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Fournier's Gangrene and Thigh Myonecrosis due to *Candida Tropicalis*: Case Report and Literature Review of a Rare Clinical Entity

Mark Shafarenko and Siba Haykal*

University of Toronto, University Health Network, Toronto Ontario, Canada

Corresponding Author*

Siba Haykal

University of Toronto, University Health Network, Toronto Ontario, Canada

E-mail: sibahaykal@gmail.com

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Abstract

Polymicrobial bacterial infections account for a majority of cases of Fournier's gangrene, although fungal etiologies are becoming increasingly common. We report the first case of Fournier's gangrene caused solely by *Candida tropicalis* in a 68-years-old diabetic female. The infection ultimately proved fatal despite maximal medical and surgical therapy. We recommend fungal sources and empiric antifungal therapy is considered in high-risk individuals.

Keywords: Fournier gangrene • *Candida albicans* • *Candida tropicalis* • Fournier's gangrene • Trauma • Necrotizing fasciitis • Myonecros

Introduction

Fournier's gangrene is an uncommon necrotizing infection of the groin and perineal region, most commonly due to mixed aerobic and anaerobic bacterial etiology [1, 2]. Certain comorbidities predispose susceptible individuals to the development of Fournier's gangrene, including immunosuppression, chronic kidney disease, and diabetes. Management involves prompt recognition, institution of broad-spectrum parenteral antibiotics, and surgical debridement [3, 4]. Despite these interventions, mortality remains high and ranges from 7.5% to 50% in some series [3]. While fungal etiologies of these infections are quite rare, they are becoming increasingly reported in the literature. In the setting of compromised host defenses, fungal species may lead to necrotizing soft tissue infections and disseminated disease [2]. *Candida albicans* has been described as the most common fungal agent in widespread disease although other non-*C albicans* species account for roughly 50% of these infections at present [2]. We present a rare case of Fournier's gangrene and myonecrosis of the thigh due to *Candida tropicalis* infection and review the existing literature surrounding management recommendations.

Case Report

A 68-year- old female from an assisted living facility was admitted to the internal medicine service with a fall in the context of subjective weakness, fevers, and a 3-day history of left upper leg pain. She had a history of hypertension, schizophrenia, and poorly controlled type 2 diabetes. At the time of presentation, she was mildly tachycardia with significant leukocytosis (WBC of 20.2) and a fluctuant lesion in the left inguinal fold. She was promptly started on ceftriaxone before broadening to piperacillin-tazobactam in light of worsening systemic symptoms and urine cultures that were positive for *Escherischia coli*. The patient refused any additional imaging to characterize the symptoms in the left upper leg and to rule out any deeper infection. A small blister measuring roughly 4 cm^2 was noted by the team in the left inguinal region although the patient was uncooperative with a full groin and genital examination. Wound swabs of the area showed only scant growth of mixed flora. Despite hemodynamic stability, the patient continued to endorse pain in the left inguinal region which was concomitant with a WBC count that rose to 26.3 on post-admission day 5.

However, she continued to refuse further imaging of the region. On post-admission day 7, the wound was found to extend from the inguinal fold to the left perianal region, with foul-smelling purulent discharge. CT scan demonstrated a rim-enhancing collection measuring 5.7 cmx2.9 cmx9 cm with extensive air locules extending from the left pubic tubercle along the left adductor musculature. Extensive emphysema was also noted in the subcutaneous tissues overlying the gracilis muscle to the level of the knee. Clinical examination revealed a large necrotic ulcer extending from the left inguinal fold posteriorly to the gluteal fold and crepitus on palpation in keeping with Fournier's gangrene. Vancomycin and clindamycin were added to the antibiotic regimen before emergent surgical debridement. The necrotic tissues of the left labia, inguinal region, buttock, and medial thigh were widely debrided. The gracilis was also found to be largely non-viable and was resected to healthy tissue. Multiple wound and tissue samples were sent for culture and gram stain. The wound was packed open with betadine-soaked gauze before transfer to the ward.

On the morning of postoperative day 1, the patient was found to have rapidly rising lactate, acidemia, and tachypnea. She was rapidly transferred to the intensive care unit and intubated. Her WBC count rose to 91 and she had severe lactate acidosis with a pH of 6.94. A bicarbonate infusion and multiple vasopressor agents were started together with the initiation of Sustained Low-Efficiency Dialysis (SLED) for oliguria and recalcitrant acidosis. The clinical deterioration continued despite no change in the appearance of her wounds on serial reassessment and the lack of an intra-abdominal source identified on the CT scan. Gram stains from the previous day failed to reveal any organisms. In consultation with the critical care and infectious disease teams, Intravenous Immune Globulin (IVIG) was started and meropenem was added in place of piperacillin-tazobactam. She was booked for emergent re-exploration of the wounds as an unidentified necrotic tissue burden may have accounted for the lack of improvement (Figure 1).

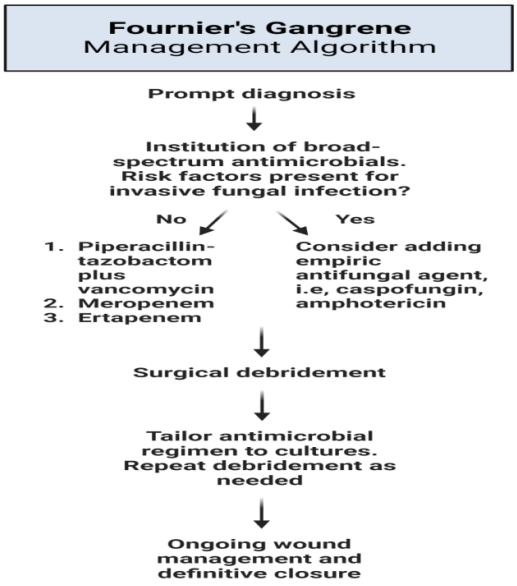

Figure 1. Fournier's gangrene management algorithm.

In the operating room, the distal portion of the gracilis muscle was found to be necrotic and was excised in its entirety. The adductor magus muscle

similarly was non-viable and was resected to healthy tissue. The remainder of the posterior and medial thigh compartments appeared healthy and only minimal further debridement was carried out in the perineum before transfer back to the intensive care unit. Cultures from the first debridement returned positive for *Candida tropicalis* on the second day following the initial debridement. Caspofungin was added to the antimicrobial regimen in consultation with infectious disease. Unfortunately, her overwhelming sepsis and circulatory shock progressed to multi-organ failure involving her liver and kidneys. She passed away later the same day.

Discussion

Fournier's gangrene is a life-threatening infection and often occurs due to the synergistic interplay between aerobic and anaerobic bacteria in susceptible hosts [2–4]. Fungi as the etiologic agent in Fournier's have been infrequently reported, although one recent series reported rates as high as 22% [5]. In almost all documented cases, underlying medical conditions predisposing to invasive fungal infections were found, including diabetes, alcoholism, and immunosuppression [1, 3, 5-7].

Candida albicans account for the majority of Fournier's infections due to fungi, although other non-*C albicans*. *Candida* species have seen increasing emergence as causative agents [3, 6], including *Candida glabrata*. To our knowledge, this is only the fourth report of *Candida tropicalis* as a causative organism in Fournier's gangrene [4, 5], and the only such report in which *C. tropicalis* was identified as the sole agent. In a report by Eisen and Brown, *C. tropicalis* was identified in conjunction with *C. albicans* in a 51-years-old male with a necrotizing infection of the scrotum, abdomen, and thigh following emergency laparotomy after a motor vehicle collision [4]. He recovered well with prompt surgical debridement and treatment with intravenous fluconazole. In a series of 143 patients with Fournier's gangrene reported by Castillejo Becerra et al., *C. tropicalis* was identified in one patient although the outcome and any other infectious organisms isolated were not known. *Candida glabrata* and *C. tropicalis* were both identified in addition to multiple bacterial species in a 59-year-old renal transplant patient who had superficial penile necrosis complicated by Fournier's gangrene. He recovered well with serial debridements and treatment with caspofungin. *Candida tropicalis* is the most prevalent pathogenic *Candida* spp of the non-*albicans* group. Particularly amongst immunocompromised patients, there has been a marked increase in global infection rates due to this pathogen [6]. It has also shown substantial resistance to fluconazole, although this is still under investigation [6, 8].

As noted by multiple groups, *Candida*-induced necrotizing infections can clinically resemble gas gangrene caused by *Clostridium perfringens* [1, 4] with respect to the presence of soft tissue gas and myonecrosis. The clinical presentation of our patient would fit such a description given the subcutaneous emphysema on CT scan and widespread myonecrosis seen intraoperatively. The cause of gas formation has been suggested to be glucose fermentation by these pathogenic species [1, 4]. Some authors have suggested that *Candida* species be considered in the early differential of gas-forming necrotizing soft tissue infections in diabetic patients [1, 9]. In addition, urinary tract infections have also been commonly found in Fournier's gangrene patients with fungal etiologies [1-3] and these patients may be chronically colonized with *Candida* species. While no fungal growth was isolated on urine or blood cultures, bacterial translocation and urinary incontinence may help explain the progression of *C. tropicalis* infection in our patient.

Given the increasing prevalence of necrotizing infections due to fungal sources, some have suggested empiric therapy with antifungals in addition to broadspectrum antibiotics and surgical debridement [3, 10]. As resistance to azole therapy is high among *Candida* species, caspofungin may be an appropriate first-line antifungal in those susceptible to invasive fungal infections, although this is yet to be determined [3].

Conclusion

In summary, we present the first report of *Candida tropicalis* as the sole causative organism in a fatal case of Fournier's gangrene and thigh myonecrosis. While rare, fungal etiologies of this clinical entity are being reported with increasing frequency. Particularly in high-risk patients, such as those with underlying diabetes and immunosuppression, early consideration should be given to fungal sources in Fournier's gangrene. Prompt initiation of empiric caspofungin should be considered as part of the treatment algorithm in addition to the standard of care.

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