

CASE REPORTS

Fournier's gangrene in Patients Operated for Hemorrhoidal Prolapse in the Surgical Emergency Department.

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Abstract

Fournier's gangrene (FG) is a well known often fatal fasciitis of the pelvic floor following ano-rectal, urologic and gynecologic infections. Although rarely it is described as a complication of operative anal procedures and predisposing factors such as diabetes, alcoholism, immune-defects and consumptive diseases.

Current literature only briefly mentions the potential risk of FG after such a common surgical procedure. However, devastating complications occur more often than expected. This catastrophic complication without a predisposing factor is discussed along with a review of the literature.

The objective of this article is to provide updated and relevant information regarding the recognition, diagnosis and management of FG, from the general surgeon to the emergency department.

This article refers to two complicated cases of Fournier's gangrene. The patients underwent emergency surgical intervention with the diagnosis of hemorrhoidal prolapse with rectal bleeding and accompanying anemia...

Conclusion; The gold standard for treatment was found to be a combination of surgical debridement, broad-spectrum antibiotics, and the administration of intravenous fluids. Further, patient survival was found to be directly related to the time from diagnosis to treatment when they underwent surgical debridement.

The General surgeon must be vigilant for this condition and be aware of risk factors, prognostic indicators, and proper treatment protocols to recognize FG early and initiate appropriate management.

Keywords: Fournier's gangrene, necrotizing fasciitis, emergency department

Introduction

Fournier's gangrene is a serious inflammatory pathology with a very high risk to life. It is also known as necrotizing fasciitis or acute necrotizing inflammation. [1, 2]

It was first described in 1764 by Baurienne and in 1877 by Avicenna. [3]

The precise definition was later given by Jean Alfred Fournier, a Parisian venereologist. It affects older males more than females. [4]

The incidence is 1.6 males per 100,000 inhabitants. Cases have also been observed in children. It is mainly localized in the perianal and perineal areas and often involves the genital area as well. [5] The clinical presentation is characterized by fever, pain, foul odor, necrosis, and subcutaneous crepitus, rapid progression, and general deterioration leading to multiorgan failure. [6]

The cause of gangrene is a result of thrombosis of the blood vessels localized in these areas. There is still no exact cause for this pathology. The infectious and inflammatory process spreads quickly along the Dartos, Colles, and Scarpa's fascias, allowing for the early involvement of the

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abdominal wall. [7]

Many studies suggest that the causes may be anorectal infections, genitourinary infections, and various anorectal interventions. [8]

These infections can compromise the immune system, allowing toxins and enzymes from microorganisms to cause necrosis. The origin of the microorganisms appears to be from the digestive tract (30%-50%), the genitourinary tract (20%-40%), and the skin (20%). [9]

Microbial cultures have revealed both aerobic and anaerobic microbes. The synergy between the microbes is a crucial factor in this pathology. [10]

The most common associated pathologies are diabetes mellitus, hepatic cirrhosis, malignant pathologies, obesity, and immunodeficient patients. [11]

Since 1950, more than 1,800 cases for study have been reported in English language medical literature. This disease occurs worldwide and, although it is recognized more frequently among male adults, has been identified also among women and children. Treatment usually consists of the surgical removal (debridement) of extensive areas of dead tissue (necrosis, necrotic) and the administration of broad-spectrum intravenous antibiotics. Surgical reconstruction may follow where necessary.

This article refers to two complicated cases of Morbus Fournier. The patients underwent emergency surgical intervention with the diagnosis of hemorrhoidal prolapse with rectal bleeding and accompanying anemia.

First Patient

Patient A.N., 59 years old, presented to the surgical emergency department with a diagnosis of active hemorrhoidal prolapse with rectal bleeding. He reported a 7-year history of hemorrhoidal disease, and in the past two months, rectal bleeding had become almost daily. No accompanying pathology was found during rectoscopy. Occasionally, the patient used oral therapy with daflon 500mg. Blood tests showed: red blood cells - 3,780,000 10⁶/mL, hemoglobin - 8.9 g/dl, leukocytes - 6.40 10⁹/mL. Blood group - 0 (Rh+). Biochemical analysis was within normal limits. Urine analysis showed 30-35 leukocytes per field and 9-10 red blood cells per field. The only accompanying diseases were prostatitis, left kidney calculi, and previous urinary tract infections.

After consultation with the anesthetist, a Milligan-Morgan hemorrhoidectomy was performed under spinal anesthesia. During the hospital stay, the patient was treated with fluids, oral metronidazole, ciprofloxacin, intravenous omeprazole 40 mg, subcutaneous enoxaparin, and analgesics. The patient started an oral liquid diet immediately after the spinal anesthesia wore off. The patient was discharged from the hospital in good condition 48 hours after the surgery. After leaving the surgical clinic, the patient was recommended a high-fiber diet, local toilet with potassium permanganate, oral ciprofloxacin 500 mg, subcutaneous enoxaparin, and analgesics.



Figure 1. First Patient after surgery

Six days after the surgery, the patient presented to the emergency department with unbearable pain in the anal area, a temperature of 38.8 degrees Celsius, pallor, profuse sweating, fatigue, fecal incontinence, a pulse rate of 112 beats per minute, and a rhythmic pattern. Locally, edema of the perianal area, crepitus, intraanal necrosis, and extension towards the scrotal area with foul-smelling and dark-colored secretions were observed. The anorectal examination was very painful. Rectoscopy revealed a dark purple mucosa in the anal area with clearly demarcated necrotic zones, extending about 2 cm above the dentate line.

The patient was transferred to the intensive care unit where a urinary catheter was placed. Intravenous therapy with fluids, electrolytes, imipenem, metronidazole, gastroprotective agents, and analgesics was initiated. Blood tests performed upon arrival at the hospital showed the following deviations: red blood cells - 3,620,000 10⁶/mL, hemoglobin - 8.3 g/dl, leukocytes - 4.3 10⁹/mL, SGOT - 46 U/L, SGPT - 56 U/L, PCR - 7 mg/L. Viral tests were negative.

Abdominal ultrasound showed edema of the perineum, perianal edema, thickened rectal walls up to 1 cm, air bubbles in the perianal and perirectal soft tissues, and minimal fluid in both scrotums. The patient underwent surgery under general anesthesia 6 hours after arriving in the emergency department. Initially, six deep elliptical incisions were made, four perianal and two in each scrotum. The necrotic tissue was then excised until the borders of healthy macroscopic tissue were reached. These borders were considered the zone where there was no necrosis and red blood was visible. The procedure was mainly performed with scissors and cold bistoury, with very limited use of electrocautery. Suture with 3-0 vicryl was used in two moments. Attempt was made to achieve perfect hemostasis. The wounds were treated with oxygenated water and 0.9% physiological saline after excision.

Finally, the wounds were covered with compresses soaked in 2% lidocaine gel and chlorhexidine acetate. After the surgery, the patient continued the initiated therapy,

adding subcutaneous enoxaparin and human albumin. Intravenous therapy lasted for 12 days and was discontinued two days after the patient became afebrile.

The operative wounds were treated three times a day with isotonic saline solution, oxygenated water, and 5% betadine solution every 8 hours. Necrotomy was performed twice at the patient's bedside. Oral fluid, liquid diet, and hyperprotein diet started 7 hours after the operation. On the 15th day after the second intervention, the beginning of wound granulation was evident, the secretion from the wounds decreased, there was no foul odor, the patient was afebrile, active, with a pulse rate of 87 beats per minute, leukocyte count resulted in 9.20 10/ml, erythrocytes - 3,780,000 per mL, hemoglobin - 8.3 g/dL. Biochemical analyses were within normal limits. On the 23rd day, the patient was discharged from the hospital in good condition. It was recommended that the wound be treated daily by the respective nurse, and a high-fiber, hyperprotein, and fluid-rich diet was advised.

Epithelialization of the wounds was completed after 95 days. After wound epithelialization, the patient reported two complaints: urinary urgency and occasional incontinence of gas and liquid stools.

Second Patient

Patient M.S, 66 years old, is transferred to the surgical emergency department from the endocrinology ward. The patient was previously diagnosed with non-insulin-dependent diabetes mellitus (type 2), arterial hypertension, and morbid obesity. He had been on oral therapy with metformin and antihypertensive medication for three years.

For the past six years, he has been undergoing conservative therapy for hemorrhoidal disease and constipation. In the past week, significant rectal bleeding and hemorrhoidal prolapse during defecation were daily occurrences.

In the last 24 hours, rectal bleeding and hemorrhoidal prolapse have occurred outside the act of defecation. Anorectal examination in the surgical emergency department reveals prolapsed hemorrhoids with active bleeding. The anal canal was tamponaded with a sponge

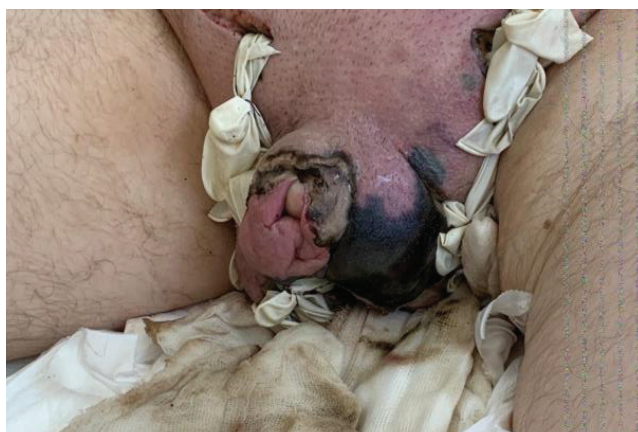


Figure 2. Second Patient after surgery

Deviations in the blood tests performed upon arrival at the surgical emergency department show: erythrocytes - 3,910,000 per mL, hemoglobin - 11.1 g/dL, blood glucose - 205 mg/dL.

Arterial blood pressure is 150/91 mm/Hg, and the body mass index is 36 kg/m². After consultation with the anesthesiologist, the patient underwent surgery 4 hours after presenting to the surgical emergency department.

The Milligan-Morgan procedure was performed with the help of spinal anesthesia. Postoperative therapy includes two liters of fluids, flagyl, ciprofloxacin, omeprazole, enoxaparin, oral antidiabetic therapy, and intravenous analgesics. Oral feeding commenced immediately after the recovery of motor block. The patient stayed in the surgical clinic for 48 hours and was transferred to the endocrinology clinic without early postoperative complications.

The surgeon recommended a high-fiber diet, local wound toilet with potassium permanganate, oral ciprofloxacin 500 mg, subcutaneous enoxaparin for 10 days, antidiabetic therapy, and paracetamol. On the eighth postoperative day, the patient reports a temperature of 37.6 degrees Celsius, pulsating and burning pain in the anal and perianal area, perianal itching, difficulty urinating, anorexia, abdominal discomfort, and putrid secretions in the wound dressing.

The pain radiates posteriorly to the sacrum and anteriorly to the scrotum. Locally, there is evident extensive perianal cellulitis spreading posteriorly and anteriorly. No signs of necrosis are observed on the skin. Crepitus is detected along the cellulitis area, and the skin appears reddish and inflamed. Only at the site of the operative wound are necrotic hemorrhoidal bridges evident. A rectoscopy is performed under anesthesia, revealing that the anorectal mucosa has a dark reddish color. This area extends approximately 4 cm above the dentate line.

There is a strong foul odor. The following abnormalities were detected in the laboratory examinations at the surgical emergency department: erythrocytes - 3,810,000 per mL, hemoglobin - 10.8 g/dL, leukocytes - 3.90 10/mL, blood glucose - 490 mg/dL. The pulse rate was 112 beats per minute, and the blood pressure was 105-79 mm/Hg.

The blood type is group 0 (Rh-). An abdominal CT-scan with intravenous contrast revealed the presence of air bubbles in the subcutaneous perianal area with wide spread, double-layered walls of the rectum, disruption of the subcutaneous adipose tissue structure, and free fluid in both testicular sacs. In these conditions, the patient was transferred to the intensive care unit for monitoring and preparation for intervention.

A urinary catheter was inserted. Fluid therapy and intravenous imipenem were initiated. The intervention took place 4 hours after arrival in the emergency department. With the help of general anesthesia, eight wide elliptical incisions were made: four perianal incisions, two scrotal incisions, and two anococcygeal incisions. Necrotomy was performed until macroscopically healthy tissue margins were reached. Electrocautery was used sparingly.

The intervention was performed with the help of a retractor and cold bistoury. The wounds were covered with gauze soaked in chlorhexidine acetate and lidocaine gel. After the surgery, the patient was treated with three liters of fluid, imipenem, flagyl, omeprazole, subcutaneous enoxaparin, insulin, and antihypertensive medication. An oral fluid diet rich in fiber and protein started 6 hours after the intervention.

The operative wounds were treated three times a day, every 8 hours, with isotonic saline solution, abundant oxygenated water, and 5% betadine solution. On the 14th day, the patient felt better and was active. The temperature has normalized. Blood glucose level is 140 mg/dL. Erythrocytes count is 3,880,000 per mL. White blood cells count is 9.20 10/mL. Blood pressure is 150-86 mm/Hg, and pulse rate is 89 beats per minute. Locally, the wounds continue to have secretions, but without a strong odor. During this period, necrotomy was performed 8 times at the patient's bedside.

On the 14th day, insulin was replaced with metformin, flagyl and imipenem were discontinued, and intravenous ciprofloxacin was started. The patient was discharged from the hospital on the 30th day after the second intervention. Laboratory analyses showed: erythrocytes count - 3,980,000 per mL, white blood cells count - 9.50 10/mL, blood glucose - 170 mg/dL, blood pressure - 150-80 mm/Hg. Other biochemical analyses were within normal range.

During the patient's stay in the surgical ward, they lost 10 kg of body weight. The wounds were closed 4 months after discharge from the hospital.

Discussion

Both cases mentioned above are rarely reported in the literature. FG is a serious surgical problem with high mortality and morbidity, still there is a male predominance [10].

Vick R et al. have written that there are so many predisposing factors described by various authors as seen in literatures. Out of them, diabetes, old age, alcoholism, obesity, paraplegia, and renal insufficiency are commonly seen. However, it is interesting to note that in almost 30% to 50% cases no definite predisposing factor is found [13].

Agranulocytosis is mentioned as a cause in one reported case [14].

Fournier's gangrene is described as a synergistic polymicrobial infection of the soft tissues in the perineal area. This synergy is believed to be the cause of the spread of inflammation, pronounced edema, and progressive necrosis. Anorectal infections, genitourinary infections, and traumatic wounds are referred to as the most common causes of Fournier's gangrene. [10]

Inflammatory anal pathologies, malignant tumors of the sigmoid and rectum, perforated diverticulitis, rectal trauma, acute transverse appendicitis, and transanal procedures are thought to be contributing factors to the development of the pathology. [15]

Chernyadyev SA et al. [16] note that the cause of Fournier's gangrene can be established in 95% of cases; the disease usually occurs because of infectious processes of the urogenital tract, anorectal area, or skin of the genitals. Fournier gangrene develops commonly in immunocompromised patients with diabetes, obesity, and malignant neoplasms. Kincius et al. [17] who conducted a study among patients with Fournier's gangrene found a high proportion of people with diabetes mellitus. Also, our cases was with non-insulin-dependent diabetes mellitus (type 2), arterial hypertension, and morbid obesity.

Yoshino et al. [18] described the case of an anal fistula in a patient with type 2 diabetes mellitus, resulting in an abscess of pararectal tissue complicated by Fournier gangrene.

Cases of Fournier's gangrene have been reported even after minimal interventions in the anal canal. In the literature, only one case has been reported as complicated with Fournier's gangrene following hemorrhoidectomy.[19]

Fournier's gangrene is usually associated with pathologies that reduce the immune system's powers. These include diabetes, malignant pathologies, renal insufficiency, malnutrition, cirrhosis, AIDS, corticosteroid users, and dialysis patients. Both patients we referred to had accompanying pathologies. [20, 21]

Patient A presented with anemia and recurrent urinary infections. Patient B had diabetes and obesity. These comorbidities affect the patient's immune system.

The Fournier's gangrene rate due to proctological procedures is extremely rare.[22]

However, in the literature, necrotizing perineal soft tissue infections after anal dilatation, hemorrhoidal band ligation, and open hemorrhoidectomy, which require radical debridement, have been described. [23]

Although hemorrhoidectomy is a common surgical procedure, the appearance of FG is unpredictable.[24]

The most important point in the treatment of this extremely severe pathology is the early diagnosis. Diagnosis is usually made by physical examination. Perineal necrotizing fasciitis is clinically characterized by, severe perineal pain and high fever. In the early phase of FG, clinical findings may be indistinguishable from other soft tissue infections such as erysipelas or cellulitis, but the presence of indeterminate borders and sensitivity outside of the affected area is in favor of FG. [25]

Early diagnosis and aggressive surgical intervention with broad-spectrum antibiotics can reduce mortality in FG. [26]

We surgically treated the patients after resuscitating them with fluids and broad-spectrum antibiotics, aiming to normalize the parameters as much as possible.

The second intervention was performed under general anesthesia to avoid the complications of spinal anesthesia.

The second intervention in both patients was performed with the help of retractors and cold scissors. Electrocautery was used very sparingly. Vicryl 3-0 sutures were occasionally used.

The incisions were wide and elliptical in shape.

The removal of all the devitalized tissue is important to stop the progress of the infection and simultaneous elimination of systemic effects of toxins and bacteria [27].

Necrectomy was performed aggressively, aiming to reach the level of healthy tissue in terms of color and arterial blood supply.

Hemostasis was attempted to be as close to perfect as possible. Antibiotic therapy should be regulated in double or triple combinations to include broad-spectrum agents for possible pathogens as soon as possible, without antibiogram result [28, 29]

During their stay in the surgical clinic, the patients were treated three times a day, every 8 hours, with ample oxygenated water, 5% Betadine solution, physiological saline, and Flagyl solution. Every day, temperature, pulse, blood pressure, and diuresis were evaluated.

A suggested regime would include a third-generation cephalosporin or broad-spectrum penicillin, gentamicin, metronidazole, or clindamycin [30].

Every 4 or 5 days, laboratory and biochemical analyses were performed. As soon as necrosis spread was observed, it was excised. Fluid and protein balance were maintained not only intravenously but also orally. Early diagnosis and timing of the first debridement is an important determinant of mortality. [31, 32]

The patients were allowed to eat through the mouth 5 or 6 hours after the intervention. The patients were mobilized 48 hours after the intervention. We followed the course of releasing incisions with aggressive necrectomy during the intervention. The second intervention was performed with the help of endotracheal anesthesia. This anesthesia method was chosen to prevent infection. Local treatment three times a day accompanied by necrectomy was also a significant advantage in the path to recovery.

Other surgical techniques are referred to when the pathology progresses and biochemical, laboratory parameters, and the general condition of the patient do not stabilize. Temporary colostomy technique is recommended in such cases. It was not necessary in our two cases. The presentation of the patients in a relatively short time also led to their early diagnosis.

On the other hand, the assessment of the overall condition, resuscitation, correction of biochemical and vital abnormalities, and the initiation of broad-spectrum antibiotic therapy are believed to have influenced the good postoperative course after the second intervention. Microbiological studies show that *Pseudomonas aeruginosa* predominates over other microbial strains in Fournier's pathology.

Hakkarainen TW et al. shown that medical intervention revolves around the initiation of empiric broad-spectrum antibiotics while awaiting culture sensitivities. Antibiotic therapy has historically involved triple therapy in covering gram-positive, gram-negative, and anaerobic organisms associated with Fournier gangrene. These typically include

staphylococcus, streptococcus, coliforms, *Pseudomonas*, *Bacteroides*, *Clostridium*, and possibly yeast. The combination of a third-generation cephalosporin, aminoglycoside, penicillin, and metronidazole, has classically been used as triple therapy antibiotic coverage. Antibiotic therapy typically requires a duration of at least two weeks. [33, 35]

It is thought that this strain is also the cause of leukopenia in the preoperative period. The synergistic activity of aerobic and anaerobic microbes enables the release of exotoxins and enzymes such as collagenase, heparinase, hyaluronidase, streptokinase, and streptodornase. All these elements lead to tissue destruction and the spread of inflammation. Aerobic microorganisms cause complement fixation and platelet aggregation. Anaerobic microorganisms produce heparinase and collagenase. Park et al. [34], in an analysis of factors related to mortality from necrotizing soft tissue infections, found that patients with a high body mass index or abnormal leukocyte, C-reactive protein and platelet counts reflecting the severity of the infectious process or impaired renal function have an unfavorable outcome.

Both of these factors contribute to microvascular thrombosis, resulting in necrosis of soft tissues. On the other hand, impairment of phagocytic activity in necrotic tissues leads to rapid spread of infection. Inflammation spreads along fascial planes, usually limited by the Colles' fascia of the perineum. Inflammation can spread to the scrotum, penis, and anterior abdominal wall. Testicular involvement indicates widespread inflammation, including retroperitoneum.

Urogenital infections progress posteriorly along the Buck's and Dartos fascia. Anorectal infections begin and spread in the perianal and anorectal areas. [1]

The severity index of the pathology depends on temperature, respiratory rate, pulse, blood pressure, and sodium level in the blood. [36]

It is reported that when this index is greater than 9, the mortality rate reaches 73%. In addition to the risk factors for each patient, the fact that the Milligan-Morgan technique in patients with large hemorrhoidal prolapse causes significant wounds and local necrosis should not be overlooked.[37]

It is still inconclusive whether a change in surgical technique would have an impact on the non-appearance of this severe postoperative complication. In both cases, leukopenia, anemia, and mild alterations in biochemical parameters are evident.

Conclusion

The gold standard for treatment was found to be a combination of surgical debridement, broad-spectrum antibiotics, and the administration of intravenous fluids. Further, patient survival was found to be directly related to the time from diagnosis to treatment when they underwent surgical debridement.

The General surgeon must be vigilant for this condition and be aware of risk factors, prognostic indicators, and proper treatment protocols to recognize FG early and initiate appropriate management.

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