risk factors
Any condition that is likely to decrease the host immunity can predispose a patient to Fournier’s gangrene. Common precipitants include diabetes and chronic alcohol abuse, present in 20–70% and 20–50% of cases, respectively.⁷ Other risk factors include obesity, steroid use and malnutrition.⁷ Low socio-economic status has also been found to be an important predisposing factor.⁸
Diabetes
Patients with diabetes are more likely to develop Fournier’s gangrene; however, the overall length of hospital stay and mortality does not appear to be affected.\textsuperscript{2,5,10} Diabetes appears to be associated with younger age at presentation\textsuperscript{6} and patients may require significantly more debridements than those without diabetes.\textsuperscript{10} Patients with poorly controlled diabetes, indicated by a high HbA1c, have been found to require longer hospital stays, larger surface area involvement and higher severity index scores.\textsuperscript{11} Fungal organisms should be considered in diabetic patients as an important causative pathogen.\textsuperscript{12}

Alcoholism
Chronic alcoholism is consistently indicated as a risk factor for developing Fournier’s gangrene. Low albumin, an indicator of synthetic liver failure, has shown a trend toward worse prognosis.\textsuperscript{8} However, alcoholism appears to have no bearing on outcome.\textsuperscript{2,8}

Causative organisms
Once considered to be the result of streptococcal infection alone, Fournier’s gangrene is now understood to be polymicrobial in nature\textsuperscript{13,14} and is typically caused by the opportunistic invasion of commensal bacteria. Commonly found organisms include \textit{Staphylococcus aureus}, \textit{Streptococcus species}, \textit{Escherichia coli} and \textit{Acinetobacter}.\textsuperscript{6,8,13} In this regard, antibiotic resistance may be a growing concern in Fournier’s gangrene with methicillin-resistant \textit{Staphylococcus aureus} (MRSA) being the most commonly cultured resistant organism.\textsuperscript{15} Patients with multiple drug resistant organisms have shown a trend towards having poorer outcomes.\textsuperscript{15}

Risk stratification and laboratory investigations
Wong \textit{et al.} described the laboratory risk indicator for necrotising fasciitis using white cell count, C-reactive Protein, haemoglobin, sodium, glucose and creatinine measurements, with a cumulative score >6 having a 92\% positive predictive value for necrotising infection versus cellulitis, with a narrow confidence interval.\textsuperscript{16}

The Fournier’s gangrene severity index (FGSI) (Table 1) utilises physiological and laboratory parameters, which are individually graded 0–4. A cumulative score of greater than 9 was associated with a 75\% chance of mortality.\textsuperscript{17} The reliability of this score as an outcome predictor has been validated in several studies since its conception.\textsuperscript{13,18,19} Although widely quoted in the literature, the FGSI has a limited clinical role and, due to its cumbersome nature, it is difficult to calculate quickly at the bedside. Lin \textit{et al.} proposed a simplified FGSI including only haematocrit, potassium and creatinine, which was demonstrated to be a similarly reliable predictor of outcome.\textsuperscript{19}

<table>
<thead>
<tr>
<th>Variables</th>
<th>High abnormal values</th>
<th>Low abnormal values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Temperature (°C)</td>
<td>+4</td>
<td>39–40.9</td>
</tr>
<tr>
<td>Heart rate</td>
<td>&gt;180</td>
<td>140–179</td>
</tr>
<tr>
<td>Respiration rate</td>
<td>&gt;50</td>
<td>35–49</td>
</tr>
<tr>
<td>Serum Na (mmol/L)</td>
<td>&gt;180</td>
<td>160–179</td>
</tr>
<tr>
<td>Serum K (mmol/L)</td>
<td>&gt;7</td>
<td>6–6.9</td>
</tr>
<tr>
<td>Serum creatinine (mg/100mL) x2 for acute renal failure</td>
<td>&gt;3.5</td>
<td>2–3.4</td>
</tr>
<tr>
<td>Haemotocrit (%)</td>
<td>&gt;60</td>
<td>50–59.9</td>
</tr>
<tr>
<td>White blood cell count (x1000/mm(^3))</td>
<td>&gt;40</td>
<td>20–39.9</td>
</tr>
<tr>
<td>Serum bicarbonate (mmol/L)</td>
<td>&gt;52</td>
<td>41–51.9</td>
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Table 1. Fournier’s Gangrene Severity Index\textsuperscript{17}
In addition, other factors such as chronic kidney disease, surface area involved and serum albumin have all been found to differ significantly between survivors and non-survivors of Fournier’s gangrene. The Uludag FGSI is an updated scoring system that utilises age and extent of disease parameters. Patients scoring over 9 have a 94% incidence of mortality.

**Imaging**

Imaging plays a limited role in Fournier’s gangrene. If necrotising infection is suspected then urgent surgical debridement should be performed. A plain X-ray has low sensitivity and specificity but it may show gas in the soft tissues (Figure 2).

**Ultrasound**

Ultrasound may be beneficial in patients where the diagnosis is unclear. It is readily available, quick to perform and can even be utilised at the bedside. Hyperechoic foci with reverberation artifact and ‘dirty’ shadowing represents gas in the soft tissue and is highly suggestive of Fournier’s gangrene. However, 10% of patients will not have subcutaneous emphysema. Furthermore, although an ultrasound may allow quick diagnosis and rapid definitive treatment, its success is operator dependent.

**Computed tomography and magnetic resonance imaging**

Computed tomography and magnetic resonance imaging (MRI), unlike ultrasound, will demonstrate the nidus of infection and delineate the extent of soft tissue and fascial involvement. The main findings include soft tissue and fascial thickening with or without subcutaneous emphysema (Figure 3). MRI results in greater soft tissue detail and resolution; however, its use is seldom reported due to its cost and relative lack of availability.

**Management**

**Antibiotics**

Empirical broad-spectrum antibiotics are administered when a diagnosis of Fournier’s gangrene is suspected. Given the polymicrobial nature of the disease, the typical regimens will include a penicillin-based agent and anaerobic cover with metronidazole or clindamycin. The European Association of Urology (EUA) has suggested antimicrobial regimens (Table 2).

**Surgical debridement**

Overlying skin changes may not necessarily reflect fascial or deep tissue involvement and surgical debridement is the mainstay of successful management of Fournier’s gangrene. Urgent debridement should be performed within 24 hours to prevent worsening mortality. Tissue resection should include a cuff of healthy tissue and as a result will hopefully halt disease progression. However, patients may require multiple debridements, with an average of 1.5 debridements per admission. Repeated procedures have been associated with increased mortality, and correlate with a high FGSI score.

To encourage perineal wound healing and prevent any faecal contamination a diverting colostomy may be indicated and is performed in the presence of anal sphincter involvement and/or faecal incontinence. The formation of a diverting stoma has been found to increase mortality and overall length of hospital stay and therefore the risk/benefit of this procedure should be made on a case-by-case basis.

An alternative to a diverting stoma is a faecal management system – a catheter inserted into the rectum that navigates faeces away from the wound. These devices have been shown to decrease hospital stay while successfully maintaining wound integrity.

Urinary contamination can be prevented by urethral catheterisation in the majority of cases, though (rarely) cystectomy may be indicated.

**Vacuum assisted closure**

Negative pressure dressings are thought to reduce oedema, increase blood flow and hence improve wound healing when compared to conventional healing by secondary intention. Negative pressure therapy encourages the formation of
granulation tissue by removal of bacterial contamination and exudate, and may be used to decrease the size of broad defects prior to definitive reconstructive surgery. Although Vacuum assisted closure therapy (Figure 4) reduces wound surface area significantly quicker than conventional therapy, the total length of hospital stay does not appear to be significantly altered. Hyperbaric oxygen therapy Hyperbaric oxygen therapy (HBOT) involves exposing patients to increasing atmospheric pressure while inhaling 100% oxygen. Hypoxia due to small vessel thrombosis provides an ideal environment for proliferation of anaerobic bacteria, hence providing tissues with a surplus of oxygen aims to counteract this. Furthermore, HBOT is thought to up-regulate the body’s immune system, stimulate collagen formation and increase intra-cellular transport of antibiotics. Indeed, HBOT has been found to reduce mortality in a study of 341 patients. However, overall the evidence is inconsistent and HBOT is not recommended in the EUA guidelines. Reconstructive procedures Ideally, wounds following debridement would be primarily closed; however, in large defects this is unlikely to be possible. Healing by secondary intention, although feasible, can lead to skin contracture and poor cosmesis. Though it may be considered for wounds confined to less than 50% of the scrotal area. However, for the majority of cases the defect is too large and reconstruction is generally achieved using a full thickness skin graft, or flap reconstruction.

Summary
Fournier’s gangrene remains a surgical emergency with a high mortality if it is not diagnosed early in its progression. The scoring systems predict mortality accurately but play a small role in clinical practice. Rapid administration of broad-spectrum antibiotics and urgent surgical debridement remain the mainstay of successful treatment, despite the emergence of adjuvant therapy such as HBOT. Survival rates of >70% are achievable depending on the patient group and availability of higher level care.

Declarations of interests: none declared

References