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Original Article

Fournier's gangrene: ten-year experience in a medical center in northern Taiwan

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Background and Purpose: Fournier's gangrene is a life-threatening infection. The mortality is still high despite the rapid advancement of modern intensive care and surgical technique. In this study, we present our institution's recent experience with a large series of patients with Fournier's gangrene.

Methods: A retrospective chart review was performed including 44 consecutive patients with Fournier's gangrene over a 10-year period.

Results: The 44 cases comprised 39 males and 5 females, with a mean age of 55.5 years. The mean duration of hospitalization was 27.9 days. Overall mortality was 22.7%. Diabetes mellitus, hypertension, chronic liver disease, liver cirrhosis and chronic renal insufficiency were the 5 leading predisposing factors. Liver cirrhosis was highly related to mortality (p=0.009). The etiologic origin of the gangrene was colorectal, urological and dermatological in 52.3%, 25.0%, and 11.4% of patients, respectively. The most common isolated pathogens were *Escherichia coli*, *Bacteroides fragilis*, *Klebsiella pneumoniae*, *Enterococcus* spp., and *Proteus mirabilis*. There were a total of 74 debridements. Other related surgical procedures were reconstruction surgery (n = 18), colostomy (2), cystostomy (1), vasectomy (1), orchiectomy (1) and penectomy (1). Major complications of Fournier's gangrene, including respiratory failure, renal failure, septic shock, hepatic failure and disseminated intravascular coagulopathy, were significantly to mortality (p<0.05).

Conclusions: Early diagnosis, intensive medical care (aggressive resuscitation and broad-spectrum antibiotics), and prompt and repeated surgical intervention are the mainstays of treatment. Liver cirrhosis in particular is a poor prognostic factor. Reconstructive surgery should also be a consideration once the acute condition has improved. Patients with comorbid condition, serious infection, and major complications should be treated carefully and aggressively.

Key words: Fasciitis, necrotizing; Fournier gangrene; Infection

Introduction

Fournier's gangrene, a fulminant necrotizing fasciitis involving the genital, perianal and perineal region, was first reported by Baurienne in 1764 [1]. A French dermatologist and venereologist, Jean Alfred Fournier, gave this infectious disease its eponymous name in 1883 [2]. The infection developed abruptly in 5 young healthy males and progressed rapidly to gangrene. Three characteristics were emphasized: (1) sudden onset in a healthy young male; (2) rapid progression to gangrene; and (3) absence of a definite cause. Since then, the epidemiology and clinical features of the disease have changed significantly.

Despite the development of medical therapy and intensive care technique, Fournier's gangrene still carries a high mortality [3]. Although conservative treatment without surgery may be adequate in some patients, broad-spectrum antibiotics, aggressive debridement, and intensive care technique are recognized as cornerstone measures for avoiding fatal outcome [4].

In this study, we retrospectively investigated the data on etiology, underlying diseases, bacteriology, surgical management, reconstructive surgeries, and complications in a series of 44 patients with Fournier's gangrene during a 10-year period.

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Methods

Patients

We reviewed the medical charts of patients with Fournier's gangrene between 1994 and 2003 at Mackay Memorial Hospital, a medical center and teaching hospital comprising about 2000 beds in northern Taiwan. We used the International Classification of Diseases-9 codes (1 = Fournier's gangrene; 2 = perianal abscess; 3 = anal fistula and necrotizing fasciitis) to identify all patients. Forty four patients were identified and included. The inclusion criteria were: (1) clinical picture compatible with rapid progressive fulminant infection initially appearing in the genital, perineal and perianal region; (2) surgical findings of gangrenous and necrotic tissue, and purulent drainage, etc.; and (3) pathologically proven necrotizing fasciitis. Statistical analysis was performed using the Statistical Package for the Social Sciences for Windows (Version 11.0; SPSS Chicago, IL, USA) software package. F-test, *t* test and chi-squared tests was used to analyze the clinical data. A p value <0.05 was considered statistically significant.

Definitions

Respiratory failure was defined as a failure of gas exchange (partial pressure of oxygen <60 mm Hg or partial pressure of carbon dioxide >45 mm Hg) leading to mechanical ventilation support; renal failure was defined as a progressive rise of serum creatinine and blood urea nitrogen; septic shock was defined as sepsis-induced hypotension not responsive to fluid challenge; hepatic failure was defined as a progressive rise of serum bilirubin level; disseminated intravascular coagulopathy (DIC) was defined as thrombocytopenia (100×10^9/L), elevated fibrinogen degradation products and D-dimer, reduced fibrinogen level, prolonged prothrombin time, activated partial thromboplastin time and thrombin time; upper gastrointestinal bleeding was defined as positive occult blood test in the stool and gastric juice and gastroscopy revealing peptic ulcer.

Results

Age and gender

There were 39 males and 5 females in this series. The mean age of the patients was 55.5 years (range, 25 to 82). The patients had a mean hospital stay of 27.9 days (range, 1 to 94). The overall mortality rate was 22.7% (10 of 44 patients). The clinical variables of these patients were recorded (Table 1).

Etiology

The source of infection was colorectal in 23 patients (52.3%), urological in 11 (25.0%), and dermatological in 5 (11.4%). Unknown infection source was noted in 5 patients (11.4%). Six patients (13.6%) developed septic infections from postoperative complications, including perianal or anal abscess post-debridement (3 patients), post-hemorrhoidectomy (2 patients), and post-subtotal hysterectomy (1 patient). No specific etiology was significantly related to mortality (Table 2).

Comorbidities

Some diseases predisposed to Fournier's gangrene. The most frequent pre-existing comorbidities were diabetes mellitus (24/44, 54.5%), hypertension (6/44, 13.6%) and chronic liver disease (5/44, 11.4%). Most patients (84.0%) had at least one predisposing factor. Only 7 patients had no predisposing factors. Liver cirrhosis (p=0.009) was the most significant predictor of mortality (Table 3).

Microbiology

A variety of organisms had been cultured from necrotic tissue or pus during surgery or at the bedside. Only 1 organism was identified in 13 patients (29.5%), while culture results revealed a polymicrobial infection in 28 patients (63.6%). In 3 patients (6.8%), wound cultures were negative. Positive blood culture was noted in 4 patients (9.1%). The most commonly isolated organisms from wound were *Escherichia coli* in 26 patients (59.1%), *Bacteroides fragilis* in 17 patients (38.6%), *Klebsiella pneumoniae* in 16 patients (36.4%), *Enterococcus* spp. in 14 patients (31.8%), and *Proteus mirabilis* in 10 patients (22.7%). Mortality was not related to the specific isolated organism (Table 4).

Medical treatment

Patients were treated with aggressive fluid resuscitation, broad-spectrum antibiotics and hemodynamic support, with slight variation depending on the individual situation.

Surgical management

After proper medical treatment, prompt and aggressive debridement should be performed. All visible devitalized skin, affected subcutaneous tissue, necrotic fascia and even muscle were excised. A total of 98 operations were performed, including 74 debridements (1 in 24 patients, 2 in 9 patients, 3 in 5 patients, 4 in 3 patients, and 5 in 1 patient). The other 24 operations included 2 cases with colostomy, 1 cystostomy, 1 vasectomy, 1 penectomy, 1 orchiectomy and other 18 reconstructive

Table 1. Clinical data of 44 patients with Fournier's gangrene

Patient no.	Age/gender	Etiology	Debridement	Reconstruction	Hospital stay (day[s])	Outcome
1	57/M	Skin infection	Nil	Nil	6	Death
2	45/M	Perianal abscess	1	Nil	1	Death
3	59/M	UTI post-Foley	3	Nil	17	Death
4	32/M	Perianal abscess	1	Nil	1	Death
5	42/M	Colon cancer	2	Nil	51	Death
6	65/M	Perianal abscess	4	Nil	44	Death
7	45/M	Scrotal abscess	1	Secondary repair	9	Death
8	68/M	Scrotal abscess	1	Nil	23	Death
9	82/F	Perianal abscess	Nil	Nil	10	Death
10	29/M	Perianal abscess	1	Nil	1	Death
11	41/M	Scrotal abscess	2	Secondary repair	77	Survival
12	68/M	BPH post-Foley	1	Nil	13	Survival
13	38/M	Postoperative anal fistula	1	Nil	17	Survival
14	37/M	Anal fistula	1	Nil	3	Survival
15	47/M	Unknown	1	Primary closure	15	Survival
16	43/M	Anal fistula	3	Nil	19	Survival
17	43/M	Anal fistula	1	Primary closure	16	Survival
18	70/F	Rectovaginal fistula	1	Nil	13	Survival
19	70/M	Post-hemorrhoidectomy	1	Nil	65	Survival
20	55/M	Unknown	2	Secondary repair	15	Survival
21	67/M	Postoperative perianal abscess	3	Secondary repair	25	Survival
22	25/M	Perianal abscess	1	Nil	19	Survival
23	42/M	Scrotal abscess	1	Secondary repair	39	Survival
24	73/M	Urethral stricture post-Foley	2	Secondary repair	48	Survival
25	69/M	Unknown	2	Secondary repair	39	Survival
26	37/M	Perianal abscess	2	Nil	17	Survival
27	74/M	Unknown	1	Nil	7	Survival
28	69/M	Post-hemorrhoidectomy	1	Nil	43	Survival
29	71/F	Epidermoid cyst	3	Secondary repair	76	Survival
30	70/M	Postoperative anal fistula	5	Secondary repair	94	Survival
31	35/M	Perianal abscess	1	Primary closure	19	Survival
32	51/M	Bed sore	1	Nil	15	Survival
33	66/M	Unknown	1	Nil	9	Survival
34	53/F	Post-subtotal hysterectomy	2	Secondary repair	35	Survival
35	70/M	Perianal abscess	2	Nil	24	Survival
36	41/M	Perianal abscess	1	Nil	8	Survival
37	77/M	Scrotal abscess	3	Secondary repair	38	Survival
38	66/M	Anal fistula	4	Secondary repair	39	Survival
39	42/M	Groin wound	1	Nil	15	Survival
40	74/M	Scrotal abscess	1	Flap	62	Survival
41	72/M	Scrotal abscess	4	Flap	45	Survival
42	64/F	Perianal abscess	2	Nil	55	Survival
43	57/M	UTI post-Foley	1	Primary closure	21	Survival
44	42/M	Perianal abscess	1	Nil	20	Survival

Abbreviations: M = male; F = female; UTI = urinary tract infection; BPH = benign prostatic hyperplasia

surgeries. Two of the 10 non-survivors did not receive debridement due to rapid disease course. All of the 34 survivors received debridement.

Fecal and urinary diversion

Two patients (4.6%) underwent fecal diversion with colostomy and one patient (2.3%) underwent urinary diversion with suprapubic cystostomy to prevent wound contamination. There was no significant relation between these procedures and mortality on chi-squared analysis.

Reconstruction

There may be massive genital skin loss after debridement. Eighteen patients received reconstructive surgery.

Table 2. Etiologic causes in 44 patients with Fournier's gangrene

Cause	Number of non-survivors	Number of survivors	Total (no. [%])	*p*
Colorectal portion	6	17	23 (52.3)	0.694
Perianal abscess or anal fistula	5	11	16 (36.4)	0.308
Perianal abscess or postoperative anal fistula	0	3	3 (6.8)	0.331
Post-hemorrhoidectomy	0	2	2 (4.5)	0.432
Colon cancer	1	0	1 (2.3)	0.062
Rectovaginal fistula	0	1	1 (2.3)	0.583
Urological portion	3	8	11 (25.0)	0.678
Scrotal abscess	2	5	7 (15.9)	0.687
UTI post-Foley	1	1	2 (4.5)	0.346
Urethral stricture post-Foley	0	1	1 (2.3)	0.583
BPH post-Foley	0	1	1 (2.3)	0.583
Dermatological portion	1	4	5 (11.4)	0.328
Bed sore	0	1	1 (2.3)	0.583
Groin wound	0	1	1 (2.3)	0.583
Contact dermatitis	1	0	0 (2.3)	0.062
Epidermoid cyst	0	1	1 (2.3)	0.583
Post-subtotal hysterectomy	0	1	1 (2.3)	0.583
Unknown	0	5	5 (11.4)	0.328
Total	10	34	44 (100.0)	

Abbreviations: UTI = urinary tract infection; BPH = benign prostatic hyperplasia

Wound healing with secondary repair was performed in 12 patients. Wound healing with primary closure was performed in 4 patients. Wound healing with flap was performed in 2 patients.

Complications

Complications developed in 13 patients; these included respiratory failure (11 patients), renal failure (9 patients), septic shock (7 patients), pneumonia (6 patients), hepatic failure (4 patients), DIC (2 patients) and upper gastrointestinal bleeding (1 patient). All of the complications except pneumonia and upper GI bleeding were significantly related to mortality ($p<0.05$) [Table 5].

Discussion

Fournier's gangrene is a serious disease, with mortality as high as 80% [5]. The natural course of this infection

Table 3. Comorbidity in 44 patients with Fournier's gangrene[a]

Comorbidity	Number of non-survivors	Number of survivors	Total (no. [%])	*p*
Diabetes mellitus	6	18	24 (54.5)	0.694
Hypertension	1	5	6 (13.6)	0.703
Chronic liver disease	1	4	5 (11.4)	0.877
Liver cirrhosis	3	1	4 (9.1)	0.009
Chronic renal insufficiency	2	2	4 (9.1)	0.172
Old cerebrovascular accident	0	3	3 (6.8)	0.331
Malignancy	1	2	3 (6.8)	0.650
Benign prostatic hyperplasia	0	3	3 (6.8)	0.331
Alcoholism	1	0	1 (2.3)	0.062
Uremia	1	0	1 (2.3)	0.062
Gastric ulcer	1	0	1 (2.3)	0.062
Valvular heart disease	1	0	1 (2.3)	0.062
Chronic obstructive pulmonary disease	0	1	1 (2.3)	0.583
Congestive heart failure	0	1	1 (2.3)	0.583
Chorea	0	1	1 (2.3)	0.583
None	0	7	7 (15.9)	0.118

[a]Each may have more than 1 comorbid condition.

Table 4. Bacteriology in 44 patients with Fournier's gangrene

Organisim	Number of non-survivors	Number of survivors	Total (No. [%])	*p*
Aerobes				
Gram-positive				
Enterococcus spp.	5	9	14 (31.8)	0.160
Streptococcus spp.	2	7	9 (20.5)	0.968
Staphylococcus epidermidis	0	2	2 (4.5)	0.432
MSSA	1	1	2 (4.5)	0.346
Gram-negative				
Escherichia coli	4	22	26 (59.1)	0.162
Klebsiella pneumoniae	4	12	16 (36.4)	0.786
Proteus mirabilis	2	8	10 (22.7)	0.815
Pseudomonas aeruginosa	1	3	4 (9.1)	0.909
Morganella morganii	1	3	4 (9.1)	0.909
Enterobacter spp.	0	2	2 (4.5)	0.432
Proteus vulgaris	1	0	1 (2.3)	0.062
Anaerobes				
Bacteroides fragilis	4	13	17 (38.6)	0.920
Prevotella spp.	0	3	3 (6.8)	0.331
Peptostreptococcus spp.	0	1	1 (2.3)	0.583
Candida albicans	1	1	2 (4.5)	0.346
Mixed infection			28 (63.6)	
Monobacterial infection			13 (29.5)	

Abbreviation: MSSA = methicillin-susceptible *Staphylococcus aureus*

is rapid and fulminant, ultimately resulting in death. The age of most patients was between 30 and 60 years [6]. The mean age of our patients in this study was 55.5 years (range, 25 to 82). Fournier's gangrene was considered as a male disease in the past; however, it is generally believed that it also occurs in women, but at a lower incidence [7]. In our study, the male-to-female ratio was 7.8:1.

Although the etiology of Fournier's gangrene was described as idiopathic originally, that is not the case today [8]. The source of infection may be identified as of colorectal, urological, or cutaneous origin. The distribution of suspected infection source in one study (including 55 patients) was urological in 35%, colorectal in 29%, and cutaneous in 29% [9]. Another study (including 29 patients) reported the distribution of colorectal, urological and cutaneous origin as 48%, 21%, and 31%, respectively [10]. In our study, colorectal sources (52.3%) were the most frequent etiology, including perianal abscess or fistula in 16 patients, perianal abscess or postoperative fistula in 3, post-hemorrhoidectomy in 1, colon cancer in 1 and recto-vaginal fistula in 1.

Perianal abscess, colorectal carcinoma, ruptured appendicitis or diverticulitis, post-hemorrhoidectomy or rectal mucosal biopsy, etc., have also been implicated [11]. Urological sources include bladder carcinoma, urethral stricture or calculi, indwelling catheters or post-prostate biopsy, etc. [9]. Urological sources were responsible for 25% of our cases, including scrotal abscess in 7 patients; urinary tract infection post-Foley in 2; urethral stricture post-Foley in 1; and benign prostatic hyperplasia post-Foley in 1. The infection source can even arise from the surrounding local skin such as via superficial

Table 5. Complications in 44 patients with Fournier's gangrene

Complication	Number of non-survivors	Number of survivors	Total (No. [%])	*p*
Respiratory failure	8	3	11 (25.0)	<0.001
Renal failure	9	0	9 (20.5)	<0.001
Septic shock	4	3	7 (15.9)	0.018
Pneumonia	3	3	6 (13.6)	0.086
Hepatic failure	4	0	4 (9.1)	<0.001
Disseminated intravascular coagulopathy	2	0	2 (4.5)	0.008
Upper gastrointestinal bleeding	1	0	1 (2.3)	0.062

skin abscess or Bartholin's abscess [12]. The source was dermatologic in 5 patients in our study (bed sore wound in 1 patient, groin wound in 1, contact dermatitis in 1, epidermoid cyst in 1, and post-hysterectomy in 1). Fournier's gangrene might be a complication of operation. In our series, 6 patients developed Fournier's gangrene after operation (3 post-perianal abscess and anal fistula, 2 post-hemorrhoidectomy, and 1 post-hysterectomy). The infection source did not affect mortality in our series; however, another 2 reports concluded that patients with a colorectal source tend to have a worse prognosis [10,13].

Many attempts were made to identify predisposing factors of Fournier's gangrene. Diabetes mellitus was the most common comorbid condition, affecting 40-60% of patients who developed Fournier's gangrene [11]. Chronic alcoholism was believed to be another common comorbid condition, which affects 25 to 50% of cases [3]. Other predisposing factors, such as advanced age, malignancy, and immunocompromised status, have also been reported [14]. In our study, 24 patients (54.5%) were diabetics, 6 patients (13.6%) were hypertensive, 5 patients (11.4%) had chronic liver disease, 4 patients (9.1%) had liver cirrhosis, and 3 patients (6.8%) had malignancy. Whether the presence of diabetes mellitus influences prognosis is still controversial [10,13]. Liver cirrhosis was significantly related to mortality (p=0.009) in our series. Malnutrition and immunocompromised status may be the reasons for high mortality in liver cirrhosis. Although chronic renal insufficiency and uremia were not significantly related to mortality in our series, clinical renal function impairment may be a prognostic factor according to another report [15].

Fournier's gangrene is considered to be a polymicrobial infection [16]. Both aerobic and anaerobic bacteria are usually present, but anaerobes are isolated less frequently. Some patients are infected by one pathogen only (either an aerobe, anaerobe or fungi) [17, 18]. The causative pathogens may have low virulence but act synergistically [16]. Thrombosis of the small vessels, known as obliterative endarteritis, is thought to be a key pathophysiological event [14]. The underlying thrombosis results in a cutaneous and subcutaneous vascular necrosis [3,11]. *E. coli*, *Bacteroides* and *Streptococcus* spp. were the most common organisms in one study [19]. However, *E. coli*, *Streptococcus* spp., *Staphylococcus* and *Enterococcus* were isolated more commonly in other study [15]. In our series, *E. coli*, *Bacteroides fragilis*, *K. pneumoniae*, *Enterococcus* spp., and *P. mirabilis* were the leading 5 pathogens. Positive blood culture was noted in 4 patients in our series but it did not influence prognosis. However, positive blood culture was an adverse prognostic factor in 2 studies [13,20].

We found that survivors had a mean of 1.76 (60/34) surgical debridements compared to 1.4 (14/10) in non-survivors. Numerous reasons may exist for this. We speculate that non-survivors did not live long enough to receive another debridement. Another possibility is that the condition of non-survivors precluded debridement. Another factor that could account for the greater rate of debridement among survivors is the aggressiveness of the surgeon treating the patient.

Additional surgical procedures are sometimes necessary. Temporary fecal diversion can be achieved by colostomy to prevent wound contamination, especially in cases of large complex wounds [16]. Suprapubic cystostomy is sufficient for urinary diversion; however, Foley catheters may be enough in selected patients [19]. Orchiectomy should be considered when severe epididymoorchitis is noted. As in 2 cases in our study, penectomy or vasectomy may be sometimes necessary if the wound area is diffuse.

Reconstruction surgery should be considered once all necrotic tissue has been removed and the wound base is clean. Many factors, such as the patient's age, sexual activity and erectile status, the location of skin loss and the amount of genital skin remaining, etc., should be taken into account [21]. For example, direct closure or a split-thick skin graft may be enough in partial penile skin loss.

Although there are some reports of hyperbaric oxygen therapy in managing Fournier's gangrene, it was not utilized in our series. The report of Pizzorno et al showed no mortality in 11 male patients managed with hyperbaric oxygen therapy [22]. Korhonen et al reported that lack of hyperbaric oxygen therapy is an adverse outcome factor [23]. However, in most series it has not been shown to affect outcome significantly [24,25]. Hyperbaric oxygen therapy should be used as an adjunct to aggressive surgical debridement and broad-spectrum antibiotics, rather than to delay or replace these.

The major complications of Fournier's gangrene (including respiratory failure, renal failure, septic shock, hepatic failure and DIC) were significantly related to mortality in our series. Deviation of hemodynamic homeostasis may be the cause of high mortality [3]. Scar cosmesis and contracture problems are other common complications. Chintamani et al reported a rare case

who developed squamous cell carcinoma in the scar of Fournier's gangrene [26].

In conclusion, Fournier's gangrene is truly a surgical emergency. Both genders can be affected and the mortality is high. In our 44-case series, liver cirrhosis was highly correlated with mortality. Some major complications can also be lethal. The clinical presentation in many patients in early stage may not be prominent. Thus, rapid and accurate diagnosis remains a key component to successful treatment. Fluid, hemodynamic and nutritional support, use of appropriate broad-spectrum antibiotics, and prompt and repeated surgical intervention are the cornerstones of treatment.

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