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Fournier Gangrene: Nursing Assessment and Management-An Updated Review

Fahad Mohammed Ali Kariri [(1)] , Hanin Mohammed Abdu Mashraqi [(2)] , Saud Faisal Almutairi [(3)] , Ali Mhthal Faleh Alqahtani [(4)] , Zuhur Ali Hassan Majrashi [(5)] , Sultanah Essa Hussein Almaslamani [(6)] , Mohrah Hamoud Dughaylib Alrashdi [(7)] , Rehab Saleh Alenezi [(8)] , Mushyirah Ahmed A Hadadi [(9)] , Laila Mohammed A Alnaji [(10)] , Khulud Mohammed Saleh Zaybi [(11)] , Saleh Talea Sarar ALqahtani [(12)]

(1) Primary Health Care – Ahad Al Marsha, Jazan,Ministry of Health, Saudi Arabia,
(2) King Fahad Central Hospital,Ministry of Health, Saudi Arabia,
(3) Al-Mithnab General Hospital,Ministry of Health, Saudi Arabia,
(4) Ruwaidat Al-Ardh General Hospital – Al-Quwayiyah Ministry of Health, Saudi Arabia,
(5) Jazan Health Cluster,Ministry of Health, Saudi Arabia,
(6) Alwajh Hospital – Alwajh ,Ministry of Health, Saudi Arabia,
(7) Al-Dawadmi – Al-Shamiyah Primary Health Care Center,Ministry of Health, Saudi Arabia,
(8) Prince Sultan Medical Maternity Hospital,Ministry of Health, Saudi Arabia,
(9) ALKARBOS PRIMARY HEALTH CARE CENTER,Ministry of Health, Saudi Arabia,
(10) Mushaliha Primary Health Care Center,Ministry of Health, Saudi Arabia,
(11) Ministry Of Health, Saudi Arabia,
(12) Al-Farsha General Hospital,Ministry of Health, Saudi Arabia

Abstract

Background: Fournier gangrene is a rare but life-threatening form of necrotizing soft tissue infection affecting the perineal and genital regions, associated with rapid progression, sepsis, and high mortality despite modern advances in care.

Aim: This review aimed to provide an updated overview of Fournier gangrene with a specific focus on nursing assessment, early recognition, and multidisciplinary management to improve patient outcomes.

Methods: A narrative literature review was conducted, synthesizing current evidence on etiology, epidemiology, pathophysiology, clinical presentation, diagnostic evaluation, treatment strategies, nursing considerations, and prognosis of Fournier gangrene.

Results: Fournier gangrene is characterized by polymicrobial infection spreading along fascial planes, leading to extensive tissue necrosis and systemic toxicity. Diabetes mellitus, immunosuppression, and advanced age are major risk factors. Early symptoms are often subtle, resulting in frequent misdiagnosis. Prompt surgical debridement, broad-spectrum antibiotics, aggressive resuscitation, and vigilant nursing care are critical determinants of survival. Delayed intervention is strongly associated with increased mortality.

Conclusion: Early recognition, rapid surgical management, and comprehensive nursing assessment play a vital role in reducing morbidity and mortality in Fournier gangrene.

Keywords: Fournier gangrene, necrotizing fasciitis, nursing management, sepsis, surgical debridement.

Introduction

Fournier gangrene is a life-threatening necrotizing infection that primarily affects the perineal, genital, scrotal, and perianal regions. This condition is characterized by rapid progression along fascial planes, resulting in extensive soft tissue destruction, vascular thrombosis, and subsequent ischemic necrosis. First described by Dr. Alfred Fournier in 1883, the initial report documented five previously healthy men who developed acute, rapidly advancing gangrene of the external genitalia and perineal tissues. The disease's aggressive nature distinguishes it from superficial infections, as it can extend beneath apparently normal skin, rendering early detection difficult and often delaying intervention [1][2][3]. The pathophysiology involves polymicrobial infection, usually consisting of a combination of aerobic and anaerobic bacteria that synergistically contribute to tissue destruction. The infection produces enzymes and toxins that facilitate rapid spread along the Dartos, Colles, and Scarpa fascial planes, potentially reaching the abdominal wall and anterior thigh. Thrombosis of subcutaneous vessels leads to ischemia, further accelerating necrosis and predisposing affected tissue to secondary infections. Clinically, early signs are frequently subtle, often mimicking cellulitis or minor soft tissue infections, which complicates prompt recognition [6][7][8]. Patients often present with systemic manifestations of sepsis, including fever, tachycardia,

*Corresponding author e-mail: Fahad_kuriry@hotmail.com (Fahad Mohammed Ali Kariri).
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hypotension, and malaise. Local symptoms may include erythema, edema, tenderness, and crepitus, though these findings can be absent in the initial stages. Due to the rapid progression and high mortality rate, which can reach 40%, early clinical suspicion is critical, particularly in patients with predisposing factors such as diabetes mellitus, immunosuppression, chronic alcoholism, or local trauma [4][5]. Prompt recognition and aggressive management are essential to improve survival. Surgical debridement, broad-spectrum intravenous antibiotics, hemodynamic support, and close monitoring form the cornerstone of treatment. Delays in diagnosis or intervention are associated with rapid deterioration and extensive tissue loss. Because the initial presentation may be deceptively mild, clinicians must maintain a high index of suspicion in at-risk individuals presenting with perineal or genital discomfort, systemic signs of infection, or rapidly progressing soft tissue changes [2][3][8][9][10]. Early intervention remains the most significant factor influencing prognosis in this otherwise fatal disease.

Etiology

Fournier gangrene arises from a synergistic polymicrobial infection involving both aerobic and anaerobic bacteria that invade the fascia and subcutaneous tissues of the perineum, genitalia, and surrounding areas. Microbiological analyses of affected patients frequently identify mixed bacterial populations, including gram-positive organisms such as group A Streptococcus and Staphylococcus aureus, alongside gram-negative bacteria like Escherichia coli and Pseudomonas aeruginosa, emphasizing the polymicrobial etiology of the disease [11][12]. These pathogens gain entry through a variety of portals, often related to the urinary, gastrointestinal, or dermal systems. Urinary tract infections, perianal abscesses, and minor dermatologic lesions in the perineal region may serve as initiating foci, allowing bacteria to infiltrate deeper tissues [6]. Traumatic events and surgical procedures in the perineal or genital regions are additional predisposing factors. Surgical interventions, catheterizations, or other manipulations can disrupt local tissue integrity, facilitating bacterial invasion. Similarly, minor skin injuries, lacerations, or ulcers may allow pathogens to penetrate subcutaneous tissues, setting the stage for rapid infection progression [6][7][13][14][15]. Despite these known risk factors, approximately one-quarter of cases present without an identifiable source, highlighting the sporadic and sometimes idiopathic nature of the condition [16]. Emerging evidence suggests a potential association between the use of sodium-glucose cotransporter-2 (SGLT2) inhibitors and the development of Fournier gangrene. Although the link remains under investigation and somewhat controversial, clinicians are advised to discontinue SGLT2 inhibitors immediately if Fournier gangrene is suspected and to promptly initiate broad-spectrum antimicrobial therapy and supportive care [17][18][19]. Early recognition of these risk factors and predisposing conditions is critical, as timely identification and intervention significantly impact patient outcomes and reduce the high morbidity and mortality associated with this aggressive soft tissue infection.

Epidemiology

Fournier gangrene is an uncommon but severe form of necrotizing soft tissue infection, representing less than 0.02% of all hospital admissions. The condition predominantly affects males, with a male-to-female ratio estimated at 10:1, although affected females experience disproportionately higher morbidity and mortality rates [20][21][22]. The overall incidence among men is approximately 1.6 cases per 100,000 population, with peak prevalence observed in men aged 50 to 79 years, who experience an incidence rate of 3.3 cases per 100,000 [12][23]. Regional variations exist, with the southeastern United States reporting the highest frequency at 1.9 cases per 100,000 population [24]. Globally, these patterns appear consistent, although the disease remains rare in women, who often present with more severe clinical manifestations, including a higher likelihood of requiring mechanical ventilation, dialysis, prolonged hospitalization, and a greater risk of mortality compared to male patients [22][24]. While Fournier gangrene can develop in otherwise healthy individuals, approximately 70% to 74% of cases occur in patients with underlying comorbidities or immunocompromised states [5]. Diabetes mellitus is the most frequently associated condition, reported in 20% to 70% of cases [26], and is often accompanied by other risk factors such as chronic alcohol use, atherosclerosis, and obesity. Additional predisposing conditions include chronic steroid therapy, HIV infection, inflammatory bowel disease, renal failure, liver cirrhosis, and malignancies, particularly colorectal or prostate cancer [7][27][28][29][30]. Trauma or surgical procedures in the perineal or urogenital region, including prostate biopsy, catheterization, and cystoscopy, are recognized as potential initiating events [29]. Use of sodium-glucose cotransporter-2 (SGLT2) inhibitors, recently implicated in increasing the risk of Fournier gangrene, represents a newer pharmacologic association requiring clinical vigilance [31]. Other contributing factors include malnutrition, neurogenic bladder, urethral strictures, and spinal cord injury. The presence of multiple comorbidities significantly correlates with more severe disease and poorer outcomes [5]. Despite advances in critical care and sepsis management, Fournier gangrene continues to carry a high fatality rate of approximately 40% [5][32][33]. Delays in diagnosis and surgical intervention substantially increase mortality, with delayed recognition associated with death rates rising up to 88% [11][25][33]. Timely surgical debridement remains the most critical determinant of survival, as early operative management can halve the mortality

rate [11][34][35]. Patients with severe diabetes, cardiac disease, or renal failure are particularly vulnerable, demonstrating significantly elevated risk of death compared to healthier individuals [16]. These epidemiologic patterns highlight the importance of early recognition, prompt surgical intervention, and aggressive management of underlying comorbidities to improve survival in patients affected by Fournier gangrene.

Pathophysiology

Fournier gangrene develops through a polymicrobial infection in which aerobic and anaerobic bacteria interact synergistically to produce extensive tissue damage. The infection typically originates from a breach in the protective barriers of the perineum, scrotum, or genital area. Common initiating insults include urinary tract infections, perianal or perineal abscesses, localized cellulitis, minor skin trauma, or recent surgical procedures in the urogenital region. In these scenarios, bacteria gain access to the subcutaneous tissues and fascia, creating an environment conducive to rapid microbial proliferation [36][37]. The bacterial synergy results in the production of potent tissue-destructive enzymes, including collagenases and lipases, as well as endotoxins. These substances induce obliterative endarteritis characterized by microvascular thromboses, leading to ischemia and necrosis of the subcutaneous tissues and fascia. This vascular compromise limits the delivery of immune cells and systemic antibiotics to the affected area, accelerating the progression of necrosis [36][37]. The infection spreads along fascial planes, including the Dartos, Colles, and Scarpa fascia, permitting extension to the scrotum, perineum, and even the lower abdominal wall. The speed of tissue invasion is notable, with reports indicating that the infection can advance as much as one inch per hour, highlighting the aggressive nature of Fournier gangrene [38]. Host factors significantly influence disease progression. Comorbidities such as diabetes mellitus, immunosuppression, chronic renal or hepatic disease, and vascular compromise impair the body's ability to mount an effective immune response and exacerbate ischemia. Additionally, the hypoxic and nutrient-depleted microenvironment created by microthromboses further enhances bacterial growth and promotes the synergistic activity of anaerobic and aerobic organisms. In combination, these processes result in the characteristic rapid tissue destruction, systemic inflammatory response, and high mortality associated with Fournier gangrene [36][37][38].

Histopathology

Histopathological evaluation of Fournier gangrene, while not always required for diagnosis, provides insight into the extent and nature of tissue damage and can help distinguish it from severe cellulitis. Characteristic findings include widespread necrosis of the epidermis, dermis, and subcutaneous tissues, with the destruction often extending to the fascial planes. Thrombosis of small and medium-sized vessels is commonly observed, reflecting the obliterative endarteritis caused by bacterial toxins and host inflammatory responses [39]. Inflammatory infiltrates are typically dominated by neutrophils, often forming dense exudates within necrotic tissue. Bacterial colonies may be visible microscopically, particularly when Gram staining highlights mixed populations of aerobic and anaerobic organisms. Cellular debris, edema, and hemorrhage are frequently present, reflecting ongoing tissue destruction and ischemia. Ulceration of the overlying epidermis is common, while deeper layers exhibit coagulative necrosis. In severe cases, necrosis may extend into skeletal muscle and adjacent soft tissue, although muscle involvement is less common due to relative resistance [39]. Histopathology also demonstrates the presence of bacterial enzymes and endotoxins mediating local tissue destruction, highlighting the pathophysiologic mechanism of rapid spread. These findings correlate with the clinical observation of systemic toxicity, sepsis, and hemodynamic instability, which frequently accompany Fournier gangrene. While imaging and clinical assessment often suffice for diagnosis, histological confirmation may be indicated in atypical or early-stage presentations, particularly when differentiation from cellulitis, pyoderma gangrenosum, or other necrotizing soft tissue infections is necessary [39].

History and Physical

Patients with Fournier gangrene commonly present with pain localized to the perineal, scrotal, or genital regions, although the intensity and visibility of early signs can vary significantly. Initial manifestations may be subtle, with mild erythema, tenderness, or edema, which can easily be mistaken for benign conditions such as cellulitis or erysipelas [6][12]. Clinicians should maintain a high index of suspicion when evaluating perineal or scrotal infections, particularly when reported pain is disproportionate to the visible lesion. The rapid progression of infection, combined with the potential for systemic toxicity, underscores the importance of early recognition and timely intervention [8][44]. The patient's medical history often provides critical clues to risk stratification. Comorbidities such as diabetes mellitus, hypertension, malignancy, chronic alcohol use, or immunosuppression are frequently reported and are established predisposing factors [14][40][41][42]. Approximately half of all patients with Fournier gangrene have diabetes, highlighting the significance of metabolic dysregulation in disease development. Additional historical factors may include recent trauma to the genital or perineal area, surgical interventions, urinary tract infections, or other genitourinary or gastrointestinal insults that may introduce pathogens into the subcutaneous tissues [43]. Systemic manifestations often accompany local

symptoms. Patients may report fever, chills, malaise, nausea, vomiting, and urinary retention. Early local signs include itching, localized tenderness, erythema, and edema. Clinically, the overlying skin may initially appear unremarkable, which can delay diagnosis [12]. As the infection progresses, more definitive features emerge, including bullae formation, purulent drainage, crepitus, and patchy areas of black or dusky discoloration [8][44][45]. In a representative cohort, scrotal swelling was reported in 79% of patients, tachycardia in 61%, purulent drainage in 60%, crepitus in 54%, and fever in 41% [45]. Early recognition of subtle signs such as small perineal lesions or unexplained tenderness is crucial for preventing rapid deterioration.

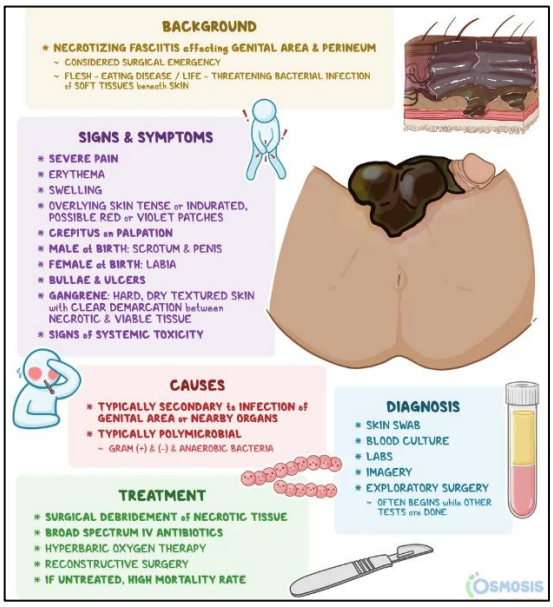

Fig. 1: Fournier Gangrene.

While Fournier gangrene predominantly affects males, the incidence in females is rising. In women, the infection typically presents with labial or vulvar involvement and is more likely to occur in patients with morbid obesity [45]. Notably, early diagnosis is challenging, as up to 40% of patients may initially be asymptomatic, and approximately 75% of cases are initially misdiagnosed, contributing to the persistently high mortality rate of around 40% [4][40][47]. Severe pain out of proportion to physical findings is a hallmark feature, and the presence of crepitus on palpation, purpura, bullae, or black tissue discoloration indicates progression. Subcutaneous gas formation, often due to anaerobic organisms such as Clostridia, is frequently detected and correlates with rapid fascial involvement [48][49]. The infection typically spreads along fascial planes, including the Dartos, Colles, and Scarpa fascia, and may extend to the lower anterior abdominal wall. Late-stage infection is marked by systemic manifestations of sepsis, including hypotension, tachycardia, tachypnea, fever, and multi-organ dysfunction [12][50]. Although testicular and spermatic cord structures are generally spared due to independent blood supply, they may become involved if the infection originates in these structures [7]. Fournier gangrene can progress rapidly, with the average time from initial symptom onset to hospital admission reported as approximately five days [52]. The natural history can be divided into five stages: prodromal symptoms such as lethargy and fever; localized genital tenderness and mild erythema; intense pain with worsening erythema; subcutaneous crepitation with dusky skin changes; and purulent drainage with overt tissue necrosis [53]. Special attention during examination is required for patient populations in which perineal and genital areas are infrequently inspected, including patients who are elderly, frail, morbidly obese, nonverbal, obtunded, or neurologically impaired due to spinal cord injury or other disabilities [12][54][55]. Thorough history taking and meticulous physical examination in these populations are essential for early detection and prompt intervention, which are critical for improving outcomes in this aggressive and potentially fatal condition.

Evaluation

The evaluation of patients with suspected Fournier gangrene requires a comprehensive approach that integrates clinical assessment, laboratory studies, and imaging while prioritizing prompt recognition and timely surgical intervention. Fournier gangrene remains primarily a clinical diagnosis, as early signs are often subtle, and the disease can progress rapidly along fascial planes, leading to life-threatening sepsis. Clinicians should maintain heightened vigilance in patients with known risk factors, including diabetes mellitus, immunosuppression, chronic alcohol use, recent trauma or surgery to the perineal or genital region, and older males, as these individuals are particularly susceptible to aggressive soft tissue infections [6][14][40]. Laboratory evaluation plays a crucial role in supporting the diagnosis and identifying systemic involvement. A complete blood count (CBC) typically reveals leukocytosis with a left shift, reflecting the acute inflammatory response. Electrolyte disturbances such as hyponatremia, hyperglycemia, and metabolic acidosis may be evident on a comprehensive metabolic panel (CMP), particularly in patients with pre-existing renal impairment or sepsis [56]. Measurement of serum lactate, C-reactive protein (CRP), and procalcitonin provides additional information regarding the degree of systemic inflammation, sepsis, and tissue hypoperfusion [56]. Arterial blood gases may assist in evaluating oxygenation and acid-base status, particularly in patients with respiratory compromise or extensive systemic involvement [57]. Blood and wound cultures are essential for identifying the polymicrobial pathogens typically implicated in Fournier gangrene, guiding empiric and targeted antibiotic therapy [57][58]. Imaging serves as a valuable adjunct to clinical evaluation by confirming disease extent and identifying subcutaneous gas or necrosis. Ultrasound is particularly useful for

assessing the scrotum and perineum, revealing thickened scrotal walls, linear hypoechoic fluid streaks, increased vascularity on Doppler, and subcutaneous gas [60][62]. Ultrasonographic findings such as cobblestoning, the snow globe effect, and dirty shadowing help distinguish Fournier gangrene from other soft tissue infections [12][49][61][62][64]. X-ray imaging can detect subcutaneous emphysema along fascial planes in up to 90% of cases, although the absence of visible gas does not exclude the diagnosis [12][47].

Computed tomography (CT) is considered the most sensitive and specific imaging modality for Fournier gangrene, with sensitivity of 88.5% and specificity of 93.3% [65]. CT findings typically include fat stranding, asymmetrical fascial thickening, fluid collections, abscess formation, and subcutaneous emphysema [64][66][67][68][69]. CT is particularly useful for identifying the source of infection, evaluating deep fascial involvement, and ruling out intra- or retroperitoneal pathology [70][71]. Postoperative CT may be used to assess for residual infection or wound healing [72]. While magnetic resonance imaging (MRI) provides excellent soft tissue contrast and detail, it is not routinely recommended for initial evaluation due to extended acquisition times, cost, and limited practicality in emergent settings [45][48][73]. Importantly, the presence of gas on imaging, while highly specific, has limited sensitivity, particularly in early or deep fascial infections, and should not be relied upon exclusively [12][65][67]. Because Fournier gangrene can progress rapidly, clinical judgment takes precedence: in unstable or septic patients, surgical intervention must be initiated immediately without waiting for confirmatory laboratory or imaging studies. Delays in operative management are associated with significantly increased mortality, emphasizing that early recognition and prompt debridement are the most critical factors in optimizing patient outcomes [12][56][59]. Overall, the evaluation of Fournier gangrene requires an integrated, multidisciplinary approach that balances thorough assessment with urgent surgical intervention. Laboratory markers, imaging studies, and microbiological cultures are crucial for supporting diagnosis, assessing disease severity, guiding treatment, and monitoring systemic involvement. However, the cornerstone of evaluation remains careful clinical assessment and prompt recognition of subtle early signs, particularly in high-risk populations. Failure to act decisively in the face of suspected Fournier gangrene can result in rapid tissue destruction, sepsis, and death, highlighting the life-saving importance of immediate surgical management.

Treatment / Management

Fournier gangrene is a surgical emergency that demands immediate intervention due to its rapid progression, high mortality rate, and frequent association with sepsis and multi-organ dysfunction [43]. Management involves a coordinated combination of medical resuscitation, broad-spectrum antibiotic therapy, aggressive surgical debridement, and adjunctive supportive care, with the overarching goal of halting the spread of infection, preserving viable tissue, and stabilizing the patient hemodynamically. Medical management begins immediately upon suspicion of Fournier gangrene. Empiric broad-spectrum antibiotics should be administered without delay, even before culture results are available. The infection is typically polymicrobial, involving aerobic and anaerobic bacteria, including gram-positive organisms such as Streptococcus and Staphylococcus species, gram-negative coliforms like Escherichia coli and Pseudomonas aeruginosa, anaerobes such as Bacteroides and Clostridia, and occasionally yeast [48]. Historically, triple therapy has been recommended to cover all relevant pathogens. Current guidelines suggest regimens such as carbapenems (imipenem or meropenem 1 g IV every 6–8 hours, or ertapenem 1 g IV every 24 hours), piperacillin-tazobactam (3.375 g IV every 6 hours or 4.5 g IV every 8 hours), in combination with clindamycin (600–900 mg IV every 8 hours) and vancomycin (15–20 mg/kg IV every 8–12 hours) to provide coverage for resistant gram-positive organisms [12][44][61]. Alternatives to vancomycin include daptomycin or linezolid. Antifungal therapy with amphotericin B or fluconazole may be added in cases of suspected fungal involvement [44]. Patients with exposure to aquatic environments may require doxycycline to cover Vibrio or Aeromonas species.

Aggressive fluid resuscitation is a cornerstone of initial management, as patients often present with hypotension, septic shock, and electrolyte imbalances. Balanced crystalloids such as Lactated Ringer solution are preferred for correcting hypovolemia and electrolyte disturbances [12][75][76][77]. Vasopressors may be required if hypotension persists despite adequate fluid replacement [75]. Blood glucose management is particularly important in diabetic patients, as hyperglycemia can exacerbate infection and impair wound healing [66]. Definitive management of Fournier gangrene is prompt and aggressive surgical debridement, which remains the single most critical intervention in reducing mortality [4][34][78]. Surgical exploration must be immediate, even if imaging or laboratory results are pending, to prevent further tissue necrosis and systemic compromise. Delays of more than 12 hours from initial presentation, or even incremental delays of a few hours, significantly increase mortality [79]. Surgical intervention requires radical resection of all necrotic tissue, often extending beyond visibly affected skin.

Multiple surgical specialties may be involved, including urology, general surgery, plastic surgery, colorectal surgery, and obstetrics-gynecology, depending on the extent and origin of infection. Urology typically leads cases involving the scrotum or urethra, whereas colorectal or general surgery manage infections extending into the anorectal region or abdominal wall. Multidisciplinary coordination ensures comprehensive debridement without unnecessary delays [5][80][81].

The testicles are typically spared due to independent blood supply via the spermatic cord. Preservation techniques include temporary placement in subcutaneous thigh pouches, with permanent positioning achieved through scrotal advancement or pudendal thigh flaps [5][80][81]. Penile corpora cavernosa and urethra are usually preserved unless initially involved, while penile skin may require resection [82]. Severe cases often require staged debridement, sometimes necessitating three or more operations [66][83]. Vacuum-assisted closure (VAC) systems are frequently used postoperatively to optimize wound healing, reduce fluid accumulation, and promote granulation tissue formation [39][84][85][86][87]. Reconstructive surgery is indicated once infection is controlled and granulation tissue has formed. Techniques include local or distant skin flaps, split-thickness grafts, and in selected cases, stem cell therapy. Colostomy or fecal management systems may be required for perineal involvement to divert stool and support healing. While fecal management systems shorten hospitalization compared to colostomy, colostomies are still necessary when the anal sphincter, rectum, or perineal area is significantly involved [89][90][91]. Decisions regarding fecal diversion should be individualized, as these interventions do not improve mortality and are associated with increased morbidity [92][93]. Adjunctive therapies such as hyperbaric oxygen therapy (HBOT) may improve tissue oxygenation, enhance antibiotic efficacy, and reduce morbidity, although its use should never delay surgical intervention [94][95][96][97][98][100]. Other potential adjuncts include honey application, intravenous immunoglobulins, and therapeutic plasma exchange, though current evidence remains insufficient to recommend these routinely [47][66][102][103][104].

The principles of effective management focus on early recognition, rapid hemodynamic stabilization, immediate initiation of broad-spectrum antibiotics, and aggressive surgical debridement. Preservation of vital structures such as the testicles, urethra, and penile corpora is prioritized, with staged reconstruction tailored to tissue loss. VAC therapy and hyperbaric oxygen may aid recovery, while fecal diversion is selectively applied. Successful outcomes rely on multidisciplinary coordination, timely intervention, and vigilant postoperative monitoring to minimize complications, reduce morbidity, and improve survival [59]. In conclusion, the treatment of Fournier gangrene integrates rapid surgical intervention, aggressive medical resuscitation, broad-spectrum antimicrobial therapy, and supportive postoperative care. Early diagnosis and prompt management are critical in reducing the high mortality rate associated with this condition. The combination of hemodynamic stabilization, staged debridement, and reconstruction, along with adjunctive therapies as appropriate, represents the current standard of care for optimizing outcomes in patients affected by this life-threatening infection.

Differential Diagnosis

Fournier gangrene presents with nonspecific early signs such as perineal or genital pain, swelling, erythema, and systemic symptoms, making accurate differentiation from other conditions essential. Misdiagnosis can delay treatment and worsen outcomes. Clinicians must consider a broad range of infectious, inflammatory, vascular, and urologic conditions that mimic the early manifestations of Fournier gangrene [43][66]. Infectious causes include cellulitis, erysipelas, epididymitis, orchitis, gangrenous balanitis, gangrenous vulvitis, scrotal abscess, and perianal or periurethral abscess. These conditions can produce localized erythema, tenderness, and systemic symptoms such as fever, similar to Fournier gangrene, but typically lack extensive fascial involvement and rapid progression. Viral infections such as herpes simplex and syphilis, or fungal infections including invasive candidiasis and mucormycosis, should also be considered when lesions are ulcerative or necrotic. Non-infectious causes include scrotal edema, deep vein thrombosis, acute renal colic with referred pain, testicular torsion, and pyomyositis, which can present with pain and swelling in the genital or perineal region. Autoimmune or dermatologic conditions such as pyoderma gangrenosum, vasculitis, Stevens-Johnson syndrome, and toxic epidermal necrolysis may mimic the necrotic and erythematous lesions seen in advanced Fournier gangrene. Toxic shock syndrome should also be considered in patients with systemic inflammatory response and rapid progression. Accurate differentiation relies on careful clinical evaluation, history, and adjunctive imaging or laboratory testing when necessary. Early recognition of the rapid progression, disproportionate pain, subcutaneous emphysema, and systemic toxicity can help distinguish Fournier gangrene from these alternative diagnoses and guide prompt surgical and medical intervention to reduce morbidity and mortality.

Prognosis

The prognosis of Fournier gangrene is influenced by multiple factors, with overall mortality remaining high at approximately 40% despite advances in antibiotics and surgical care.[5][32][33][105] Mortality increases in patients with underlying comorbidities such as poorly controlled diabetes, renal or hepatic failure,

cardiovascular disease, and immunosuppression. Septic shock on presentation, delayed diagnosis, extensive tissue involvement, and infection spread into the abdominal or retroperitoneal space are strongly associated with worse outcomes.[105][106][107] Female patients, patients with fungal infections, and those with multidrug-resistant organisms also tend to have poorer prognoses.[108] Several scoring systems have been developed to help predict outcomes in patients with Fournier gangrene. The Fournier Gangrene Severity Index (FGSI) incorporates vital signs and laboratory values, including temperature, heart rate, respiratory rate, serum sodium, potassium, creatinine, bicarbonate, hematocrit, and white blood cell count. A FGSI score above 9 correlates with a mortality rate exceeding 75%, while scores below 9 predict survival in approximately 78% of cases.[109] Electrolyte disturbances such as elevated calcium or low magnesium levels further increase mortality risk.[57] Age and extent of tissue involvement are additional key prognostic factors. The Uludag FGSI (UFGSI) incorporates these variables, showing a score above 9 is associated with a 94% mortality risk, whereas a score below 9 predicts an 81% chance of survival.[110] General surgical scoring systems, such as the age-adjusted Charlson Comorbidity Index (ACCI) and the surgical APGAR score (sAPGAR), provide additional prognostic guidance. The ACCI assigns points to 19 comorbid conditions, with higher scores linked to increased mortality. The sAPGAR score evaluates estimated blood loss, lowest mean arterial pressure, and lowest heart rate, with lower scores indicating higher postoperative complication risk. Comparisons suggest ACCI offers high sensitivity and specificity for predicting outcomes while remaining clinically practical.[111][112] Timing of intervention is critical. Early recognition and emergent surgical debridement significantly improve survival. Delays in surgery increase mortality, with even three-hour delays associated with worse outcomes.[4][34] Glycemic control is also a factor, as diabetic patients with HbA1c above 7% demonstrate worse prognosis.[109][113] Biomarkers such as a high neutrophil-to-lymphocyte ratio (>9) and a low lymphocyte-to-CRP ratio may serve as indicators of increased mortality risk, reflecting systemic inflammation and immune dysregulation.[114][115][116] In summary, prognosis depends on comorbidities, extent of infection, timing of surgery, age, and systemic response to sepsis. Early diagnosis and aggressive multidisciplinary management remain the most effective strategies to improve survival in patients with Fournier gangrene.

Complications

Fournier gangrene carries a high risk of both systemic and local complications due to its rapidly progressive and destructive nature. Short-term systemic complications primarily result from sepsis and the host's inflammatory response. Acute renal failure is common, especially in patients with preexisting kidney disease or those who develop hypotension and hypoperfusion. Acute respiratory distress syndrome (ARDS) may develop secondary to systemic inflammation, while cardiac complications such as arrhythmias, heart failure, and myocardial injury are frequently observed.[117] Multiple organ dysfunction can rapidly evolve, with bacteremia contributing to thromboembolic events, including strokes and peripheral arterial occlusion, potentially leading to limb loss. Ileus is another complication, often resulting from prolonged hospitalization, critical illness, or repeated surgical interventions. Postoperative wound infections remain a concern, although adjunctive therapies like hyperbaric oxygen have demonstrated reductions in infection rates.[117] Local complications are significant due to extensive tissue necrosis. Involvement of the anal sphincter or perineum may lead to fecal incontinence, often necessitating a diverting colostomy to protect the wound from contamination. Colostomy carries potential complications including stoma evisceration, infection, or obstruction. Urinary retention is common when periurethral edema is present, requiring catheterization or, in severe cases, suprapubic cystostomy. Urinary tract infections may arise secondary to instrumentation or obstruction. Penile involvement may result in tissue loss, scarring, and, in some cases, partial penile amputation if necrosis is extensive.[66] Long-term complications include profound psychological and sexual consequences. Patients frequently report persistent genital pain, reduced quality of life, and increased risk of depression. Reconstructive surgery may leave disfiguring scars, contributing to emotional distress. Sexual dysfunction is also prevalent, with impaired erectile function, penile deviation, torsion, or loss of sensitivity commonly reported. Pain during erections or intercourse can further exacerbate sexual and psychological morbidity.[7][118] Overall, the complications of Fournier gangrene are extensive, ranging from acute, life-threatening systemic effects to chronic physical, functional, and psychological sequelae. Close monitoring, early intervention, and multidisciplinary care are essential to limit both immediate and long-term consequences of this devastating disease.

Conclusion:

Fournier gangrene remains a devastating and rapidly progressive surgical emergency with persistently high mortality rates despite advances in antimicrobial therapy, critical care, and reconstructive surgery. The disease is frequently associated with systemic comorbidities such as diabetes mellitus, immunosuppression, and chronic organ dysfunction, which significantly worsen outcomes. A major challenge in management is the subtlety of early clinical signs, leading to frequent misdiagnosis and dangerous delays in treatment. This highlights the

essential role of healthcare professionals—particularly nurses—in early detection, continuous assessment, and rapid escalation of care. Effective management relies on a multidisciplinary approach combining immediate hemodynamic stabilization, broad-spectrum antibiotic therapy, and aggressive surgical debridement. Nursing care is central throughout this process, encompassing early risk identification, meticulous monitoring of vital signs, wound management, glycemic control, pain management, psychosocial support, and coordination of interdisciplinary care. Postoperative and rehabilitative nursing interventions are equally important in preventing complications, promoting wound healing, and addressing long-term physical and psychological sequelae. In conclusion, improving survival in Fournier gangrene largely depends on early recognition, timely surgical intervention, and high-quality nursing care. Ongoing education, clinical vigilance, and adherence to evidence-based protocols are essential to enhance patient outcomes and reduce the significant morbidity and mortality associated with this life-threatening condition.

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