SJIF Impact Factor: 3.458

WORLD JOURNAL OF ADVANCE HEALTHCARE RESEARCH

ISSN: 2457-0400
Volume: 3.
Issue: 5.
Page N. 43-45
Year: 2019

Original Article www.wjahr.com

FOURNIER'S GANGRENE: AN EXPERIENCE IN TERTIARY CARE HOSPITAL

Dr. Shweta Kumari[1], Dr. (Prof) Vimal Mukesh[2]*, Dr. Md Sarfaraz Alam[3], Dr. Pranay Kumar[4] and Dr. Babar Ali[5]

[1]Junior Resident, Dept. of General Surgery, PMCH Patna Bihar India.
[2]Professor, Dept. of General Surgery, PMCH Patna Bihar India.
[3]Assistant Professor, Dept. of General Surgery, PMCH Patna Bihar India.
[4]Senior Resident Dept. of General Surgery, PMCH Patna Bihar India.
[5]Junior Resident, Dept. of General Surgery, PMCH Patna Bihar India.

Received date: 01 July 2019 **Revised date:** 22 July 2019 **Accepted date:** 12 August 2019

***Corresponding author: Dr. (Prof) Vimal Mukesh**

Professor, Dept. of General Surgery, PMCH Patna Bihar India.

ABSTRACT

Objective: To determine the clinico-pathological features of Fournier's gangrene and to identify risk factors associated with the mortality. **Material and Methods:** This retrospective study was done at Patna Medical College and Hospital, Patna from May 2018 to April 2019. All consecutive patients with a diagnosis of Fournier's Gangrene were studied for clinico-pathological features, management and outcome. **Result:** There were 58 patients; all were male. The source of infection was urological in 30 patients, anorectal in 10 and cutaneous in 7, while no cause was found in 11 patients. Ma- jority presented with erythema and swelling of genitalia, while 33 had fever, 55 had pain, 24 crepitus, and 18 septic shock. E.coli was the most common organism isolated in 29 patients. Other organisms included Acinetobacter in 10, Klebsiella in 5 and Candida in 3 patients. The overall mortality rate was 30%. 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 man- agement with aggressive debridement, broad spectrum antibiotics and intensive supportive care has improved the outcome in recent years.

KEYWORDS: Fournier's gangrene, co-morbid, intensive treatment.

INTRODUCTION

Fournier's gangrene (FG) is a fulminant form of infective necrotising fascitis of the perineal, genital, or perianal regions, which commonly affects men. FG is a synergistic polymicrobial infection that leads to thrombosis of small subcutaneous vessels which ultimately lead to gangrene of the overlying skin.[1] Even though this clinical entity is eponymously credited to the Parisian venerologist Jean-Alfred Fournier, who described it as a fulminant gangrene of the penis and scrotum in young men,[2] Baurienne in 1764 and Avicenna in 1877 had described the same disease earlier.[3] Initially, FG was defined as an idiopathic entity, but diligent search will show the source of infection in the vast majority of cases, as either perineal and genital skin infections. The most common foci include the gastrointestinal tract (30%–50%), followed by the genitourinary tract (20%–40%), and cutaneous injuries (20%). Comorbid systemic disorders are being identified more and more in patients with FG, the commonest being diabetes mellitus and alcohol misuse. Diabetes mellitus is reported to be present in 20%–70% of patients with FG and chronic alcoholism in 25%–50% patients. The emergence of HIV into epidemic proportions has opened up a huge population at risk for developing FG. Table below lists the commonest causes and the comorbid risk factors.

Table:

Aetiology	Risk Factors
Perianal abscess	Diabetes
Rectal biopsy	Alcohol misuse
Diverticulitis	Immunosupression
Genital piercing	Chemotherapy
Perianal trauma	HIV
Hydrocele aspiration	Chronic steroid use
Prostatic biopsy	

The central principal of 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.

MATERIALS AND METHOD

Th is is a retrospective study done at Patna Medical College and Hospital, Patna from May 2018 to April 2019. 58 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 comorbids, they were shifted to the ward. Blood sample was taken for routine blood examinations like complete blood count (CBC), urea, creatinine, electrolytes and blood glucose. 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.

RESULTS

There were 58 male patients. The source of infection was urological in 30 patients (51.7%), anorectal was in 10 patients (17.2%) and cutaneous causes were found in 7 patients (12%), while no cause was found in 11 (18.9%) patients (Figure 1).

Majority of patients presented with erythema and swelling of genitalia, while 33 (56.8%) had fever, 55 (94.8%) had pain, 24 (41.3%) had crepitus, while 18 (31.0%) patients presented with full blown septic shock.

The most common co-morbidities included diabetes mellitus followed by renal failure, cerebrovascular accidents, hypertension, hepatitis C.

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 29 patients (50%). Other organisms included Acinetobacter in 10 (17.2%), Klebsiella in 5 (8.6%) and Candida in 3 (5.1%) patients. Mixed bacterial growth was found in 11 (18.9%) patients (Figure 2).

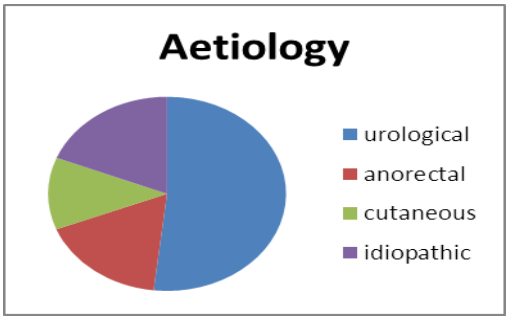

Figure-1.

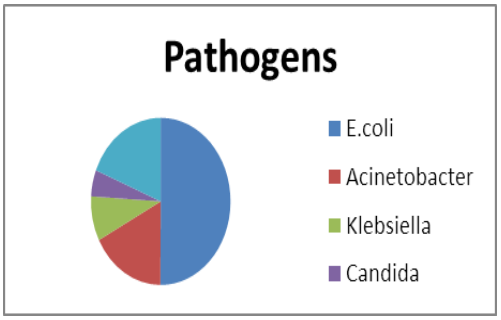

Figure-2.

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. 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 and anti-septic dressings. The overall mortality rate was 30%.

DISCUSSION

Fournier's gangrene, caused by synergistic aerobic and anaerobic organisms, is a life-threatening disorder in which infection of the perineum and scrotum spreads along fascial planes, leading to soft-tissue necrosis. This infectious was described by Jean Alfred Fournier, French dermatologist as a syndrome of unexplained sudden onset and rapidly progressing gangrene in the penis and scrotum of 5 young men with no other pathology basis of sudden onset and rapid progression. In its early reports Fournier's gangrene was described as an idiopathic entity, but in most cases a perianal infection, urinary tract and local trauma or skin condition at that level can be identified.[4] The mortality rate for FG is still high, (20–50%) in most contemporary series,[5,6] despite an increased knowledge of the etiology, diagnosis and treatment, and intensive-care techniques. The high mortality reflects both the aggressive nature of the infection and the destructive effects of accompanying predisposing factors. Several factors affecting the mortality were studied such as increasing age, primary anorectal infections, existence of diabetes, delay in treatment, evidence of systemic sepsis at presentation, extent and depth of involvement, a low haematocrit, a high leukocytosis and blood urea nitrogen, a high alkaline phosphatase and serum albumin, and many others. These and other studied variables that influence the outcome of patients with FG, in large part, remains controversial. In this purpose, the FGSI was developed to help clinicians predict the outcome of patients with FG and remains an objective and simple method to quantify

the extent of metabolic aberration at presentation in patients with FG. Numerous factors have been implicated at the onset of FG, in particular, those involving the immune system.[7] Diabetes mellitus was the most reported co-morbid disease associated with this pathology. Despite of being a risk factor for FG and associated with a more progressive and fatal outcome (decreased phagocytic and intracellular bactericidal activity and neutrophil dysfunction). Ultimately, occurrence of septic shock and need for postoperative mechanical ventilation, have been demonstrated as a powerful (even late) factors of mortality. Furthermore, Yanar *et al*. found that the presence of sepsis was as the only significant independent risk factor for mortality in FG.[8]

FG is commonly associated with a polymicrobial infection.[9] 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.[10] The causative pathogens act synergistically and release diff erent proteins and enzymes which cause thrombosis of small vessels, known as obliterative endarteritis. This is the key patho-physiological event. The underlying thrombosis results in a cutaneous and subcutaneous vascular necrosis. Most commonly isolated organism is E coli, followed by Bacteroides and Streptococcal species. Staphylococci, Peptostreptococci and Clostridia are also frequently isolated.[11] In the present study, we found E.coli was the most common organism found in tissue culture, i.e., in 29 patients (50%). Other organisms included Acinetobacter in 10 (17.2%), Klebsiella in 5 (8.6%) and Candida in 3 (5.1%) patients. Mixed bacterial growth was found in 11 (18.9%) patients.

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

Traditionally, aggressive haemodynamic stabilization, parentral broad spectrum antibiotics and repeated surgical debridement are the most crucial steps in the treatment of patients with Fournier's Gangrene.[12] 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 man- agement. In this study, suprapubic cystostomy was done in 70% of patients. Although most authors suggest that defect should be closed by secondary healing but there are many who prefer reconstructive surgery. In our study, secondary wound healing occurred in majority of our patients. Death rate is high ranging from 15-50% in literature. In our study, death rate was 30%.

CONCLUSION

FG is still a life threatening condition with unacceptably high death rates despite insights gained regarding the disease process. Diagnosis should be prompt with early surgical intervention, along with antibiotics and good supportive care. Radiography can be helpful when the clinical picture is not straightforward. Continued medical care in the form of a multidisciplinary approach is necessary as these patients may require reconstructive procedures in the future. Proactive management of the diabetic and immunosuppressed patients with perineal infections is of extreme importance to prevent the development of the condition in the first instance as this condition in the presence of such comorbidities is associated with high mortality.

REFERANCES

1. EKE N. Fournier's Gangrene: 4 review of 1726 cases; Br. J. Surg, 20; 87: 718-28.
2. Fournier J - A. Gangrene foudroyante de la verge. Semaine Medicale, 18833345–348.
3. Nathan B. Fournier's gangrene: a historical vignette. (Letter). Can J Surg, 19984172.
4. Sorensen MD, Krieger JN, Rivara FP, Klein MB, Wessells H: Fournier's gangrene: management and mortality predictors in a population based study. J Urol, 2009, 182: 2742-2747.
5. Eke N: Fournier's gangrene: a review of 1726 cases.
6. Morua AG, Lopez JA, Garcia JD, Montelongo RM, Guerra LS: Fournier's gangrene: our experience In 5 Years, bibliographic review and assessment of the Fournier's gangrene severity index.
7. Malik AM, Sheikh S, Pathan R, Khan A, Sheikh U: The spectrum of presentation and management of Fournier's gangrene-An Experience of 73 Cases. J Pak Med Assoc, 2010; 60: 617-619.
8. Yanar H, Taviloglu K, Ertekin C, Guloglu R, Zorba U, Cabioglu N, Baspinar I: Fournier's gangrene: risk factors and strategies for management. World J Surg, 2006; 30: 1750-1754.
9. Morpurgo E, Galandiuk S. Fournier's Gangrene. Surg Clin North Am, 2002; 82: 1213-24.
10. 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.
11. Adams JA, Culkin DJ, Mata JA Bocchini JA, Venable DD. Fournier's Gangrene in Children. Urology, 1990; 35: 439-41.
12. Corman JM, Moody JA, Aronsan WJ. Fournier's Gangrene in a modern surgical sett ing: improved survival with aggressive management. BJU int., 1999; 84: 85-88.