Background: Fournier's gangrene is known to have an impact in the morbidity and despite antibiotics and aggressive debridement, the mortality rate remains high.

Objectives: To assess the morbidity and mortality in the treatment of Fournier’s gangrene in our experience.

Methods: The medical records of 14 patients with Fournier’s gangrene who presented at the University Hospital Center "Mother Teresa" from January 1997 to December 2006 were reviewed retrospectively to analyze the outcome and identify the risk factor and prognostic indicators of mortality.

Results: Of the 14 patients, 5 died and 9 survived. Mean age was 54 years (range from 41-61): it was 53 years in the group of survivors and 62 years in deceased group. There was a significant difference in leucocyte count between patients who survived (range 4900-17000/mm³) and those died (range 20.300-31000/mm³). Mean hospital stay was about 19 days (range 2-57 days).

Conclusion: The interval from the onset of clinical symptoms to the initial surgical intervention seems to be the most important prognostic factor with a significant impact on outcome. Despite extensive therapeutic efforts, Fournier’s gangrene remains a surgical emergency and early recognition with prompt radical debridement is the mainstays of management.

Key words: Fournier gangrene, contamination, debridement, mortality, rate

INTRODUCTION

In 1883, J.A. Fournier, a French venerealogist, described an idiopathic gangrene at genital level, of sudden presentation and rapid development, presenting in previously healthy young males. From them on, much has been talked about this entity. It’s pathologic characteristics are well defined. For this reason, it is not correct to consider it as "idiopathic", but nowadays, their pathophysiological mechanisms are precisely known, as well as the bacteria involved1-3.

This entity affects not only young males: in several reports the age at presentation included early infancy up to adulthood; cases in women have also been reported 4. Nevertheless, the majority of cases are in man, between the third and sixth decades of life, with 51 years as the average age at presentation5.

In the majority of cases of Fournier’s gangrene (FG), aerobic and anaerobic bacteria are synergistically involved, as a result of local trauma, extended periurethral infection or extended perianal infection1,3.

Nowadays, FG is defined as an infective necrotizing fasciitis, which affects perianal, perineal and genital regions, leading to thrombosis of subcutaneous vasculature and skin necrosis.

Main predisposing factor are diabetes mellitus, alcoholism, immunosuppression, liver and renal disease 6,7. The high morbidity and mortality of FG are attributed to delayed integrated diagnosis and to inadequate management of underlying disease.8,9

There are significant differences between patients who survive and those who die. These differences are important to elaborate a prognostic scale to be applied in all patients with FG10,11. We present data related to our experience in the management of FG and compare the clinical presentation, predisposing factors, and bacteria involved and administrated therapies between surviving and deceased patients.
hospitals. We registered the following data: age, gender, clinical presentation, predisposing factors, underlying disease, number of surgical procedures, presence of colostomy or cystostomy, and bacterial species. The following paraclinical data, recorded upon admission, were also reviewed: complete blood count (CBC), levels of hemoglobin, blood gases and serum glucose, and ELISA test results for HIV.

**RESULTS**

A total of 14 patients, 10 were male and 4 female were registered with clinical and pathological diagnosis of FG (Table 1). The diagnosis was delayed in some cases due to the fact that they were referred from other hospitals. Of the 14 patients, 5 died and 9 survived. Mean age was 54 years (range from 41-61): it was 53 years in the group of survivors and 62 years in deceased group. There was a significant difference in leukocyte count between patients who survived (range 4900-17000/mm³) and those died (range 20.300-31000/mm³). Mean hospital stay was about 19 days (range 2-57 days).

Predisposing principal factors were identified in 14 patients (Table 2).

Ischiorectal abscess was the most frequent clinical presentation (Table 3).

Gram staining was performed on all tissue samples and demonstrated red blood cells, gram-positive cocci and gram-negative bacilli. The most frequently cultivated organism were Escherichia coli, Bactericides sp., Staphylococcus sp., Streptococcus sp., Clostridium.

Treatment was started at the moment of admission, the number of surgical procedures per patient ranged between 1 and 3. Of these, 5 died. Surgical procedure included extensive debridement of necrotic tissue. Only two cases have received reconstruction of the scrotum by plastic surgery.

Twelve patients received triple antibiotic therapy (metronidazole, ceporin, and gentamicin). Other patients received metronidazol, amikacin, cefotaxime.

Regarding nutrition: 11 patients received total parenteral nutrition (TPN); 2 received, both enteral and parenteral nutrition, and 1 did not receive nutritional support because their hospital stay was less than 3 days.

**DISCUSSION**

Technological advances allow us to identify the etiologic agent in FG in about 70%-100% of cases. Ischiorectal abscess is the most frequent cause of FG, in the present study it represents 79% of the cases, Diverticular perforation, cancer, rectal perforation, perforated acute appendicitis, anal dilatation, internal hemorrhoids ligated with rubber bands and strangulated inguinal hernia have also been reported in the etiology of FG.

Recently, FG has been observed after stapled hemorrhoidectomy.

<table>
<thead>
<tr>
<th>TABLE 1</th>
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<tbody>
<tr>
<td>CHARACTERISTICS OF THE 14 PATIENTS DIAGNOSED WITH FOURNIER’S GANGRENE IN THE PERIOD 1997-2006, BY OUTCOME OF THE DISEASE</td>
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<tr>
<td>Age, years</td>
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<td>Leukocyte count, mm³</td>
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<table>
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<tr>
<th>TABLE 2</th>
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<tbody>
<tr>
<td>Predisposing factor</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
</tr>
<tr>
<td>Leukemia</td>
</tr>
<tr>
<td>Alcoholism</td>
</tr>
<tr>
<td>Obesity</td>
</tr>
<tr>
<td>Chronic renal failure</td>
</tr>
<tr>
<td>Unknown</td>
</tr>
</tbody>
</table>

Regarding the genitourinary tract, urethral stenosis and urethral instrumentation are the most frequent etiologies; others causes are infection of periurethral glands, surgery of the penis and scrotum, transrectal prostate biopsy, bladder cancer infiltrating the urethra and phlebitis of dorsal penis vein. This infection begins with bacteria inoculation in the perineal area, which makes a localized infection and, helped by the scanty vascularization of subcutaneous fat, disseminates through the aponeurotic layers, resulting in perianal gangrene. It can be facilitated by an altered immune system; diabetes mellitus, cancer, alcoholism, leukemia, treatment with steroids, systemic lupus erythematosus, AIDS, renal failure, and hemodyalisis are associated factors in the etiology of FG.

In the present study, the most frequent associated pathology was diabetes mellitus (43%); a total of 9 patients were alcoholic, obese, or had ischemic cardiopathy. The bacteriological reports for our patients are not different from those in the medical literature: Escherica coli, Bacteroids and Streptococcus sp., Staphylococcus, Peptostreptococcus and Clostridium sp. are frequently identified. If the portal of entry is the anorectal area, it produces perianal pain and swelling. If the infection originates in the genitourinary tract, it may present with urinary retention and testicular or scrotal pain.
TABLE 3
ETIOLOGY IN 14 PATIENTS WITH FOURNIER’S GANGRENE

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Patients (n)</th>
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<tbody>
<tr>
<td>Ischiorectal abscess</td>
<td>11</td>
</tr>
<tr>
<td>Anal fistula</td>
<td>2</td>
</tr>
<tr>
<td>Urethral stenosis</td>
<td>1</td>
</tr>
</tbody>
</table>

Carefully physical exam should exclude other intraabdominal diseases. Digital rectal examination and sigmoidoscopy are necessary in order to rule out colorectal etiology.

In the genital region, one must rule out sexually transmitted diseases, balanitis, orchitis, and etc.21

Often FG can present with fever, tachycardia, water and electrolyte imbalance, hypocalcemia, hypoalbuminemia, anemia, low platelet count, acidosis, hyperglycemia with coagulation disorders, elevated urea and creatinine, and leukocytosis, except in immunosuppressed and elderly patients.23

Laor et al.11 proposed a way to distinguish patients who will die from those who will survive; the significant variables were: age, hematocrit, blood urea nitrogen, albumin, alkaline phosphatase and cholesterol on the day of admission, as well as leukocyte count, platelet level, potassium, bicarbonate, total protein, albumin and lactate dehydrogenase on the seventh day.

The most remarkable difference between patients who survived and those who died was found in the leukocyte count on the day of admission: it was significantly higher in patients who died, without taking into account those cases in which the underlying disease produced leukopenia.

Once FG is diagnosed, treatment should be started promptly:

a) Intensive reanimation in two peripheral vein and Foley’s catheter, correct water and acid-base balance;
b) Intravenous antibiotics, especially broad spectrum antibiotics, for gram-positive skin, enteric and gram-negative genitourinary tract microorganisms, as well as for anaerobic bacteria. Tetanus vaccine and hyperimmune gammaglobulin should be administered1,12

c) Surgical treatment consists of extensive debridement of damaged and necrotic tissues far as needed to find normal tissue, regardless of the size of the defect produced, since an unsatisfactory debridement technique will definitively cause gangrene to progress, at the rate of approximately 2.5cm² per hour ¹

Colostomy should be done in patients with damage to the anal sphincter and therefore with fecal incontinence, which frequently produces contamination, as well as in cases of rectal perforation or if any fistula is found1,12. It is necessary to do a cystostomy if FG is secondary to or if it is related to stenosis or ruptured urethra; otherwise, a Foley’s catheter is sufficient12

Care of surgical wound after proper debridement includes frequent irrigation (every 6-8 hours) and changing the wounds dressings at bedside. Once the wound is clean and granulating tissue is present, one must decide whether to perform reconstructive surgery or allow closure by secondary intention1,13

Different studies have recommended hyperbaric oxygenation as treatment, since it produces an antibacterial effect on anaerobic bacteria, favors phagocytic function, eliminates alpha-toxins produced by clostridium and helps wound healing and scarring by promoting fibroblast development.24

In our patients it was not used, because we do not have such equipment at our institution.

In conclusion, an aggressive multidisciplinary management is mandatory in FG, because of its complexity and high morbidity.

SUMMARY
DA LI JE FURNIJEROVA GANGRENA I DALJE OPASNA?

Uvod: Poznata je činjenica da Furnijerova gangrena ima uticaj na morbiditet i da uprkos antibioticima i agresivnom debridmanu mortalitet ostaje visok.

Cilj: Procena morbiditeta i mortaliteta u tretmanu Furnijerove gangrene iz našeg iskustva.


Rezultati: Od 14 bolesnika, 5 je umrlo, a 9 preživelo. Prosečna starost bila je 54 godine (opseg od 41-61): 53 godine u grupi preživelih i 62 godine u grupi umrlih. Postojala je značajna razlika u broju leukocita izmedju bolesnika koji su preživeli (opseg 4900-17000/mm³) i onih koji su umrli (opseg 20300-31000/mm³). Prosečan ostanak u bolnici bio je oko 19 dana (opseg 2-57 dana).


Ključne reči: Furnijerova gangrena, debridman

REFERENCES