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Clinical Reviews in Emergency Medicine

FOURNIER GANGRENE: A REVIEW FOR EMERGENCY CLINICIANS

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☐ **Abstract—Background: Fournier gangrene (FG) is a rare, life-threatening infection that can result in significant morbidity and mortality, with many patients requiring emergency department (ED) management for complications and stabilization. Objective: This narrative review provides an evidence-based summary of the current data for the emergency medicine evaluation and management of FG. Discussion: Although originally thought to be an idiopathic process, FG has been shown to have a strong association for male patients with advanced age and comorbidities affecting microvascular circulation and immune system function, most commonly those with diabetes or alcohol use disorder. However, it can also affect patients without risk factors. The initial infectious nidus is usually located in the genitourinary tract, gastrointestinal tract, or perineum. FG is a mixed infection of aerobic and anaerobic bacterial flora. The development and progression of gangrene is often fulminant and can rapidly cause multiple organ failure and death, although patients may present subacutely with findings similar to cellulitis. Laboratory studies, as well as imaging including point-of-care ultrasound, conventional radiography, and computed tomography are important diagnostic adjuncts, though negative results cannot exclude diagnosis. Treatment includes emergent surgical debridement of all necrotic tissue, broad-spectrum antibiotics, and resuscitation with intravenous fluids and vasoactive medications. Conclusions: FG requires a high clinical level of suspicion, combined with knowledge of anatomy, risk factors, and etiology for an accurate diagnosis. Although FG remains a clinical diagnosis, relevant laboratory and radiography investigations can serve as useful adjuncts to expedite surgical management, hemodynamic resuscitation, and antibiotic administration. Published by Elsevier Inc.**

☐ **Keywords—necrotizing soft tissue infections; infectious disease; Fournier gangrene**

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INTRODUCTION

Although necrotizing soft tissue infections (NSTIs) comprise a wide variety of severe infections, the term necrotizing fasciitis, in particular, refers to any NSTI involving the fascial planes (1). An NSTI extending into the perineal, perianal, and genital area is termed Fournier gangrene (FG) (2). Although the understanding of the underlying pathophysiology of NSTIs, including FG, continues to improve, the mortality of this disease remains alarmingly high, at 20–50% in most contemporary series (3,4). Delayed diagnosis and consequently delayed operative debridement increase morbidity and mortality (5–7). This is understandable—the greater the delay, the greater the tissue loss and the greater the mortality. One of the principal reasons for the continued high mortality in FG and associated NSTIs

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is a failure to diagnose the condition early due to the paucity of specific clinical signs and symptoms early in its course (8,9). It is therefore imperative that emergency physicians not only have a high level of suspicion but also possess knowledge regarding the broad range of epidemiological risk factors, clinical presentations, and diagnostic adjuncts at their disposal when confronted with such clinical uncertainties. This narrative review evaluates the relevant pathophysiology, presenting features, laboratory and imaging adjuncts, and treatment strategies to aid diagnosis and allow timely surgical management of FG.

METHODS

Authors searched PubMed and Google Scholar for articles using the keywords "necrotizing fasciitis" and "emergency" as well as "Fournier gangrene" and "emergency" for production of this narrative review. Authors included case reports and series, retrospective and prospective studies, systematic reviews and meta-analyses, clinical guidelines, and other narrative reviews. The literature search was restricted to studies published in English, with focus on emergency medicine and critical care literature. Emergency physicians with experience in critical appraisal of the literature decided which studies to include for the review by consensus.

RESULTS

A total of 117 resources were selected for inclusion in this review. Of these 117 articles, 79 evaluate the epidemiology, presentation, laboratory and imaging assessment, and management of FG; 7 articles evaluate diagnosis and scoring systems concerning general NSTI; 11 articles examine imaging tests in NSTI; 9 studies evaluate general management of NSTI; 6 studies discuss management of sepsis; 3 articles investigate potential complications and triggers of FG (e.g., diabetic complications and alcohol withdrawal); and 2 investigate transfer systems for NSTI.

DISCUSSION

Epidemiology

FG remains a rare clinical entity, representing < 0.02% of all hospital admissions (10). FG has an incidence of 1.6 per 100,000 men in the United States, which peaks between the ages of 50 and 79 years (3.3 per 100,000), with the highest rates occurring in the South (1.9 per 100,000) (10). In comparison, women have an incidence of 0.25 per 100,000 (10). However, the incidence of FG is rising, likely due to an increase in the mean age of the population, in addition to increasing numbers of patients on immunosuppressive therapy or suffering from human immunodeficiency virus infection (10,11). The overall case fatality rate in contemporary literature ranges from 7.5% to upwards of 50% (3,10,12). This is likely due to the fact that much of the published literature tends to be from tertiary referral centers, reflecting a more severely ill population (12). Most hospitals (approximately 66% overall) care for no patients with FG during a given year, and only 1% of hospitals cared for more than 5 cases per year (12).

Primarily an infectious process, FG has several predisposing factors, and theoretically, any condition that impairs host immune response or microcirculation may predispose a patient to FG (Table 1) (10,13–20). In certain areas, near-epidemic proportions of human immunodeficiency virus place a significant proportion of the population at risk for FG (21). The use of nonsteroidal anti-inflammatory drugs or pain medication can suppress fever and pain, potentially hampering the diagnosis of FG (22). Advanced age is another risk factor for higher incidence and associated mortality, although this is debated within the literature (22). Large population-based studies have shown that age is a strong, independent predictor of mortality in NSTIs, although some studies have found this to be true only when accompanied by other risk factors such as renal failure or delay to surgical treatment (10,15,23). Whereas predisposing factors play an important role in risk stratification, it is important to note that their absence does not exclude FG. In one population-based study, up to 26% of patients had no comorbidities, and 23% were younger than 40 years (10).

Pathophysiology

Anatomy. Understanding the fascial anatomy of the perineum allows a better understanding of how NSTIs that originate in the urogenital and perianal region (e.g., FG) spread to involve the abdomen, flank, and chest. As FG spreads across the superficial and deep fascial planes of the urogenital and perianal regions, this deep tissue infection leads to local vascular occlusion, ischemia, and tissue necrosis (24). This hypoxia subsequently leads to infarction of surrounding nerves that initially is painful, but eventually causes localized anesthesia (22). The superficial skin is often spared during the initial stages of infection, whereas the necrotizing process spreads along the fascial planes at a rate reaching 2–3 cm/h, masking the true extent of the disease (25).

The infection in FG tends to spread along the fascial planes, with initial involvement of the superficial (Colles fascia) and deep fascial planes of the genitalia (26). FG

Table 1. Risk Factors for Fournier Gangrene (10,13–17)

Risk Factor	Frequency
Diabetes mellitus	20–70%
Alcohol misuse disorder	25–50%
Obesity	10–60%
Immunocompromised state	30–40%
Hypertension	Up to 25%
Peripheral vascular disease	Up to 25%
HIV infection	Up to 16%
Tobacco use	Up to 15%
Chronic renal failure	Up to 15%
Hematologic disorders	Up to 14%
Congestive heart failure	Up to 11%
Chronic liver disease	Up to 11%
Malignancy	Up to 8%
Recent surgery	Up to 5%
No risk factors	Up to 10%

HIV = human immunodeficiency virus.

preferentially spreads to the overlying perineum and subcutaneous tissue, sparing the deeper muscle layers, which are protected by the fascial planes themselves (27). For instance, the Colles fascia attaches to the pubic ramus, as well as the sphincter urethrae and deep transverse perineal muscles, thereby limiting bacterial spread outside of these areas (24). It is important to note, however, that Colles fascia remains continuous with other surrounding fascial planes, facilitating rapid spread toward the abdomen and thorax (via Scarpa's fascia), as well as the scrotum (via Buck's and Dartos fascia). Deeper infection extending below the fascial plane involving myonecrosis is not classically thought to be a feature of FG, although it has been described (28).

In addition to fascial spread, vascular supply plays an important role in local involvement. For instance, the external and internal pudendal arteries arise from the retroperitoneum and supply the testicles, which accounts for the limited testicular involvement seen in FG (2,29). Conversely, the inferior epigastric and circumflex iliac arteries must pierce Camper's fascia to supply the abdominal wall, and are therefore at increased risk of thrombosis (27,30). Thus, involvement of the testicles suggests a retroperitoneal origin or spread of infection, and involvement of the abdominal wall implies involvement of Camper's fascia (3,31). Although thrombosis of the corpus spongiosum and cavernosum have been reported, corpora and urethral involvement are rare, as both lie deep to Buck's fascia (2,32).

Etiology. Historically, FG was thought of as an idiopathic entity, though recent literature suggests that less than a quarter of contemporary FG cases are now considered idiopathic (33,34). The most common sources arise from the gastrointestinal tract (30–50%), genitourinary tract (20–40%), and cutaneous injuries (20%) (3). Local trauma is frequently associated with the underlying source of infection (3). Colorectal sources are varied, but include local infection, surgical site infections, abscesses (particularly in the perianal, perirectal, and ischiorectal regions), anal fissures, colonic perforations, diverticulitis, hemorrhoidectomy, and rectal carcinoma (13,27,35,36). Reported genitourinary sources may include neurogenic bladder, hydrocele aspiration, vasectomies, prostatic biopsies, urethral calculi, intracavernosal injections, instrumentation, acute epididymitis, urethral stenosis/stricture, penile implants, genital piercings, indwelling catheters, malignancies, chronic prostatitis, and urinary tract infections (24,37,38). In women, further sources include Bartholin gland or vulvar abscess, episiotomy, hysterectomy, and septic abortion (4). Cutaneous sources are generally limited to furuncles, pressure ulcers, genital piercings, and superficial soft tissue infections (15,16,39). Special attention must be paid to insect bites, burns, and circumcision as sources of pediatric FG (27,40).

Microbiology. The predisposing and etiologic factors of FG provide a favorable environment for necrotizing infections by impairing host immunity and providing a route of inoculation for the microorganisms into the perineum. Characteristic of many NSTIs, FG severity is derived from the synergism of multiple bacteria that are not highly aggressive when encountered alone, with up to 80% of cases being polymicrobial in nature (41). Typically, upwards of four organisms are cultured from each FG patient (24,42). The organisms most commonly found in FG are species that normally exist below the pelvic diaphragm in the perineum and genitalia (3). These include aerobic microorganisms (e.g., *Escherichia coli, Klebsiella pneumoniae,* and *Staphylococcus aureus*) as well as anaerobic microorganisms (e.g., *Bacteroides fragilis, Clostridium* species) (41,43,44). Other species isolated include *Vibrio, Streptococcus, Enterococcus, Pseudomonas*, *Proteus, Klebsiella pneumonia,* and *Corynebacterium* (2). More recently, resistant strains of bacteria, including methicillin-resistant *Staphylococcus aureus*, as well as extended-spectrum beta lactamase resistant *E. coli* have been reported (45). Likewise, fungal sources such as *Candida albicans* and *Zygomycetes* have been reported (46–50). Within the subset of patients with monomicrobial FG, Group A streptococcus is the most commonly isolated organism (39).

Whereas FG is primarily a mixed infection consisting of both anaerobic and aerobic bacteria, the presence of their associated exotoxins and enzymes play a crucial role (27). For instance, heparinase produced by aerobic bacteria encourages platelet aggregation and complement fixation, which leads to coagulation and subsequent thrombosis of surrounding vessels (27,51). The

resultant tissue hypoxia facilitates growth of facultative anaerobes and microaerophilic organisms (27). These species produce enzymes leading to digestion of fascial barriers, thus fueling the rapid extension of the infection. Furthermore, organisms like *Bacteroides* inhibit the phagocytosis of aerobic bacteria, aiding in further spread of the infection (4,27). As FG lies within the larger umbrella of NSTIs, it is important to understand the classification scheme for NSTIs that consist of four microbiological subtypes (Table 2) (1,22,52).

Considerations in History and Physical Examination

FG is a clinical diagnosis based on the presence of fluctuance, crepitus, exquisite tenderness, and wounds of the genitalia and perineum (27). Although the diagnosis is straightforward in the classic presentation, failure to examine the perineal area, especially in the older or obtunded patient, can result in misdiagnosis. Furthermore, the early symptoms of FG and NSTIs are not characteristic; hence, it is often misdiagnosed as cellulitis or abscess up to three quarters of the time (53,54).

The clinical presentation of FG varies widely depending on the extent of infection as well as patient comorbidities. Typically, the infection begins as a localized cellulitis adjacent to the portal of entry, commonly in the perineum or perianal region, with an insidious presentation. Like many NSTIs, the early presenting features are often nonspecific and common to other infectious etiologies. In one study of NSTIs, the most common initial chief complaints were swelling (80.8%), pain (79%), and erythema (70.7%) (53). Bullae (26%), overlying skin necrosis (24%), and crepitus (20%) were less common upon initial examination, but associated with later stages of necrotizing fasciitis (53). Subcutaneous gas and crepitus are highly specific for clostridial infections (1,55). Fever and tachycardia are present in 40% and 61% of these patients, respectively (15,53,56). The affected area may also appear swollen, dusky, or present with a characteristic purulent "dishwater" discharge with associated feculent odor, attributable to the presence of anaerobes (57,58). The presence of hypotension and septic shock is a late and ominous sign, occurring in roughly 21% of patients with NSTIs, associated with high specificity (93.3%) (53,59). This has a strong correlation with mortality, and along with associated multiorgan failure, is the principal cause of death in patients with NSTIs (27,60).

The presenting tenderness, erythema, and swelling may mimic less severe infections, including erysipelas and cellulitis. However, a key feature of FG is pain out of proportion to physical examination, which should alert the clinician to the possibility of FG. Furthermore, cellulitis and erysipelas often present with well-demarcated areas of inflammation and erythema. In contrast, FG may present with areas of poorly demarcated erythema, as well as blisters and bullae during the later stages of infection (2). Although cellulitis and erysipelas may present with symptoms of generalized infection, including malaise and fever, FG can result in severe systemic toxicity with associated multiorgan failure. Furthermore, as FG may spread rapidly along fascial planes, areas of tenderness and erythema may extend as far cephalad as the clavicle (61). The extent of necrosis is an important prognostic factor, as some studies have shown that patients with a necrotic area < 3% of total body surface area rarely die, whereas patients presenting with involvement of 5% total body surface area or more have a worse prognosis, although this is not universally accepted (19,31,62,63).

Although FG is thought of as an acute process, patients may present in a subacute manner occurring over days to weeks (2). In one study, the mean interval between initial symptoms and arrival at the hospital was 5.1 $\pm$ 3.1 days (58). The early clinical course of the subacute form of FG mirrors that of the initial condition leading to the infection. The patient often presents with local cellulitis, folliculitis, or abscess with surrounding erythema

Table 2. Necrotizing Fasciitis Types

Type	Incidence	Etiology	Organisms
I	70–80%	Immunocompromised Abdominal surgery Perianal or genitourinary process	Polymicrobial Mixed anaerobes, aerobes, *Pseudomonas*, & *Bacteroides*
II	20–30%	Skin or throat derived After trauma or direct inoculation	Monomicrobial Most commonly from group A β-hemolytic *Streptococcus*
III	Rare	Marine related organisms Seafood ingestion Contaminated wounds	*Vibrio vulnificus* *Aeromonas* *Clostridium*
IV	Rare	Trauma or burns Immunocompromised	Fungal species *Cryptococcus* *Candida* *Zygomycetes*

(22,64). The patient usually feels pain out of proportion to the clinical examination, a strong diagnostic clue for the presence of FG (2). However, this is not always present, as local anesthesia may develop secondary to local nerve ischemia or due to a preexisting neuropathy (e.g., diabetic neuropathy) (22).

Considerations in Laboratory Investigations

Although no single laboratory test has adequate sensitivity and specificity to discern NSTIs from other soft tissue infections, The Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) score may suggest the presence of NSTI (Table 3); however, it should not be used to exclude the diagnosis (65). The original study examined age, gender, serum potassium, platelet count, C-reactive protein, leukocyte count, hemoglobin, sodium, creatinine, and glucose from 89 consecutive patients with NSTIs, compared with 225 control patients (65). Patients were stratified into low-risk (≤5), intermediate-risk (6-7), and high-risk (≥8) categories, corresponding to a probability of < 50%, 50–75%, and > 75% for the development of a NSTI, respectively. A score ≥ 6 was found to have 92% positive predictive value and a 96% negative predictive value for presence of an NSTI (65). Little has been published regarding the LRINEC score to aid diagnosis of FG, as much of the literature has surrounded the diagnosis of other NSTIs. In one of the few studies examining the LRINEC score's applicability to FG, 16 male spinal cord injury patients with FG were retrospectively assessed (16). The median LRINEC value at admission was 6.5 (with a range of 2–9), with 11 patients scoring ≥ 6, 1 patient scoring ≥ 9, and the remainder scoring < 6 (16,65). Of note, the aim of the aforementioned study was to determine if a LRINEC score ≥ 6 predicted longer time to wound closure and time in the hospital, rather than determining mortality risk or sensitivity for diagnosis.

Table 3. The Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) score (65)

Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC)		
CRP (mg/dL)	< 15	0
	≥ 15	4
WBC (per mm^3)	< 15	0
	15–25	1
	> 25	2
Hemoglobin (g/dL)	> 13.5	0
	11–13.5	1
	< 11	2
Sodium (mEq/L)	≥ 135	0
	< 135	2
Creatinine (mg/dL)	≤ 1.6	0
	> 1.6	2
Glucose (mg/dL)	≤ 180	0
	> 180	1
Composite score	Score < 6	Low risk
	Score 6–7	Intermediate
	≥ 8	High risk

CRP = C-reactive protein; WBC = white blood cell.

Criticisms of the LRINEC scoring system include its retrospective development and poor sensitivity among emergency department (ED) patients (66). The score was not explicitly designed to exclude necrotizing fasciitis in patients with a low-risk score, and subsequent studies externally validating the score have thus far failed to replicate the high sensitivity and negative predictive value reported in the initial paper. The LRINEC score, applied in isolation to ED patients, may miss over 20% of cases of NSTIs, with an associated sensitivity between 68% and 80% (59,66–68). There also have been cases of NSTI with LRINEC scores of 0 (69). As the LRINEC score has limited sensitivity, it should not be used as the sole determinant of clinical decision-making for the diagnosis of FG.

Although the common laboratory findings in FG are nonspecific, they may aid in risk stratification for mortality and morbidity (27). The laboratory values most often portending a worse prognosis include elevated leukocyte counts, creatinine, creatine kinase, urea, lactate dehydrogenase, and alkaline phosphatase, as well as decreased levels of hematocrit, bicarbonate, sodium, potassium, calcium, total protein, and albumin (19,31,70–74). There are multiple scoring systems to predict severity of illness, including the Fournier Gangrene Severity Index, which may be useful to predict mortality, but it has not been shown to aid in the diagnosis of FG (75–78). The Fournier Gangrene Severity Index requires further study before routine use can be recommended (75–78).

Considerations in Imaging

The diagnosis of FG is primarily clinical, and in many cases, imaging is not necessary, nor is it desirable if it leads to a delay in surgical management (27). Imaging is a useful adjunct in those cases in which the presentation is atypical, or when there is concern regarding the true extent of the disease.

Conventional radiography can be used to detect the presence of soft tissue swelling as well as gas in the perineal fascial planes before crepitus is noted on physical examination (27). Subcutaneous emphysema may extend from the scrotum and perineum to the inguinal regions, anterior abdominal wall, and thighs. Evidence of gas formation is present in nearly half of all patients with FG and is highly specific (94%) (22,59,79). However, the absence of gas formation on imaging should not exclude the diagnosis due to poor sensitivity (49%) (22,59,79). Another weakness of conventional radiography in the diagnosis and evaluation of FG is the lack of detection

of deep fascial gas, which may lead to a falsely negative study (80).

As imaging evaluation in patients with FG may be limited by several factors, including presence of concurrent acute renal failure or patient hemodynamic instability, making transport to the imaging department unsafe, point-of-care ultrasound (POCUS) has emerged as a useful bedside tool. POCUS allows for the evaluation of soft tissue inflammation, collections/abscesses, and subcutaneous gas (81,82). Characteristic findings include thickened perineal tissue caused by inflammation and edema, as well as a "cobblestone" appearance throughout the subcutaneous tissue. Acoustic shadowing of subcutaneous gas secondary to bacteria may result in a "snow globe" or "dirty shadowing" appearance caused by hyperechoic foci demonstrating reverberation artifacts (Figure 1) (83). Although the testicular blood supply is usually preserved in patients with FG due to their retroperitoneal blood supply from the aorta, the testicles may be negatively affected with a retroperitoneal or abdominal source of infection. As such, Doppler ultrasound plays an important role in evaluating testicular viability in these patients (2,81). Although POCUS has been shown to be highly specific for NSTIs (up to 93%), it has insufficient sensitivity to exclude a diagnosis with such a high morbidity and mortality (84,85). POCUS has also been reported to accurately diagnose an NSTI in a patient with negative computed tomography (CT) and magnetic resonance imaging (MRI) studies (86).

CT imaging plays an important role in the diagnosis of FG and to evaluate the extent of the disease to further guide appropriate surgical treatment. Although there remains a paucity of data regarding the benefit of intravenous contrast, it may further characterize the soft tissue and should be used if possible (87,88). Characteristic findings include asymmetric fascial thickening, fluid collections, abscess formation, fat stranding around involved structures, and evidence of subcutaneous emphysema (83,89,90). CT may uncover the underlying etiology of FG, such as perineal abscess, fistula formation, or any infectious process in the intra-abdominal or retroperitoneal spaces (83,89). It assists in differentiating FG from less severe infections and allows evaluation of both the superficial and deep fascial planes (27). CT has a sensitivity approaching 90% for diagnosis of NSTIs, in addition to high specificity (93.3%) (59,87–90).

MRI with gadolinium contrast is an excellent imaging modality to characterize soft tissues (91). However, in the ED setting, it is of limited value due to its high cost, extended time of examination, and the fact that it requires a clinically stable patient (2). Characteristic findings include involvement of the deep intermuscular fascia, fascial thickening, and partial or complete absence on postgadolinium images of signal enhancement of the thickened fascial planes (92). Some authors contend that MRI is more helpful in surgical planning compared with CT, as it is more useful for specifying the true extent of infection (93,94). Although it has been shown to have a high sensitivity (100%) and specificity (86%) to diagnose NSTIs, MRI has a limited role for evaluation of FG in the acute care setting (Table 4) (2,92).

Considerations in Management

The cornerstones of treatment of FG include emergent surgical debridement of all necrotic tissue, broad-spectrum antibiotics, and hemodynamic resuscitation with intravenous fluids as well as vasoactive medications as needed (3,26,27). As the rate of fascial necrosis has

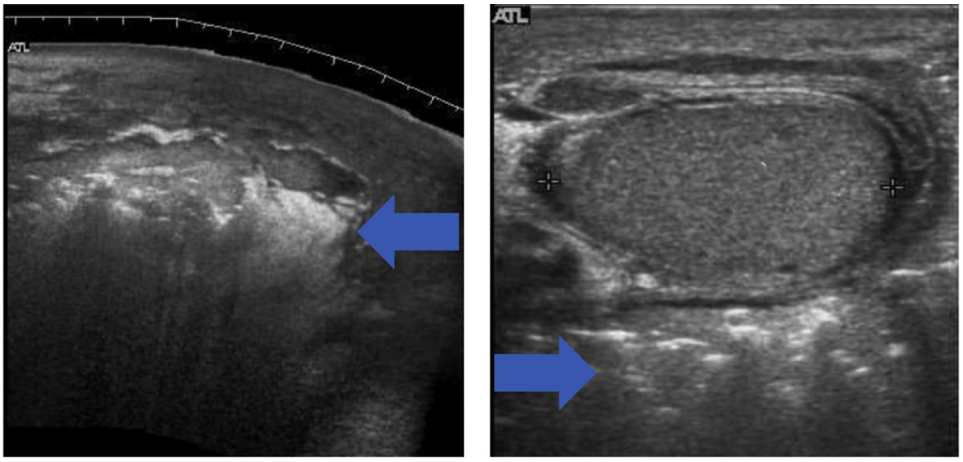

Figure 1. Scrotal ultrasonography of Fournier gangrene demonstrating echogenic debris and shadowing (blue arrows), indicative of air and infectious material. From https://commons.wikimedia.org/wiki/File:Scrotal_ultrasonography_of_Fournier_gangrene.jpg.

Table 4. Imaging Modalities in Fournier Gangrene

Imaging Modality	Test Characteristics	Findings in FG
Radiograph	Sensitivity 49% Specificity 94%	- Hyperlucencies representing subcutaneous emphysema - Soft tissue swelling
POCUS	Sensitivity 88% Specificity 93%	- Thickened, edematous soft tissue with "cobblestoning" - Reactive unilateral or bilateral hydroceles - Acoustic shadowing of subcutaneous gas resulting in a "snow globe" or "dirty shadowing" appearance
CT	Sensitivity 88.5% Specificity 93.3%	- Asymmetric fascial thickening - Underlying etiology (e.g., abscess, fistula) - Fluid collections with possible air/fluid levels - Subcutaneous emphysema - Fat stranding
MRI	Sensitivity 100% Specificity 86%	- Asymmetric fascial thickening - Partial/complete absence of signal enhancement of the thickened fascial planes on postgadolinium images - Fluid collections with possible air/fluid levels - Subcutaneous emphysema - Fat stranding

FG = Fournier gangrene; POCUS = point-of-care ultrasound; CT = computed tomography; MRI = magnetic resonance imaging.

been noted as high as 2–3 cm per hour, FG is considered a surgical emergency necessitating early involvement of the appropriate surgical teams, which decreases mortality (6,25,95). However, as up to 21% of patients present with symptoms of hypotension and septic shock, hemodynamic resuscitation and patient optimization prior to surgical intervention are important aspects of acute management (53,59).

Broad-spectrum antibiotic coverage. Broad-spectrum parenteral antibiotic therapy is initiated empirically upon diagnosis of FG and then subsequently tailored based on culture results (27). The initial antibiotic regimen must have a broad range of activity against commonly implicated organisms, most notably staphylococcal and streptococcal species, as well as coliforms, Gram-negative bacteria, *Clostridium*, *Bacteroides*, and *Pseudomonas* (Table 5) (15). Empiric antibiotics must cover for methicillin-resistant *Staphylococcus aureus*, typically with linezolid or vancomycin, which is combined with a carbapenem or beta-lactam-beta-lactamase inhibitor (96). Clindamycin should be added, as it can suppress toxin production and modulate cytokine production, as well as decrease mortality from NSTIs (15,97). In those patients with severe penicillin hypersensitivity, clindamycin or metronidazole combined with an aminoglycoside or fluoroquinolone should be administered (96). Additionally, many have suggested adding penicillin for treatment of streptococci and, in particular, when *Clostridium* species are suspected (96).

Table 5. Empiric Antibiotic Regimen for Patients with Fournier Gangrene

Empiric Antibiotic Regimen for Fournier Gangrene
Carbapenem (Imipenem, meropenem, or ertapenem)
OR
Beta lactam-beta lactamase inhibitor (piperacillin-tazobactam, ampicillin-sulbactam, or ticarcillin-clavulanate) for activity against Gram-negative bacilli and anaerobes
PLUS
Clindamycin (600–900 mg i.v. q8h in adults) for activity against Gram-positive organisms and anaerobes, as well as its antitoxin effects
PLUS
Vancomycin, daptomycin, or linezolid for activity against Gram-positive organisms and MRSA
In patients with severe hypersensitivity to carbapenems or beta lactam-beta lactamase inhibitors, consider substituting:
Aminoglycoside
OR
Fluoroquinolone
PLUS
Metronidazole
In patients with salt or freshwater exposure and significant risk for *Vibrio vulnificus* or *Aeromonas hydrophila* involvement, consider adding:
Doxycycline
In patients with significant risk for fungal involvement, consider adding:
Amphotericin B or fluoroconazoles

MRSA = methicillin-resistant *Staphylococcus aureus*.

Doxycycline should be considered for those patients with significant risk for *Vibrio vulnificus* and *Aeromonas hydrophila* involvement, including exposure to marine exposure and exposure to seafood (96). Special consideration should be given to initiating early antifungal therapy with amphotericin B or fluoroconazoles in those patients with a history of fungal infections and immunocompromised patients, as fungal sources are an emerging cause of NSTIs (98,99). Although high-dose intravenous immunoglobulin has recently been described as a potential option for neutralizing streptococcal toxins, this has yet to be demonstrated in randomized studies, and should be considered based on consultation with the primary surgical team (100–102). Therapeutic plasma exchange has been proposed as a method to improve organ function and mortality by removing inflammatory mediators (103). However, based on current evidence, therapeutic plasma exchange is not recommended as an adjuvant therapy in NSTIs (103). Surgical treatment is key, as antibiotic delivery to fascial tissue is imperfect due to poor vascularization and degradation as a consequence of infection, making it a substandard solitary treatment option.

Hemodynamic resuscitation and patient optimization. Hemodynamic resuscitation and optimization of the patient's comorbidities play an integral role in ED management of FG. Patients with FG may present hypotensive or in septic shock characterized by hypoperfusion, which can result in organ dysfunction. As a result, aggressive fluid resuscitation and hemodynamic support are often required, as evidence of end-organ dysfunction is associated with increased mortality (104). The Surviving Sepsis Campaign guidelines recommend resuscitation with intravenous crystalloid fluid within the first 3 h for patients with sepsis or septic shock and evidence of hypoperfusion (105). These fluids should preferably be a balanced crystalloid such as lactated Ringer solution or PLASMA-LYTE A (Baxter Healthcare Corporation, Deerfield, IL) (106). Fluid resuscitation should be guided by hemodynamic parameters (heart rate, blood pressure, urine output, and lactate clearance), and patients should be frequently monitored using dynamic indices (passive leg raise) to gauge response to fluid treatment (105,107,108). Vasoactive medications may be used to support end-organ perfusion in patients with continued hypoperfusion after fluid administration, with a preference toward early initiation of norepinephrine (105,109,110).

As many patients with FG have underlying comorbidities that may exacerbate FG, most commonly diabetes mellitus, it is important to treat these comorbidities. Although few data exist concerning optimizing patient comorbidities in those with FG, a treatment strategy may be extrapolated from current critical care literature. Insulin therapy for glycemic control for a target blood glucose level of 140 to 200 mg/dL has been suggested in critically ill patients, which is reasonable for patients presenting with hyperglycemia (111). These patients may also present with an episode of diabetic ketoacidosis secondary to FG and should be managed in accordance with current guidelines (112). Up to 50% of patients with FG have an underlying alcohol use disorder and may be suffering from concomitant alcohol withdrawal or delirium tremens. These patients should be treated with adequate supportive care, as well as benzodiazepines administered in a symptom-triggered fashion, guided by the Clinical Institute Withdrawal Assessment of Alcohol scale, revised (113).

Surgical debridement. Prompt surgical consultation is recommended for all patients in whom FG is suspected and should not be delayed by laboratory or imaging investigations (Figure 2) (27,96). Depending on local practice patterns, as well as individual patient characteristics, this may involve physicians from the general surgery, urology, or obstetrics and gynecology services, or any combination thereof. The most important variable affecting mortality in patients with NSTIs is time to admission and debridement (95). One study suggests that survival decreases from 93.2% to 75.2% with a delay in debridement from 24 to 48 h (53). Similarly, in another study, the average time from admission to operation was 90 h in nonsurvivors vs. 25 h in survivors, making early surgical intervention imperative (5).

Hyperbaric oxygen therapy (HBOT). HBOT is used as an adjunctive therapy for the optimization of infected tissue oxygenation and for its bactericidal and bacteriostatic effects, especially in the postsurgical period (114). However, the lack of randomized controlled studies limits the use of HBOT to patients unresponsive to conventional surgical and intensive care management (9,115). As such, HBOT is not routinely recommended prior to surgical debridement, and consultation with a hyperbaric specialist is typically not a consideration in the acute care setting.

Disposition. Early surgical consultation and involvement of the critical care team facilitate timely management and coordinated handoff to ensure the best patient care possible, as prolonged boarding in the ED has been associated with increased risk of mortality (54). Level of care is dependent on the patient's underlying pathophysiology, hemodynamic status, need for invasive monitoring, and underlying comorbidities, but typically, these patients will require admission to the intensive care unit. Additional considerations for under-resourced hospitals include transfer to a higher level of care, as up to 66%

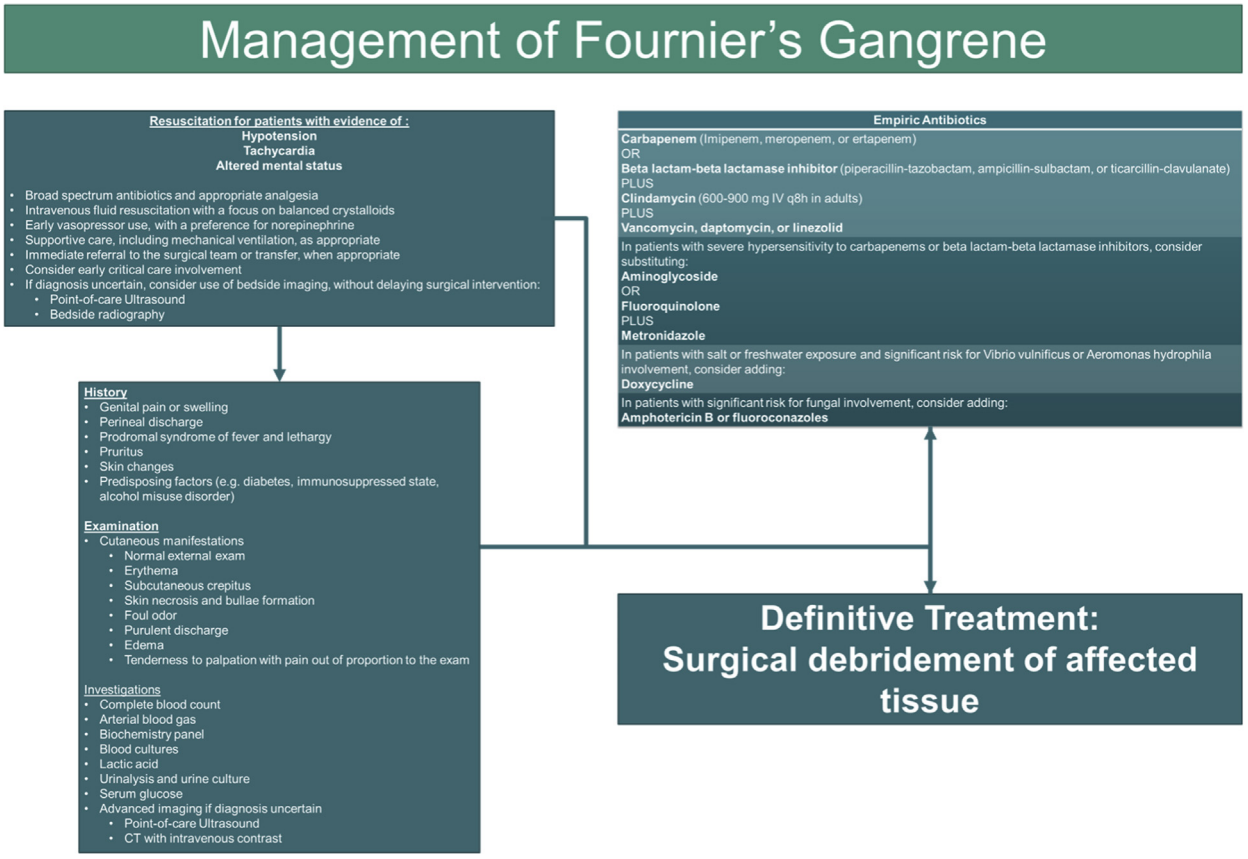

Figure 2. Management algorithm for patients with Fournier gangrene. CT = computed tomography.

of hospitals in the United States do not encounter cases of FG, and adequate surgical capabilities may not be present (10). However, the decision to transfer should be made in consultation with the receiving facility's surgical team, as well as the transferring facility's surgeon, as interhospital transfer has been associated with increased mortality, although this remains controversial (116,117).

CONCLUSIONS

FG is a rare, life-threatening necrotizing infection that requires early diagnosis to reduce morbidity and mortality. A high clinical level of suspicion, combined with knowledge of anatomy, risk factors, and etiology are necessary for an accurate diagnosis and management. Although FG remains a clinical diagnosis based on history and physical examination findings, relevant laboratory and radiography investigations can serve as useful adjuncts. The cornerstones of treatment of FG include emergent surgical consultation for debridement of necrotic tissue, broad-spectrum antibiotics, and hemodynamic resuscitation with intravenous fluids and vasoactive medications as needed.

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ARTICLE SUMMARY

1. Why is this topic important?

Fournier gangrene (FG) is a life-threatening disease requiring emergent diagnosis and treatment.

2. What does this review attempt to show?

This narrative review provides an evidence-based summary of the current literature concerning the evaluation and management of FG.

3. What are the key findings?

FG is a necrotizing soft tissue infection associated with several risk factors including male gender, diabetes, immunosuppression, and alcohol use, though it may be present with no risk factors. FG typically begins in the genitourinary tract, gastrointestinal tract, or perineum with a mixed infection of aerobic and anaerobic bacterial species. The infection develops rapidly and progresses to gangrene with multiple organ failure and death. Findings may be subtle early in infection. Imaging can include ultrasound, radiography, or computed tomography, but no imaging test can exclude the diagnosis. Treatment includes fluid resuscitation, antibiotics, and surgical debridement.

4. How is patient care impacted?

Emergency clinicians play an integral role in the evaluation and management of FG, and knowledge of this condition is essential for optimal care of these patients.