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Fournier's gangrene

Gangrena de Fournier

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Fournier's gangrene, necrotizing fasciitis, debridement.

Palabras clave:
Gangrena de Fournier, fascitis necrosante, desbridamiento.

ABSTRACT

Fournier's gangrene is a rare, life-threatening surgical emergency consisting of necrotizing fasciitis of the external genitalia, perineal, and perianal region. It often arises from an infection in the anorectal area that progresses rapidly and has a mortality of up to 40%, even with adequate treatment. Treatment consists of one or more emergency surgical interventions with debridement of devitalized tissue and specific antibiotic therapy.

RESUMEN

La gangrena de Fournier es una emergencia quirúrgica rara que pone en peligro la vida, consiste en una fascitis necrosante de los genitales externos, de la región perineal y/o perianal. Con frecuencia proviene de una infección en la región anorrectal que progresa rápidamente y tiene una mortalidad de hasta 40%, incluso con tratamiento adecuado. El tratamiento consiste en una o varias intervenciones quirúrgicas de urgencia con desbridamiento del tejido desvitalizado y antibioticoterapia específica.

INTRODUCTION

Fournier's gangrene was first described by the French venereologist Jean-Alfred Fournier in 1883 when he recorded a case of sudden-onset idiopathic gangrene in a previously healthy young man.

It is a rare disease that represents < 0.02% of total hospital admissions. As described by Auerbach et al, it occurs in approximately 1.6 cases per 100,000 people and in 0.25 women per 100,000 people per year. This condition affects both sexes, but usually occurs in men, with a 10:1 ratio with respect to women. It occurs less frequently in women because venous and lymphatic drainage of the perineum occurs vaginally.

The average age of presentation is 50 years, but it manifests in an extensive range from 42 to 70 years of age, and the risk of presentation increases with age.[1-3]

It is considered a medical-surgical emergency due to its rapid progression, 2 to 3 cm per hour. It is characterized as a very aggressive type 1 necrotizing fasciitis of the perineal, genital, and perianal regions, with a high morbimortality reported in the literature to be up to 80% in the absence of timely treatment. For a long time, it was considered an idiopathic condition; however, as described by Singh and collaborators and Chernyadyev and his team, less than a quarter of the cases are classified in this way since most of them are caused by an underlying infection that can be found in the anorectal region in 30-50%, in the urogenital region in 20-40% and skin of external genitalia in 20%; it has also been described that trauma in these areas can be a predisposing cause.[4-7]

There are several predisposing factors for the development of Fournier's gangrene, which have as underlying cause an alteration

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of the immune system that creates a favorable environment for the development of infections such as diabetes (more frequent), chronic alcoholism, malnutrition, immunosuppression (chemotherapy, steroids, malignancy), disease by human immunodeficiency virus, lymphoproliferative syndromes, among others.[5,7]

It is a pathology that frequently requires admission to the Intensive Care Unit (ICU), multiple surgical interventions, and involves high economic costs. Jiménez-Pacheco and collaborators calculated the approximate global health care cost of a patient with Fournier's gangrene in a hospital in Granada, Spain, concluding that it is more than 25,108.67 euros, that is, 627,716.75 Mexican pesos, according to the current exchange rate.[8]

ETIOLOGY

Fournier's gangrene is a polymicrobial infection in 54 to 80% of cases, according to the studies by Luján et al. and Gadler. The most isolated microorganisms are those found in the perineum and external genitalia, which are aerobic and anaerobic bacteria that act synergistically, causing tissue necrosis *(Table 1)*. The most reported bacterium is *Escherichia coli*.[9-13]

Table 1: Microorganisms most frequently involved in Fournier's gangrene.

According to oxygen requirements	Bacteria (%)
Anaerobes	*Bacteroides spp.* (38.6) *Streptococcus spp.* (37.1) *Enterococcus spp.* (27.1) *Staphylococcus spp.* (25.7) *Proteus spp.* (18.6)
Aerobes	*Escherichia coli* (40.0) *Pseudomonas spp.* (24.3) *Klebsiella pneumoniae* (20.0)

Source: Yilmazlar T et al[13] and Ersay A et al.[14]

Several predisposing factors have been significantly related to the development of Fournier's gangrene, which has as a common basis an alteration in the immune system that creates a favorable environment for the development of infections; these factors include diabetes, systemic arterial hypertension, obesity (BMI > 30), smoking and immunosuppression, mainly.[2,6,13,14]

Diabetes is considered the most prevalent comorbidity in these patients, reported in up to 60%, as described by Voelzke and colleagues, Vargas and his team, and Hatipoglu and colleagues.[7,15-19]

Fournier's gangrene has a multifactorial origin; local factors include urological pathology (surgery, urinary tract infection, paraphimosis, urethral stricture, traumatic catheterization), anorectal pathology (abscesses, surgery, rectal trauma), dermatologic (purulent skin infections, allergic reactions), proctologic (perirectal abscess, perianal abscess), scrotal or vulvar cellulitis or abscess, hidradenitis, Bartholinitis, and pressure ulcers.[17-19]

Depending on the microbiological agent, necrotic soft tissue infections can be categorized into four groups: **type 1 (polymicrobial)** is the most common type and accounts for more than 50% of infections. The synergistic action of aerobic, anaerobic, and facultative anaerobic bacteria exists. It usually affects immunocompromised patients or those with severe comorbidities, affecting the trunk and perineum; **type 2 (monomicrobial)** may be more aggressive than type 1 and is less common; group A ß-hemolytic *Streptococcus* is the most common agent and occurs more frequently with a history of trauma or recent surgery; **type 3 (clostridial myonecrosis)**, responsible for less than 5% of infections, is related to *Clostridium perfringens* and *Aeromonas hydrophila*, *Clostridium perfringens* is the bacterium most frequently involved in traumatic injuries. A common clinical finding in these cases is crackled due to gas production by these bacteria. It affects extremities, trunk, and perineum and spreads rapidly, resulting in multiple organ failure and mortality within 24 hours without treatment. **Type 4 (fungal)**, secondary to *Candida spp.* and *Zygomycetes*, usually in immunocompromised patients

and after trauma, affects extremities, trunk, and perineum. It is aggressive and rapidly progressive.[2,11,18]

PATHOPHYSIOLOGY

A localized primary infection allows the entry of commensal bacteria into the perineum, causing an inflammatory reaction that causes obliterative endarteritis in the affected area, resulting in thrombosis of small subcutaneous vessels and necrosis of the affected tissue that will subsequently cause low oxygen concentrations and growth of anaerobic bacteria. Aerobic and anaerobic bacteria act synergistically, producing enzymes such as collagenase, heparinase, hyaluronidase, streptokinase, and streptodornase, which destroy the affected tissue. Aerobic microorganisms produce vascular thrombosis and dermal necrosis due to heparinase and collagenase activity. The impaired activity of phagocytic leukocytes due to hypoxia in necrotic tissue is responsible for the spread of infection since oxygen is necessary to produce antibacterial substances by leukocytes.[5,19]

CLINICAL MANIFESTATIONS

The presentation depends on the stage of infection, the patient's comorbidities, and general health status. There may be a prodromal period with symptoms such as genital discomfort, pruritus, and fever for days, sometimes weeks, before more severe symptoms occur.

Fournier's disease has an insidious course, in most cases presenting with scrotal or vulvar pain that usually does not correspond to clinical findings, edema, cellulitis, and erythema, which may be accompanied by a foul odor, crepitus, and systemic data such as fever, hypotension, and tachycardia; pruritus, pain, and malaise usually worsen three to five days before patients go to the hospital, progressing to blistering, ischemia and necrotic lesions. Initially, the superficial skin is intact while the necrotizing process spreads into the fasciae, making timely diagnosis difficult. The infection spreads 2.5 cm per hour without showing changes in the skin. After a few hours, hyperthermia in the genitals and tissue necrosis begin. As described by Hernandez and collaborators, urination becomes painful and difficult.[20,21]

Subsequently, it is characterized by skin, subcutaneous tissue, and muscle necrosis, which can cause sepsis and multiorgan failure that can lead to death.[10,19] Local hypoxia causes infarction of the regional nerves so that initially there is pain and later anesthesia of the area, as shown in *Figures 1 and 2*.[6,18]

The superficial fascia of the perineum or Colles' fascia covers the region's muscles, is continued by Dartos' fascia of the penis and scrotum in men and the vulva in women, and by Scarpa's fascia of the anterior abdomen. These fascial planes are united and facilitate the rapid spread of infection. In the male, the internal and external spermatic fasciae, and the vessels of the retroperitoneum, independent of the vessels of the urogenital and anogenital region, protect the testis from infection. Buck's fascia lining the urethra and the corpus cavernosum give additional protection to this area.[18]

DIAGNOSIS

Diagnosis is based on clinical findings of inflammation and necrosis of the affected area, crepitus, foul odor, and fever.[12]

Plain radiography may show subcutaneous emphysema extending from the perineum and external genitalia to the inguinal region, thigh, and anterior abdominal wall. Ultrasound shows subcutaneous emphysema and echogenic areas with a "dirty shadow" in the scrotal or perineal region. The computed tomography (CT) shows subcutaneous air and heterogeneous density in the area with a thickened and edematous scrotal or vulvar wall. These studies help to differentiate a necrotizing infection from other pathologies. MRI shows subcutaneous emphysema, thickening of the scrotal wall, and fluid accumulation and helps to determine the extent of the disease. Ultrasound or CT are sufficient diagnostic methods.[5,12,19]

Imaging studies can help establish the extent of the necrotic process but should not delay the initiation of treatment, as this is associated with increased mortality.[5]

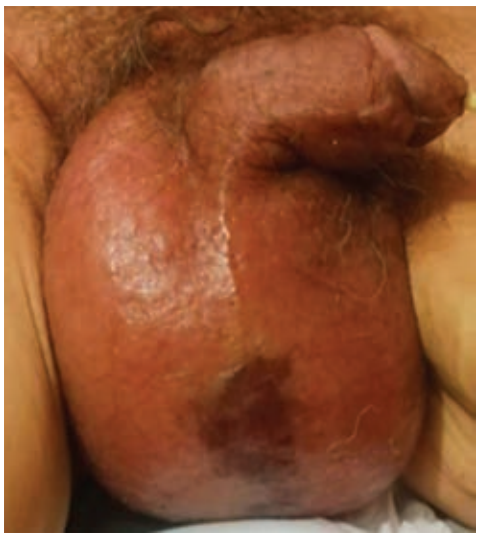

Figure 1: *Diabetic patient with early-stage Fournier gangrene.*

Of laboratory studies, blood biometry evaluates the degree of systemic inflammatory response and infection or concomitant anemia, thrombocytopenia, or thrombocytosis; the blood chemistry is important to evaluate renal function, C-reactive protein, blood cultures, and, as reported by Mehanic and collaborators and Novoa-Parra and his team, procalcitonin is very useful in the prediction of septic shock in patients with Fournier's gangrene, and has even proved to be a more effective method than the scales currently used for its diagnosis; a low procalcitonin level would help us to rule out its diagnosis early.[6,10,22,23]

The histological examination will reveal necrosis of the superficial and deep fasciae, fibrinoid coagulation in the vascular lumen, infiltration of polymorphonuclear cells in the tissues, and necrotic detritus. Venous thrombosis of the affected tissues is very significant.[6]

Scoring systems have been developed for the diagnosis and prognosis of Fournier gangrene, such as the LRINEC (Laboratory Risk Indicator for Necrotizing Fasciitis) scale, which includes variables that will help differentiate necrotizing fasciitis from other soft tissue infections; values for diagnosis range from 0-13 and prognosis: low risk equal to or less than five points, intermediate six to seven points, high risk similar to or greater than eight points, with a probability of necrotizing soft tissue infection of < 50%, 50-75% and > 75% respectively *(Table 2)*.[24-26]

The Fournier gangrene severity index (FGSI) determines the risk of mortality; it was created in 1995 by Laor et al. and is the most used scale, with a sensitivity of 65-88% and specificity of 70-100% *(Table 3)*. This index assesses body temperature, heart rate, respiratory rate, sodium, potassium, creatinine, bicarbonate, leukocytes, and hematocrit; a score > 9 is associated with a 75% probability of death, equal to or less than nine is associated with a 78% probability of survival.[12,13,20,27,28]

Differential diagnoses should be made with dermatological and systemic pathologies such as scrotal cellulitis, testicular torsion or abscess, acute epididymitis, balanitis, strangulated inguinoscrotal hernia, vasculitis, occlusive vascular syndromes, polyarteritis nodosa, erythema necrolytic migrans, herpes simplex, and warfarin necrosis.[5,29]

TREATMENT

The key to treatment consists of three fundamental principles: 1) hemodynamic stabilization (urgent resuscitation with intravenous fluids, acid-base, and metabolic stabilization), 2) empirical broad-spectrum

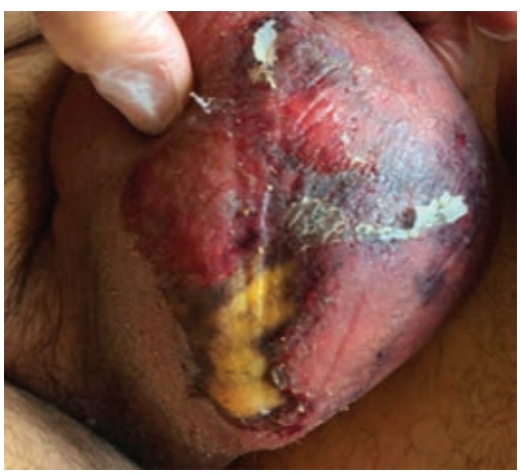

Figure 2: *Advanced stage of Fournier's gangrene. Source: Caliskan S et al.*[11]

antibiotic therapy, and 3) debridement of necrotic tissue. The goals of treatment are to reduce systemic toxicity, halt the progression of necrosis, and eliminate the causative microorganism.[5,6]

All patients should undergo surgical debridement within the first 12 hours of admission; this step is crucial to stop the progression of the infection, according to Singh and collaborators, and a delay of a few hours to initiate debridement has been associated with a significant increase in mortality in these patients, so it is considered the most important factor for survival.[5,6,12,17,19,20] On average, 3.5 surgical procedures per patient are required for adequate infection control.[5]

Empirical antibiotic therapy directed to the most frequently involved microorganisms should be initiated. According to the recommendations of Carruyo and his team, initial management should be done with three groups of antibiotics: 1) third-generation cephalosporins or aminoglycosides to cover Gram-negative aerobic microorganisms, 2) benzathine penicillin or amoxicillin to cover *Streptococcus* type microorganisms and 3) metronidazole or clindamycin to cover anaerobic microorganisms; in contrast to Chennamsetty and collaborators who likewise recommend a triple scheme of empirical antibiotic therapy, but include: 1) penicillin or third-generation cephalosporins, 2) aminoglycosides, and 3) metronidazole or clindamycin; they also recommend adding vancomycin in case of suspected *S. aureus* infection.[30-32] It is recommended to perform a culture and antibiogram of the lesion to modify the antibiotic therapy or continue with the established one.

Table 2: LRINEC (Laboratory Risk Indicator for Necrotizing Fasciitis) scale for the diagnosis and prognosis of Fournier's gangrene.

Variable	Score
C-reactive protein (mg/dl)	
< 15	0
> 15	4
Leukocyte count (per mm^3)	
< 15,000	0
15,000-25,000	1
> 25,000	2
Hemoglobin (g/dl)	
> 13.5	0
11-13.5	1
< 11	2
Sodium (mmol/l)	
> 135	0
< 135	2
Creatinine (mg/dl)	
< 1.6	0
> 1.6	2
Glucose (mg/dl)	
< 180	0
> 180	1

Modified from: Liao C et al.[24]

Debridement continues until all necrotic tissue has been removed and healthy granulation tissue is established in the wound, removing all necrotic and devitalized tissue as soon as possible; postponing increases the risk of death. Characteristic features during debridement include the absence of bleeding secondary to thrombosis of blood vessels, foul odor, grayish discoloration of soft tissues due to necrosis, fluid such as "dirty water" pus, and detachment of tissues with digital dissection.[5,6,12,17,19,20,30-32]

The overall mortality of Fournier's gangrene has been described as 20-40% in most follow-ups, but Sorensen reported it at 88%.[4] Caliskan et al. report that adequate debridement reduces mortality by up to 16%. A second look surgery at 24 hours is recommended; in case of deterioration of the patient's clinical condition, it should be performed earlier.[6,12,15,17,19]

Alternative methods such as VAC therapy® (*vacuum-assisted closure*) or therapy with recommended negative pressure of 50 to 125 mmHg, which is used in the treatment of many chronic wounds, as it stimulates the blood supply in the affected region and promotes the migration of inflammatory cells with the formation of granulation tissue. Hyperbaric oxygen therapies may accelerate

Table 3: Fournier gangrene severity index.

Variable	High abnormal values				Normal	Low abnormal values			
Score	**+4**	**+3**	**+2**	**+1**	**0**	**+1**	**+2**	**+3**	**+4**
Temperature (ºC)	> 41	39-40.9	-	38.5-39	36-38.4	34-35.9	32-33.9	30-31.9	< 29
Heart rate	> 180	140-179	110-139	-	70-109	-	55-69	40-54	< 39
Respiratory frequency	> 50	35-49	-	25-34	12-24	10-11	6-9	-	< 5
Serum sodium (mmol/l)	> 180	160-170	266-159	350-354	130-149	-	120-129	111-119	< 110
Serum potassium (mmol/l)	> 7	6-6.9	-	5.5-5.9	3.5-5.4	3-3.4	2.5-2.9	-	< 3.5
Serum creatinine (mg/100/ml × 2 for acute renal failure)	> 3.5	2-3.4	1.5-1.9	-	0.6-1.4	-	< 0.6	-	-
Hematocrit	> 60	-	50-59.9	46-49.9	30-45.9	-	20-29.9	-	< 20
Leukocytes (total/mm^3 × 1,000)	> 40	-	20-39.9	15-19.9	3-14.9	-	1-2.9	-	< 1
Serum bicarbonate (venous, mmol/l)	> 53	41-51.9	-	32-40.9	22-31.9	-	18-21.9	15-17.9	< 15

Source: Laor E et al.[27]

the speed of wound healing; oxygen therapy reduces leukocyte dysfunction caused by hypoxia and has a direct antibacterial effect against anaerobes; it has been observed to help some antibiotics penetrate bacteria better.[7,20,33] According to the findings of Dr. Devia and collaborators, hyperbaric therapy, together with the negative pressure system, was shown to decrease mortality by up to 11.4%; however, Hatipoglu mentions that hyperbaric treatment has a high risk of cerebral and pulmonary complications, as well as increased costs, which limits its use.[33,34]

The debridement area is usually located in regions close to the anus, so the wound must be protected from contamination by fecal matter, diverting the fecal matter to keep the wound clean. Usually, the fecal diversion is performed by colostomy when the scars are near the perianal region or by the fecal management system Flexi-Seal™, an alternative method that consists of the placement of a rectal tube that allows the exit of the matter through it to a collection bag. It is an economical and comfortable alternative for the patient and avoids the need for colostomy.[33,35]

PROGNOSIS

Despite treatment, mortality is reported to be 20-40%.[4] The causes of death in these patients are severe sepsis, coagulopathies, acute renal failure, diabetic ketoacidosis, and multiorgan failure.[20,35]

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