



FOURNIERS GANGRENE: A STUDY OF 50 PATIENTS

General Surgery

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ABSTRACT

Fournier's gangrene (FG) is a potentially life threatening progressive infection of the perineum and the genital. The objective of this study was to analyse etiological, epidemiological and clinical factors laboratory parameters that affects the progression and prognosis of the disease. This is a retrospective study of 50 patients admitted in hospital from 2010 to 2019. Patients were divided into survivors and non survivors. Of 50 patients, all were male. The median age of all patients was 61 years. The mortality rate was 31.6% (12/50 patients). Microbiological cultures were done in all patients. Besides clinical and laboratory parameters included in the FGSI calculation, body surface area and presence of other comorbidities on admission were also associated with increased mortality.

KEYWORDS

fourniers gangrene, necrotizing fasciitis

INTRODUCTION:

Fournier's gangrene (FG) is a potentially life threatening progressive infection of the perineum and the genital. It was named by the French dermatologist Jean-Alfred Fournier. He described fulminant gangrene of the penis and scrotum in five young men in 1883^[1]. Histopathologic examination of the affected tissues shows cell necrosis, small vessel thrombosis, plenty of inflammatory cells, bacterial infiltration, and occasionally free air^[2]. Most cases of Fournier's gangrene are caused by mixed bacterial flora, which include gram positive, gram negative and anaerobic bacteria. E.coli, bacteroids spp, S.pyogenes, and S.aureus are common etiologic pathogens. Risk factors for developing FG include underlying alcoholism, diabetes, cancer, malnutrition, advanced age, recent urogenital or colorectal instrumentation or trauma, and pre-existing peripheral vascular disease. Treatment includes combination of broad-spectrum antibiotics, extensive surgical debridement. However, despite aggressive modern management, the mortality of FG may be as high as 16% to 40%.

OBJECTIVE:

The objective of this study was to analyse etiological, epidemiological and clinical factors laboratory parameters that affects the progression and prognosis of the disease.

METHODS:

We conducted a retrospective study of patients diagnosed with FG in Sree Balaji Medical College And Hospital, Chromepet, Chennai from 2010 to 2019. Patient data was obtained by from the electronic medical record database. A total of 50 patients were found. The diagnosis of FG was established based on history and clinical examination. Collected data included: age, sex, risk factors, etiology, clinical signs and symptoms, clinical parameters like blood pressure, heart rate, temperature, respiratory rate, laboratory findings include complete blood picture, serum electrolytes, RFT's, urine routine examination, total body surface area affected, and number of surgical debridements.

The affected total body surface area was calculated using nomograms which are routinely used to assess the extent of burns. The penis, scrotum and perineum each account for surface area of 1%, and each ischiorectal fossa accounts for 2.5%. For assessment of Fourniers Gangrene severity on admission, we used the FG severity index (FGSI) score, introduced by Laor et al.^[3] in 1995, and presence of sepsis, severe sepsis or septic shock on admission. We calculated FGSI from clinical parameters including temperature, heart and respiratory rate and laboratory parameters including serum sodium, potassium, creatinine and bicarbonate, and hematocrit and leukocyte count obtained on admission, as suggested by Laor et al.^[3]: each parameter has 0-4 points, and FGSI is calculated by summing up the points of each parameter. The cut off 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 PaCO₂ <32 mm Hg (4.3 kPa), and leukocyte count <4,000 cells/mm³ or >12,000 cells/mm³, 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^[6,7].

RESULTS

Of 50 patients, all were male. The median age of all patients was 61 years. Patients were treated by repeated surgical debridement's and broad-spectrum antibiotics in the intensive treatment unit, and all were operated within 12-24 hours of admission. The mortality rate was 31.6% (12/50 patients). Age and predisposing factor analysis is shown in table 1. Severe sepsis or septic shock on admission was noted in 12 of 12 (100%) patients i. e all patients who died and in 10 of 38 (26.3%) patients who survived. Laboratory parameters and FGSI analysis, number of operations, duration of symptoms before admission and skin surface affection is shown in table 2. FGSI >9 was noted in 15 patients and FGSI <9 in 35 patients. The mortality rate was 83.33% in the group of patients with FGSI >9 (10/12 patients) and 5.3% in the group of patients with FGSI <9 (2/38).

Microbiological cultures were done in all patients. A single microorganism was isolated from culture in 10 patients (20%), and multiple microorganisms were found in 40 patients (80%). The most common organisms were Streptococcus, Bacteroides, Escherichia coli, klebsiella, Staphylococcus, Proteus, Clostridium, acinetobacter and Pseudomonas. There was no significant difference between survivors and nonsurvivors in type or number of isolated microorganisms.

TABLE 1: Analysis Of Age And Predisposing Factors

	survivors(n:38)	non survivors(n:12)
Age, median	56	70
Heart disease	8(21.05)	5(41.6)
Lung disease	7(18)	7(58.3)
Liver disease	7(18.42)	5(41.6)
Kidney disease	4(10.5)	7(58.3)
Diabetes	26(68.42)	9(75)
Hypertension	22(57.8)	10(83.3)
>1 predisposing factor	30(78.9)	12(100)
Severe sepsis on admission	10(26.3)	10(83.3)
Septic shock on admission	0	2

TABLE 2: Analysis of age and predisposing factors

	Survivors(n:38)	Non-survivors(n:12)
Temperature,C	37.8	38.6
Pulse,bpm	84	114
Respiratory rate,	18	22
Leukocyte count, 1000/mm ³	14.5	17.5
Hematocrit	0.37	0.37
Serum sodium,	136	129
Serum potassium,	4	4.3
Serum creatinine,	137	270
Serum bicarbonate,	23	17
FGSI	6	11
Duration of symptoms before admission	2	5
Number of operations	2	3
Affected skin surface %	3	11

**Fig 1: Preoperative Image****Fig 2: Post Surgical Debridement****Fig 3: Implantation Of Testis In Thigh With Primary Closure**

DISCUSSION

FG requires immediate treatment. Several reports stated that tissue necrosis can progress as rapidly as 2 cm/hr^[8,9]. Age of patients in our study were similar to other reports, with no significant difference between survivors and nonsurvivors. The mortality rate was 24%, similar as in some previous reports^[4,5]. It is found that the presence of kidney disease was associated with increased mortality. Abnormal laboratory and clinical findings, increased heart and respiratory rates, high serum creatinine, and low serum bicarbonate were associated with higher mortality. In different reports, changes in serum levels of 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^[5,10,12]. One study suggested that female sex is related to higher mortality^[16]. We found that severe sepsis on admission, with hypotension Below 90 mm Hg, 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^[14,15]. Also, no difference in isolated microorganisms was confirmed between survivors and nonsurvivors. No specific bacterial pathogen has been linked with a higher tendency to cause FG or as a cause of higher mortality. The median number of operative debridements was higher in nonsurvivors (3 compared to 2),

but this was not predictive for mortality in our report. This result is consistent with several other reports^[5,12,16], although there are series in which the higher number of operative debridement's negatively affected survival^[11]. This is most likely due to the fact that Patients who require more debridement's have a increased severity and extent of disease and hence worse prognosis. Aggressive, early debridement and intervention is still considered the most effective treatment for FG. It was found that the median extent of affected body surface was significantly higher in nonsurvivors. Involvement of abdominal and lower extremity skin leading to Increased extent of affected body surface area was associated with higher mortality in our study. The median duration of symptoms before admission was longer in nonsurvivors (5 days compared to 2), but this was not associated with higher mortality. One other study reported a similar finding^[14], but a positive correlation between the longer duration of symptoms and higher mortality has also been reported^[17]. There were no cases of testicular necrosis in our study and there was no need for orchidectomy. Some authors reported occurrence of testicular gangrene, up to 20%, but the cause of is still not clear because the anatomy of fascial layers in the perineum should prevent the spread of the infection to the testicular tissue or damage to its blood supply^[18,19]. Several possible causes of testicular gangrene have been proposed, this includes either the primary cause of the FG was epididymo orchitis^[20] that resulted in testicular destruction, or the infection spread deep into or from the retroperitoneum and then to the testis^[5]. It is also possible that Hypercoagulation and DIC, which can occur in sepsis, can lead to occlusion of the small vessels of the testes^[21].

CONCLUSION:

We conclude that FG is a life-threatening form of necrotizing soft tissue infection with high mortality. Repeated and aggressive surgical debridements and intravenous broad-spectrum antibiotic therapy are the mainstay of treatment. Along with the standard clinical and laboratory parameters included in the FGSI calculation, extent of affected body surface area, presence of severe sepsis and hypotension on admission are also associated with higher mortality. Detailed analysis of the FG aetiology, path in which the infection spreads should be made clear and done in patients that develop testicular necrosis during FG.

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