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Gangrene: The Prognostic Factors and Validation of Severity Index in Fournier's Gangrene

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1. Introduction

Fournier's gangrene (FG) is a fulminant and life-threatening disease characterized by necrotizing fascitis of the perineal and genitourinary area resulting from polymicrobial infection. The polymicrobial organisms cause ascending reactions, activating various proteins and enzymes, leading to platelet aggregation, intravascular coagulation, tissue ischemic tissue change. This disease rapidly progresses, causing thrombosis and irreversible necrosis.

Most of patients had predisposed or concomitant diseases such as diabetes mellitus, alcoholism, hepatic diseases, renal diseases, and cardiac diseases.

It is a surgical emergency and requires prompt surgical debridement in most cases. For the treatment of Fournier's gangrene, aggressive wide necrotic tissue debridement for survival and the proper use of antibiotics, post-operative wound management, and proper reconstruction are required.

High mortality rates in Fournier's gangrene range from 6.3 to 50%, which indicates that the variable outcome of patients with the disease is multifactorial. In general, disease related factors and host-related factors are important prognostic factors.

To investigate clinical features and prognostic factors in patients who underwent the treatments of Fournier's gangrene, Acute Physiology and Chronic Health Evaluation (APACHE) II, and the Fournier's Gangrene Severity Index (FGSI) score which was first reported by Laor et al in 1995 were used and other scoring system. Among them, the FGSI is very useful and it can predict mortality and survival with a high probability for patients with Fournier's gangrene according to many authors. The quantification of the extent of the disease may help determine the outcome more precisely predictions for patients with Fournier's gangrene.

We analyzed 27 patients who underwent treatments due to Fournier's gangrene in our institution and evaluated predictive factors for mortality and survival based on pathogenesis, causative factors, and the subjects of progression. The result of this study showed that sepsis and FGSI of nine points or over at the time of hospitalization were significant risk factors for mortality.

2. History and pathophysiology of Fournier gangrene

2.1 History

Fournier's gangrene was first described by Jean Alfred Fournier (1832-1914) in 1883, a French dermatology/venereologist, a series in which previously healthy young men. He used the term-'**fulminant gangrene**-sudden onset necrotizing disease, rapid progression to gangrene and absence of a definite cause' of the penis and scrotum and his description was based on five young men with scrotal gangrene. Although this disease has been still called Fournier's gangrene to date, its concept has been changed and its causes have been identified in most cases. This condition is described as infective necrotizing fascitis which occurs in perineal, perianal, and genitourinary areas due to polymicrobial organisms regardless of gender and age.

Since Fournier's gangrene was first described, various changes have been made in the definition of the disease and its treatment methods.

2.2 Pathophysiology

Localized infection adjacent to a portal of entry is the inciting event in the development of Fournier gangrene. The polymicrobial organisms cause ascending reactions, activating various proteins and enzymes, leading to platelet aggregation, intravascular coagulation, tissue ischemic change. This disease rapidly progresses, causing thrombosis and irreversible necrosis in perineal and genitourinary areas.

It has been revealed that Fournier gangrene is a polymicrobial infection with an average of 2~4 isolates per case at wound cultures from patients. The bacteria involved act synergistically, via collagenases, hyaluronidases, and other enzymes to invade and destroy fascial planes

Ultimately, an obliterative endarteritis develops, and the ensuing cutaneous and subcutaneous vascular necrosis leads to localized ischemia and further bacterial proliferation. Rates of fascial destruction as high as 2-3 cm/h have been described in some reports. Infection of superficial perineal fascia (Colles fascia) may spread to the penis and scrotum via Buck and Dartos fascia, or to the anterior abdominal wall via Scarpa fascia, or vice versa. Perineal fascia is attached to the perineal body and urogenital diaphragm posteriorly and to the pubic rami laterally, thus limiting progression in these directions. Testicular involvement is rare, as the testicular arteries originate directly from the aorta and thus have a blood supply separate from the affected region.

2.3 Outcome/prognosis

Despite the development of modern intensive care and medical therapy, mortality rate from Fournier gangrene remains still high. The mortality rate for Fournier gangrene widely varies from 30 to 50%.

Prognosis may be affected by various factos, that include disease-related and host-related ones. The outcome of patients with the disease is indicated multifactorial.

Factors associated with high mortality include an anorectal source, advanced age, extensive disease (involving abdominal wall or thighs), shock or sepsis at presentation, renal failure, and hepatic dysfunction. Death usually results from systemic illness, such as sepsis, coagulopathy, acute renal failure, diabetic ketoacidosis, or multiple organ failure.

Most studies were conducted to investigate clinical features and prognostic factors in patients who underwent the treatments of Fournier's gangrene at a single institution. Progression to single-organ or multiorgan failure (MOF, MODF) may occur, usually as a

result of gram-negative sepsis and is typically the cause of death. (Include acute renal failure and adult respiratory distress syndrome).
After recovering from a threatening condition, large scrotal, perineal, penile, and abdominal wall skin defects may require reconstructive procedures. Fatal tetanus associated with Fournier gangrene has been reported in the literature.

3. Clinical feature

3.1 Frequency

Fournier gangrene is relatively uncommon. The true incidence of the disease is unknown. A retrospective case review revealed 1726 cases documented in the literature from 1950 to 1999. An average of 97 cases per year was reported from 1989 to 1998. Poor socioeconomic conditions contribute to development of Fournier's Gangrene. However, regional prevalence and Ethnicity were not identified as relevant factors.

3.2 Age and sex

Mostly male-to-female ratio is mostly approximately 10:1 in large series. Rare reports including women, especially with postpartum perineal necrotizing fasciitis, but, the lower incidence in females may be caused by better drainage of the perineal region through vaginal secretions. Homosexual men may be at a higher risk of contracting Fournier gangrene; especially for infections caused by community-associated methicillin-resistant *Staphylococcus aureus* (MRSA).
Most cases occur in patients aged 30-60 years. When Fournier's gangrene was first described by Alfred Fournier, 'young age and male gender'were identified. The reported age of patients with the disease has progressively increased in the published data. In 1945, It was reported an average age of 40.9 years was reporeted; in 1979, Jones reported 51.3 years; Laor and colleagues reported an average age of 61 years old. In our analysis, we found an almost identical average of 57.3 years.
In our study, the male subjects composed 25 cases (92.6%) and the mean age of the subjects was 52.8 years. The age bracket of patients with Fournier's gangrene has commonly been found to be between 30 and 60 years. In this study, the mean age was 52.8 years and the patients with an age of less than 65 years accounted for 70.4 % of all subjects.
Regarding determinants of survival in older FG patients, it is now known that older patients have a lower survival rate. Clayton *et al*. statistically found that patients who survived were younger statistically than those who died of Fournier's gangrene (52 and 69 years old, respectively). Yilmazlar et al calculated a threshold age of 60 years in the ROC analysis (area under ROC curve: 0.709, 95%CI: 38.5–81.8). Logistic regression analysis identified age as an independent risk factor for mortality in large patients with Fournier's gangrene.

3.3 Predisposition to disease

Many predisposing factors have been reported, including systemic disease such as diabetes mellitus, alcoholism, chronic renal failure, chronic steroid use, malnutrition, HIV infection, and malignancy in FG. Any condition with decreased cellular immunity may predispose to the development of Fournier gangrene theoretically.
In our series, the concomitant diseases included diabetes mellitus in 29.6%; liver cirrhosis and alcoholic liver disease in 14.8% in our study. Diabetes mellitus was the most common comorbidity associated with FG and was present in 50% (24-72) of patients at the time of admission. **Table 1**

		No of Cases	Mean Age (yrs)	Gender	Origin		Fecal diversion (%)	DM (%)	Mortality (%)	FGSI *	
				M:F	Anorectal (%)	Urogenital (%)				**Survival**	**Mortality**
Laor et al	*1995*	30	61.0	NA	16.7	20.0	10.0	30.0	43.3	**6.9 ± 0.9**	**13.5 ± 1.5**
Villanueva-Saenz et al	*2002*	28	57.8	28	89.3	10.7	50.0	64.3	35.7		
Baek et al	*2003*	16	62.0	14:2	75.0	18.8	75.0	62.5	6.3		
Korkut et al	*2003*	45	54.6	NA	57.8	13.3	40.0	55.6	20.0		
Yeniyol et al	*2004*	25	51.7	NA	0.0	0.0	4.0	72.0	24.0	**3.0 ±1.8**	**12.0± 2.4**
Kim SK et al	*2006*	11	60.0	8:3	36.4	0.0	36.4	NA	27.3		
Yanar H et al	*2006*	35	58.6	25:10	17.1	8.6	14.3	45.7	40.0		
Basoglu et al	*2007*	45	54.0	44:1	48.9	2.0	46.7	24.4	8.9		
Corcoran et al	*2008*	68	55.8	54:14	38.2	11.8	26.5	52.9	10.3	**5.1± 3.4**	**10± 4.5**
Erol et al	*2009*	18	57.0	NA	NA	NA	22.2	55.6	22.2	**5.0± 2.9**	**13.5± 2.6**
Yilmazlar et al	*2010*	80	57.0	57:23	40.0	56.3	22.5	57.5	21.3	**4**	**14**
Kim KM et al	***2010***	27	52.8	25:2	59.3	40.7	51.9	29.6	14.8	**4.7± 0.4**	**9.3± 3.2**
mean		35.7	57.3		39.9	15.2	33.3	50.0	22.8	**4.0**	**14.0**

*Fournier's gangrene severity index

Table 1. Clinical feature and Outcome in patients with Fournier's gangrene

Diabetes has always been associated with an increased incidence of FG. Many authors reported the prevalence of diabetes as 50~73 percent, respectively. The high incidence of diabetics in FG was explained by the increased propensity to tissue ischemia caused by small-vessel disease. On the other hand, diabetes is associated with worse outcome and increased mortality, which could be explained by mutifactorial immunological system dysfunction, that included decreased phagocytosis ability, neutrophil dysfunction.
Although this association of unfavorable outcome and underlying diabetes has previously been mentioned, numerous review articles have failed to demonstrate a statistically significant difference.
One of the 15 nondiabetic patients died; however, the mortality rate among diabetics was higher (3 of 12 patients, 25 percent).
For pathogenesis, anorectal diseases were the most frequent causes of the infection.
In our retrospective review of 27 consecutive patients treated for FG at a single institution, factors such as the presence of sepsis, high FGSI and the initial surgical intervention affected outcome in univariate analyses.
The concomitant diseases of Fournier's gangrene have been known to include diabetes mellitus, alcoholism, chronic liver disease, various cancers, and immune suppression.
In this study, diabetes mellitus was found in eight patients; liver cirrhosis and alcoholic liver disease in four patients; and hypertension in nine patients. Bed ridden status due to paraplegia was also found in some patients.

3.4 Causative factors

Anorectal, genitourinary, and dermatologic sources are implicated in the pathogenesis of the disease. Localized infection adjacent to a portal of entry is often the inciting event in the development of Fournier gangrene. **Table 2.**
In men, anal intercourse may increase risk of perineal infection, either from blunt trauma to the area or by spread of anorectal microbes.
In women, septic abortions, hysterectomy, and episiotomy, vulvar or Bartholin gland abscesses are also documented sources.
Poor perineal hygiene or the presence of chronically indwelling catheters, such as in paraplegic patients, poses an increased risk in box sex.

Anorectal
Trauma
Ischiorectal, perirectal, or perianal abscesses
Perianal fistulotomy
Anal fissures; colonic perforations
steroid enemas for radiation proctitis
Rectal cancer
Genito urinary
Trauma
Urethral strictures with urinary extravasation
Urethral catheterization or instrumentation, penile implants
Periurethral infection ; chronic urinary tract infections;
Epididymitis or orchitis
Penile artificial implant, Foreign body

	Hemipelvectomy
	Cancer invasion to external genitalia
	Septic abortion
	Bartholin's duct abscess
	Episiotomy
Dermatologic sources	
	Scrotal furuncle
	Genital toilet (scrotum)
	Blunt perineal trauma ; intramuscular injections, genital piercings
	Perineal or pelvic surgery /Inguinal herniography
Idiopathic; more than 75%	

Table 2. Causative Factors in Patients With Fournier's Gangrene

3.5 Clinical presentation

In general, most patients were reported to visit hospitals due to itching or discomfort of the external genitals. It was reported to take approximately 5 days from symptom expression to visiting the hospital. In this study, most patients suffered from perianal/scrotal swelling and pain as a main symptom. In addition, fever and chill, perianal/scrotal necrosis, purulent discharge, and voiding difficulty were also accompanied. The mean duration from the initiation of symptom expression to visiting the hospital was 99.8 hours, i.e. 4-5 days. When the patients were divided into anorectal and genitourinary groups and the characteristics of the subject groups were compared, no significant difference except for fecal diversion was found between the two groups. For anorectal diseases, fecal diversion is thought to be frequently conducted as wound management was difficult due to fecal contamination and the surgery site was perianal area.

All patients had at least one of the following early symptoms or signs: perianal or perineal pain, hyperemia, and fever.

The clinical presentation of the disease starts with a prodromal period of genital discomfort or pruritus, followed by genital erythema, swelling, crepitation and revealing subcutaneous gas formation.

Skin overlying the affected region may be normal, erythematous, edematous, cyanotic, bronzed, indurated, blistered, and/or frankly gangrenous in progression.

However, skin appearance often underestimates the degree of underlying disease. A feculent odor may be present secondary to infection with anaerobic bacteria. The gangrenous process will lead to drainage of the affected areas and demarcation between viable and dead tissue. The extent of the involved area may reach the abdominal wall, axilla, and thighs.

Crepitus may be present, but its absence does not exclude the presence of *Clostridium* species or other gas-producing organisms. Systemic symptoms (eg, fever, tachycardia, and hypotension) may be present.

In Fournier gangrene, obtain a thorough review of systems, including history of diabetes, alcohol abuse, cancer, colorectal or urogenital disease or surgery, steroid use, sexual history, and HIV status.

Sepsis at presentation was found in seven cases (25.9%). The mean duration from the expression of the symptoms to visiting the hospital was 99 hours.

3.6 Bacteriology

Both anaerobic and aerobic organisms isolated from wound cultures have been cited as an important bacteriologic principle in Fournier's gangrene. Paty and Smith found *E. coli, Bacteroides,* and streptococci to be the most common organisms.

Laor *et al.* determined the most common organisms were *E. coli* and *Streptococcus* species, with *Staphylococcus* and *Enterococcus* more commonly isolated than *Bacteroides*.

The mean microbial number of two was identified in microbial culture tests. *Streptococcus. species* was the most common microbial organism, accounting for 48.1%. *Enterococcus* and *Escherichia Coli* were found in 29.6% and 25.9%, respectively in our study. **Table 3.**

Bacterial organism	%
Streptococcus. Species	48.1
Enterococcus	29.6
Escherichia Coli	25.9
Unspecified G(+) rods	25.9
Klebsiela pneumoniae	18.5
Unspecified G(-) rods	14.8
Bacteroides species	1.1
Unspecified G(+) cocci	11.1
Coagulase-negative staphylococcus	7.4
Enterobacter species	7.4
Acinetobacter	3.7
Candida albicans	3.7

Table 3. Causative Bacterial organism of FG

The mean numbers of isolated microorganism per patient was reported to be four, and *Escherichia Coli* and Bacteroides were reported to be the most common microbes. In addition, Proteus, Staphylococcus, Pseudomonas, and Klabsiella were also reported. According to the results of this study, one to three microbes were identified. *Streptococcus species* and *Enterococcus* were common microbes, and Klebsilla and Bacteroides were also commonly identified as shown in previous study results.

Wound culture results from our series were similar to prior reported results with predominantly polymicrobial infections. It reveals a polymicrobial infection with an average of 4 isolates per case. *Streptococcus* species is the predominant aerobe, and *Bacteroides* is the predominant anaerobe.

Other microflora includes *Proteus, Staphylococcus, Enterococcus,* aerobic and anaerobic *Streptococcus, Pseudomonas, Klebsiella,* and *Clostridium.* Incidence of methicillin-resistant *Staphylococcus aureus* (MRSA) may be increase in being mentioned in literature

FG has always been considered a surgical emergency. Some articles have so far highlighted the poor prognosis of FG in patients with a delay in presentation and treatment.
In most studies the course of these patients was characterized by a more advanced disease necessitating more aggressive debridement with fecal diversion.
There are limitations in the design and interpretation of this study. First, we still have relatively few cases that were treated during a long period. Second, the retrospective study, the extent of the disease in terms of surface area and other prognostic variables were not included.

4. Differential diagnoses

- Balanitis, Epididymitis, Orchitis
- Testicular Torsion
- Hernias, Hydrocele
- Cellulitis, Gas Gangrene and
- Necrotizing Fasciitis

Cellulitis is used to indicate a nonnecrotizing inflammation of the skin and subcutaneous tissues, a process related to acute infection that does not involve the fascia or muscles. Cellulitis was classically considered to be an infection without formation of abscess and without purulent drainage or ulceration.
Gas gangrene, a subset of necrotizing myositis, is an emergent infectious disease. Organisms in the spore-forming clostridial species, including *Clostridium perfringens*, *Clostridium septicum,* and *Clostridium novyi,* cause most of the cases. A nonclostridial form is caused by a mixed infection of aerobic and anaerobic organisms. Disease has rapid onset of myonecrosis with muscle swelling, severe pain, gas production, and sepsis.
For more than a century, many authors have described soft tissue infections. Their occurrence has been on the rise because of an increase in immunocompromised patients with diabetes mellitus, cancer, alcoholism, vascular insufficiencies, organ transplants, HIV, or neutropenia.
Necrotizing fasciitis can occur after trauma or around foreign bodies in surgical wounds, or it can be idiopathic, as in scrotal or penile necrotizing fasciitis.
Necrotizing fasciitis has also been referred to as hemolytic streptococcal gangrene, Meleney ulcer, acute dermal gangrene, hospital gangrene, suppurative fascitis, and synergistic necrotizing cellulitis. Fournier gangrene is a form of necrotizing fasciitis that is localized to the scrotum and perineal area.
Necrotizing fasciitis is a progressive, rapidly spreading, inflammatory infection located in the deep fascia, with secondary necrosis of the subcutaneous tissues. Because of the presence of gas-forming organisms, subcutaneous air is classically described in necrotizing fasciitis. The speed of spread is directly proportional to the thickness of the subcutaneous layer. Necrotizing fasciitis moves along the deep fascial plane, rapidly progress. They require aggressive treatment to combat the associated high morbidity and mortality.

5. Diagnostic methods

5.1 Laboratory studies

The following studies are indicated in patients Fournier gangrene:

- CBC with differential count

- Electrolytes, BUN, creatinine, blood glucose levels: Acidosis with hyperglycemia or hypoglycemia may be present. Dehydration occurs as the disease progresses.
- ABG sampling to provide a more accurate assessment of acid/base disturbance
- Blood and urine cultures
- Disseminated intravascular coagulation (DIC) panel (coagulation studies, fibrinogen/fibrin degradation product levels) to find evidence of severe sepsis
- Cultures of any open wound or abscess

5.2 Imaging studies Fournier gangrene

Diagnosis of Fournier gangrene is primarily is based on clinical findings. Sensitivities and specificities of different radiologic modalities are not established.

Conventional radiography

Conventional radiography may demonstrate soft-tissue gas collections (manifest as areas of hyperlucency), even4 before they are clinically apparent. Scrotal tissue edema may be observed on radiographs. Absence of air on plain films does not exclude the diagnosis.

Ultrasonography

Ultrasonography may reveal other causes of acute scrotal pain, including intratesticular injury, scrotal cellulitis, epididymoorchitis, testicular torsion, and inguinal hernia.
Gas in the scrotal wall is the "sonographic hallmark" of Fournier gangrene. Air may be appreciated in perineal and/or perirectal areas. Scrotal wall edema may be seen. Testes and epididymides are usually normal.

Computerized tomography

Findings include soft-tissue and fascial thickening, fat stranding, and soft-tissue gas collections. CT scans defines the extent of the disease more specifically. CT scan often identifies the underlying cause of the infection (eg, perirectal abscess). This modality may assist in surgical planning.

MRI

MRI use is not well described in the literature. MRI may define soft-tissue pathology more distinctly than CT scan but should not delay operative intervention if the diagnosis is highly suspected.

6. Treatment & management

6.1 Resuscitation & early care

The following treatment is indicated in patients with Fournier gangrene:

- Initially, aggressive resuscitation in anticipation of surgery - Airway management if indicated, crystalloid replacement if dehydrated or displaying signs of shock
- Supplemental oxygen, intravenous (IV) access, and continuous cardiac monitoring

Early, broad-spectrum antibiotics are indicated, including the following:

- Ampicillin/sulbactam
- Ticarcillin/clavulanate
- Piperacillin/tazobactam
- Penicillinase-resistant penicillin, aminoglycoside, and metronidazole or clindamycin
- Coverage for methicillin-resistant *Staphylococcus aureus* (MRSA), such as vancomycin

Tetanus prophylaxis is indicated if soft-tissue injury is present.
Irrigation with superoxidized water and packing with gauze soaked with zinc peroxide and hydrogen peroxide may be helpful.
Surgical consultation is imperative. Immediate urologic, colorectal consultation is mandatory.

6.2 Medication summary

The goals of pharmacotherapy in Fournier gangrene are to reduce morbidity and to control the infection.

Antibiotics

Initiate early broad-spectrum antibiotics as soon as possible. Providing coverage for gram-positive, gram-negative, aerobic, and anaerobic bacteria is essential. Penicillins and beta-lactamase inhibitors or triple antibiotics are potential choices.

Vancomycin

Ampicillin-sulbactam sodium

Ticarcillin and clavulanate potassium

Piperacillin/tazobactam

Gentamicin

Metronidazole

Clindamycin

6.3 Immunizations

Patients with fatal tetanus associated with Fournier gangrene have been documented in literatures. Patients with noncurrent tetanus status require immunization in the emergency department.

Diphtheria and tetanus toxoid (Decavac)

Tetanus toxoid is manufactured by first culturing *Clostridium tetani* and then detoxifying the toxin with formaldehyde. This toxoid is commonly combined with diphtheria toxoid, and both serve to induce production of serum antibodies to toxins produced by the bacteria.

6.4 Surgical management

Aggressive surgical debridement may have a positive effect on survival. Although Clayton e*t al.* and Laor *et al.* suggested that the extent of disease was not predictive of outcome, Spirnak *et al.* associated the greater mortality rate for patients who underwent more frequent operations to the presence of a greater extent of the disease. Others found that the extent of body surface area involved in the necrotizing process was directly related to mortality.
Surgeon or urologist may order further diagnostic tests in patients with Fournier gangrene, including cystourethroscopy, retrograde urethrography, sigmoidoscopy, barium enema, tissue biopsy, and examination under anesthesia.
Urinary and/or fecal diversion (eg, suprapubic catheterization, ileostomy or colostomy) may be required depending on the source of infection.[5]

If the initial facility does not have the capability to provide operative therapy in a timely fashion, arrange for **transfer** once the patient has been stabilized and resuscitative efforts have begun. Patients often require a multidisciplinary team, including urologist, general surgeon, and team for intensive care. Transfer to a tertiary facility may be required if these resources are not available at the initial facility.

Multiple surgical debridements in the operating room may be required to effectively remove all necrotic tissue. Patients with Fournier gangrene undergo an average of 2-4 operative procedures during their initial hospitalization. In our study, 17 cases (63%), required surgical treatments of fecal or urinary diversion. Orchiectomy and/or penectomy are rarely required.

Reconstructive surgery due to wide wound defects was required in 11 cases (40.7%). The mean length of stay in hospital was 70.8 days.

Hyperbaric oxygen therapy (HBO) has been used as an adjuvant to surgical and antimicrobial therapy, especially in patients for whom conventional treatment failed, in those with documented clostridial involvement, or in those with myonecrosis or deep tissue involvement. HBO is postulated to reduce systemic toxicity, prevent extension of necrotizing infection, and inhibit growth of anaerobic bacteria. However, in one series, there was actually a trend toward increased mortality in patients undergoing HBO therapy. Decisions regarding hyperbaric therapy must be made on an individual basis and should be an adjuvant to debridement and antimicrobial therapy.

7. Outcome and prognosis

There is no consensus on which clinical variables predict a poor outcome in FG. Retrospective studies have implicated increasing age, diabetes mellitus, delay in presentation / treatment and extent of involvement (BSA, Body Surface area). While BSA was suggestive of a poor prognosis of all the operative characteristics examined, only lower extremity or abdominal wall involvement was associated with inpatient mortality.

Previous reports suggest that older, debilitated or bedridden patients with multiple comorbidities presenting with advanced FG are more likely to have poor outcomes. Factors associated with an improved prognosis include age younger than 60 years, localized clinical disease, absence of systemic toxicity, and sterile blood cultures.

Four patients (14.8%) died during the treatment; three patients due to sepsis and one patient who had scrotal abscess accompanied with incarcerated inguinal hernia died due to renal failure. However, our results indicate that with, age, comorbidity, use of early aggressive therapy and time to presentation do not affect prognosis.

For host related factors, the novel scoring system should be validated through other prospective studies and independent observations and can be applied in clinical practice

The number of patients with FG is significant and the mortality ranges between 15 to 50%, showing various prognoses. Higher mortality was reported to be seen in the cases of anal diseases, the elderly, diabetes mellitus, invasion to the abdominal wall and the thigh, higher FGSI, shock and sepsis at the time of hospitalization, and accompanying hepatic failure and renal failure.

The FGSI was developed to help clinicians predict outcome in patients with FG. **Table 4**. A score of 0-4 is assigned to each of the following parameters: temperature; heart rate; respiratory rate; serum sodium, potassium, bicarbonate, and creatinine levels; hematocrit; and WBC count. Its modified scoring system also has been developed, which has been shown to aid in prognosis. **Table 5.**

	High Abnormal Values				Normal	Low Abnormal Values			
Physiologic Variables / Point assignment	+4	+3	+2	+1	0	+1	+2	+3	+4
Temperature (C) [	>41	39–40.9	—	38.5–38.9	36–38.4	34–35.9	32–33.9	30–31.9	<29.9
Heart rate (bpm)	>180	140–179	110–139	—	70–109	—	55–69	40–54	<39
Respiratory rate	>50	35–49	—	25–34	12–24	10–11	6–9	—	<5
Serum potassium (mmol/L)	>7	6–6.9	—	5.5–5.9	3.5–5.4	3–3.4	2.5–2.9	—	<2.5
Serum sodium (mmol/L)	>180	160–179	155–159	150–154	130–149	—	120–129	110–119	<110
Serum creatinine (mg/100 ml) (x2 for acute renal failure)	>3.5	2-3.4	1.5-1.9	—	0.6-1.4	—	<0.6	—	—
Hematocrit (%)	>60	—	50–59	46–49	30–45	—	20–29	—	<20
White blood count (91000/mm3)	>40	—	20–39.9	15–19.9	3–14.9	—	1–2.9	—	<1
Serum bicarbonate, (venous) (mmol/L)	>52	41–51	—	32–40	22–31	—	18–21	15–17	<15

Table 4. Fournier's gangrene severity index

Laor et al reported that a FGSI score greater than 9 indicated a 75% probability of mortality while a score of 9 or less was associated with a 78% probability of survival. This cutoff point has subsequently been validated in other small retrospective series. However, Tuncel et al of 20 men with FG demonstrated no association between FGSI and mortality, and stated that specific metabolic parameters (serum albumin and alkaline phosphatase), predisposing factors and disease extent should be assessed together to predict treatment outcome and survival.

In our study, the mean FGSI was 9.25 in patients who had died and 4.69 in patients who survived. Of the factors affecting the mortality, sepsis and FGSI of 9 points or over at the time of hospitalization were statistically significant. **Table 6**.

The morbidity of FG has been gradually increasing and its causal diseases and causal microbes have also varied. For the treatment of Fournier's gangrene, active wound managements such as early diagnosis, wide excision for necrotic tissue, the proper use of antimicrobials, and continuous postoperative aseptic dressing are required.

Variables	+4	+3	+2	+1	0	+1	+2	+3	+4
a. Physiological parameters									
Temperature (C) [	>41	39–40.9	—	38.5–38.9	36–38.4	34–35.9	32–33.9	30–31.9	<29.9
Heart rate	>180	140–179	110–139	—	70–109	—	55–69	40–54	<39
Respiratory rate	>50	35–49	—	25–34	12–24	10–11	6–9	—	<5
Serum potassium (mmol/L)	>7	6–6.9	—	5.5–5.9	3.5–5.4	3–3.4	2.5–2.9	—	<2.5
Serum sodium (mmol/L)	>180	160–179	155–159	150–154	130–149	—	120–129	110–119	<110
Serum creatinine (mg/100 ml) (x2 for acute renal failure)	>3.5	2-3.4	1.5-1.9	—	.6-1.4	—	<0.6	—	—
Hematocrit (%)	>60	—	50–59	46–49	30–45	—	20–29	—	<20
White blood count (91000/mm3)	>40	—	20–39.9	15–19.9	3–14.9	—	1–2.9	—	<1
Serum bicarbonate, (venous) (mmol/L)	>52	41–51	—	32–40	22–31	—	18–21	15–17	<15
b. Dissemination score									
Fournier's gangrene confined to the urogenital and/or anorectal region, add ''1''									
Fournier's gangrene confined to the pelvic region, add ''2''									
Fournier's gangrene extending beyond the pelvic region, add ''6''									
c. Age score									
Age ≥60 years, add ''1''									
Age <60 years, add ''1''									

UFGSI = A+B+C

Table 5. The Uludag Fournier's gangrene severity index Yilmazlar T et al (2007)

		Total N=27	Mortality N=4(%),	Odds ratio(95%, CI)	p
Age (years)	<65	19	3 (15.8)	0.762 (0.067-8.665)	1.000
	≥65	8	1 (12.5)		
Duration	<48 hours	9	1 (11.1)	1.600(0.142-18.000)	1.000
of symptoms	>48 hours	18	3(16.7)		
Infection source	Anorectal	16	2 (12.5)	1.556 (0.185-13.108)	1.000
	Genitourinary	11	2 (18.2)		
Sepsis	Present	7	3 (42.9)	14.250 (1.162-174.801)	*0.042*
	Absent	20	1 (5.0)		
FGSI*	≤9	24	2 (8.3)	22.000(1.334-362.916)	*0.049*
	>9	3	2 (66.7)		
DM or Liver	Present	12	3 (25.0)	4.667 (0.418-52.121)	0.294
disease	Absent	15	1 (6.7)		
No of	<2	9	3 (33.3)	0.118 (0.010-1.359)	0.093
Debridement	≥2	18	1 (5.6)		
Diversion	Performed	17	2 (11.8)	0.533 (0.063-4.531)	0.613
	Not performed	10	2 (20.0)		

*FGSI, Fourier's gangrene severity index

Table 6. Prognostic factors for mortality of Fournier's gangrene *Kim KM et al 2010*

	Survival vs. Nonsurvival (N)	p	References
Age (years)	63 vs. 17	*0.002*	*Yilmazlar et al 2010*
FGSI(median)		*<0.001*	
Extent of the disease (Grade I,II, III)		*<0.001*	
Need for ICU		*<0.001*	
Need for Ventilator		*<0.001*	
Length of Hospital stay (days, median)		*0.002*	
RR	14 vs. 4	*0.003*	*Erol et al 2009*
Tempaerature		*0.046*	
Median BSA*		*0.018*	
Charlson Comorbidity Index		*0.008*	
Life expectancy for 10 y (%)		*0.008*	
RR	61 vs. 7	*0.021*	*Corcoran et al 2008*
Serum bicarbonate		*0.001*	
FGSI		*0.006*	
Serum lactate		*0.001*	
Serum calcium		*0.032*	
Time to consult	19 vs. 6	*0.002*	*Yeniyol et al 2004*
BSA		*0.0001*	
FGSI		*0.0001*	
Sepsis	23 vs. 4	*0.042*	*Kim et al 2010*
FGSI* ≤9 vs. >9		*0.049*	

BSA _ body surface area.

Table 7. Prognostic factors for mortality of Fournier's gangrene

Aggressive and early surgical débridement continues to be the mainstay of treatment of FG in most series. It was reported that the number of operative débridements negatively affects survival, speculating that patients requiring multiple débridements had greater extent of disease, were less healthy at baseline and had progressed to systemic sepsis despite aggressive surgical therapy. Factors confounding the significance of the number of débridement necessary for disease control among survivors include total surface area involved, variation in the extent of the initial resection and whether the patient is healthy enough to survive multiple procedures

The result of our study showed that sepsis and FGSI of nine points or over at the time of hospitalization were statistically significant as factors affecting mortality. The patients included in the aforementioned criteria could show poor prognoses such as DIC, acute renal failure, acute renal failure, and multiorgan failure.

If necessary, hyperbaric oxygen therapy can be helpful, and furthermore, reconstruction surgery may be necessary later.

In this study, FG was investigated in relatively many cases at a single institution compared to other studies conducted in Korea. Further studies with a larger subject population will be required.

8. Conclusion

Despite the development of modern intensive care and medical therapy, Fournier's gangrene remains a fulminant and life-threatening disease, mortality rates have improved as a result of advances in surgical and critical care. Early diagnosis and surgical treatment of Fournier's gangrene are required.

Comprehensive evaluation of metabolic and physiological parameters, predisposing factors, and the extent of disease are also essential for early diagnosis and treatment.

There is no current consensus regarding the use of individual patient admission characteristics or laboratory values as prognostic indicator, sepsis at presentation and lower extremity/abdominal wall involvement were associated with disease severity and inpatient mortality in large series.

The FGSI remains a simple method of assessing severity of presentation and predicting outcome in this complex patient population.

Poor prognoses were seen in the cases of sepsis and FGSI of nine points or over at the time of hospitalization. Our results support previous findings that a FGSI threshold of 9 is a sensitive and specific predictor of mortality during initial assessment. Therefore, the careful observation of vital signs and active treatments are required to treat Fournier's gangrene.

9. References

Ayan F, Sunamak O, Paksoy SM et al (2005) Fournier's gangrene: a retrospective clinical study on forty-one patients. ANZ J Surg 75:1055–1058

Baek JH, Yoon SJ, Oh JH. (2003) Surgical management of Fournier's gangrene.J Korean Soc Coloproctol 19:349-53.

Baskin LS, Carroll PR, Cattolica EV, et al. (1990) Necrotising soft tissue infections of the perineum and genitalia: bacteriology, treatment and risk assessment. *Br J Urol*. 65:524-529.

Basoglu M, Ozbey I, Atamanalp SS et al. (2007) Management of Fournier's gangrene: review of 45 cases. Surg Today 37:558–563

Clayton MD, Fowler JE, Sharifi R. (1990) Causes, presentation and survival of fifty-seven patients with necrotizing fasciitis of the male genitalia. *Surg Gynecol Obstet*. 170:49-53.

Corcoran AT, Smaldone MC, Gibbons EP, et al. (2008) Validation of the Fournier's gangrene severity index in a large contemporary series.*J Urol*. 180:944-948.

Eke N (2000) Fournier's gangrene: a review of 1726 cases. Br J Surg 87:718–728

Ersay A, Yilmaz G, Akgu¨n Y et al (2007) Factors affecting mortality of Fournier's gangrene: review of 70 patients. ANZ J Surg 77:43–48

Fournier JA. (1883) Gangrene foudroyante de la verge. Medecin Practique 4:589–97.

Kim KM, Seong SH, Won DY, Ryu H, Kim IY (2010) The Prognostic Factors and Severity Index in Fournier's Gangrene. J Korean Soc Coloproctol 26:29-33.

Kim SK, Park JI, Joo YT, Park ST, Ha WS, Hong SC, et al. (2006)Fournier's gangrene: clinical analysis of 11 patients. J Korean Surg Soc 71: 274-8.

Korkut M, Ic¸o¨z G, Dayangac¸ M et al (2003) Outcome analysis in patients with Fournier's gangrene: report of 45 cases. Dis Colon Rectum 46:649–652

Laor E, Palmer LS, Tolia BM, Reid RE, Winter HI (1995) Outcome prediction in patients with Fournier's gangrene. J Urol 154:89–92

Lin E, Yang S, Chiu AW et al (2005) Is Fournier's gangrene severity index useful for predicting outcome of Fournier's gangrene? Urol Int 75:119–122

Palmer LS, Winter HI, Tolia BM, Reid RE, Laor E (1995) The limited impact of involved surface area and surgical de´bridement on survival in Fournier's gangrene. Br J Urol 76:208–212

Quatan N, Kirby RS. (2004) Improving outcomes in Fournier's gangrene. *BJU Int*. 93:691.

Spirnak JP, Resnick MI, Hampel N. Fournier's gangrene: report of 20 patients. *J Urol*. 1984;131:289-292.

Tahmaz L, Erdemir F, Kibar Y et al (2006) Fournier's gangrene: report of thirty-three cases and a review of the literature. Int J Urol 13:960–967

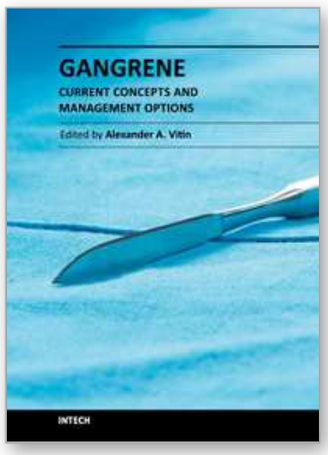
Tuncel A, Aydin O, Tekdogan U et al (2006) Fournier's gangrene: three years of experience with 20 patients and validity of the Fournier's gangrene severity index score. Eur Urol 50:838– 843

Villanueva-Sa´enz E, Martinez Herna´ndez-Magro P, Valde´s Ovalle M et al (2002) Experience in management of Fournier's gangrene. Tech Coloproctol 6:5–10

Yanar H, Taviloglu K, Ertekin C et al (2006) Fournier's gangrene: risk factors and strategies for management. World J Surg 30:1750–1754

Yeniyol CO, Suelozgen T, Arslan M, Ayder AR (2004) Fournier's gangrene: experience with 25 patients and use of Fournier's gangrene severity index score. Urology 64:218–222

Yilmazlar T, Ozturk E, Alsoy A, Ozguc H (2007) Necrotizing soft tissue infections: APACHE II score, dissemination, and survival. World J Surg 31:1858–1862



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Gangrene is the term used to describe the necrosis or death of soft tissue due to obstructed circulation, usually followed by decomposition and putrefaction, a serious, potentially fatal complication. The presented book discusses different aspects of this condition, such as etiology, predisposing factors, demography, pathologic anatomy and mechanisms of development, molecular biology, immunology, microbiology and more. A variety of management strategies, including pharmacological treatment options, surgical and non-surgical solutions and auxiliary methods, are also extensively discussed in the bookâ€™s chapters. The purpose of the book is not only to provide a reader with an updated information on the discussed problem, but also to give an opportunity for expert opinions exchange and experience sharing. The book contains a collection of 13 articles, contributed by experts, who have conducted a research in the selected area, and also possesses a vast experience in practical management of gangrene and necrosis of different locations.

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