

Fournier gangrene as a polymicrobial infection complication in a diabetic patient: a case report

Gangrena de Fournier como complicación de una infección polimicrobiana en un paciente diabético: informe de un caso

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SUMMARY

Introduction: Fournier gangrene (FG) is a rare but life-threatening necrotizing fasciitis involving the genital and perineal regions, with mortality rates reaching up to 88%. The disease is typically caused by a polymicrobial infection involving both aerobic and anaerobic bacteria. Diabetes mellitus is the most common predisposing factor and is present in more than half of FG cases. **Case overview:** We report the case of a 58-year-old man with progressive painful scrotal swelling that initially appeared as a small lesion but gradually enlarged with discharged pus. The patient had a history of uncontrolled diabetes mellitus. Laboratory investigations showed hemoglobin 10.5 g/dL, leukocytosis ($22.03 \times 10^3/\mu\text{L}$), thrombocytosis ($583 \times 10^3/\mu\text{L}$), hyperglycemia (334 mg/dL), hypoalbuminemia (2.2 g/dL), elevated HbA1c (10.3%), and increased procalcitonin (3.36 ng/mL). Urinalysis demonstrated hematuria, leukocyturia, proteinuria, and significant bacteriuria. Urine culture grew *Pseudomonas aeruginosa*, while pus cultures

yielded *Escherichia coli* and *Proteus mirabilis*. The patient received empirical cefotaxime and metronidazole therapy followed by surgical debridement. Antibiotic treatment was subsequently adjusted to ceftazidime according to culture and susceptibility results. Following six days of treatment, including adequate glycemic control, marked clinical improvement was observed, and the patient was discharged on oral ciprofloxacin. **Conclusion:** FG requires prompt management to achieve favorable outcomes and prevent severe complications.

Keywords: Fournier gangrene, polymicrobial, antibiotics

RESUMEN

Introducción: La gangrena de Fournier (GF) es una fascitis necrosante poco frecuente pero potencialmente mortal que afecta las regiones genital y perineal, con tasas de mortalidad de hasta el 88%. La enfermedad suele deberse a una infección polimicrobiana que involucra bacterias aerobias y anaerobias. La diabetes mellitus es el factor predisponente más común y se observa en más de la mitad de los casos de GF. **Presentación del caso:** Presentamos el caso de un hombre de 58 años con inflamación escrotal dolorosa y progresiva que inicialmente se manifestó como una pequeña lesión que aumentó gradualmente

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*con secreción purulenta. El paciente tenía antecedentes de diabetes mellitus no controlada. Los análisis de laboratorio mostraron hemoglobina de 10,5 g/dL, leucocitosis ($22,03 \times 10^3/\mu\text{L}$), trombocitosis ($583 \times 10^3/\mu\text{L}$), hiperglucemia (334 mg/dL), hipoalbuminemia (2,2 g/dL), HbA1c elevada (10,3 %) y procalcitonina aumentada (3,36 ng/mL). El análisis de orina demostró hematuria, leucocituria, proteinuria y bacteriuria significativa. El urocultivo reveló *Pseudomonas aeruginosa*, mientras que los cultivos de pus resultaron positivos para *Escherichia coli* y *Proteus mirabilis*. El paciente recibió tratamiento empírico con cefotaxima y metronidazol, seguido de desbridamiento quirúrgico. Posteriormente, el tratamiento antibiótico se ajustó a ceftazidima según los resultados del cultivo y de la prueba de sensibilidad. Tras seis días de tratamiento, con control glucémico adecuado, se observó una notable mejoría clínica y el paciente fue dado de alta con ciprofloxacino por vía oral. Conclusión: La gangrena de Fournier requiere un manejo inmediato para lograr resultados favorables y prevenir complicaciones graves.*

Palabras clave: Gangrena de Fournier, polimicrobiana, antibióticos

INTRODUCTION

Fournier gangrene (FG) is a form of necrotizing fasciitis involving the genital, perineal, and perianal regions. It is a rare condition, accounting for approximately 0.02% of all hospitalized patients, but is associated with a high mortality rate ranging from 20% to 40%, with some reports documenting mortality rates as high as 88%. The disease is caused by bacterial infection, with the primary source of infection typically originating from the genitourinary tract, anorectal region, or surrounding soft tissues of the genital area (1,2). This disease originates from a soft tissue infection caused by a mixture of aerobic and anaerobic bacteria. These organisms produce enzymes such as collagenases that promote rapid tissue destruction and facilitate the spread of infection along fascial planes. Consequently, the infection may progress rapidly from the genital and perineal regions to the anterior abdominal wall and adjacent vital structures (3,4).

FG commonly occurs in elderly patients and in those with comorbid conditions such as diabetes mellitus, alcoholism, atherosclerosis, peripheral arterial disease, malnutrition, prostate cancer, human immunodeficiency virus (HIV) infection, leukemia, and liver disease. Among these, diabetes mellitus is the most frequently identified risk factor, being present in more than half of patients with FG. Furthermore, diabetes may exacerbate the disease course through impaired immune function, tissue ischemia, and microvascular complications, thereby promoting rapid infection progression and tissue necrosis (3,5,6).

Prompt diagnosis and appropriate treatment, including surgical intervention and targeted antibiotic therapy, are essential components in the management of FG, as recovery is often prolonged and the disease is associated with significant morbidity (7). Early and adequate treatment may also prevent long-term urological complications and disability (3). In addition, optimal glycemic control is a critical aspect of management in patients with diabetes mellitus, as uncontrolled hyperglycemia can impair immune function and delay wound healing (8). In this article, we present the case of a 58-year-old man with FG complicated by a polymicrobial infection and discuss the comprehensive multidisciplinary management of this condition.

Case overview

A 58-year-old man was referred from Pasuruan Regional General Hospital with a chief complaint of scrotal swelling. The swelling initially appeared as a small lesion but progressively enlarged, became blackish in color, and was associated with purulent and bloody discharge. The patient also reported severe scrotal pain. He had a history of poorly controlled diabetes mellitus for the past year and had never received treatment for the condition. Physical examination of the right scrotum revealed edema, hyperemia, fluctuation,

and active bleeding. The affected area was warm to palpation and markedly tender, as shown in Figure 1.



Figure 1. Clinical appearance of the infected scrotal area at presentation. The lesion demonstrated marked edema, hyperemia, fluctuation, and active bleeding, consistent with severe soft tissue infection

Laboratory analysis revealed anemia with a hemoglobin level of 10.5 g/dL. Marked leukocytosis was present, with a white blood cell count of $22.03 \times 10^3/\mu\text{L}$ and neutrophil predominance (89.1%). Thrombocytosis was also observed, with a platelet count of $583 \times 10^3/\mu\text{L}$. Clinical chemistry results demonstrated severe hyperglycemia with a blood glucose level of 334 mg/dL and poor glycemic control reflected by an HbA1c of 10.3%. Hypoalbuminemia was noted, with a serum albumin level of 2.2 g/dL, while procalcitonin was elevated at 3.36 ng/mL, suggesting a significant bacterial infection.

Urinalysis showed a specific gravity of 1.016 and a pH of 5.50, with glucose 3+, urobilinogen 2+, ketones 2+, and protein 1+. Evidence of urinary tract inflammation and infection was demonstrated by leukocytes of $203.8/\mu\text{L}$, leukocyte esterase 3+, erythrocytes of $33.24/\mu\text{L}$, and bacteriuria with 36.68 bacteria/HPF and 663.01/LP. Additional findings included epithelial cells at $16.8/\mu\text{L}$, squamous epithelial cells at 3712/LP, and hyaline casts at 3.22/LP. The results of microbial culture and antibiotic susceptibility tests of urine and pus from scrotal tissue were shown in Tables 1 and 2, respectively.

The clinical presentation and microbiological findings led to a diagnosis of FG. The patient underwent surgical management with debridement and necrectomy in the scrotal region to remove necrotic tissue and control the infection. The post-debridement result is shown in Figure 2. The patient was also given empirical antibiotic therapy with cefotaxime 4×2 gram and metronidazole 3×500 mg. After the culture and sensitivity results became available, the antibiotic regimen was changed to ceftazidime 3×1 gram.

Blood glucose control was managed with subcutaneous NovoRapid 3×6 IU and Levemir 1×12 IU in the morning. The patient also received a 20% albumin infusion to correct hypoalbuminemia. On the seventh postoperative day, clinical improvement was observed. Therefore, on the next day, the antibiotics were switched to oral ciprofloxacin 2×500 mg, and the patient was subsequently discharged.

DISCUSSION

FG involves a synergistic infectious process between aerobic and anaerobic bacteria, resulting in a localized infection of the perineum, scrotum, or genitalia that can rapidly progress to extensive tissue necrosis. The bacterial infection often begins with an initial source such as a urinary tract infection, perianal abscess, local cellulitis, a history of surgical procedures in the genital or perineal area, or even minor skin injuries such as scratches, abrasions, or acne lesions. The synergistic activity of these bacteria leads to the production of tissue-damaging enzymes, collagenase, and endotoxins, resulting in obliterative endarteritis with microvascular thrombosis in the subcutaneous vessels. This ultimately causes ischemic gangrene of the affected structures and rapid spread of infection to adjacent and surrounding tissues. The progression can occur very rapidly, even within hours. In the patient's history, the exact source of infection could not be definitively identified; however, it was likely related to a urinary tract or perianal source associated with poor hygiene (4).

Table 1. Microbiology identification and susceptibility testing from the urine specimen

Urine Culture	
Organism	<i>Pseudomonas aeruginosa</i> $\geq 10^5$ CFU/mL
Drug sensitivity test	
Sensitive	Amikacin, Amoxicillin-Clavulanate, Aztreonam, Cefepime, Cefoperazone-sulbactam, Cefotaxime, Cefoxitin, Ceftazidime, Ceftazidime-avibactam, Ceftriaxone, Chloramphenicol, Fosfomycin, Imipenem, Meropenem, Piperacillin-Tazobactam
Intermediate	Ampicillin-Sulbactam
Resistant	Ampicillin, Ciprofloxacin, Gentamicin, Levofloxacin, Moxifloxacin, Tetracycline

Table 2. Microbiology identification and susceptibility testing from the pus specimen

Pus Culture	
Organism	<i>Escherichia coli</i> and <i>Proteus mirabilis</i>
Drug sensitivity test of <i>Escherichia coli</i>	
Sensitive	Amikacin, Amoxicillin-Clavulanate, Ampicillin, Ampicillin-Sulbactam, Aztreonam, Cefepime, Cefoperazone-Sulbactam, Cefotaxime, Ceftazidime, Ceftriaxone, Ciprofloxacin, Gentamicin, Imipenem, Levofloxacin, Meropenem, Moxifloxacin, Piperacillin-Tazobactam, Tigecycline
Resistant	Tetracycline, Trimethoprim-Sulfamethoxazole
Drug sensitivity test of <i>Proteus mirabilis</i>	
Sensitive	Amikacin, Amoxicillin-Clavulanate, Ampicillin, Ampicillin-Sulbactam, Cefepime, Cefoperazone-Sulbactam, Cefotaxime, Ceftazidime, Ceftriaxone, Ciprofloxacin, Gentamicin, Levofloxacin, Meropenem, Moxifloxacin, Piperacillin-Tazobactam, Tigecycline, Trimethoprim-Sulfamethoxazole
Resistant	Tetracycline

A history of uncontrolled diabetes mellitus was a major predisposing factor for FG in this patient. Type 2 diabetes mellitus increases susceptibility to FG through immune dysfunction, microvascular complications, tissue ischemia, and impaired wound healing. These abnormalities facilitate rapid progression of infection, resulting in extensive tissue necrosis and potentially life-threatening complications. Therefore, optimal

glycemic control is a critical component of FG management. In our patient, insulin therapy was administered to achieve adequate glycemic control, which likely contributed to infection control and wound healing (6,8).

Laboratory examinations play an important role in both the diagnosis and prognostic assessment of patients with FG. The patient's laboratory results



Figure 2. Postoperative clinical appearance. The necrotic tissue has been removed, leaving viable tissue

showed elevated leukocyte count and procalcitonin levels, indicating a bacterial infection. The patient also developed anemia due to an inflammatory state in which pro-inflammatory cytokines such as IL-6 increase hepcidin production, thereby inhibiting iron release from body stores. This reduces iron availability for erythropoiesis, despite adequate erythropoietin (EPO) production. In addition, pro-inflammatory cytokines such as TNF- α , IL-1, and IL-6 can reduce the bone marrow response to erythropoietin. Thrombocytosis in this patient was a reactive thrombocytosis due to infection. During inflammation, elevated IL-6 levels stimulate hepatic thrombopoietin (TPO) mRNA expression, leading to increased plasma TPO levels and, consequently, elevated platelet counts. The patient also had hypoalbuminemia as part of the negative acute-phase protein response to infection. Urinalysis revealed hematuria, leukocyturia, proteinuria, and a high bacterial count, indicating infection as well as complications related to poorly controlled diabetes mellitus (9,10).

Microbial examination with culture and antibiotic susceptibility tests is essential for

identifying the causative microorganisms, which are typically polymicrobial infections involving both aerobic and anaerobic bacteria. The culture results serve as a guide for selecting appropriate broad-spectrum antibiotic therapy, a crucial component of FG management alongside surgical debridement (3).

Antibiotic therapy in diabetic patients with FG plays a central role in the management of this life-threatening necrotizing infection, particularly as it involves the perineal, genital, and perianal regions and progresses aggressively with a risk of small vessel thrombosis and subcutaneous tissue necrosis (3). The patient received antibiotic therapy consisting of intravenous cefotaxime 4×2 g per day and metronidazole 3×500 mg per day. This combination is used to treat the polymicrobial infection commonly seen in FG, covering both aerobic and anaerobic bacteria (3,8). Subsequently, the antibiotic regimen was adjusted to ceftazidime 1 g three times daily based on the antimicrobial susceptibility test results, as *Pseudomonas aeruginosa* is intrinsically resistant to cefotaxime (11).

Parenteral administration is preferred in the initial phase to ensure optimal drug distribution and a rapid bactericidal effect. The usual duration of therapy is at least two weeks and should be adjusted according to clinical response and microbiological culture results (3). In our patient, intravenous antibiotic therapy was administered for one week. With progressive clinical improvement, the patient was eventually discharged with oral antibiotic therapy, ciprofloxacin 2x500 mg per day (12).

The final and equally important management approach is surgical intervention in the form of debridement and necrectomy in the scrotal region, as well as incision and drainage of abscesses. These procedures are essential components of the primary treatment of Fournier's gangrene, removing necrotic tissue and controlling the spread of infection. This intervention is crucial to halt the rapid progression of infection through fascial planes and to reduce the bacterial load. Surgical intervention should not be delayed for diagnostic imaging or other supporting examinations when clinical suspicion is high, as any delay may worsen the prognosis (3). The patient was also provided with education on hygiene practices as part of the management plan to accelerate wound healing and prevent recurrent infection (13).

CONCLUSION

FG is a rapidly progressive necrotizing infection of the perineal and genital region that requires early diagnosis and immediate surgical and medical management. In our case, the patient was successfully managed with prompt surgical debridement, appropriate antibiotics adjusted based on culture results, and supportive care, including glycemic control. This case highlights the importance of early intervention and multidisciplinary management in improving outcomes.

Informed Consent Statement

Written informed consent has been obtained from the patient for the publication of this paper.

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Conflict of Interest

The authors declare no conflict of interest.

Authors' contributions

All authors made significant contributions to the article.

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