

Man, 32, With Severe Scrotal Pain and Swelling

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A 32-year-old man presents to the urgent care center at a community hospital with severe scrotal pain and swelling of five days' duration. What began as mild left scrotal discomfort is now causing increasing pain, swelling, hematuria, dysuria, low-grade fever, and nausea, prompting him to seek medical attention.

The patient, who is a pipefitter in a hospital, was at work when his symptoms began. He denies any history of scrotal trauma, and his review of systems is otherwise unremarkable. His medical history is significant for mild hypertension and morbid obesity, but he is not immunocompromised. Two months ago, he had an excision and repair of a left ureterocele, for which he was treated prophylactically with ciprofloxacin for one week. He has a 3-pack-year history of smoking and consumes three alcoholic beverages per week. He denies illicit drug use and has no report of sexually transmitted infection.

Upon arrival to urgent care, the patient appears to be in moderate distress, with a blood pressure (BP) of 111/79 mm Hg; pulse, 104 beats/min; respiratory rate, 18 breaths/min⁻¹; temperature, 100.1°F; and SpO₂, 94%. Physical exam reveals left scrotal erythema, severe tenderness upon palpation, marked scrotal edema, and a slight amount of foul-smelling discharge seeping from a pinpoint opening in the left perineum (see Figure 1a). Given his scrotal presentation, he is quickly transferred to a regional emergency department (ED) for a urology consult.

In the ED, lab testing yields significant findings (see Table 1, page 36). His ECG demonstrates sinus tachycardia at 126 beats/min without rhythm or ST changes. His urinalysis reveals a cloudy appearance,

a protein level of 100 mg/dL, and trace leukocyte esterase.

Urgent CT with contrast is obtained; it shows significant soft-tissue inflammatory changes in the left groin and scrotum that extend into the left thigh. In addition, a collection of fluid is seen in the inferior aspect of the left scrotal wall, indicating a probable abscess. There is no free air or lymphadenopathy.

Given the patient's worsening condition and his apparent advancement to a systemic inflammatory response syndrome, surgical consult is obtained. He is diagnosed with a scrotal abscess and cellulitis; two blood and two scrotal cultures are obtained, and the patient is empirically started on IV ampicillin and gentamicin.

Two hours later, he has a BP of 122/74 mm Hg; pulse, 112 beats/min; respiratory rate, 20 breaths/min⁻¹; and temperature, 103.1°F. His genital inflammation has advanced to the perineum and the left lower abdomen. The purulent, bloody, foul-smelling drainage from the opening in the left perineum is increasingly apparent. The patient is taken emergently to surgery for an incision and drainage, along with exploration of the scrotal abscess. During surgery, the patient is discovered to have Fournier's gangrene.

DISCUSSION

Fournier's gangrene (FG) is a necrotizing fasciitis of the perineal, perianal, and/or genital areas involving the superficial and deep fascial planes while sparing the deep muscular structures and overlying skin.¹ A rare but potentially fatal disease, FG spreads at a rate of up to 3 cm/h.^{2,3}

Mortality rates range from 7.5% to 88%,

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with the highest mortality occurring within the first 96 hours of hospitalization.^{1,4-7} Mortality is often related to the onset of sepsis.^{4,5} Survival requires early recognition; immediate, aggressive surgical debridement of all necrotic tissue; and concomitant, early administration of appropriate antibiotics.^{1,4,5,8} Mortality risk and prognosis are improved in patients younger than 60 with localized disease and no toxicity, along with sterile blood cultures.¹

Risk Factors

FG is most commonly seen in males between the ages of 50 and 70, with a 10:1 male-to-female ratio.^{3,9} Impaired immunity typically increases a patient's susceptibility to FG, with type 2 diabetes having the highest incidence (85% of patients).^{1,4,6,8,10} Other conditions that can increase the risk for FG include obesity, alcoholism, cirrhosis, cardiac disease, tobacco use, peripheral vascular disease, malignancy, chronic steroid use, renal insufficiency, IV drug abuse, and HIV.^{1,4,6,8,9,11}

Trauma frequently initiates the infectious process, with urogenital trauma (eg, placement of urethral instrumentation, surgery, and urinary tract infection) being the main cause of bacterial introduction.^{1,3} Localized infection causes the development of an obliterative endarteritis, resulting in subcutaneous vascular ischemia, necrosis, and bacterial proliferation.^{3,7,9}

Presentation and Diagnosis

Presenting symptoms of FG include intense, abrupt genital pain that is disproportionate to the physical exam findings.⁹ This rapidly escalates to include extreme swelling, erythema, bullae, discolored skin, and tissue crepitus with eventual necrosis.^{2,10} Lab results typically show leukocytosis $> 18.0 \times 10^9/L$.⁴ The testicle and spermatic cord are generally unaffected (as in this patient), due to the anatomic relationship between the

FIGURE
Presentation and Urgent Management of Case Patient



Physical exam findings included left scrotal erythema, severe tenderness upon palpation, and marked scrotal edema (a). CT with contrast revealed significant soft-tissue inflammatory changes in the left groin and scrotum that extended into the left thigh. The patient's condition ultimately necessitated extensive debridement down to healthy tissue in all affected areas (b).

various layers of fascia within the scrotum and the anterior abdominal wall, as well as the independent blood supply of the compartmentalized testicular tissue.¹⁻³

During an exam of the acute scrotum, the differential diagnosis includes cellulitis, scrotal abscess, acute epididymitis, and testicular torsion, with scrotal abscess being most frequently diagnosed (57% of patients).^{9,11,12} The distinguishing features of these diagnoses can be found in Table 2 (page 37). Necrotizing fasciitis in the form of FG tends to be an unexpected, rare finding usually only diagnosed during the surgical draining of an abscess.¹²

CT is the test of choice to detect FG and determine the extent of its spread by identifying subcutaneous air/gas within the involved fascial planes.^{10,13} However, an incisional biopsy with culture is needed to confirm the diagnosis.^{3,9} Most patients with FG require an average of four surgeries (eg, reconstruction, skin grafting, and possibly

TABLE 1
Patient Lab Values

Lab Test	Reference range	Initial ED results	Postoperative results
WBC count (x 10 ⁹ /L)	4.0-11.0	11.3	9.0 with 13% bands
Hematocrit (%)	40-52	37	26
Platelets (x 1,000 μ L)	150-450	126	106
Lactate (mmol/L)	0.4-2.0	1.5	Not tested
Troponin (ng/mL)	< 0.01	2.29	16.23

Abbreviation: WBC, white blood cell.

colostomy if the infection has entered the peritoneal cavity) in order to eradicate the disease and achieve the best functional and cosmetic outcome.⁴

Etiology

About 83% of FG cases are polymicrobial infections comprised of enterobacter, enterococci, *Escherichia coli*, group A streptococci, pseudomonas, and clostridium, with symptoms evolving two to four days following the initial insult.^{4,7,11,14,15} Monomicrobial infections are much less common, but the symptoms progress even more rapidly.¹⁵ Methicillin-resistant *Staphylococcus aureus* (MRSA) necrotizing fasciitis infections occur in about 3% of monomicrobial cases.¹² MRSA emerged in the early 2000s as an additional causative pathogen for polymicrobial necrotizing fasciitis infections.^{12,14,15} Prior to that time, *S aureus* strains were almost uniformly susceptible to penicillinase-resistant β lactams.¹²

A distinction should be made between health care-associated (HA) MRSA and community-acquired (CA) MRSA due to treatment considerations. HA-MRSA infections are contracted through previous health care exposure (within the past year) and are less resistant to treatment.^{16,17} In contrast, CA-MRSA, which comprises 29% of MRSA cases, causes infections in previously healthy young patients without prior health care contact within the past year.¹⁶ CA-MRSA strains

are more robust than HA-MRSA strains and can cause sepsis and other invasive, rapidly progressive, and possibly life-threatening infections due to the amount of tissue destruction and necrosis.^{16,18} Transmission of CA-MRSA is often associated with crowded environments, frequent skin-to-skin contact, compromised skin integrity, contaminated items or surfaces, and lack of cleanliness.¹⁶ Over the years, CA-MRSA has developed resistance to multiple antimicrobials; providers should therefore consider CA-MRSA on initial evaluation of necrotizing infections, to ensure appropriate initiation of treatment.^{12,16}

CASE CONTINUED

Extensive debridement was completed down to healthy tissue in all affected areas (see Figure 1b, page 35). The necrotizing fasciitis had spared the left testicle and spermatic cord, and a colostomy was not required.

The patient's initial postoperative vital signs were unremarkable, except for his BP (86/54 mm Hg). The patient was taken postoperatively to the surgical intensive care unit (SICU) with the diagnosis of FG. Aggressive IV fluids were administered for resuscitation, and he was closely monitored for increasing sepsis. Metronidazole was added for anaerobic and gram-positive coverage. His postoperative lab results can also be found in Table 1.

His ECG showed a normal sinus rhythm without ST changes, and he denied any cardiac symptoms. His physical exam was significant for mild pallor, dry mucus membranes, and a left scrotal and pelvic packed dressing. He was given two units of packed red blood cells for acute postoperative blood-loss anemia. The preliminary tissue culture results showed gram-positive cocci consistent with a staphylococcal infection; his antibiotics were then changed to IV ampicillin/sulbactam and clindamycin.

Approximately five hours postoperatively, an ECG suddenly showed acute ST elevation in leads II, III, and aVF, with reciprocal changes. The patient was diagnosed with an acute myocardial infarction (AMI). He denied any chest pain, short-

TABLE 2
Differential Diagnoses

Diagnosis	Description	Clinical features	Risk factors
Acute epididymitis	Infection from retrograde spread of organism (chlamydia or gonorrhoeae) via the vas deferens; gradual onset of pain and swelling	Marked scrotal swelling and pain, urethral discharge, irritative symptoms; heaviness; pain into spermatic cord and flank; + Prehn sign; +/-fever, chills	Sexually active men younger than 35, heavy physical strain
Cellulitis	Non-necrotizing infection, inflammation of skin and SQ tissue; expands over 6-36 h	Local erythema, pain, warmth, swelling; no fluctuance; usually lower extremity; +/- fever, chills, malaise	History of venous insufficiency, IV drug use, open wounds, diabetes, trauma, obesity
Fournier's gangrene	Necrotizing fasciitis of perineal, perianal, genital areas; can spread up to 3 cm/h; high mortality	Sudden, intense pain out of proportion to physical findings; severe swelling, erythema, bullae, tissue crepitus, eventual necrosis	Males ages 50-70; impaired immunity, especially diabetes; urogenital trauma
Scrotal abscess	Collection of pus in dermis and deeper tissues; often at hair follicle; grows over 4-14 days; +/- multiple in one area	Local swelling, pain, warmth, erythema; round or conical nodule; fluctuance; +/- drainage; +/- fever, chills, malaise	History of diabetes, shaving, IV drug use, therapeutic injections, HIV, immunosuppression
Testicular torsion	Testicle rotates, twisting the spermatic cord and reducing blood flow to the scrotum	Sudden, severe pain; high-riding testicle; erythema, swelling; +/- nausea, vomiting; painful urination; absent cremasteric reflex	Prepubertal boys (ages 12-16); direct trauma; history of cryptorchism

Sources: Norton et al. *Am Surg.* 2002¹; Agostini et al. *Indian J Plast Surg.* 2014²; Cabrera et al. 2016³; Cottrill. *Urol Nurs.* 2013¹⁰; Papadakis et al. 2015²⁵; Erye RC.²⁶

ness of breath, or diaphoresis. The SICU team initiated aspirin therapy and immediately contacted cardiology for an emergent coronary angiogram.

The angiogram and cardiac catheterization revealed an elevated left ventricular end diastolic (LVED) volume, inferior wall hypokinesis, a low-normal ejection fraction, and a 30% lesion in the first diagonal of his left anterior descending artery. A postprocedure echocardiogram demonstrated left ventricular (LV) ejection fraction of 50%, with LV hypokinesis in the inferior base and mild left atrial enlargement. The patient was started on metoprolol for myocardial protection and recovery.

Complications

Perioperative complications of FG, including AMI, must be considered due to the

physiologic stress on the body.¹⁹ Most patients with perioperative AMI after noncardiac surgery do not experience ischemic symptoms.²⁰

Growing evidence suggests the pivotal role of acute inflammation (postoperatively or from infection) as a precipitating event in AMI.^{20,21} Chemical mediators, such as inflammatory cytokines, endotoxins, and nitric oxide, may play a role in the development of an AMI.²²

If cardiovascular disease and/or significant cardiovascular risk factors (ie, older age, male, cigarette smoking, cardiac family history, acute kidney injury) are present, the risk for AMI increases in the first two days following surgery.^{21,23} Acute infections and sepsis also initiate or increase systemic inflammatory activity via these same chemical mediators.²¹

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Most suspected infectious agents also produce coronary artery sheer stress and destabilization of vulnerable plaques, leading to plaque rupture and thrombosis.^{19,24} Proinflammatory cytokines promote enhanced platelet activation and contribute to this thrombotic environment.^{21,23} Thrombus leads to obstructed coronary blood flow, myocardial ischemia, and finally, infarction.²¹

A reversible myocardial depression, cardiomyopathy, or myocardial ischemia may occur in patients with acute systemic infection or sepsis when the myocardium is functionally and structurally injured by these inflammatory chemical mediators.^{19,22-24} Characteristics of such a cardiomyopathy include left ventricle dilation with a low filling pressure, an abnormal increase in LVED

➤ **Patients with soft-tissue infections require constant review of symptoms, since early diagnosis with prompt debridement and antibiotic therapy are crucial to survival.**

volume, and a depressed ejection fraction.²²

An acute infectious or septic process can raise troponin levels in 43% to 85% of patients.^{22,24} Troponin biomarkers can assist in predicting myocardial injury and events after surgery with nearly absolute myocardial tissue specificity.²⁰ Cardiovascular involvement caused by myocardial injury-related sepsis is observed in up to 70% of patients in the ICU for these reasons.²³ Therefore, providers should consider measuring troponin biomarkers during such infectious and septic processes, as this team did for the case patient. The providers were able to diagnose his AMI early and institute appropriate treatment measures to avoid extensive myocardial tissue damage.

Several studies have already demonstrated a correlation between pneumococcal pneumonia and an increased risk for AMI, and the same mechanisms are presumed responsible for any severe acute infectious state.²¹ More research is needed to understand the pathophysiology of AMI in sepsis and acute systemic infections.²³

OUTCOME FOR THE CASE PATIENT

On postoperative day 2, the patient's vital signs and lab results were normal. Additional lab results included an AIC of 5.2%. His ECG showed a resolving ST-elevation myocardial infarction (STEMI). The surgical wound had initiation of early granulation tissue without any further signs of necrosis.

A postoperative acute STEMI was unexpected in this patient, as his only risk factors included being male, mild hypertension, obesity, and tobacco use. At the time of his initial elevated troponin level, he had no cardiac symptoms or ECG changes. This initial high troponin level may have been stress-induced from the acute infectious process, and his acute inferior wall STEMI may have been secondary to a transient thrombotic event. The STEMI may then have resolved on its own during the cardiac catheterization with the administration of heparin, IV fluids, blood products, aspirin, or dye infiltration, thus enhancing reperfusion of the coronary artery system.

The final tissue culture showed MRSA. Given his job and his history of a genitourinary procedure, as well as the less fulminant form of disease and relatively quick recovery, it was likely HA-MRSA (rather than CA-MRSA). Only clindamycin was used for treatment.

The wound continued to have decreasing erythema, a reduction in tenderness, and evidence of viable, pink granulation tissue. HIV testing was not completed during his admission. The remainder of the patient's hospital course was unremarkable, and he was discharged home with wound care, urology, and cardiology follow-up services.

CONCLUSION

Multiple factors contribute to a delayed or mistaken diagnosis of FG; it may be overlooked in the initial working diagnoses because of its low incidence and manifestations similar to those of other soft-tissue infections (eg, cellulitis, scrotal abscess). The cutaneous signs of FG often lag behind the disease manifestation, with minimal or no external presence while extensive internal tissue destruction is occurring.

Constant review of symptoms is required when treating patients with soft-tissue infections, and early signs—such as pain out of proportion to physical findings—should prompt a clinician to include FG in the differential.

Early diagnosis with prompt debridement and antibiotic therapy are crucial to patient survival. Detecting FG within the first 24 hours is critical. Further differentiation between CA-MRSA and HA-MRSA can assist in patient recovery and survival by guiding appropriate antibiotic therapy. Perioperative risk assessment and serial troponin biomarkers may identify patients in need of intensive monitoring and management postoperatively to avoid an AMI, since patients may not experience ischemic symptoms. **CR**

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