



Review Article

Fournier Gangrene Microbiology and Risk Factors

Zhong Li Titus Lim^{1*}, Yuyang Liu¹, Kit June Chan², Jason Jae Yeun Kim¹, Eric Chung¹, Handoo Rhee¹

¹Department of Urology, Princess Alexandra Hospital, 199 Ipswich Road, Woolloongabba, Queensland, Australia

²Mater Hospital Brisbane Raymond Terrace, South Brisbane, Queensland, Australia

***Corresponding author:** Zhong Li Titus Lim, Department of Urology, Princess Alexandra Hospital, 199 Ipswich Road, Woolloongabba, Queensland, Australia

Citation: Lim ZLT, Liu Y, Chan KJ, Kim JJY, Chung E, et al. (2025) Fournier Gangrene Microbiology and Risk Factors. J Surg 10: 11263 DOI: 10.29011/2575-9760.011263

Received Date: 22 February 2025; **Accepted Date:** 28 February 2025; **Published Date:** 03 March 2025

Abstract

Fournier Gangrene Microbiology and risk factors

Purpose: Fournier gangrene, a form of necrotizing fasciitis, is an emergent urological condition that affects the perineal, anal, scrotal and genital superficial and deep tissues. The purpose of this study is to provide a contemporaneous report on the polymicrobial organisms associated with Fournier Gangrene in the region of Queensland, Australia and the potential risk factors associated with this condition.

Method: A retrospective study was conducted to analyse admissions at the Princess Alexandra Hospital regarding Fournier gangrene from November 2012 to November 2023. A total of 44 patients were diagnosed with this condition and operated on during this time. Standard demographics, past medical history, as well as urine, tissue and swab microscopy, cultures and sensitivities were reviewed.

Results: From these 44 patients, a total number of 231 debridements and reconstruction surgeries were performed. 36 patients had polymicrobial infections, while 7 had single organisms causing their condition. The risk factors that contributed the most were diabetes (n=19, 43.2%), hypertension (n=23, 52.3%) and a current smoker status (n=27, 61.4%). The most common organisms were *Staphylococcus aureus*, followed by *Streptococcus pyogenes* and *Escherichia coli*.

Conclusion: This report serves to provide an ongoing review and examination of the organisms and risk factors that contribute to Fournier gangrene. This is clinically important due to ongoing changes in human bacterial flora which are impacted by the increasing use of antibiotics in modern medicine.

Keywords: Fournier Gangrene; Microbiology; Necrotizing fasciitis

Abbreviations: EMR: Electronic Medical Record; PAH: Princess Alexandra Hospital

Introduction

Fournier gangrene is a severe, rapidly progressing form of necrotizing fasciitis that primarily involves the perineal, anal,

scrotal, and genital regions. [1] This life-threatening condition is characterized by the widespread death of soft tissues due to bacterial infection, and it typically affects both superficial and deep tissue layers. [1,2] Fournier gangrene requires urgent medical intervention, as it can lead to systemic toxicity, septic shock, and multi-organ failure if not treated promptly. [3] Although it is a rare condition, it often presents in patients with underlying comorbidities such as diabetes, immunosuppression, or chronic alcoholism, making early diagnosis and aggressive management

critical for improving outcomes [4].

The purpose of this study is to provide a contemporaneous report on the polymicrobial organisms associated with Fournier Gangrene in the region of Queensland, Australia and the potential risk factors associated with this condition.

Method

A retrospective observational analysis was conducted at a major quaternary Princess Alexandra Hospital (PAH) in Southeast Queensland, Australia between 1 November 2012 and 31 November 2023. Local ethics approval was obtained through the PAH Human Research Ethics Committee. A database captured patients under the ICD-10 code of ‘N49.8’ and ‘N76.8’ displayed anywhere in their hospital coding’ - ‘Fournier’s Gangrene’ in the hospital admission coding. Basic demographics, time of presentation, length of stay, surgery performed were obtained via independent review of the Electronic Medical Record (EMR). Further sub-analysis of the data was conducted to review various risk factors, as well as urine, tissue and swab microscopy, culture and sensitivities. Baseline characteristics will be expressed as mean ± standard deviation for continuous variables or as numbers and proportions for categorical variables. Differences between categorical variables will be assessed using the chi-squared test,

while differences between continuous parametric variables will be evaluated using the Student’s t-test. For continuous non-parametric variables, the Mann-Whitney U test will be employed.

Results

Clinical and Microbiological Characteristics of Patients

In this cohort of 44 patients, a total of 231 debridement and reconstruction surgeries were performed. The average number of debridements required per patient was 4.1 (range: 0-15), and the average number of plastic reconstruction procedures was 1.1 (range: 0-4). The mean age of the patients was 59.34 years (range: 34-84). The male to female ratio was 41:3. The mean length of stay (LOS) in the hospital was 39.05 days (range: 1-266), and the mean ICU LOS was 2.66 days (range: 0-61). Five out of the 44 patients (11.4%) succumbed to Fournier’s Gangrene. Microbiological analysis revealed that 36 patients (81.8%) had polymicrobial infections, while 7 patients (15.9%) had infections caused by a single organism. The 1 patient who had no microbiology succumbed to his illness before any cultures were done. The most frequently isolated organisms were *Staphylococcus aureus*, followed by *Streptococcus pyogenes* and *Escherichia coli*. The predominant risk factors identified were diabetes (n=19, 43.2%), hypertension (n=23, 52.3%), and current smoker status (n=27, 61.4%).

Characteristics	
No of Patients	44
No of surgeries	231
Average debridement per patient (Range)	4.1 (0-15)
Average plastic reconstruction procedures per patient (Range)	1.1 (0-4)
Mean age (Range)	59.34 (34-44)
Male to female ratio	41:3
Mean hospital LOS (Days, Range)	39.05 (1-266)
Mean ICU LOS (Days, Range)	2.66 (0-61)
Mortality (% , No. of patients)	11.4 (5)
Polymicrobial infections (% , No. of patients)	81.8 (36)
Single organism infections (% , No. of patients)	15.9 (7)
Common organisms	<i>Staphylococcus aureus</i> <i>Streptococcus pyogenes</i> <i>Escherichia coli</i>

Table 1: Summary of Clinical and Microbiological Characteristics.

Comorbidities	No. of patients	%
Diabetes	19	43.18
HTN	23	52.27
Cardiac Disease	13	29.55
BMI > 35	15	34.09
Vasculopathy	9	20.45
Alcohol	12	27.27
Smoker	27	61.36
Immunocompromised States	11	25.00

Table 2: Co-morbidities and Risk Factors.

Discussion

Fournier’s Gangrene is a rapidly progressing, life-threatening soft tissue infection that predominantly affects the genital, perineal, and lower abdominal regions. [2] It is characterized by the necrosis of tissues due to a synergistic infection caused by a mix of aerobic and anaerobic microorganisms [5]. First described by the French surgeon Jean-Alfred Fournier in 1883, the condition remains a critical and urgent clinical scenario requiring immediate intervention, typically involving broad-spectrum antibiotics and often surgical debridement. [3,6] Despite its relatively rare occurrence, it has significant implications in terms of public health, as it often involves underlying systemic conditions and requires multidisciplinary management.

Clinical Presentation

The clinical presentation of Fournier’s Gangrene is typically dramatic and severe. [7] Patients often experience an acute onset of severe or excruciating pain in the perineal or genital region, accompanied by swelling and erythema. [8] As the condition progresses, signs of necrosis such as skin discoloration, bullae formation, and crepitus from gas production become apparent. [8] Systemic symptoms including fever, chills, tachycardia, hypotension, and signs of sepsis may develop, with systemic toxicity becoming more pronounced over time. [8] The extensive tissue breakdown often leads to the discharge of foul-smelling pus. [8] In severe cases, patients may exhibit altered mental status, including delirium or confusion, due to septic shock [9].

Risk Factors for Fournier’s Gangrene

Fournier’s Gangrene tends to occur in patients with various predisposing factors, particularly those that compromise the immune system or alter the integrity of the skin and mucous membranes. [10] Key risk factors include diabetes mellitus, which accounts for 50-60% of cases and is associated with hyperglycemia, immune dysfunction, poor circulation, and peripheral neuropathy. [11,12] Diabetes can lead to neuropathy, which reduces sensation

and the ability to detect early signs of infection. Additionally, diabetes often compromises the immune system, making it harder for the body to fight off infections. Poor blood circulation, another common issue in diabetic patients, further exacerbates the risk by limiting the delivery of immune cells and antibiotics to the affected area. Immunocompromised states such as HIV/AIDS, cancer, organ transplantation, and the use of immunosuppressive medications increase the risk by impairing the immune response. [13] The weakened immune response in these individuals hampers their ability to fight off infections effectively, allowing bacteria to proliferate unchecked. Additionally, immunosuppressed patients might not exhibit the typical inflammatory responses, delaying diagnosis and treatment. This delay can lead to extensive tissue destruction, systemic infection, and increased mortality rates.

Chronic alcoholism, linked to poor nutrition, immune dysfunction, and liver disease, increases the risk of Fournier’s Gangrene. [14] Alcohol abuse leads to various physiological changes that can predispose individuals to this devastating condition. It weakens the immune system, making it harder for the body to fend off infections. Additionally, alcohol abuse often results in poor nutritional status and liver disease, further compromising the body’s defenses. Chronic alcoholics might also neglect personal hygiene and delay seeking medical care, which can exacerbate the condition. These combined factors create an environment where bacteria can easily invade and spread, leading to the rapid tissue destruction characteristic of Fournier’s gangrene. Obesity, which is associated with metabolic syndrome and diabetes, is also another key risk factor. [15,16] Obesity impairs circulation and skinfolds create environments conducive to bacterial growth. Trauma leading to wounds in the perineal or genital region is also a significant risk factor. [12] Any wound or laceration can serve as a portal of entry for pathogens. Chronic skin conditions like psoriasis and eczema, which compromise the skin’s barrier function, heighten susceptibility to infections. [17,18] Additionally, certain infectious diseases such as syphilis, gonorrhea, and tuberculosis, poor hygiene, smoking, and lack of access to medical care contribute to the development of Fournier’s Gangrene [19].

Microbiology of Fournier’s Gangrene

Fournier’s Gangrene is a polymicrobial infection, meaning it involves multiple types of bacteria, both aerobic and anaerobic, which act synergistically to produce tissue necrosis. The exact pathogens involved can vary depending on the source of infection and the patient’s underlying health status. Gram-positive cocci are frequently isolated in Fournier’s Gangrene. Among these, *Streptococcus pyogenes* (group A streptococcus) and *Staphylococcus aureus*, including methicillin-resistant *Staphylococcus aureus* (MRSA), are prominent pathogens. [20,21] These bacteria are known to produce toxins that exacerbate

tissue damage and systemic toxicity. Additionally, *Enterococcus faecalis* is often implicated, particularly in infections originating from the gastrointestinal or genitourinary tracts. [20,22] Gram-negative bacteria are also significant contributors. *Escherichia coli* is one of the most isolated species from wound cultures of patients with Fournier's gangrene. [20] It is a common species of facultative anaerobes found in the human gastrointestinal tract. [23] Other enteric gram-negative rods, including *Klebsiella pneumoniae*, *Proteus mirabilis*, and *Pseudomonas aeruginosa*, may also play a role in the disease process. [20] Anaerobes are critical to the pathophysiology of Fournier's Gangrene, as they contribute to the extensive tissue necrosis observed. [5] Common anaerobic pathogens include *Bacteroides species*, *Clostridium species* (e.g., *Clostridium perfringens*, often associated with gas gangrene), *Fusobacterium species*, and *Peptostreptococcus species*. [8] Although less commonly involved, fungal organisms such as *Candida species* may contribute to infections, especially in immunocompromised individuals [20].

Synergistic Effects

The polymicrobial nature of Fournier's Gangrene is significant, as the combined activity of these organisms exacerbates tissue damage. [8] For instance, *Clostridium perfringens* produces alpha-toxin, which breaks down cell membranes, while other bacteria produce gas and destructive enzymes that promote the spread of infection. [1,8] This synergistic interaction accelerates the progression of the disease, leading to severe necrosis and systemic toxicity [1].

Pathogenesis

The infection typically begins in the subcutaneous tissue, spreading rapidly along fascial planes and causing extensive tissue necrosis. [24] Aerobic bacteria initiate tissue degradation, while anaerobes create anoxic conditions that support their growth and further enhance tissue destruction. [24] These pathogens secrete a range of enzymes, including proteases, lipases, and collagenases, which disrupt tissue integrity. [24] This enzymatic activity, coupled with toxin production, leads to widespread ischemia, necrosis, and potentially fatal sepsis. [8] Understanding the microbiology of Fournier's Gangrene underscores the importance of early, broad-spectrum antimicrobial therapy targeting both aerobic and anaerobic pathogens. Prompt surgical debridement and multidisciplinary management are essential to mitigate the devastating effects of this aggressive infection.

Management and Treatment

Early and aggressive surgical debridement of necrotic tissue is crucial for improving survival and outcomes in patients with Fournier's Gangrene. [1] Multiple surgeries may be necessary, depending on the infection's extent. [25] Empiric broad-spectrum

antibiotic therapy should be initiated promptly to cover both aerobic and anaerobic organisms. [20,24] Common regimens include combinations such as piperacillin-tazobactam or meropenem for broad coverage, including anaerobes, and vancomycin or clindamycin to cover MRSA and toxin-producing organisms like *Streptococcus pyogenes*. [1,8] Supportive care is essential and includes fluid resuscitation, vasopressors in cases of septic shock, and monitoring for organ dysfunction. Patients may also require nutritional support and wound care. [1,8] Some studies suggest that hyperbaric oxygen therapy may enhance the killing of anaerobic organisms and promote tissue healing, although its role remains debated. [8,26] Given the severity and complexity of Fournier's Gangrene, a multidisciplinary team approach involving urologists, surgeons, infectious disease specialists, and critical care providers is essential for optimal management [8].

Conclusion

Fournier's Gangrene is a rare but life-threatening condition with high mortality rates, particularly when not promptly recognized and treated. Identifying the risk factors, such as diabetes, immunosuppression, trauma, and urological or genital conditions, is crucial for early diagnosis and intervention. The microbiology of the disease is polymicrobial, with both aerobic and anaerobic bacteria contributing to the rapid tissue necrosis seen in affected areas. Early surgical debridement and appropriate antibiotic therapy are essential components of management, while supportive care plays a vital role in improving patient outcomes. This report serves to provide an ongoing review and examination of the organisms and risk factors that contribute to Fournier gangrene. This topic holds significant clinical importance due to the ongoing shifts in human bacterial flora, driven by the increasing use of antibiotics in modern medical practice. Additionally, It facilitates the early identification of high-risk populations, enabling timely intervention and effective management.

References

1. Shyam DC, Rapsang AG (2013) Fournier's gangrene. The Surgeon 11: 222-232.
2. Misiakos EP, Bagias G, Patapis P, Sotiropoulos D, Kanavidis P, et al. (2014) Current concepts in the management of necrotizing fasciitis. Front Surg 1: 36.
3. Thwaini A, Khan A, Malik A, et al. (2006) Fournier's gangrene and its emergency management. Postgrad Med J 82: 516-519.
4. Sorensen MD, Krieger JN, Rivara FP, et al. (2009) Fournier's Gangrene: population based epidemiology and outcomes. J Urol 181: 2120-2126.
5. Uluğ M, Gedik E, Girgin S, Çelen MK, Ayaz C (2009) The evaluation of microbiology and Fournier's gangrene severity index in 27 patients. International Journal of Infectious Diseases 13: e424-e430.

6. Norton KS, Johnson LW, Perry T, Perry KH, Sehon JK, et al. (2002) Management of Fournier's gangrene: an eleven year retrospective analysis of early recognition, diagnosis, and treatment. *Am Surg* 68: 709-713.
7. Kuzaka B, Wróblewska MM, Borkowski T, et al. (2018) Fournier's Gangrene: Clinical Presentation of 13 Cases. *Med Sci Monit* 24: 548-555.
8. Auerbach J, Bornstein K, Ramzy M, Cabrera J, Montrieff T, et al. (2020) Fournier Gangrene in the Emergency Department: Diagnostic Dilemmas, Treatments and Current Perspectives. *Open Access Emerg Med* 12: 353-364.
9. Stevens DL, Bisno AL, Chambers HF, et al. (2005) Practice Guidelines for the Diagnosis and Management of Skin and Soft-Tissue Infections. *Clinical Infectious Diseases* 41: 1373-1406.
10. Tosun Y, Akıncı O, Küçük HF (2022) Risk factors for mortality in Fournier's gangrene of anorectal origin. *Ulus Travma Acil Cerrahi Derg* 28: 1128-1133.
11. Lewis GD, Majeed M, Olang CA, et al. (2021) Fournier's Gangrene Diagnosis and Treatment: A Systematic Review. *Cureus* 13: e18948.
12. Zhang N, Yu X, Zhang K, Liu T (2020) A retrospective case series of Fournier's gangrene: necrotizing fasciitis in perineum and perianal region. *BMC Surg* 20: 259.
13. Crowell W, Roberts R, Tarry S (2016) Fungal Fourniers Gangrene in an Immunocompromised Patient. *Urol Case Rep* 4: 1-3.
14. Hughes T, Bowen D, Saeed K, Juliebø-Jones P, Somani B (2023) Management of Fournier's gangrene: a practical guide for clinicians. *Br J Hosp Med (Lond)* 84: 1-9.
15. Czymek R, Hildebrand P, Kleemann M, et al. (2009) New insights into the epidemiology and etiology of Fournier's gangrene: a review of 33 patients. *Infection* 37: 306-312.
16. Tang L-M, Su Y-J, Lai Y-C (2015) The evaluation of microbiology and prognosis of fournier's gangrene in past five years. *Springer Plus* 4: 14.
17. Livia Hoyer Garcia Miranda JAS, Isabella Cristina Chiamolera, José Moacir Mierzva FDdS. Fournier's gangrene in a patient with psoriatic arthritis: a case report. *Rev Soc Bras Clin Med* 21: 118-121.
18. Çalışkan S, Özsoy E, Sungur M, Gözdaş HT (2019) Fournier's gangrene: Review of 36 cases. *Ulus Travma Acil Cerrahi Derg* 25: 479-483.
19. You Q, Guan J, Wu B, et al. (2024) Fournier's Gangrene: clinical case review and analysis of risk factors for mortality. *BMC Surg* 24: 251.
20. Yilmazlar T, Gulcu B, Isik O, Ozturk E (2017) Microbiological aspects of Fournier's gangrene. *International Journal of Surgery* 40: 135-138.
21. He X, Xiang X, Zou Y, et al. (2022) Distinctions between Fournier's gangrene and lower extremity necrotizing fasciitis: microbiology and factors affecting mortality. *International Journal of Infectious Diseases* 122: 222-229.
22. He R, Li X, Xie K, Wen B, Qi X (2022) Characteristics of Fournier gangrene and evaluation of the effects of negative-pressure wound therapy. *Front Surg* 9: 1075968.
23. Shaked H, Samra Z, Paul M, et al. (2012) Unusual "flesh-eating" strains of *Escherichia coli*. *J Clin Microbiol* 50: 4008-4011.
24. Chernyadyev SA, Ufimtseva MA, Vishnevskaya IF, et al. (2018) Fournier's Gangrene: Literature Review and Clinical Cases. *Urol Int* 101: 91-97.
25. Izadi D, Coelho J, Gurjal S, Salim F (2016) Fournier's Gangrene and the Reconstructive Challenges for the Plastic Surgeon. *Eplasty* 16: ic38.
26. Kindwall EP, Gottlieb LJ, Larson DL (1991) Hyperbaric oxygen therapy in plastic surgery: a review article. *Plast Reconstr Surg* 88: 898-908.