**INTRODUCTION**

Fournier's gangrene (FG) is the necrotizing fasciitis of the perineum and genital area with possible involvement of the abdominal wall (Figure 1). It is a serious surgical emergency. Intense pain and tenderness in the genitalia are hallmarks of this infection. Early diagnosis, prompt antibiotic administration and surgical debridement are essential. It was named after French dermatologist Jean-Alfred Fournier who described fulminant gangrene of the penis and scrotum in five young men in 1883. Histopathologic examination of the affected skin and subcutaneous tissue shows cell necrosis, thrombosis of small vessels, infiltration with bacteria and inflammatory cells, and occasionally free air. The cause of FG is mostly polymicrobial aerobic and anaerobic synergistic infection originating from a colorectal, genitourinary or skin infection site. Although the disease is rare and accounts for 0.02% of all admissions to general surgical wards, it usually occurs in patients who are immunosuppressed due to comorbidities and who develop a primary infection site such as an abscess. Despite a multidisciplinary approach that includes broad-spectrum antibiotics, radical surgical debridement (Figure 2) and hemodynamic support in an intensive care unit (ICU), mortality rates are still very high and can be as high as 88%. The aim of this study was to analyze clinical and laboratory parameters in patients with FG, and to report factors that determine, influence or predict mortality.

**METHODOLOGY**

We conducted a retrospective study of all patients with FG in our hospital from January 2007 to December 2011. Patient's records were reviewed and a total of 82 patients were included in the study. They were divided into two groups: survivors and non survivors. The diagnosis was based on history and clinical examination. Collected data included age, gender, risk factors, etiology, clinical signs and
symptoms, clinical parameters (heart rate, temperature, respiratory rate and blood pressure), laboratory findings (serum sodium, potassium, creatinine and bicarbonate, hematocrit and leukocyte count), duration of symptoms before admission, total extent of affected body surface, and number of surgical debridements.

The extent of involvement, total body surface area, was calculated using charts routinely used to assess the extent of burn injuries. The penis, scrotum and perineum each account for 1% surface area and each ischiorectal fossa accounts for 2.5%. For assessment of FG severity on admission, we used the FG severity index (FGSI) score,\(^8\) and presence of sepsis, severe sepsis or septic shock on admission. We calculated FGSI from clinical (temperature, heart and respiratory rate) and laboratory parameters (serum sodium, potassium, creatinine and bicarbonate, hematocrit and leukocyte count) obtained on admission, as suggested by Laor E et al.\(^8\) Each parameter is given 04 points, and FGSI is calculated by summing up the points of each parameter. The cutoff point is 9 so that when FGSI is >9, the probability of death is 75% and when it is 9, the probability of survival is 78%. Sepsis is defined as infection with systemic inflammatory response syndrome, which is manifested with two or more of the following findings: body temperature <36°C (97°F) or >38°C (100°F), heart rate >90 beats/min, respiratory rate >20 breaths/min or, on blood gas, a PaCO\(_2\) <32 mm Hg, and leukocyte count <4,000 cells/mm\(^3\) or >12,000 cells/mm\(^3\), or >10% immature forms. Severe sepsis is defined as sepsis combined with organ dysfunction, hypoperfusion or hypotension. Septic shock is defined as sepsis with refractory arterial hypotension or signs of systemic hypoperfusion in spite of fluid resuscitation.\(^9,10\)

Statistical analysis was performed with SPSS v 17. P<0.05 was considered statistically significant.

**RESULTS**

Of 82 patients, 78 were males, and 4 females. The median age of all patients was 59 years. Patients were treated by repeated surgical debridements and broad-spectrum antibiotic therapy in (ICU), and all were operated within 24 hours of admission. The mortality rate was 36.6% (30/82 patients). Age and predisposing factor analysis is shown in Table 1.

<table>
<thead>
<tr>
<th>Predisposing factor</th>
<th>Survivors (n=52)</th>
<th>Non survivors (n=30)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, Median</td>
<td>58 (47.66)</td>
<td>69 (45.78)</td>
<td>0.12</td>
</tr>
<tr>
<td>Heart disease</td>
<td>12 (23.10)</td>
<td>10 (33.30)</td>
<td>0.73</td>
</tr>
<tr>
<td>Lung disease</td>
<td>12 (23.10)</td>
<td>12 (40.00)</td>
<td>0.43</td>
</tr>
<tr>
<td>Peripheral arterial disease</td>
<td>12 (23.10)</td>
<td>16 (53.30)</td>
<td>0.10</td>
</tr>
<tr>
<td>Liver disease</td>
<td>10 (19.20)</td>
<td>12 (40.00)</td>
<td>0.28</td>
</tr>
<tr>
<td>Kidney disease</td>
<td>04 (07.70)</td>
<td>16 (53.30)</td>
<td>0.0038</td>
</tr>
<tr>
<td>Hypertension</td>
<td>32 (61.50)</td>
<td>20 (66.60)</td>
<td>0.99</td>
</tr>
<tr>
<td>Diabetes</td>
<td>14 (26.90)</td>
<td>12 (40.00)</td>
<td>0.60</td>
</tr>
<tr>
<td>Malignant disease</td>
<td>08 (15.40)</td>
<td>06 (20.00)</td>
<td>0.96</td>
</tr>
<tr>
<td>More than one factor</td>
<td>22 (84.60)</td>
<td>15 (100.00)</td>
<td>0.29</td>
</tr>
<tr>
<td>Severe sepsis on admission</td>
<td>12 (23.10)</td>
<td>22 (73.30)</td>
<td>0.0049</td>
</tr>
<tr>
<td>Septic shock on admission</td>
<td>0</td>
<td>02 (13.30)</td>
<td>0.25</td>
</tr>
</tbody>
</table>

Severe sepsis or septic shock on admission was noted in 26 of 30 (86.7%) patients who died and in 12 of 52 (23.1%) patients who survived (p=0.001). Analysis of the infection source is shown in Table 2. FGSI >9 was noted in 26 patients and FGSI <9 in 56 patients.

<table>
<thead>
<tr>
<th>Source</th>
<th>Survivors (n=52)</th>
<th>Non survivors (n=30)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Colorectal</td>
<td>30 (57.7)</td>
<td>16 (53.4)</td>
<td>0.95</td>
</tr>
<tr>
<td>Genitourinary</td>
<td>14 (26.9)</td>
<td>04 (13.3)</td>
<td>0.53</td>
</tr>
<tr>
<td>Skin (trauma etc)</td>
<td>08 (15.4)</td>
<td>06 (20.0)</td>
<td>0.96</td>
</tr>
<tr>
<td>Unknown source</td>
<td>0</td>
<td>04 (13.3)</td>
<td>0.25</td>
</tr>
</tbody>
</table>

The mortality rate was 84.6% in the group of patients with FGSI >9 (22/26 patients) and 14.3% in patients with FGSI <9 (8/56) (p=0.0001). Overall, 16 patients (19.5%) required suprapubic cystostomy, and 12 (14.6%) required diverting colostomy, but the difference among survivors and nonsurvivors was not significant.
The most common organisms were Streptococcus (n=22; 29.7%), Bacteroides (n=20; 27.0%), Escherichia coli (n=14; 18.9%), Staphylococcus (n=12; 16.2%), Proteus (n=8; 10.8%), Clostridium (n=6; 8.1%) and Pseudomonas (n=4; 5.4%). There was no significant difference between survivors and nonsurvivors in type or number of isolated microorganisms.

**DISCUSSION**
FG is an aggressive and rapidly spreading infection of soft tissues or necrotizing fascitis that involves the deep and superficial fascia of the perineum. Several reports have stated that tissue necrosis can progress as fast as 2-3 cm/h. Thrombosis of subcutaneous and cutaneous blood vessels produces gangrene, but the fascial necrosis is usually more extensive than the visible gangrene suggests. Age and gender in our study were similar to other reports, with no significant difference between survivors and nonsurvivors and mortality rate of 36.6% was also similar to previous reports.

We found that the presence of kidney disease, elevated heart and respiratory rates, high serum creatinine, and low serum bicarbonate were associated with higher mortality. In other studies, changes in hematocrit, leukocyte count, blood urea nitrogen, creatinine, serum sodium, potassium, magnesium, calcium, serum albumin, lactate dehydrogenase and alkaline phosphatase have been reported to be predictive for higher mortality. In addition, elevated levels of fibrinogen and Factor VIII, low protein C, and positive lupus anticoagulant were found in over 90% of patients in one series, and one study suggested that female gender was related to higher mortality. We found that severe sepsis on admission, with hypotension was also predictive for higher mortality. Wound cultures were mostly polymicrobial and contained common skin, urinary tract and colonic pathogens, which is similar as in other reports. Also, no difference in isolated microorganisms was confirmed between survivors and non survivors.

Source of bacterial pathogen in FG, was no difference between survivors and non survivors. Perianal/perirectal, periurethral and scrotal abscesses are most common sources of the infection.

Microbiological cultures were done in 74 patients. A single microorganism was isolated in 12 patients (16.2%), and multiple microorganisms were found in 62 patients (83.8%).
but any skin lesion including pressure ulcer or surgical wound can act as a starting point for this disease. In our series we noted 4 cases of postoperative FG, one after hernia repair and three after open hemorrhoidectomy. The median number of debridements was higher in non survivors (3 vs 2), but this was not predictive for mortality in our report. This is consistent with several other reports. This is most likely due to the fact that patients who require more debridements have a greater extent of disease and therefore a worse prognosis.

We found that the median extent of affected body surface was significantly higher in non survivors, as expected and reported in several other series. Affection of abdominal and lower extremity skin, which is usually associated with higher extent of affected body surface, was positively associated with mortality in our series. The median duration of symptoms before admission was a day longer in non survivors (4 vs 3), but this was not associated with higher mortality. One other study reported a similar finding, but a positive correlation between the longer duration of symptoms and higher mortality has also been reported.

In our series, we did not notice any cases of testicular necrosis, and there was no need for orchidectomy. Some authors reported occurrence of testicular gangrene, up to 20%, but the cause of the necrosis is still not clear because the anatomy of fascial layers in the perineum should in theory prevent the spread of the infection to the testicular tissue or damage to its blood supply. Hyperbaric oxygen therapy increases tissue-oxygen tension, leukocyte activation, oxygen free-radical production, capillary angiogenesis, fibroblast proliferation and vasoconstriction and decreases anaerobic multiplication and helps in FG. However, prompt antibiotic administration and surgical debridement are the cornerstone of therapy.

**CONCLUSION**

Radical and repeated surgical debridements and intravenous broad-spectrum antibiotics are the mainstream therapy. Besides the standard clinical and laboratory parameters included in the FGSI calculation, a higher extent of affected body surface area and presence of severe sepsis with hypotension on admission are also linked with higher mortality.

**REFERENCES**


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Critical revision of the article for important intellectual content: Ghulam Hyder Rind
Statistical expertise: Azhar Ali Shah, Shahid Hussain Mirani
Final approval and guarantor of the article: Khush Muhammad Sohu

**Conflict of Interest:** None declared

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