





Fournier's Gangrene Following Coronary Artery Bypass Surgery: Should Extracorporeal Circulation Be Blamed?

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Background: Fournier's gangrene is a rare infection with high morbidity and mortality caused by the aerobic and anaerobic bacteria that affects the superficial tissues. It is a kind of necrotizing infection which is characterized by the necrotizing forms of cellulitis, myositis, and fasciitis. These infections are presented clinically by fulminant tissue destruction, systemic signs of toxicity, and even sepsis. Accurate diagnosis and appropriate treatment must include early surgical intervention and antibiotic therapy. Aggressive debridement, appropriate broad-spectrum antibiotic therapy, and combined enteral and parenteral nutrition are applied for the treatment. Fournier's gangrene is rarely seen after extensive surgical operations, and if untreated it may increase the surgical mortality.

Conclusion: In this article, diagnosis and the treatment of a case with a Fournier's gangrene that developed after coronary bypass surgery and its etiological relationship with the extracorporeal circulation were discussed.

Keywords: Fournier's gangrene, coronary artery bypass, extracorporeal circulation, therapy

Introduction

ournier's gangrene is a type of necrotizing gangrene usually fasciitis and affecting the perineum and genitalia. It was initially reported in 1883 by a French venereologist Jean Alfred Fournier (1). It is mostly encountered with diabetes and poor hygiene. Etiology is mainly related to comorbid disease that impairs the immune system whereas 20% of the cases are idiopathic.

Fournier's gangrene is commonly seen in men but can also affect women and children. Incidence is higher between the ages of 50 to 70. Early diagnosis, aggressive surgical debridement and parenteral infusion of wide spectrum antibiotics may reduce the mortality rates (2). Hematopoietic system alteration following open heart surgery may increase the patient's susceptibility to infections. Polymicrobial infection in perineum and genital organs can quickly disseminate and cause tissue destruction (3). In this article, we present the diagnosis and management of a case who developed a Fournier's gangrene following coronary artery bypass graft (CABG) operation.

Case Presentation

A 65-year-old man admitted to the emergency department with an acute myocardial infarction. Following coronary angiography, CABG operation was planned. In his medical history, Diabetes mellitus (DM)

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and chronic obstructive pulmonary disease (COPD) were present. Upon his complaints of pain and burning sensation, the physical examination was extended to his gluteal region that was seemingly healthy. Biochemical and hematological blood tests were normal except a slight increase in creatinine (1.7 mg/dL). His blood sugar level was 189 mg/dL and glycolyzed hemoglobin (HbA_{1C}) was 8,1%. He was receiving oral hypoglycemic treatment including metformin (2000 mg/day) and gliclazide (180 mg/day).



Figure-1: (A) Necrotizing fasciitis of the scrotum and (B) after complete recovery.

Following preoperative evaluation, CABG operation was performed successfully with full revascularization. Patient was discharged postoperative sixth day with a complete recovery. At the third day after release, (postoperative ninth day), he re-admitted with a 38 °C

fever and perineal ulcer. On admission, increased white blood cell count (WBC, 13000/mm3), erythrocyte sedimentation rate (ESR, 70mm/h), and C-reactive protein (CRP, 19 mg/dL) levels were detected in his laboratory tests. In his physical examination, he was otherwise well except the presence of a 2x2 cm decubitus ulcer over his sacral region. The patient was hospitalized, and parenteral third-generation cephalosporin and local rifampicin were initiated. After the fifth day of local and systemic antibiotherapy, ulcer was not regressed and became hard and hyperemic. Hematological parameters including WBC, ESR, and CRP showed an increase despite the antibiotic administration. WBC, ESR, and CRP have risen to 15000/mm3, 90 mm/h, and 24 mg/dL respectively. Biochemical tests, which had been previously normal were also deteriorated. Liver function tests (Alanine amino transferase: 80 U/L, Aspartate amino transferase: 60 U/L) and renal function test (creatinine:1.8 mg/dL) were slightly increased. A purulent effusion was started at the gluteal ulcer. Fever was around 38.5 °C. Smear culture was taken. Escherichia Coli and Candida Albicans were proliferated in the culture. Antibiotic (Imipenem-Cilastatin 1500 mg/day) and antifungal (Voriconazole 400 mg/day) regime was established according to the antibiogram. Human albumin was started as the serum albumin levels were decreased down to 1.7 g/dL. General status of the patient was progressively deteriorated, and liver function tests increased further. Sacral lesion was progressed towards the perineum and scrotum (Figure-1A). At the seventh day of antibiotherapy, urology and general surgery consultations were made. Extensive surgical debridement was decided with the diagnosis of Fournier's gangrene. Necrotic area in the sacrum was excised and debrided until the presacral fascia. Scrotal and penile gangrenous tissues were also operated in the same manner. Parenteral Doripenem Monohydrate (0.5 g/day) and Tigecycline (100 mg/day) were started instead of the previous antibiotic regime.

Wound care and debridement was continued every day with mild sedation. Urinary catheterisation was established. Fluid balance was monitored. Colostomy was not considered. Enteral and supportive parenteral nutrition was started. Glycemic control was made with inter mittent regular insulin keeping the blood glucose between 90-130 mg/dL. New antibiotic regime was maintained for fourteen days. WBC (8000/mm3), ESR (24 mm/h), and CRP (12 mg/dL) levels were declined. An erythrocyte suspension was replaced as the hemoglobin levels were around 8,0 g/dL. Patient started to be mobilized as the open sores become hyperemic which is a sign of recovery. Vacuum-assisted closure (VAC) system was applied and maintained for nine days. Finally, open lesions were sutured and closed primarily (*Figure-1B*). Complete recovery of the patient was achieved 35 days after his second admission.

Discussion

Fournier's gangrene is a rare and highly mortal fulminant gangrenous infection of subdermal tissues and the fascia of perineal, genital, and perianal regions (4). In the majority of patients, Fournier's gangrene is caused by a mixed infection of both aerobic and anaerobic bacteria. In most of the cases, the source of the infection is perineal and genital skin infections (5). Anorectal, urogenital, or perineal traumas also contribute the pathogenesis. The most common bacterial foci include gastro intestinal tract (30%-50%), genitourinary tract (20%-40%) and the dermal injuries (10%-20%). The most common pre disposing comorbidity is DM and chronic alcoholism (6). DM is reported in 20%-70% of the patients with Fournier's gangrene, similarly as the chronic alcoholism in 25%-50% (7). Fournier's gangrene is mostly seen over the age of sixty-five, although cases in any age have also been reported.

What was the etiology of the Fournier's gangrene, in this case? Was it a coincidence to be preceded by a CABG operation? Do CABG operations really cause an immunosuppressive state? Does the ongoing DM have a role in the development of this disease? The immune response to the open heart surgery has been the subject of interest for a long time (8). The pro-inflammatory response was well recognized with the apparent clinical and laboratory evidence in the patients (9,10). Exposure to the pro-inflammatory trigger factors is thought to be responsible for this response. These include the interaction of the blood with the foreign surface of the cardiopulmonary bypass (CPB) circuits (11). Reduction in pulmonary blood flow due to cross-clamping and hypothermia also contribute the development of the inflammatory response (12).

Endotoxemia and the surgical stress response occur during the CPB (13). This pro-inflammatory response is characterized by the increased plasma concentrations of the pro-inflammatory cytokines, tumor necrosis factor (TNF) and interleukins (IL) IL-1, IL-6 and IL-8 (14-16). Serum levels of complement products and elastase also increase with the elaboration of free oxygen radicals (17, 18). The adhesion potentials of the neutrophils also increase with the upregulation of adhesive molecules like L-selectin, CD11b and CD18 (19, 20). In brief, changes in the immune response in CABG patients mainly result from the CPB process and the surgery (8). The clinical immunosuppression occurring after surgery is described with an increased risk of infection (9,10).

The immunosuppressive impact of the open heart surgery and impairment of the perineal and overall hygiene may play a role in the development of Fournier's gangrene in CABG patients (21). In addition to the immunosuppression, renal insufficiency, existing DM, older age, steroid treatment and peripheral vascular disease are among the major etiological factors of Fournier's gangrene (22).

In pathogenesis, bacterial infections cause micro thrombosis of the subcutaneous arterioles leading to the development of gangrene of the overlying skin (23). Cultures from the lesions often reveal poly microbial proliferation, including coliforms, klebsiella, staphylococci, streptococci, clostridia, and coryne bacteria (24). Cultures may reveal more than two pathogens, most of which are normal commensal organisms of the perineum and genitalia. Because of the impaired host immunity, they become virulent and behave synergistically to destroy the tissue and lead an extensive damage (25). Epidemic emergence of HIV infection also triggered a new threat in population for developing Fournier's gangrene (26).

Although Fournier's gangrene demonstrates a wide spectrum of clinical presentation, the most common presenting symptoms are erythema, pain, fever and fatigue (3). It is mostly fulminant and with rapid onset. Mortality rates vary from 9.1% to 45% in the literature (2). The infection commonly starts as cellulitis at the entry region, mostly the perineum and genitalia. Local signs and symptoms progress so dramatically with a catastrophic erosion and gangrene of the skin that it reveals the subcutaneous tissue to appear. Computerized tomography scan is beneficial in the diagnosis as it demonstrates the extension of the necrosis by visualizing deep and superficial fascia (27).

Unless treated aggressively and appropriately, the patient can easily progress to sepsis with multiple organ failure that is the common cause of death in these patients. Surgical debridement, mobilization, enteral and parenteral feeding, appropriate antibiotherapy, and isolation decreases the morbidity and mortality. Low serum albumin levels are detected in Fournier's gangrene patients According to the status of the patient, enteral or total parenteral nutrition should be provided immediately. Early diagnosis is especially important, as the progression rate of the necrosis can easily be as rapid as 2-3 cm/h (27). Emergent surgical debridement should aim to total excision of all devitalized tissues, in order to prevent the dissemination of infection and to inhibit the development of systemic toxicity (5). Vacuum assisted closure (VAC) is considered beneficial over the sterile sore after the infection is suppressed, and purulation is stopped (28). Mortality rates related to Fournier's gangrene decreased 50% in the last 15 years by highlighting the significance of early diagnosis and emergent extensive surgical intervention in the management of the disease (5).

As a conclusion, Fournier's gangrene is a lifethreatening disease, with high death rates despite the developments in diagnostic and therapeutic tools. Early diagnosis and prompt surgical intervention are crucial for recovery. Extensive surgical operations like open heart surgery may lead to a relative state of immuno suppression especially in older and diabetic patients. Postoperative sacral and perineal hygiene should strongly be advised and any lesion developed after surgery in this region should be carefully examined to rule out possible Fournier's gangrene.

Conflict of Interest

The authors declare that no conflict of interest exists in publishing this article.

Reference

1. Eke N. Fournier's gangrene: A review of 1726 cases. Br J Surg 2000;87(6):718-28.

2. Spirnak JP, Resnick MI, Hampel N, Persky L. Fournier's gangrene; report of 20 patients. J Urol 1984;131(2):289-91.

3. Morpurgo E, Galandiuk S. Fournier's gangrene. Surg Clin North Am 2002;82(6):1213-24.

4. Smith GL, Bunker CB, Dineeen MD. Fournier's gangrene. Br J Urol 1998;81(3):347-55.

5. Thawani A, Khan A, Malik A, Cherian J, Barua J, Shergill I, et al. Fournier's gangrene and its emergency management. Postgrad Med J 2006;82(970):516-9.

6. Fournier JA. Gangrene foudroyante (overwhelming gangrene). Sem Med 1883. Dis Colon Rectum 1988;31(12):984-8.

7. Clayton MD, Fowler JE Jr, Sharifi R, Pearl RK. Causes, presentation and survival of fifty-seven patients with necrotizing fasciitis of the male genitalia. Surg Gynecol Obstet 1990; 170(1):49-55.

8. Knudsen F, Andersen LW. Immunological aspects of cardio pulmonary bypass. Journal of Cardiothoracic Anesthesia 1990; 4(2):245-58.

9. Norenberg RG, Sethi NK, Scott SM, Takaro T. Opportunistic endocarditis following open heart surgery. Ann Thorac Surg 1975;19(5):592-604.

10. Hisatomi K, Isomura T, Kawara T. Changes in lymphocyte subsets, mitogen responsiveness and interleukin-2 production after cardiac operations. J Thorac Cardiovasc Surg 1989;98(4):580-91.

11. Butler J, Chong GL, Baigrie RJ, Pillai R, Westaby S, Rocker GM. Cytokine responses to cardiopulmonary bypass with membrane and bubble oxygenation. The Annals of thoracic surgery 1992;53(5):833-8.

12. Prasad K, Kalra J, Bharadwaj B, Chaudhary AK. Increased oxygen free radical activity in patients on cardiopulmonary bypass undergoing aortocoronary surgery. Am Heart J 1992;123(1):37-47.

13. Jansen NJG, van Oeveren W, Gu YJ, van Vliet MH, Eijsman L, Wildevuur CR. Endotoxin release and tumor necrosis factor formation during cardiopulmonary bypass. The Annals of thoracic surgery 1992;54(4):744-8.

14. Lahat N, Zlotnick AY. Serum levels of IL-1, IL-6 and TNF in patients undergoing coronary artery bypass grafts or cholesystectomy. Clinical and Experimental Immunology 1992;89(2):255-60.

15. Kawamura T, Wakusawa R, Okada K, Inadat S. Elevation of cytokines during open heart surgery with cardiopulmonary bypass: participation of interleukin 8 and 6 in reperfusion injury. Canadian journal of anaesthesia 1993;40(11):1016-21.

16. Kalfin RE, Engelman RM, et al. Induction of interleukin-8 expression during cardiopulmonary bypass. Circulation 1993;88 (5 pt 2):2401-6.

17. Haeffner-Cavaillon N, Roussellier N, et al. Induction of interleukin-1 production in patients undergoing cardio pulmonary bypass. The Journal of Thoracic and Cardiovascular Surgery 1989;98(6): 1100-6.

18. Girardin E, Roux-Lombard P, Grau GE. Imbalance between tumour necrosis factor-alpha and soluble TNF receptor concentrations in severe meningococcaemia. The J5 Study Group. Immunology 1992;76(1):20-3.

19. McBride WT, Armstrong MA, Crockard AD, McMurray TJ, Lyons SM. Selective reduction in leucocyte surface marker expression following high dose fentanyl administration at cardiac surgery. British Journal of Anesthesia 1994;73(5):717-8.

20. Gillinov AM, Bator JM, Zehr KJ, Redmond JM, Burch RM, Ko C, et al. Neutrophil adhesion molecule expression during cardiopulmonary bypass with bubble and membrane oxygenators. The Annals of thoracic surgery 1993;56(4):847-53.

21. McBride WT, Armstrong MA, Crockard AD, McMurray TJ, Rea JM. Cytokine balance and immunosuppressive changes at cardiac surgery: contrasting response between patients and isolated CPB circuits. British Journal of Anesthesia 1995; 75(6):724-33.

22. Donaldson PMW, Naylor B, Lowe JW, Gouldesbrough DR. Rapidly fatal necrotizing fasciitis caused by streptecoccus pyogenes. J Clin Pathol 1993;46(7):617-9.

23. Johnin K, Nakatoh M, Kadowaki T, Kushima M, Koizumi Z, Okada Y. Fournier's gangrene caused by Candida species as the primary organism. Urology 2000;56(1):153.

24. Yaghan RJ, Al-Jaberi TM, Bani-Hani I. Fournier's gangrene: changing face of the disease. Dis Colon Rectum 2000; 43(9):1300-8.

25. Rotstein OD, Pruett TL, Simmons RL. Mechanisms of microbial synergy in polymicrobial surgical infections. Rev Infect Dis 1985;7(2):151-70.

26. Elem B, Ranjan P. Impact of immunodeficiency virus (HIV) on Fournier's gangrene: observations in Zambia. Ann R Coll Surg Engl 1995;77(4):283-6.

27. Levonsen RB, Singh AK, Novelline RA. Fournier gangrene: role of imaging. Radiographics 2008;28(2):519-28.

28. Czymek R, Schmidt A. Fornier's gangrene: VAC. Am J Surg 2009;197(2):168-76.

DOI: dx.doi.org/10.5455/umj.20150827014934

Cite article as: Usta S, Basbug HS. Fournier's Gangrene following coronary artery bypass surgery: Should extracorporeal circulation be blamed?. Ulutas Med J. 2015; 1(3):81-85

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