International Surgery Journal
Alla M et al. Int Surg J. 2024 Nov;11(11):1866-1869
http://www.ijsurgery.com
pISSN 2349-3305 | eISSN 2349-2902

DOI: https://dx.doi.org/10.18203/2349-2902.isj20243245

Case Report

A rare case of postoperative Fournier's gangrene after a hemorrhoidectomy

Mrinalini Alla[1], Chloe Bodden[1], Patrick Kamere Kiarie[2], Noman Khan[1], Martine A. Louis[1]*, Neil Mandava[1]

[1]Department of Surgery, Flushing Hospital Medical Center, Flushing, New York
[2]St. George's University, School of Medicine, Grenada, West Indies

Received: 28 August 2024
Accepted: 03 October 2024

***Correspondence:**
Dr. Martine A. Louis,
E-mail: mlouis2.flushing@jhmc.org

ABSTRACT

Fournier's gangrene (FG) is a severe and fulminant necrotizing fasciitis of the perineal, genital, and perianal regions. Extensive postoperative infections after anorectal surgeries are rare, and the development of FG after hemorrhoid surgery is extremely rare. We present the case of a 68-year-old diabetic male found to have a severe necrotizing infection of the perianal and perineal region eight days after a hemorrhoidectomy. His hospital course was complicated by septic shock, acute kidney injury, respiratory failure, and multiple strokes. The purpose of this case is to increase awareness of extensive necrotizing infections such as FG, complicating a hemorrhoidectomy, albeit rarely, especially in patients with comorbidities such as diabetes mellitus.

Keywords: Hemorrhoid surgery, FG, Necrotizing fasciitis, Postoperative complication

INTRODUCTION

Fournier's gangrene (FG), first described by Jean Alfred Fournier in 1883, is an emergent condition characterized by progressive necrotizing infection affecting the external genitalia or perineum.[1] This condition primarily afflicts males, with an incidence of 1.6 per 100,000 in US.[1,2] Etiology of FG is infectious, stemming from synergistic activity of aerobic and anaerobic bacteria.[2] Trans-anal procedures are typically not associated with infections or septic complications due to rich bacterial flora in anorectal region; incidence of FG after anal procedures remains exceedingly low.[3] Identified risk factors for FG include diabetes mellitus (DM), chronic alcohol abuse, steroid use, malignancy, HIV and CVD.[1,2,4,5]

Diagnosis of FG is infrequent, with population-based epidemiological studies indicating that only 1% of hospitals in the United States manage more than five cases of FG annually. Contemporary mortality rates range from 20-40%. Moreover, outcomes hinge on comorbidities, early recognition, administration of broad-spectrum antibiotics, resuscitation, aggressive debridement, and potentially on clinical volumes and institutional disparities between teaching and non-teaching hospitals. Additional factors, such as necessity for further procedures and organ failure necessitating mechanical ventilation and dialysis, contribute to heightened morbidity and prolonged hospital stays.[6]

Here, we present a case of postoperative necrotizing fasciitis after anorectal surgical procedures, highlighting FG's emergence following an uncomplicated closed hemorrhoidectomy. Given FG's rarity, high mortality rate, and associated complications, any insights aiding physicians in swift diagnosis and management hold significant value, potentially enhancing patient outcomes.

CASE REPORT

A 68-year-old male with a past medical history of type 2 insulin-dependent diabetes mellitus, hypertension, and

hyperlipidemia presented to the emergency department (ED) with hyperglycemia and altered mental status. As per collateral from his wife, the patient had a recently closed hemorrhoidectomy eight days prior and developed anorexia, fevers, and perineal pain since then, culminating in confusion, loss of consciousness, and seizures the morning of the presentation. The patient was afebrile and saturating at 95% but agitated. His vital signs were a blood pressure of 137/64 mmHg (mean arterial pressure of 88 mmHg), heart rate of 100 beats per minute and respiratory rate of 20 breaths per minute.

Laboratory values included a white blood cell (WBC) count of 27.3 K/uL, a platelet count of 392 K/uL, and hemoglobin of 13.2 g/dL, hyperosmolar hyperglycemic syndrome with a blood glucose of 902 mg/dL, bicarbonate of 17 mmol/L, total bilirubin of 1.2 mg/dL, a normal anion gap, lactate of 3.01 mmol/L, an elevated beta-hydroxybutyrate of 2.56 mmol/L, blood urea nitrogen (BUN) and creatinine (Cr) of 36 mg/dL and 1.7 mg/dL, respectively. C-reactive peptide (CRP) 40 mg/dL and sodium 137 mmol/L. His laboratory risk indicator for necrotizing fasciitis (LRINEC) score of 10 points, putting him at high risk for necrotizing soft tissue infection. While on a non-rebreather of 100% fractional inspired oxygen (FiO_2), arterial blood gas (ABG) showed a pH of 7.34, pCO_2 of 34, and pO2 of 132 and a P/F ratio of 132. Urinalysis showed ketones, glycosuria, and proteinuria. The patient's initial sequential organ failure assessment (SOFA) score was 7. In the context of altered mental status, the patient underwent a non-contrast computed tomography (CT) scan that showed chronic ischemic changes without acute intracranial hemorrhage; CT angiography was negative for large vessel occlusion with appropriate brain perfusion. The electroencephalogram (EEG) was mildly abnormal, and the patient was started on levetiracetam.

Examination of the perineum revealed a discoloration over the perineum and scrotum, necrotic tissue in the previous surgical site, and crepitus on the rectal exam at 11 o'clock (Figure 1). CT abdomen and pelvis (CTAP) revealed perianal gas and wall thickening of the anus, highly suspicious for necrotizing fasciitis in the pelvis involving the perianal soft tissues and extending to the lower pelvis (Figures 2 and 3). He was started on clindamycin, piperacillin-tazobactam, and vancomycin. He was admitted to the MICU and had acutely worsening mental status and increased oxygen requirements; the decision was made to intubate the patient as he was not protecting his airway.

The patient was intubated and sedated and started on an insulin drip. He underwent an operative debridement of the rectum and perineum with multiple incisions parallel to the anus, revealing copious amounts of murky, "dishwater" fluid. Penrose drains were inserted into the right and left pelvis's deeper presacral space. He developed septic shock postoperatively in the surgical intensive care unit (SICU) and received vasopressors. His antibiotic combination consisted of clindamycin, piperacillin-tazobactam, and tigecycline tailored to wound culture growth of pan-sensitive *Escherichia coli*, abnormal *Streptococcus viridians*, *Streptococcus anginous*, *and Bacteroides fragilis.*

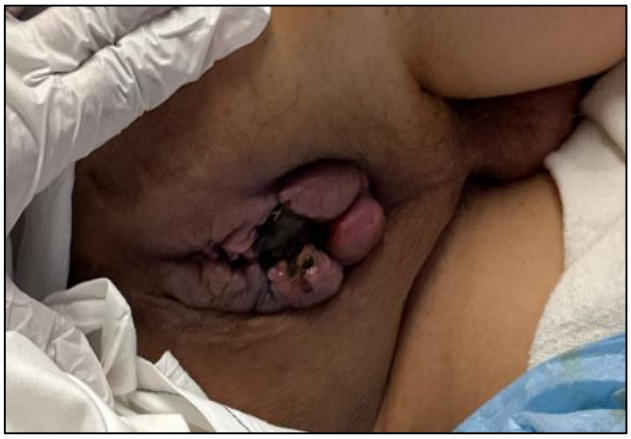
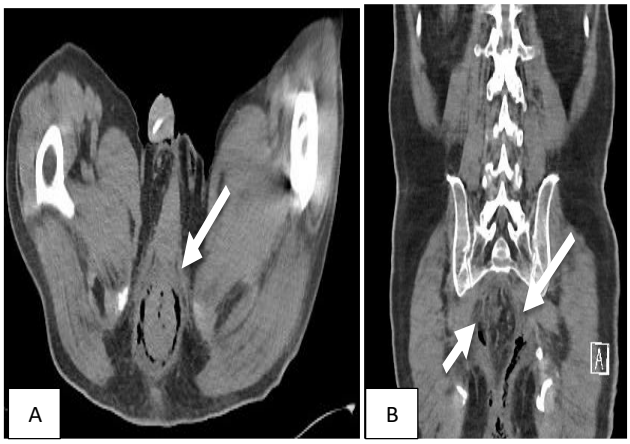


Figure 1: Visual examination of the perineal and perianal region showing discoloration over the perineum and scrotum, necrotic tissue in the previous hemorrhoid surgical site.



Figures 2 (A and B): Cross sectional and coronal CTAP showing wall thickening of the anus with arrows pointing to perianal gas and concerning for necrotizing fasciitis in the pelvis involving the perianal soft tissues and extending to the lower pelvis.

On postoperative day (POD) 5, in the context of persistent fevers and recurrent leukocytosis, a repeat CTAP showed a 7×1.5 cm fluid collection in the right pelvis and a 5×1 cm collection along the right pelvis. Clindamycin was switched with metronidazole, and micafungin was started empirically. CT-guided drainage of the pelvic collection was performed, and fluid culture was initially negative, but on POD 6, the blood culture grew actinomyces species. On POD 8, during his daily awakening and spontaneous breathing trial, the patient appeared lethargic, which prompted additional neurological imaging, which revealed bilateral subdural hygromas, a small subarachnoid hemorrhage in the right

parietal lobe with intraventricular hemorrhage, and subacute ischemic focus on corpus callosum and in the left occipital white matter. Repeat EEG also revealed subclinical seizure and a transthoracic echocardiogram ruled out septic vegetations.

By POD 12, the WBC count had reduced to 10.3 K/uL on meropenem, metronidazole, tigecycline, and micafungin. The patient was extubated to a high-flow nasal cannula (HFNC) on POD thirteen. On POD sixteen, the patient was downgraded to the telemetry unit, awake, saturating well on room air, and undergoing physical therapy, with the plan to complete a total of six weeks of piperacillin-tazobactam and metronidazole for four weeks. POD 20, he had an acute stroke of the left pons seen on MRI, and without significant clinical deterioration, was eventually discharged to a nursing home on POD 29.

DISCUSSION

FG is a fulminant form of necrotizing fasciitis of the soft tissues of the perineum, external genitalia, and perianal region.[1] FG most often affects men, with an incidence of 1.6 per 100,000 in the United States.[1,2] The disease affects men five times as commonly as women, and studies show an average age of 49 years old; however, age ranges vary from as young as 20 to as old as 76 years old.[2]

The diagnosis of FG is mainly clinical. The initial stages of the disease can present with discomfort and pruritus, and advancing disease is characterized by edema, erythema, pain, necrosis, induration, and crepitations.[3,5] Systemic signs such as fever, chills, and hemodynamic instability are associated with high mortality.[4] In our case, a hyperglycemic hyperosmolar state, septic shock, acute kidney injury, and cerebrovascular injury complicated our patient's hospital course. Despite being a clinical diagnosis, imaging aids in determining the extent of necrosis in advanced diseases and evaluating for differential diagnoses and complications.[2,5] In addition, imaging may be necessary to confirm the diagnosis since gas within soft tissues visualized on a plain radiograph has a 90-100% sensitivity for FG.[2] Our patient received a CT scan to confirm the diagnosis of FG, showing perianal gas and wall thickening of the anus concerning necrotizing fasciitis in the pelvis involving the perianal soft tissues extending to the lower pelvis.

The LRINEC is a scoring system that allows clinicians to rapidly differentiate necrotizing fasciitis from other soft tissue infections. Six metrics include C-reactive protein, white blood cell count, hemoglobin, sodium, creatinine, and glucose. Scores range from 0, denoting a low risk, to 13, representing a high risk for necrotizing fasciitis.[7] Our patient was classified as high-risk for necrotizing fasciitis with 10 points with the LRINEC. Other severity index scores that aid in prognostication include the FG severity index (FGSI), the Uludag FGSI and the Charlson comorbidities index.[8] This correlates with our case, as magnesium levels for our patient were elevated at 2.5 mEq/L on admission and remained within normal limits during the hospital stay. The clinical examination, imaging findings, history of previous surveys, and risk stratification were all crucial in determining that this patient required urgent surgery.

Despite improvements in treatment strategies for FG, advanced initial resuscitation, and antibiotic treatment, mortality of FG is high.[2,5] Factors that increase mortality are diseases of the anorectal region, advanced age, delayed diagnosis, and patients presenting with sepsis or septic shock.[5] A common risk factor is DM in 37-56% of patients presenting with FG.[1,2,4] In our case, the anorectal region was affected, and a history of poorly controlled DM with an elevated hemoglobin A1c of 10.6%. Diabetes has also been shown to increase mortality rates for patients with FG, with one study reporting 16% higher mortality rates for patients with diabetes compared to patients presenting with FG without diabetes.[4] Other risk factors include chronic alcohol abuse, steroid use, malignancy, HIV, and cardiovascular disorders.[1,5]

Hemorrhoids present in 4% of adult populations and are treated using varying surgical approaches such as rubber band ligation (RBL), sclerotherapy, and excisional surgery.[9] The anorectal region is rich in bacterial flora; however, trans anal procedures are proposed to be free of infections or associated septic complications.[3] Thus, postoperative infection rates after anorectal surgeries are low and have only been described as individual case reports and series. Furthermore, there is a limited number of described cases of postoperative FG after hemorrhoid surgery; our literature search revealed only 8 cases of patients returning to the hospital for perineal, genital, or perianal infections and in one of eight patients FG occurred after a hemorrhoid procedure in a retrospective review carried out in Taiwan.[3,10-16] Our patient had a history of hemorrhoid surgery, and due to the presentation of pain, edema, erythema, and crepitus near the anal verge, the anorectal surgery was likely a contributing factor to the development of this severe infection.

Polymicrobial infections are the usual etiology of FG, involving both anaerobic and aerobic bacteria. The implicated agents include Escheric*hia coli, Pseudomonas aeruginosa, Proteus, Klebsiella, Streptococcus species,* and *Staphylococcus aureus*; less commonly infections with multi-resistant *Staphylococcus aureus* (MRSA) and *Candida* occur in hospitalized patients.[2,5] Infection results in thrombosis of small subcutaneous vessels and tissue necrosis, leading to low oxygen concentration, allowing for anaerobic organism growth and the production of enzymes such as collagenase and hyaluronidase, leading to tissue destruction.[5] Fungal causes of FG are rare; however, especially in cases of immunocompromised patients, empirical fluconazole, caspofungin, or amphotericin B can be used if fungal species are isolated from cultures.[17]

The mainstay of treatment of FG involves immediate surgical debridement and antibiotic therapy. Early, aggressive, wide, and radical excision of necrotic and devitalized tissue is crucial to successfully treating FG. This arrests the spread of the infection, limits the extent of necrosis, and should be repeated as the necrosis progresses.[1,5] In addition, empirical broad-spectrum therapy should be initiated before surgical treatment.[5] Studies have shown that early surgical intervention improves outcomes, reduces complications, and decreases mortality rates.[1,4] Our patient, an uncontrolled diabetic with an HbA1c of 10.6, received broad-spectrum empirical antibiotics and underwent prompt extensive surgical debridement with drain placement and required a CT-guided drainage of pelvic collection, which likely contributed to his survival. Nevertheless, he still experienced a complex hospital course with septic shock, ARDS with prolonged mechanical ventilation, acute kidney injury, and two strokes, leading to functional decline resulting in discharge to a rehabilitation facility.

CONCLUSION

FG development after anorectal surgery is a rare occurrence; however, FG has a high mortality associated with disease progression and complications, making it an important clinical entity. Thus, surgeons must maintain heightened clinical awareness, especially in patients with specific comorbidities such as diabetes, that can cause FG to develop even in minor anorectal surgery, such as hemorrhoid removal. Patient education about the signs and symptoms of such a dreadful complication of anorectal surgery to prevent delay in presentation is paramount as well to allow early intervention and improve outcomes.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Maria HT, Cheema AS, McGuire MJBS, Janis JE. Practical Review of the Current Management of Fournier's Gangrene. Plastic Reconstructive Surg Global Open. 2022;10(3):e4191.
2. Bensardi FZ, Hajri A, Kabura S, Bouali M, El Bakouri A, El Hattabi K, Fadil A. Fournier's gangrene: Seven years of experience in the emergencies service of visceral surgery at Ibn Rochd University Hospital Center. Ann Med Surg. 2021;71(1):10281.
3. Guldeniz K, Oktay I, Bulent U, Kemal K. Fournier's Gangrene after Open Hemorrhoidectomy without a Predisposing Factor: Report of a Case and Review of the Literature. Case Rep Gastroenterol. 2009;3:147-55.
4. Vagholkar K, Deshmukh K, Sai H, Purandare T. Fournier's gangrene of the scrotum after inguinal hernia repair: Case report. Int Surg J. 2023;10(6):1091-3.
5. Sroczyński M, Sebastian M, Rudnicki J, Sebastian A, Agrawal AK. A complex approach to the treatment of Fournier's gangrene. Adv Clin Exp Med. 2013;22(1):131-5.
6. Sorensen MD, Krieger JN, Rivara FP, Broghammer JA, Klein MB, Mack CD, et al. Fournier's Gangrene: population-based epidemiology and outcomes. J Urol. 2009;181(5):2120-6.
7. Tarricone A, Mata K, Gee A, Axman W, Buricea C, Mandato MG, et al. A Systematic Review and Meta-Analysis of the Effectiveness of LRINEC Score for Predicting Upper and Lower Extremity Necrotizing Fasciitis. J Foot Ankle Surg. 2022;61(2):384-9.
8. Erol B, Tuncel A, Tok A, Hanci V, Sari U, Sendogan F, et al. Low magnesium levels an important new prognostic parameter can be overlooked in patients with Fournier's gangrene: a multicentric study. Int Urol Nephrol. 2015;47(12):1939-45.
9. McCloud JM, Jameson JS, Scott AND. Life-threatening sepsis following treatment for haemorrhoids: a systematic review. Colorectal Dis. 2006;8:748-55.
10. Dinc T, Kayilioglu SI, Isa S, Baris DY, Faruk C. Fournier's Gangrene as a Postoperative Complication of Inguinal Hernia Repair. Case Rep Surg. 2014;408217:3.
11. Subramaniam D, Hureibi K, Zia K, Uheba M. The development of Fournier's gangrene following rubber band ligation of haemorrhoids. BMJ Case Rep. 2013;bcr2013201474.
12. Bönner C, Prohm P, Störkel S. Fournier-Gangrän als seltene Komplikation nach Staplerhämorrhoidektomie Kasuistik und Literaturübersicht. Chirurg. 2001;72:1464-6.
13. Giebel GD. Comment on Ch. Bonner et al.: Fournier gangrene as a rare complication after stapler hemorrhoidectomy. Chirurg. 2002;73(3):288.
14. Kaya C, Bozkurt E, Ömerolu S, Yazici P, Mihmanli M. Fournier's Gangrene after Hemorrhoidectomy: Case Report and Review of Literature. Gastroenterol Hepatol. 2017;7(2):00238.
15. Gurfinkel R, Slovik Y, Glezinger R, Walfisch S. Fournier's gangrene as a delayed complication of closed hemorrhoidectomy. Harefuah. 2005;144(6):394-6.
16. Yang SC, Wu TJ. Fournier's gangrene--Taiwan experience. Zhonghua Yi Xue Za Zhi. 2001;64(4):239-43.
17. Perkins TA, Bieniek JM, Sumfest JM. Solitary Candida albicans Infection Causing Fournier Gangrene and Review of Fungal Etiologies. Rev Urol. 2014;16(2):95-8.

Cite this article as: Alla M, Bodden C, Kiarie PK, Khan N, Louis MA, Mandava N. A rare case of postoperative Fournier's gangrene after a hemorrhoidectomy. Int Surg J 2024;11:1866-9.