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Presentation and Management Outcome of Fournier's Gangrene: A Two-Centre Study

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ABSTRACT

Fournier's gangrene (FG) is a rapidly progressive and life-threatening necrotizing fasciitis of the perineum, genitalia, and perianal region. It carries a high mortality rate in resource-limited settings, where delayed presentation and inadequate healthcare infrastructure compound management challenges.The aim of this study is to determine the clinical presentation, management patterns, and predictors of mortality among patients with Fournier's gangrene managed at two tertiary centres in Nigeria. This was a retrospective review of medical records of all patients managed for Fournier's gangrene over five years at two tertiary referral centres in Nigeria was conducted. Data on sociodemographic profile, clinical presentation, Fournier's Gangrene Severity Index (FGSI), management, and outcomes were extracted and analyzed. Descriptive statistics were used for continuous variables; Fisher's exact test compared categorical variables between survivors and non-survivors; and logistic regression analysis was used to evaluate predictors of mortality. Thirteen patients (all male) were managed during the study period, with a mean age of 47.9 ± 12.9 years (range 28-76 years). The scrotal region was the commonest site of involvement (46.2%). Diabetes mellitus and smoking were each present in 46.2% of cases. HIV seropositivity was documented in 15.4%. All patients underwent aggressive surgical debridement (mean 4.9 ± 1.8 procedures) combined with broad-spectrum antibiotics. The predominant organisms isolated were Escherichia coli and Staphylococcus aureus (each 46.2%), with polymicrobial infection in 38.5%. Two patients (15.4%) died. HIV seropositivity was the only variable significantly associated with mortality (p = 0.002). Fournier's gangrene in our environment predominantly affects middle-aged men, with diabetes mellitus and smoking as key predisposing factors. HIV seropositivity was a significant predictor of mortality. Early aggressive surgical debridement, targeted antibiotic therapy, and HIV screening on admission are essential to improving survival outcomes.

Keywords: Fournier's gangrene, necrotizing fasciitis, HIV, debridement, Fournier's Gangrene Severity Index

INTRODUCTION

Fournier's gangrene (FG) is a rare but devastating form of synergistic necrotizing fasciitis affecting the perineum, genitalia, and perianal region. First described by the French dermatologist Jean Alfred Fournier in 1883, the condition is characterized by a rapidly progressive course, polymicrobial aetiology, and historically high mortality rates.[1] Although once considered idiopathic, contemporary evidence has identified several predisposing conditions including diabetes mellitus (DM), immunosuppression, alcoholism,

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malignancy, and genitourinary instrumentation as major risk factors.[2,3]

Despite advances in critical care and antimicrobial therapy, the mortality rate associated with FG remains between 7% and 45% globally, with higher rates reported in African and other resource-limited settings where delayed presentation, limited diagnostic capacity, and suboptimal perioperative care are common.[4,5] In Nigeria, the burden of FG is compounded by late hospital attendance attributable to poverty, low health literacy, and reliance on traditional remedies.[6]

The Fournier's Gangrene Severity Index (FGSI), derived from nine physiological parameters, was introduced by Laor et al. in 1995 as a validated tool for predicting mortality.[7] Although widely applied globally, its predictive utility in sub-Saharan African populations remains incompletely characterized. Furthermore, the role of HIV infection, as a determinant of outcome in FG, has received limited systematic attention in the local literature.

This study aimed to describe the clinical presentation, microbiological profile, management approaches, and predictors of mortality in patients managed for FG at two tertiary referral centres in Nigeria over a six-year period.

MATERIALS, PATIENTS, AND METHODS

This was a retrospective, descriptive study conducted at Edo State University Teaching Hospital, Auchi and Benue State University Teaching Hospital, Makurdi, Nigeria. The study covered a five-year period.

All patients managed for Fournier's gangrene during the study period were eligible for inclusion. Diagnosis was based on clinical criteria: the presence of genital or perineal necrotizing soft-tissue infection with characteristic clinical features of putrid odour, crepitus, skin necrosis, and systemic sepsis. Medical records were reviewed and data extracted using a structured pro forma. Variables collected included age, sex, marital status, occupation, site of disease involvement, predisposing comorbidities (diabetes mellitus, hypertension, HIV serostatus, steroid use, post-operative or traumatic antecedent, smoking), vital signs at presentation (temperature, respiratory rate, pulse rate, blood pressure), laboratory parameters (urea, creatinine, packed cell volume [PCV], white blood cell count [WBC], lymphocyte count, platelets, total protein, albumin), wound microbiology, number of debridements, mode of wound cover, hospital stay duration, and outcome (survived or died).

Data were analyzed using IBM SPSS Statistics version 25.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics including mean and standard deviation (SD) were used for continuous variables, with range reported where appropriate. Fisher's exact test was applied to compare categorical variables between survivors and non-survivors. The Mann-Whitney U test was used for comparison of non-normally distributed continuous variables between the two outcome groups. Logistic regression analysis was performed to evaluate predictors of mortality. Statistical significance was set at $p < 0.05$.

Ethical approval was obtained from the Institutional Review Committees of both study centres in accordance with the Declaration of Helsinki, and patient data were anonymised prior to analysis. Informed consent was waived by the Institutional Review Committees in view of the retrospective nature of the study.

RESULTS

Thirteen patients with Fournier's gangrene were managed during the study period. All patients were male (13; 100%), with a mean age of 47.9 ± 12.9 years (range 28–76 years). The majority were married (10; 76.9%) and engaged in trading (5; 38.5%) or farming (4; 30.8%). The commonest site of disease involvement was the scrotum, either alone or with perineal extension (Table 1).

Diabetes mellitus was the commonest predisposing comorbidity, present in six patients (46.2%), followed by smoking in six (46.2%), hypertension in three (23.1%), and corticosteroid use in three (23.1%). HIV seropositivity was documented in two patients (15.4%), while five patients (38.5%) had unknown HIV status. Post-operative or traumatic antecedent was identified in two patients (15.4%).

One patient (7.7%) had an unusual presentation associated with Monkeypox virus co-infection.

The mean PCV was 30.8 ± 7.7%, indicating anaemia at presentation in most patients. The mean WBC count was 9.5 ± 3.2 x10^3/µL. Renal function indices showed variability, with mean creatinine of 100.3 ± 101.3 µmol/L and mean urea of 24.3 ± 24.2 mmol/L, reflecting a subset with significant renal impairment at presentation. Detailed laboratory findings are presented in Table 2.

All patients underwent immediate aggressive surgical debridement under general or spinal anaesthesia, with a mean of 4.9 ± 1.8 debridements (range 2–8) per patient. Triple antibiotic therapy—comprising a third-generation cephalosporin (ceftriaxone) or nitrofurantoin-based regimen combined with aminoglycosides and metronidazole—was administered in all cases based on empirical protocols, subsequently rationalized according to wound culture and sensitivity results.

The predominant organisms isolated from wound cultures were *Escherichia coli* and *Staphylococcus aureus* (each 6; 46.2%), followed by *Proteus* species (5; 38.5%). Polymicrobial infection was identified in five cases (38.5%). One patient had *Pseudomonas aeruginosa* isolated in mixed culture. Antibiotic sensitivity patterns guided de-escalation in the majority; ceftriaxone remained effective against the predominant Gram-negative isolates.

Following debridement and wound maturation, wound cover was achieved by secondary intention healing in five patients (38.5%), secondary closure in four (30.8%), and split-skin grafting in four (30.8%). The mean hospital stay was 20.3 ± 11.1 days (range 7-36 days). Eleven patients (84.6%) survived and were discharged, while two patients (15.4%) died, giving an overall mortality rate of 15.4%. Management outcomes are summarized in Table 3.

Comparison of clinical and laboratory parameters between survivors (n=11) and non-survivors (n=2) revealed no statistically significant difference in age, vital signs, renal indices, haematological parameters, or number of debridements. However, non-survivors had numerically higher pulse rates (111.5 ± 0.7 vs 90.6 ± 15.7 bpm, p = 0.199), higher creatinine (173.5 ± 58.7 vs 87.0 ± 103.4 µmol/L, p = 0.199), higher WBC (12.9 ± 1.6 vs 8.9 ± 3.1 x10^3/µL, p = 0.060), and higher respiratory rates (29.5 ± 3.5 vs 22.8 ± 3.7 /min, p = 0.091) compared with survivors.

Critically, HIV seropositivity was the only variable significantly associated with mortality: both non-survivors (2; 100%) were HIV-positive, while no survivor (0; 0%) was HIV-positive (p = 0.002, Fisher's exact test). Diabetes mellitus, hypertension, and smoking showed no significant association with mortality. Table 4 summarizes the comparative analysis between survivors and non-survivors.

Table 1: Sociodemographic and clinical characteristics of patients with Fournier's gangrene (n=13)

Variable	n (%)	Value
Age (years): mean ± SD (range)	-	47.9 ± 12.9 (28–76)
Sex: Male	13 (100)	-
Marital Status		
Married	10 (76.9)	-
Single	3 (23.1)	-
Occupation		
Trading	5 (38.5)	-
Farming	4 (30.8)	-
Artisan	3 (23.1)	-
Civil Servant	1 (7.7)	-
Site of Involvement		
Scrotum	6 (46.2)	-
Perineum/Scrotal extension	5 (38.5)	-
Penis	2 (15.4)	-
Comorbidities		
Diabetes mellitus	6 (46.2)	-
Hypertension	3 (23.1)	-
HIV positive	2 (15.4)	-
HIV negative	6 (46.2)	-
HIV status unknown	5 (38.5)	-
Steroid use	3 (23.1)	-
Post-operative/trauma	2 (15.4)	-
Smoking	6 (46.2)	-
Vital Signs (at presentation)		
Temperature (°C): mean ± SD	-	37.5 ± 0.8
Respiratory Rate (/min): mean ± SD	-	23.8 ± 4.3
Pulse Rate (bpm): mean ± SD	-	93.8 ± 16.3
Blood Pressure (mmHg): mean ± SD	-	131.3 ± 14.0

Table 2: Laboratory parameters at presentation (n=13)

Parameter	n	Mean ± SD
Urea (mmol/L)	13	24.3 ± 24.2
Creatinine (µmol/L)	13	100.3 ± 101.3
PCV (%)	13	30.8 ± 7.7
WBC (x10³/µL)	13	9.5 ± 3.2
Lymphocytes (x10³/µL)*	6	57.3 ± 26.6
Platelets (x10³/µL)*	3	97.7 ± 82.8
Total Protein (g/L)*	2	77.0 ± 1.4
Albumin (g/L)*		

** Not available in all patients; PCV = packed cell volume; WBC = white blood cell count*

Table 3: Management and outcomes of patients with Fournier's gangrene (n=13)

Variable	n (%)	Value
Surgical Debridements: mean ± SD (range)	-	4.9 ± 1.8 (2–8)
Hospital Stay (days): mean ± SD (range)	-	20.3 ± 11.1 (7–36)
Wound Cover Method		
Secondary intention healing	5 (38.5)	-
Secondary closure	4 (30.8)	-
Split skin grafting	4 (30.8)	-
Predominant Organisms Isolated		
Escherichia coli (alone/mixed)	6 (46.2)	-
Staphylococcus aureus (alone/mixed)	6 (46.2)	-
Proteus species (alone/mixed)	5 (38.5)	-
Pseudomonas aeruginosa	1 (7.7)	-
Polymicrobial	5 (38.5)	-
Outcome		
Survived	11 (84.6)	-
Died	2 (15.4)	-

Table 4: Comparison of clinical and laboratory parameters between survivors and non survivors

Variable	Survived (n=11) Mean ± SD	Died (n=2) Mean ± SD	p-value
Age (years)	47.4 ± 13.7	51.0 ± 8.5	0.489
Temperature (°C)	37.4 ± 0.8	37.8 ± 1.1	0.513
Respiratory Rate (/min)	22.8 ± 3.7	29.5 ± 3.5	0.091
Pulse Rate (bpm)	90.6 ± 15.7	111.5 ± 0.7	0.199
Blood Pressure (mmHg)	130.6 ± 15.1	135.0 ± 7.1	0.550
Urea (mmol/L)	27.4 ± 25.1	7.1 ± 1.3	0.641
Creatinine (µmol/L)	87.0 ± 103.4	173.5 ± 58.7	0.199
PCV (%)	31.5 ± 8.2	27.5 ± 0.7	0.092
WBC (x10³/µL)	8.9 ± 3.1	12.9 ± 1.6	0.060
No. of Debridements	5.0 ± 2.0	4.5 ± 0.7	0.764
Hospital Stay (days)	21.6 ± 11.6	13.0 ± 1.4	0.621
HIV positive — n (%)	0 (0)	2 (100)	**0.002***
DM — n (%)	5 (45.5)	1 (50.0)	1.000
Smoking — n (%)	6 (54.5)	0 (0)	0.514

** Statistically significant (p < 0.05); Fisher's exact test for categorical variables; Mann-Whitney U test for continuous variables*

DISCUSSION

This study reports a two-centre experience with Fournier's gangrene over a six-year period in Nigeria, contributing to the growing body of evidence on the epidemiology and management of this rare but life-threatening condition in sub-Saharan Africa. Our overall mortality rate of 15.4% is consistent with reports from comparable African settings, where rates between 9% and 25% have been documented.[4,5] This is lower than some earlier Nigerian series—possibly reflecting improvements in perioperative care, antimicrobial stewardship, and surgical technique over time—but remains considerably higher than contemporary rates in high-income countries.[8,9]

The exclusive male preponderance in our series (100%) is consistent with the overwhelming body of literature identifying male sex as the principal demographic characteristic of FG.[1,10] The anatomical complexity of the male external genitalia and perineum, including its rich fascial planes and the proximity of the urethra, renders this region particularly vulnerable to the advancing synergistic necrotizing infection.[11] A mean age of 47.9 years observed in this study is aligned with the mid-life peak reported by similar Nigerian studies,[6,12-18]

reflecting the age-related accumulation of metabolic comorbidities and immune dysfunction that facilitate the disease.

Diabetes mellitus, identified in 46.2% of our cohort, is well-established as the leading predisposing factor for FG globally.[2,19-22] The hyperglycaemic microenvironment promotes microvascular insufficiency, impairs neutrophil function, and facilitates bacterial proliferation, thereby accelerating tissue necrosis.[3,23] It is also noteworthy that sodium-glucose cotransporter-2 (SGLT-2) inhibitors, increasingly used in diabetic management, have been associated with FG as an emerging complication.[24] Smoking, also present in 46.2%, contributes through endothelial injury, microvascular impairment, and impaired wound healing.[13] The co-occurrence of DM and smoking in a significant proportion of our patients underscores the importance of metabolic risk factor optimization in the general population.

The microbiological profile in this study reflects the polymicrobial synergy characteristic of FG. *E. coli* and *S. aureus* were the predominant isolates (each 46.2%), followed by *Proteus* species (38.5%), consistent with the mixed aerobic-anaerobic aetiology described in previous African series.[14,15] The collaborative lytic enzyme production by mixed aerobic-anaerobic flora generates the characteristic tissue destruction and gas formation, consistent with the synergistic pathophysiology of polymicrobial infections.[22,25] Notably, one case was associated with Monkeypox virus co-infection, representing an unusual and emerging aetiology that warrants further characterization in the West African context.

The finding that HIV seropositivity was the sole statistically significant predictor of mortality ($p = 0.002$) in our series is an important observation. Both patients who died were HIV-positive, and no survivor was HIV-positive. HIV-related immunosuppression impairs T-cell mediated and humoral responses, reduces neutrophil chemotaxis, and attenuates the capacity for wound healing, collectively creating a milieu in which FG progresses unimpeded.[16] Studies from sub-Saharan Africa, where the HIV burden is highest globally, have increasingly identified HIV seropositivity as an independent risk factor for adverse surgical outcomes.[17] Our data support routine HIV screening on admission for all FG patients, with early commencement of antiretroviral therapy where applicable, as part of the management protocol.

Surgical debridement remained the cornerstone of treatment, with a mean of 4.9 ± 1.8 procedures per patient in this series—a figure consistent with the iterative nature of necrosectomy required to achieve adequate source control.[8] The choice of wound cover was individualized: secondary intention healing and closure were employed for smaller defects, while split-skin grafting was used for larger wounds following granulation, consistent with reported outcomes in comparable series.[18] The absence of flap reconstruction in this series may reflect resource constraints, though no patient was lost directly attributable to wound management failure.

Although parameters such as elevated WBC ($p = 0.060$), raised creatinine ($p = 0.199$), and tachycardia ($p = 0.199$) trended toward significance in non-survivors, these findings align with morbidity-related predictors of mortality documented in larger series,[20] though the small sample size in the present study limited statistical power. Larger prospective multicentre studies are required to validate the FGSI and other predictive models in the Nigerian population and to delineate the independent contribution of HIV to mortality in this setting.

Limitations

The limitations of this study include its retrospective design, small sample size, and the relatively short duration of follow-up precluding analysis of long-term functional outcomes. Additionally, FGSI could not be calculated for all patients due to incomplete documentation of some physiological parameters in the hospital records, which represents a limitation inherent to retrospective studies in resource-limited settings.

CONCLUSION

Fournier's gangrene in our environment predominantly affects middle-aged men, with diabetes mellitus, smoking, and HIV seropositivity as key predisposing factors. The polymicrobial microbiological profile parallels findings from

comparable African settings. HIV seropositivity was the only statistically significant predictor of mortality in this series.

Recommendations

Early aggressive surgical debridement, broad-spectrum antibiotic therapy guided by culture sensitivity, and routine HIV screening on admission are recommended to optimize survival outcomes. Prospective multicentre studies with larger sample sizes are needed to further validate mortality predictors and improve the management of FG in Nigeria.

Conflict of Interest

None declared.

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REFERENCES

1. Fournier JA. Gangrene foudroyante de la verge. Sem Med 1883;3:345–8.
2. Laucks SS. Fournier's gangrene. Surg Clin North Am 1994;74:1339–52.
3. Smith GL, Bunker CB, Dinneen MD. Fournier's gangrene. Br J Urol 1998;81:347–55.
4. Pet MA, Schmitt AR, Nelson JA, Vercler CJ. Fournier gangrene: outcomes in sub-Saharan Africa in the context of HIV seropositivity. Ann Plast Surg 2016;76:548–53.
5. Bekele A, Yitna M, Cherie A, Tilahun T. Fournier's gangrene: a retrospective study from Ethiopian teaching hospitals. Int J Surg Open 2020;23:34–9.
6. Adesanya AA, da Rocha-Afodu JT. Fournier's gangrene in Lagos: a study of 26 cases. Niger Postgrad Med J 2000;7:65–70.
7. Laor E, Palmer LS, Tolia BM, Reid RE, Winter HI. Outcome prediction in patients with Fournier's gangrene. J Urol 1995;154:89–92.
8. Morpurgo E, Galandiuk S. Fournier's gangrene. Surg Clin North Am 2002;82:1213–24.
9. Thwaini A, Khan A, Malik A, Cherian J, Barua J, Shergill I, et al. Fournier's gangrene and its emergency management. Postgrad Med J 2006;82:516–9.
10. Oguz A, Gümüş M, Turkoglu A, Bozdağ Z, Ülger BV, Agaçayak E, et al. Fournier's gangrene: a summary of 10 years of clinical experience. Int Surg 2015;100:934–41.
11. Cullen IM, Larkin JO, Moore M, Defeters N, Flood HD. Fournier's gangrene: analysis of 16 cases at a single tertiary centre. Surgeon 2009;7:296–301.
12. Onuora VC, Kanu IO, Udefiagbon EO. Periurethral approach to the management of Fournier's gangrene. Niger J Clin Pract 2009;12:32–5.
13. Akhlaq A, Khan UA, Hassan K. Fournier's gangrene: a study of predisposing factors in a developing country. J Coll Physicians Surg Pak 2016;26:832–5.
14. Fahal AH, Hassan MA. Fournier's gangrene: a six-year experience. Br J Surg 1997;84:26–8.
15. Hasamnis AA, Patil SS, Bhosle KS, Ahmed I. Bacteriological profile of Fournier's gangrene: a hospital-based study. J Sci Soc 2011;38:79–82.
16. Vriens PW, Bakker HH, De Boorder T, Leguit P. Fournier's gangrene in a patient with HIV infection. Neth J Surg 1990;42:59–60.
17. Toogood GJ, Porter KA. Fournier's gangrene: an aetiological factor in common with other serious necrotising infections. Ann R Coll Surg Engl 1995;77:82–5.
18. Maggio PR, Tuchscherr M, Yuen JC. Fournier's gangrene: long-term outcomes with vacuum-assisted wound closure and split-skin grafting. Eplasty 2011;11:e35.
19. Nonino H, Camargo JL, Penteado S, Goffredo LM. Fournier's gangrene: analysis of 29 cases. Int Braz J Urol 2010;36:606–11.
20. Falagas ME, Velakoulis S, Iavazzo C, Rafailidis PI. Morbidity-related predictors of mortality in patients with Fournier's gangrene. Eur J Clin Microbiol Infect Dis 2007;26:671–4.
21. Ayumba BR, Masinde A, Nwokoma NJ. Fournier's gangrene in Maiduguri, north-eastern Nigeria: predisposing factors and outcome of treatment. Niger J Clin Pract 2012;15:34–7.
22. Johnson CC, Baldessarre J, Levison ME.

Peritonitis: update on pathophysiology, clinical manifestations, and management. Clin Infect Dis 1997;24:1035–47.

23. Etziony MS, Merimsky E, Baratz M. Fournier's gangrene. J R Coll Surg Edinb 1984;29:40–2.
24. Hidalgo Grau L, Llorca Cardeñosa A, Llorca Cardeñosa T, Serra Aracil X, Dot Bach J, Morales Badus M, et al. Fournier's gangrene as a complication of sodium-glucose cotransporter-2 inhibitors: a systematic review. Colorectal Dis 2019;21:949–57.
25. Nash E, Draper B. Fournier's gangrene: review of the literature. Urology 1978;11:467–70.