

Fournier's gangrene: A tertiary care hospital experience and analysis of mortality factors

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Abstract

Objective: To determine the clinico-pathological features of Fournier's gangrene (FG) and to identify risk factors associated with the mortality.

Material and Methods: This retrospective study was done at Sindh Institute of Urology and Transplantation (SIUT), Karachi, Pakistan from January 2012 to September 2015. All consecutive patients with a diagnosis of FG were studied for clinico-pathological features, management and outcome. Data were entered and analyzed by SPSS version 10.0.

Results: There were 83 patients; all were male. The mean age was 58.2 ± 12.6 years and mean duration from symptoms to admission was 3.66 ± 3.06 days. Mean hospital stay was 15.96 ± 11.19 days. The source of infection was urological in 36 patients (43.3%), anorectal in 10 (12.0%) and cutaneous in 6 (7.2%), while no cause was found in 31 (37.3%) patients. Majority presented with erythema and swelling of genitalia (85%), while 33 (39.7%) had fever, 50 (60.2%) pain, 24 (28.9%) crepitus, and 8 (9.6%) septic shock. E.coli was the most common organism isolated, i.e., in 19 patients (21.6%). Other organisms included Acinetobacter in 10 (12.0%), Klebsiella in 5 (6.0%) and Candida in 3 (3.6%) patients. The overall mortality rate was 22.8%. Sepsis, advanced age, renal failure on admission, extension of infection onto abdominal wall, full-blown shock on presentation and need for mechanical ventilation were the main risk factors contributing to mortality.

Conclusion: Fournier gangrene is a surgical emergency with high mortality rates. Early management with aggressive debridement, broad spectrum antibiotics and intensive supportive care has improved the outcome in recent years.

Keywords: Fournier's gangrene, infection, debridement, mortality, urological sepsis, Acinetobacter, E.coli

Introduction:

Fournier gangrene (FG) is a rare but potentially lethal disease.¹ FG is a synergistic polymicrobial infection of the perineal, genital and perianal region that leads to thrombosis of small subcutaneous vessels which ultimately lead to gangrene of the overlying skin.² Jean Alfred Fournier, a French dermatologist, gave the condition its eponymous name in 1883, but FG was first described by Baurienne over 100 years previously in 1764.³ Fournier gangrene is an acute urological emergency that needs an urgent diagnosis and aggressive treatment with the use of broad spectrum antibiotics and surgical debridement.^{1,2} Death rate is high ranging from 15-50%

despite the development of modern treatment techniques.^{2,4} Fournier gangrene is usually associated with malignancies, diabetes mellitus, alcoholism, liver failure, renal failure⁵ and acquired immunodeficiency syndrome.⁶ It can also arise as secondary infection resulting from different pathological conditions of the colorectal area, the lower urogenital tract and the perineum.⁵ The hallmark of Fournier gangrene is intense pain and tenderness in genitalia. Systemic effects vary from local tenderness with no toxicity to florid septic shock. Generally, the infection is caused by three or more microorganisms, the most common being the E.coli, proteus, enterococcus and anaerobes.^{7,8} The central principal of

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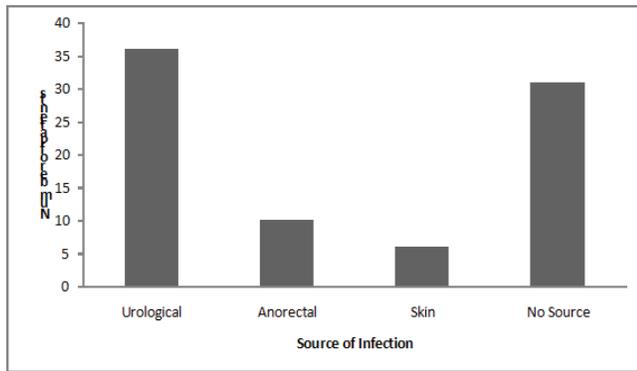


Figure 1: Sources of infection in 83 patients with Fournier's gangrene

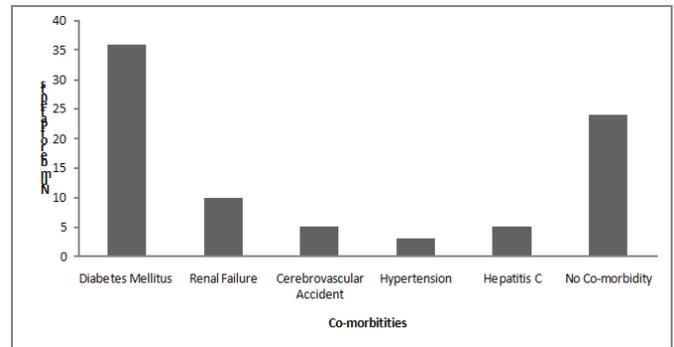


Figure 2: Co-morbidities in 83 patients with Fournier's gangrene

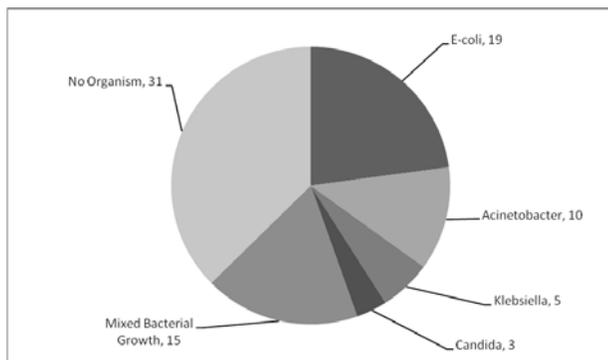


Figure 3: The type of organisms isolated from 83 patients with Fournier's gangrene.

electrolytes, blood glucose, urine and blood culture. Diagnosis of FG was established clinically on the basis of the patient's history and physical examination. Patients with a simple scrotal or perineal abscess without necrotizing infection were not included in this study.

Data were analyzed retrospectively to see the outcome of disease and to identify the risk factors which increased the mortality.

The data items were entered and analyzed by SPSS version 10.0. Simple descriptive statistics were used for computing the continuous and categorical data.

Results:

There were 83 patients, and all were male. The mean age of patients was 58.2 ± 12.6 years (range: 30-87 years). Mean duration from the onset of symptoms to hospital admission was 3.66 ± 3.06 days (range: 1-15 days). Mean hospital stay was 15.96 ± 11.19 days (range: 2-70 days). The source of infection was urological in 36 patients (43.3%), anorectal was in 10 patients (12.0%) and cutaneous causes were found in 6 patients (7.2%), while no cause was found in 31 (37.3%) patients (Figure 1). Among the urological causes, 15 (18.0%) patients had stricture urethra and 10 (12.0%) patients had long-term indwelling catheter.

Majority of patients presented with erythema and swelling of genitalia (85%), while 33 (39.7%) had fever, 50 (60.2%) had pain, 24 (28.9%) had crepitus, while 8 (9.6%) patients

management is aggressive haemodynamic stabilization, broad spectrum antibiotics and urgent surgical debridement.

The aim of the present study was to share our experience in the management of FG and to identify risk factors that influence the mortality rate.

Material and methods:

This is a retrospective study done at Sindh Institute of Urology and Transplantation (SIUT), Karachi, Pakistan from January 2012 to September 2015. Written informed consent was obtained from patients prior to the operative procedure. The study was done according to ethical guidelines as laid down in the declaration of Helsinki. 83 patients who were affected with FG were studied. All these patients were initially admitted in emergency. After assessment of pulse, blood pressure (BP), temperature and the presence of associated co-morbid, they were shifted to the ward. Blood sample was taken for complete blood count (CBC), urea, creatinine,

presented with full blown septic shock.

The most common co-morbidities included diabetes mellitus in 36 (43.3%) patients, renal failure in 10 (12.0%), cerebrovascular accident (CVA) in 5 (6.0%), hypertension in 3 (3.6%), hepatitis C in 5 (6.0%) patients, while 24 (28.9%) patients had no associated co-morbidity factor (Figure 2). A variety of organisms had been cultured from necrotic tissue or pus. *E.coli* was the most common organism found in tissue culture, i.e., in 19 patients (21.6%). Other organisms included *Acinetobacter* in 10 (12.0%), *Klebsiella* in 5 (6.0%) and *Candida* in 3 (3.6%) patients. Mixed bacterial growth was found in 15 (18.0%) patients (Figure 3).

All patients were treated with the common approach of medical management and surgical debridement. Medical management included aggressive intravenous fluids, broad spectrum antibiotics and hemodynamic support. 5 patients who presented with advanced renal failure needed dialysis support. 4 patients needed ventilator support also. Nutritional support was also given in those patients who presented in severe malnourished condition.

Surgical management included extensive and repeated wound debridement. Urinary diversion was done by means of percutaneous suprapubic cystostomy in 90% patients. Faecal diversion was also done in 2 patients.

The overall mortality rate was 22.8%. Sepsis, advanced age, advanced renal failure on admission, extension of infection on abdominal wall, full blown shock on presentation and need for mechanical ventilation were the main risk factors contributing to mortality.

Discussion:

Fournier Gangerne, first described by Jean Alfred Fournier in 1883, is a serious and rapidly progressive disease with high mortality rate. The disease is no longer restricted to young men as was initially thought but can affect a wide age range from neonates to the very elderly. Despite this, the age of most patients has been reported

between 30-60 years.⁹ The mean age of patients in our study was 58.2 ± 12.6 years. All of our patients were male, although it has also been reported in females and in children.¹⁰ The mortality rate increased in patients who had significant delay in hospital admission.¹¹ In our study, the mean duration from onset to hospital admission was 3.66 ± 3.06 days. However, Laor and colleagues found that the interval between the onset of the disease and the hospital admission does not play an important role in the prognosis and clinical outcome. Clinical signs and symptoms are similar to that described in literature i.e. pain, scrotal swelling, erythema associated with fever and crepitus.¹² Pain is usually prominent in early stage but as the disease progresses, pain subsides gradually due to destruction of cutaneous nerves.¹³ In our study, 9.6% patients presented with full blown sepsis due to advanced disease and significant delay; four of them required ventilator support. Shock, intestinal ileus, and delirium are common in this condition.¹⁴

Initially, it was thought that this disease has an idiopathic origin but it is not true.¹⁵ The source of infection may be urological, anorectal or cutaneous origin. The source of infection in one study including 55 patients was urological in 35%, colorectal in 29% and cutaneous in 29%.¹⁶ In our study, the source of infection was urological in 43.3%, anorectal in 12.0% and cutaneous causes was found in 7.2% of patients, while no cause was found in 37.3% patients. In our study, urological causes included stricture in 15 (18.0%) patients and indwelling catheter in 10 (12.0%) patients.

Multiple predisposing factors leading to FG have been reported in the literature. Most common is diabetes mellitus affecting 40-60% of patients; chronic alcoholism is another common co-morbid condition, which affects 25 to 50% of patients.¹ Other predisposing factors include advanced age; malignancy and immunocompromised status.¹⁷ In this study, 36 (43.3) patients were diabetic, 10 (12.0%) were in renal failure, 5 (6.0%) had CVA and 3 (3.6%) patients had high blood pressure. In our study, 60% patients were non-diabetic, so diabetes mellitus does not

seem to be a necessary underlying disease for FG but there is still controversy as to whether the co-existence of diabetes mellitus influences the prognosis.¹⁸

FG is commonly associated with a polymicrobial infection.¹⁸ Both aerobic and anaerobic bacteria are usually present but anaerobes are isolated less frequently. Some patients are infected by monomicrobial infection as aerobes, anaerobes or fungi.¹⁹ The causative pathogens act synergistically¹⁸ and release different proteins and enzymes which cause thrombosis of small vessels, known as obliterative endarteritis. This is the key patho-physiological event.¹⁷ The underlying thrombosis results in a cutaneous and subcutaneous vascular necrosis.¹ Most commonly isolated organism is *E coli*, followed by *Bacteroides* and *Streptococcal* species. *Staphylococci*, *Peptostreptococci* and *Clostridia* are also frequently isolated.²⁰ In the present study, we found *E.coli* in 21 (25.3%) patients, *Acinobacter* in 10 (12.0%), *Klebsiella* in 9 (10.8%) and *Candida* in 5 (6.0%) patients. Mixed bacterial growth was seen in 9 (10.8%) patients while no growth was found in 20 (24.0%) patients.

Various radiological techniques can be used to determine the extension of disease, for example, plain X-Ray abdomen, ultrasound, CT scan and MRI.²¹ In our study, these techniques were not routinely used.

Traditionally, aggressive haemodynamic stabilization, parenteral broad spectrum antibiotics and repeated surgical debridement are the most crucial steps in the treatment of patients with Fournier's Gangrene.²² All infected and necrotic tissue should be excised to the level of viable tissue and sample taken for culture. Frequent debridement is required. In our study, majority of patients (93%) required multiple debridements. Testis and spermatic cord are generally not affected by the disease, as they maintain an adequate and independent blood supply.

Literature suggests that temporary faecal diversion can be achieved by colostomy to prevent wound contamination and urinary diversion by

suprapubic cystostomy for better wound management. In this study, faecal diversion was done in 3 patients while suprapubic cystostomy was done in 90% of patients. Although most authors suggest that defect should be closed by secondary healing but there are many who prefer reconstructive surgery.²² In our study, secondary wound healing occurred in majority of our patients while skin grafting was done in 10 (12.5%) patients. Death rate is high ranging from 15-50% in literature. In our study, death rate was 22.8%. Advanced age, advanced renal failure on admission, extension of infection on abdominal wall, full blown shock on presentation and need for mechanical ventilation were the main risk factors for mortality.

Conclusion:

Fournier's gangrene is a surgical emergency with high mortality rates. Early management with aggressive debridement, broad spectrum antibiotics and intensive supportive care has improved the outcome in recent years.

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Role and contribution of authors:

Dr Rashid Hamid: Conception, designing, collection and analysis of data, primary drafting of the paper.

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Dr SAH Rizvi: Critical review and final approval of the manuscript.

References:

1. Smith GL, Bunker CB, Dinneen MD. Fournier's Gangrene Br. J. Urol. 1998; 81: 347-55
2. EKE N. Fournier's Gangrene: 4 review of 1726 cases; Br. J. Surg. 20;87:718-28
3. Quatan N, Kirby RS. Improving outcome in Fournier's Gangrene. BJU Int. 2004; 93: 691-2
4. Palmer LS, Winter HI, Reid RE, Loare E. The limited impact involved surface area and surgical debridement on survival in Fournier's Gangrene. Brit. J. Urol. 1995; 76:208-12
5. Spirnak JP, Resnick MI, Hampel N, Persky L. Fournier's Gangrene report of 20 patients. J Urol 1984; 131: 289-291
6. Mekay TC, Water WB. Fournier's Gangrene as the presenting sign of an undiagnosed Human Immunodeficiency virus infection. J Urol 1994; 152: 1552-1554
7. Paty R, Smith AD. Fournier's Gangrene. Urol clin North Am 1992; 19: 149-62
8. Baskin LS, Carroll PR, Cattolcia EV, McAninch JW. Necrotizing soft tissue infection of the perineum and genitalia. Bacteriology, treatment and risk assessment. Br. J Urol 1990; 65: 524-9
9. Xeropotamos NS, Nousias VE, Kappas AM. Fournier's Gangrene. Diagnostic approach and therapeutic challenge. Eur J surg. 2002;168: 91-5
10. Addison WA, livengood CH 3rd, Hill GB. Necrotizing fasciitis of vulvar origin in diabetic patient. Obstet gynaecol 1984; 63: 473-9
11. Laor E, Palmer LS, Tolia BM, Reid RE, Winter HI. Outcome prediction in patient with Fournier's. Gangrene. J Urol, 1995; 154: 89-92
12. Basoglu M, Gul O, Yildirgn I, Balik AA, Ozbey I, Oren D. Fournier's Gangrene. Review of 15 cases. Am Surgeon 1997; 63: 1019-21
13. Brown Gs, Jones RB, Hirshmann JV. Fournier's Gangrene. Necrotizing subcutaneous infection of the male genitalia. J. Urol. 1979; 122:279-82
14. Robert H, Hester L. Progressive Synesrgistic Bacterial Gangrene arising from an abscess of the vulva and bartholin's gland duct. Am. J. obstet gynecol. 1972;114:285-91
15. laucks SS 2nd Fournier's Gangrene. Surg clin North Am 1994; 74: 1339-52
16. Asci R. Sarikaya s, Buykalpelli R, Yilmaz AF, Yildiz S. Fournier's Gangrene. Risk assessment and enzymatic debridement with lyophilized collagenase application. Eur Urol 1998; 34:411-8
17. Vick R, Carson CC 3rd. Fournier Disease. Urol Clin North Am 1999; 26:841-9
18. Morpurgo E, Galandiuk S. Fournier's Gangrene. Surg Clin North Am 2002;82:1213-24
19. Septimus JD, Samo T, Fainstein V. Fournier's Gangrene Due to Candida Glabrata: Case report and review of the literatures. Infect Dis Clin Pract. 200;11:406-7
20. Adams JA, Culkin DJ, Mata JA Bocchini JA, Venable DD. Fournier's Gangrene in Children. Urology 1990;35:439-41
21. Sherman J, Sollday M, Paraiso E, et al. Early CT Finding of Fournier's Gangrene in a healthy male. Clin Imaging 1998;22;425-427
22. Corman JM, Moody JA, Aronsan WJ. Fournier's Gangrene in a modern surgical setting: improved survival with aggressive management. BJU int 1999; 84:85-88