A STUDY ON FOURNIER'S GANGRENE

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Certificate

This is to certify that this dissertation entitled "**A study on Fournier's Gangrene"** submitted by Dr. M.SIVARAJ, appearing for M.S Branch I (General surgery) Degree Examination in March 2008 is a bonafide record of work done by him under my direct supervision and guidance in partial fulfillment of regulations of the Tamilnadu Dr. M.G.R. Medical University, Chennai, Tamilnadu, India.

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CONTENTS

INTRODUCTION:

Fournier's gangrene first described by Fournier in 1764 is a necrotizing fascitis involving the genital, perianal or perineal regions. It can involve contiguous areas like the lower urinary tract, anus, rectum and colon. It is a fulminating, rapidly spreading infection that leads to thrombosis of subcutaneous blood vessels, resulting in gangrene of the overlying skin.

It affects all ages and has been reported in both sexes and various etiological factors have been noted. This is more commonly seen in middle aged having immunosuppressive disorder like diabetes mellitus, malignancy and chronic alcoholism.

The basic treatment involves active resuscitation, prompt excision of all non-viable tissue, limitation and abolition of any infective process, antibiotics and occasional anatomical reconstruction. Orchidectomy may be required. Methods for reconstruction of the scrotum include burying the testes in the thigh or in the abdomen, split-thickness skin grafting and wide surgical debridement with delayed suturing.

Early recognition of this condition with prompt surgical treatment and early antibiotics form the cornerstone in it is management. The course of disease is rapid and the disease can be lethal if not treated urgently

Aim of the Study:

Health negligence is common in our society. Minor injuries or infection of genitalia can lead to life threatening discover. The aim of the study is

- To study the epidemiological aspect of Fournier gangrene
- To study the clinico pathological profile of patients of Fournier gangrene
- To study the management of Fournier gangrene with due importance to the active hemodynamic stabilization and urgent surgical debridement.
- To study the outcome and prognosis of Fournier gangrene and
- To draw attention towards early appropriate management of Fournier gangrene.

Patients and Methods:

The study was conducted in department of surgery, Government Royapettah hospital, Kilpauk Medical College over a period of 3 years.

44 cases of Fournier gangrene were analyzed. Diagnosis was established from the patient's history and clinical examination. Age, etiology, predisposing factors, extent of involvement, bacteriological findings, lab investigations, therapeutic response, surgical outcome, and hospital stay were evaluated.

OBSERVATION:

44 male patients admitted with diagnosis of Fournier gangrene were included in the study. The following are the observations;

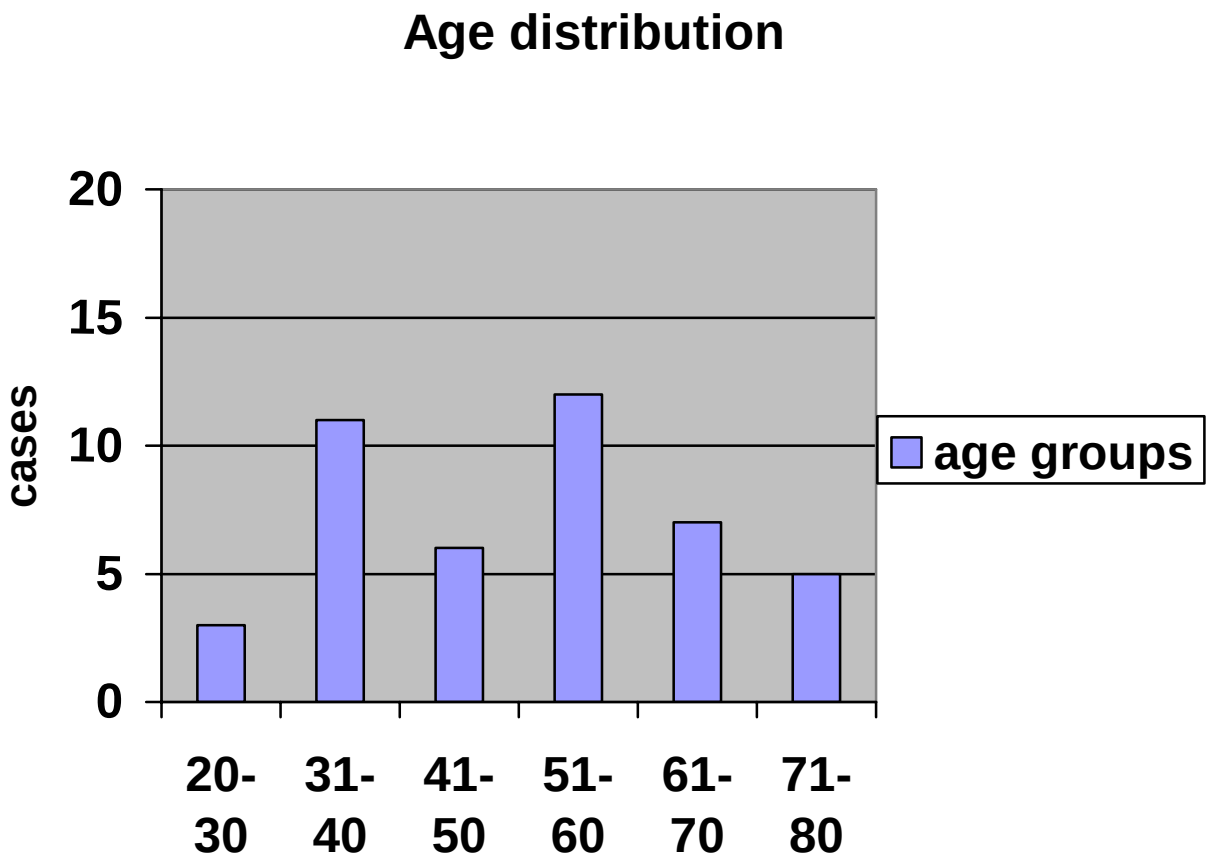
Age distribution:

The age of patients ranged from 28 to 80 yrs, with majority of patients in 51 to 60 yrs age group. This is in accordance with the reports that the mean age of patients appears to have increased from 40 yrs in cases reported before 1945 to 50 yrs or more in revert series of study. [2]

The mean age in this study is 30.8 years.

Age groups	No. of cases	percentage
20-30	3	6.8%
31-40	11	25%
41-50	6	13.6%
51-60	12	27.2%
61-70	7	15.9%
71-80	5	11.4%

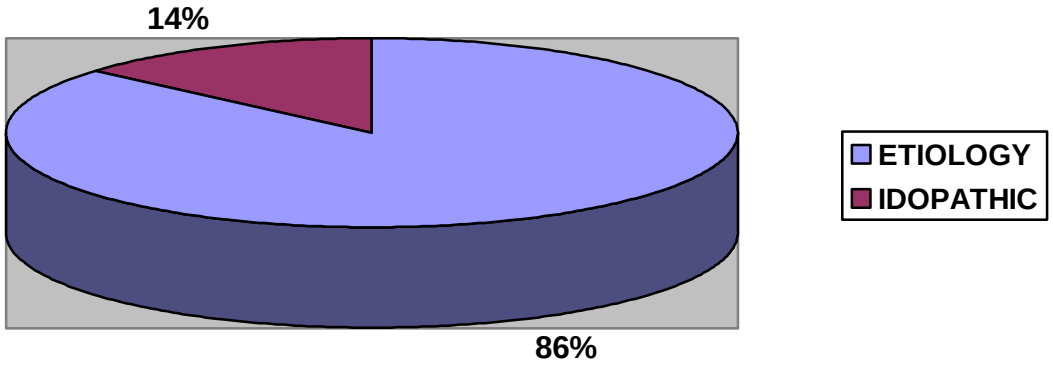
About 27.2% cases were in the age group of 51-60 years. Only 6% were below 30 years of age.


Age distribution
20
15
10
5
0
cases
age groups
20-30
31-40
41-50
51-60
61-70
71-80

ETIOLOGY AND PREDISPOSING FACTORS:

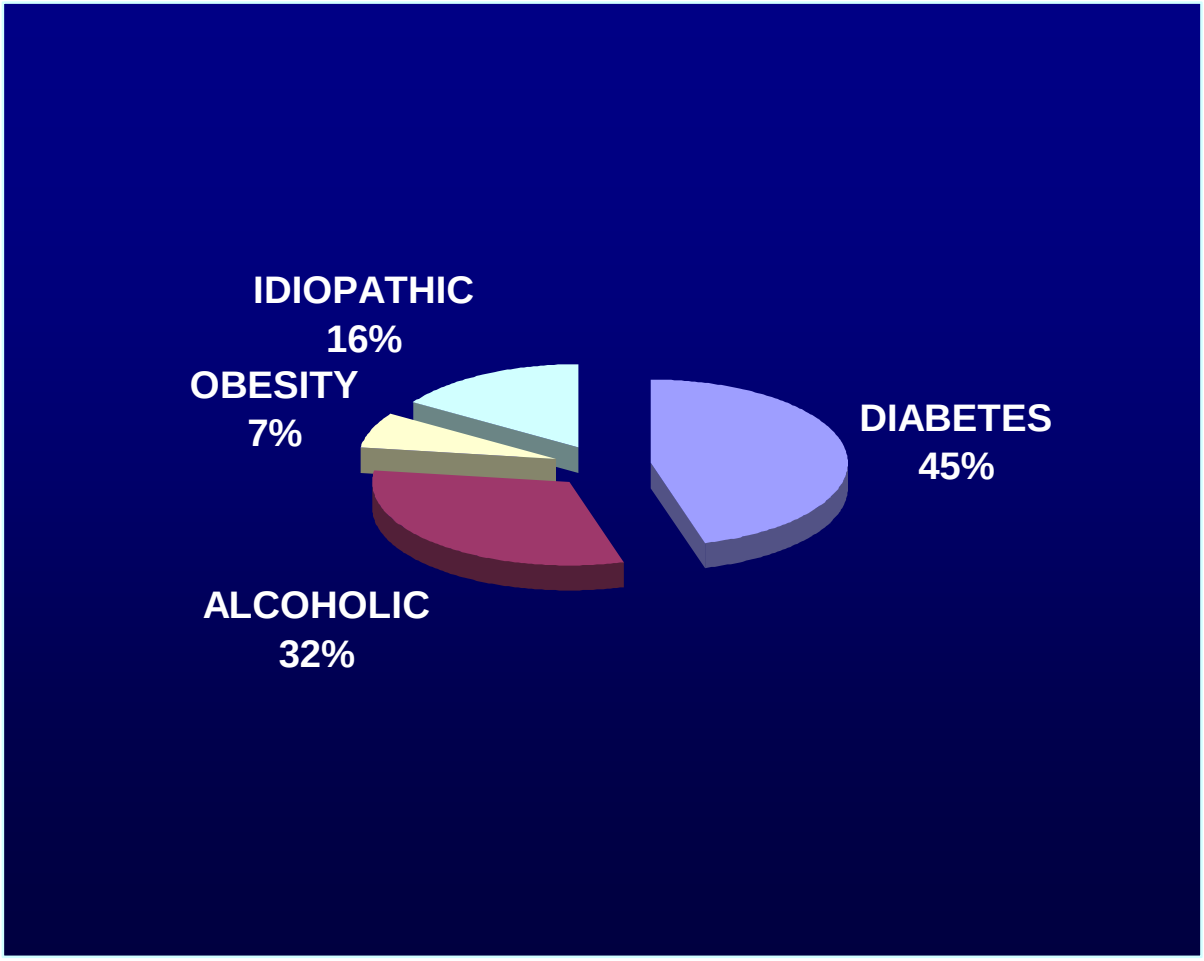
Although Fournier was unable to identify a cause in his initial series, most present day clinician would agree that the condition is rarely idiopathic. This reflects both increased understanding of condition and a more rigorous approach to investigation of established cases. When the cause is not found, it is likely that the portal of entry has been overlooked.[15]

In this study, the period of time spent before presentation to our hospital ranged from 1-8 days. 38 patients had etiological and predisposing factors and only in 6 patients, no apparent cause was found.



14 patients gave history of trauma as initiating factor. The most striking changes in presentation over revert years is that victims of disease are now almost always recognized to have an underlying systemic disorders.[38]

In this study, 16 of patients were diagnosed diabetes mellitus i.e. about 36% and 14 patients gave history of chronic alcoholism. Morbid obesity was noted in 3 patients.



Thus, in this study, Fournier gangrene has an identifiable cause in approximately 88% of cases, and the co morbid disease that compromise the immune system has been implicated as necessary predisposing factors for the development of Fournier gangrene.[12]

The common predisposing co morbidities are:

- Diabetes mellitus
- Alcoholism
- Immunosuppresion
- Liver disease
- Obesity

CLINICAL:

In this study, majority of patients were admitted with history of predisposing symptoms of fever and malaise, which varied between 1-8 days. There was history of trauma in 14 patients and 2 patients had history of painful pile mass. The patients complained of intense genital pain and tenderness, with progressive erythema of the overlying skin. Obvious gangrene of portion of genitalia and turbid discharge was noted in majority of patients.

The systemic effects varied from local tenderness with no toxicity to florid septic shock. Toxic features were more significantly noted in elderly patients, those with delayed consultation and those with co morbid disease like diabetes, alcoholism etc.

The extent of gangrene varied in each patient. Being a spreading infective process, the gangrene was either involving the part or whole of scrotum and some with extension to the penis, perineum, thigh and anterior abdominal wall. In this study, gangrene was confined to scrotum in 38 patients, with extension into penis in 4 patients. 2 patients had spreading extensive gangrene to the perineum, medial aspect of thigh.

Fluctuation, soft tissue crepitations, localized tenderness; turbid, foul smelling discharge was noted in approximately 80% of patients.

LAB STUDIES:

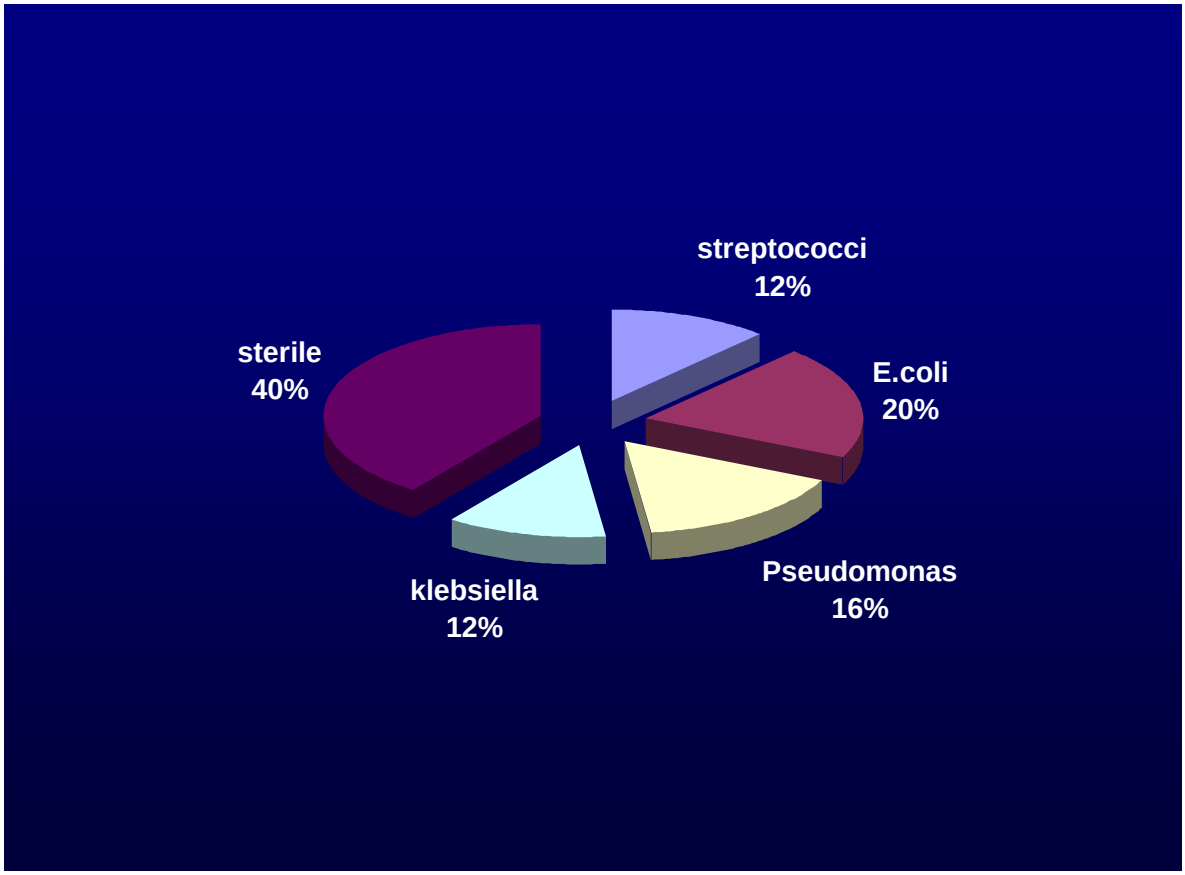
After complete history and physical examination, blood investigation to evaluate possible electrolyte disturbances, to look for laboratory evidence of dehydration (urea/creatinine) and to determine glucose intolerance.

Approximately 80% of patients had raised urea/creatinine values, suggestive of pre renal azotemia, on the day of admission.

Blood sugar values >120 mg% was noted in 16 patients. Complete blood count was done to assess the immunologic stress induced by infective process.

Chest x ray, ECG was taken to assess the cardio respiratory status and to assess exacerbation of a co morbid condition. ECG changes suggestive of coronary artery disease were observed in 6 of the patients admitted.

Wound swab and pus culture and sensitivity were sent for all patients. In the study, the culture was sterile in 10 cases. This could be attributed to inadequate antibiotic therapy received from outside before coming to our hospital or due to fastidious anaerobes. The organism isolated were Streptococci [3], E.coli [5], Pseudomonas [4], and Klebsiella [3].


streptococci
12%
E.coli
20%
Pseudomonas
16%
klebsiella
12%
sterile
40%

Treatment:

The mainstay of treatment of patients with Fournier gangrene is three fold.

Firstly, patients were actively resuscitated especially, when they presented with signs of systemic toxicity or septic shock.

Secondly, patients were commenced on antibiotics. Almost for all patients intravenous cefotaxime and metronidazole was administered.

Thirdly, patients were shifted to emergency operation theatre and under IV sedation, prompt debridement was done in almost 90% patients. Patient in a lithotomy position, all visible necrotic skin and affected subcutaneous tissues were excised aggressively. The extent of excision was until fresh oozing was noted in edges of cut tissues and where separation of skin and subcutaneous tissue do not occur easily.[3]

In patients with extensive involvement of scrotum, penis, thigh, urinary catheterization was done. Thus, soiling of wound was avoided and urinary output was monitored.

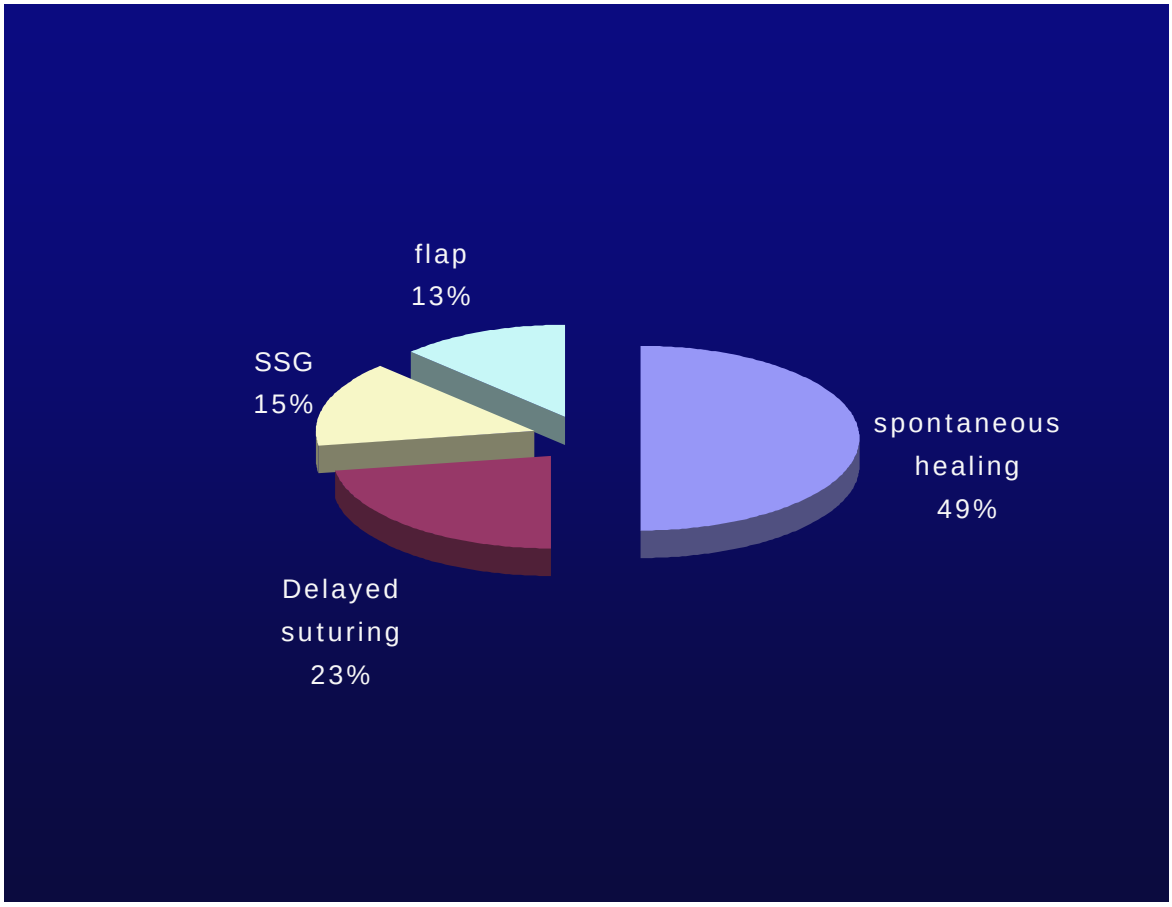
Debridement resulted in exposure of testicles in patients with extensive involvement of scrotum. In 4 patients, Orchidectomy was done. In these patients there was history of trauma to the scrotum and testis viability was doubtful.

Regular cleaning and dressing with hydrogen peroxide and povidone iodine solution was done for all patients, until healthy granulation appeared. ***fig.6, 7***

Healing and spontaneous closure of wound was achieved in 20 patients. Delayed suturing was done in 9 patients. Fig 8

With large ulcer area, split skin grafting was done. Fig 9 Mesh skin grafting was done which offered the advantages of covering a large area with a small sheet of donor skin and avoids the serous collections beneath the graft. Penile raw area was also grafted similarly. In 2 patients, circumcision was done.

In patients with exposed testicle, medial thigh implantation of testis was done in 4 cases and in 1 patient medial thigh flap fig 10 was done a reconstructive procedure.[8, 50]


flap
13%
SSG
15%
spontaneous
healing
49%
Delayed
suturing
23%

Complication:

Major systemic complication of acute renal failure was observed in 3 patients, gastro intestinal bleeding in 1 patients and heart failure in 2 patients. 4 patients died of septic shock despite vigorous resuscitation. These patients presented late to our hospital with extensive gangrene and septic shock with raised renal parameters.

Complications of severe acute illness (e.g., line sepsis, bed sores, pneumonia, atelectasis} were also noted and managed appropriately.

Outcome:

- Mortality rates: 9%
- The following are the poor prognostic factors noted:
    - Age
    - Delay in presentation and treatment
    - Diabetes mellitus
    - Liver disease/alcoholism
    - Immunosuppresion
    - Extent of involvement

Outcome:

Outcome of Fournier gangrene in different studies: [23]

Study	country	Number of cases	Mortality Rate (%)	Mean duration of hospital stay	% of survivors not in need of skin graft
Kouadio et al	Ivory coast	30	27	45	100
Brissiand et al	Senegal	44	34	42	48
Clayton et al	Chicago	57	18	48	_
Palmer et al	New York	30	43	41	-
Study in GRH	Chennai	44	9	31.5	66

GRH-Government Royapettah Hospital

RESULTS:

The following are the results of my study of Fournier gangrene;

- The median time elapsed between the onset of infection and consultation is 3.3 days.
- Majority of patients were from low socio economic status
- The peak incidence is among the age group of 50-60 years
- The port of entry and predisposing factors were identified in 16 and 31 patients respectively.
- The major predisposing factors are diabetes mellitus and alcoholism.
- Raised urea/creatinine ratio suggestive pre renal azotemia was noted in more than 80% patients.
- The predominant microbial agents isolated are E.coli and pseudomonas. More than 40% of cases, pus culture is sterile.
- All patients were treated with antibiotics and early debridement.
- Healing was achieved without skin graft in 16 patients.
- Secondary suturing was done in 9 patients.
- Skin grafting done in 6 patients. Flap cover was done in 5 patients.
- Percentage of patients not in need of skin grafting was 66%.
- Orchidectomy was done in 4 patients.
- 4 Patients died from septic shock.

- Mean duration of hospital stay was 31 days. In patients with poor prognostic factors, the duration of stay was longer.
- Delayed consultation, shock at presentation, predisposing factors like diabetes, alcoholics, and liver disease gave poor prognosis.

DISCUSSION:

Fournier gangrene is a necrotizing infection that involves the soft tissues of the male genitalia. In modern-day vernacular, Fournier gangrene is a specific form of necrotizing fascitis, a general term introduced in 1951 by Wilson to describe infection of soft tissue that involves the deep and superficial fascia, regardless of location.

Originally, the term Fournier gangrene was used to describe idiopathic gangrene of the genitalia; however, it has also has been used to describe most soft tissue necrotizing infections of the perineum, independent of cause. Modern-day use of the term Fournier gangrene should be restricted to describe infections that primarily involve the genitalia. The indiscriminate use of this eponym makes comparing the results of clinical series or defining a reliable occurrence rate difficult.

HISTORICAL BACKGROUND:

Jean Alfred Fournier, a Parisian dermatologist and venereologist in 1883 was the first to be associated with this condition in a specific region of the body, namely scrotum. His initial description was based on 5 young men with scrotal gangrene. The cardinal points of that description included (1) sudden onset in a healthy young man, (2) rapid progression to gangrene and (3) absence of a definite cause.

1764: Bauriene

1924: Meleney (streptococcal gangrene)

1952: Wilson (necrotizing fascitis)

1990s: "Flesh-eating" bacteria

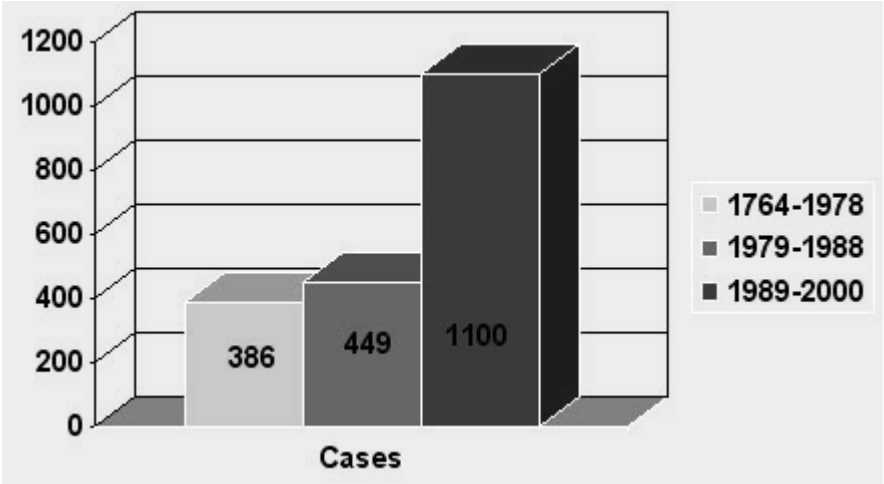
Although Jean Alfred Fournier gave the condition its eponymous name in 1883, Baurienne first described Fournier's gangrene over 100 years previously in 1764. Meleny in 1924, while in China, described a more generalized form of the disease and termed it "streptococcal gangrene". In the 1990s sensational medical journalists dramatized the whole process of necrotizing fascitis by associating it with flesh-eating bacteria.

EPIDEMIOLOGY:

Epidemiology
· Sex- Male: Female = 10:1

The epidemiology has changed from the original description in that the disease is no longer restricted to young men but may affect a wide age range from neonates to the very elderly. Fifty-five cases have been reported in the pediatrics literature, two-thirds of who were younger than 3 months.

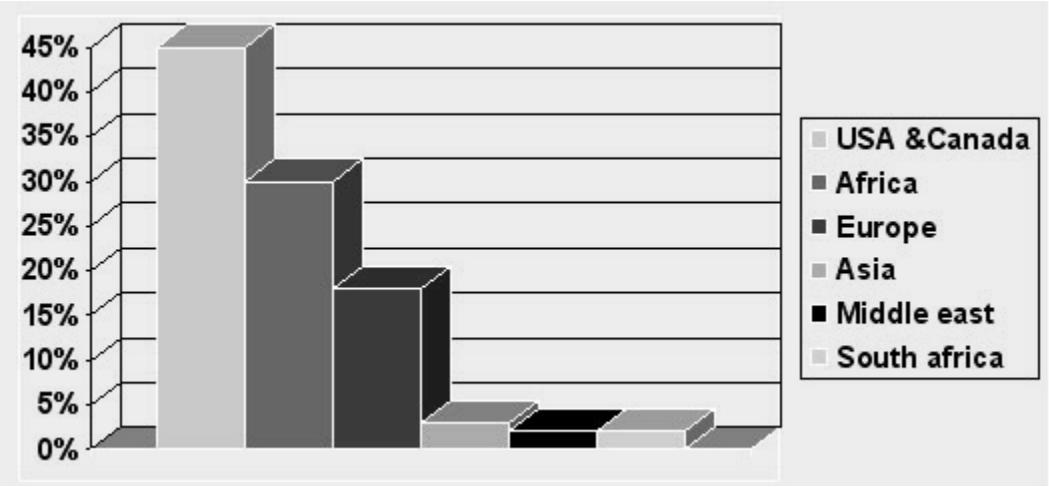
The mean age of patients appears to have increased from 40 years in cases reported before 1945 to 50 years in more recent series. Though this disease process is more common in males, there are also several reports of genital gangrene occurring in women.



The chart above shows the there has been an increase in the number of cases reported in the literature over the last decade. This probably is because of better recognition of this condition and an increase in the reporting of this condition in the medical literature.

There has been some suggestion that poor socioeconomic conditions contribute to the development of Fournier's gangrene. Though a report from Europe stated that the disease is not so frequent in the civilized world,

Fournier's gangrene does occur in affluent as well as poor communities, as evidenced by many reports from affluent regions of the USA and Europe.



There also have been claims that the condition is especially prevalent in Africa and Asia, and the pattern of this disease in Africa and India differs from that in Europe and America. Skin as the source of sepsis is more common in the developing world and the mortality rate is much lower. It has been speculated, without proof, that there may be a strong resistance to infection in the Negro race or that the organisms involved in Africa lack sufficient virulence for more severe sepsis.

Associated disorder
· Diabetes: 40 - 60% Chronic alcoholism: 25 - 50%
· Immunosuppresion

The most striking change in presentation over the recent years is that victims of the disease are now almost always recognized to have an underlying systemic disorder. The most common associations are diabetes and chronic alcoholism. Immunosuppresion, either after organ transplantation or caused by chemotherapy for malignant disease, has also been associated with an increased risk.

With the emergence of HIV, a new group of patients at risk has been recognized in both in Africa and the developed world, and Fournier's gangrene has been reported as a presenting sign of undiagnosed HIV infection.

ETIOLOGY:

Although originally described as idiopathic gangrene of the genitalia, Fournier gangrene has an identifiable cause in approximately 95% of cases. The necrotizing process commonly originates from an infection in the anorectum, the urogenital tract, or the skin of the genitalia.

- Anorectal causes include infection in the perianal glands, manifesting as a consequence of colorectal injury or as a complication of colorectal malignancy, colonic diverticulitis, or appendicitis.
- Urogenital tract causes include infection in the bulbourethral glands, urethral injury, iatrogenic injury secondary to urethral stricture manipulation, or lower urinary tract infection.
- Dermatologic causes include hidradenitis suppurativa, ulceration from scrotal pressure, trauma, intentional trauma (skin popping or piercing), or complications from surgery.
- Other causes, although less common, include Fournier gangrene developing as a consequence of bone marrow malignancy (acute promyelocytic leukemia, acute nonlymphoid leukemia, and acute myeloblastic leukemia), systemic lupus erythematosus, Crohn’s disease, or HIV infection. Additionally, Fournier gangrene may result from iatrogenic or noniatrogenic injury in the perineum.

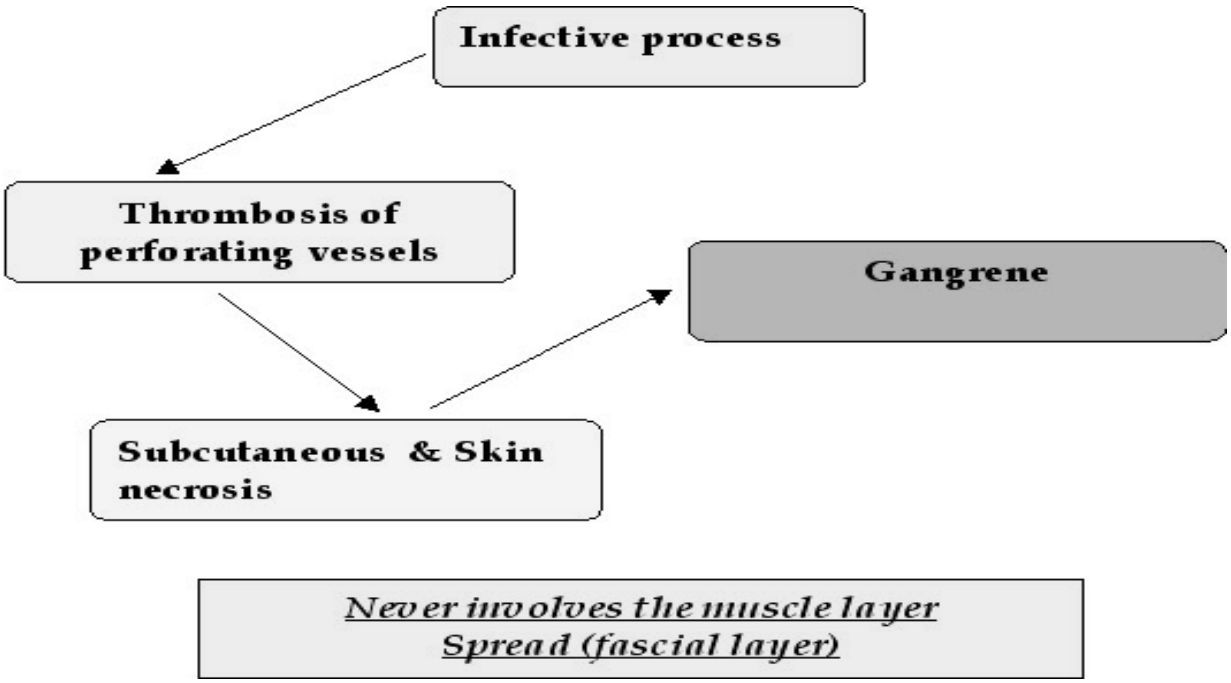
Co morbid diseases that compromise the immune system have been implicated as necessary predisposing factors for the development of Fournier gangrene. The following are common predisposing co morbidities:

- Diabetes mellitus (cited most often)
- Morbid obesity
- Cirrhosis
- Vascular disease of the pelvis
- Malignancies
- High-risk behaviors implicated as promoters of Fournier gangrene (e.g., alcoholism, intravenous)
- Immune suppression from systemic disease or steroid administration.

Portal of entry		
Urogenital (45%) Periurethral infections Urethral strictures Bladder carcinoma Indwelling catheter Traumatic catheterisation Epididymo-orchitis Prostatic biopsy	Anorectal causes (33%) Perianal abscess Complications of: *Colorectal cancers* *Haemorrhoidectomy* *Anal dilatation* *Rectal biopsy* *Appendicitis* *Diverticulitis* *Rectal perforation* *Crohn's disease*	Cutaneous (21%) Occult trauma Complications: *Vasectomy,* *Orchidectomy,* *Herniorraphy* *Superficial skin abscess*

PATHOPHYSIOLOGY:

Pathology



The following are pathognomonic findings upon pathologic evaluation of the involved tissue:

- Necrosis of the superficial and deep fascial planes
- Fibrinoid coagulation of the nutrient arterioles
- Polymorphonuclear cell infiltration
- Microorganisms identified within the involved tissues
- Air in the perineal tissues

Infection represents an imbalance between (1) host immunity, which is frequently compromised by one or more of the above co morbid systemic processes, and (2) the virulence of the causative microorganisms. The etiologic factors allow the portal for entry of the microorganism into the perineum, the compromised immunity provides a favorable environment to initiate the infection, and the virulence of the microorganism promotes the rapid spread of the disease.

Microorganism virulence results from the production of toxins or enzymes that create an environment conducive to rapid microbial

multiplication. In a 1924 series of Chinese men with necrotizing infections, Meleney reported that streptococcal species were the predominant organisms recovered from cultures. Meleney attributed the necrotizing infection to this sole organism; however, subsequent clinical series have emphasized the multiorganism nature of most cases of necrotizing infection, including Fournier gangrene. Presently, recovering only streptococcal species is unusual; rather, streptococcal organisms are cultured along with as many as 5 other organisms.

The following are common causative microorganisms:

- Streptococcal species
- Staphylococcal species
- Enterobacteriaceae species
- Anaerobic organisms
- Fungi

Microbiology	
Polymicrobial (Aerobic + anaerobic) Type 1 infections Type 2 infections	Common organisms: E. Coli*, P. Mirabilis, K. Pneumoniae, Bacteroides* Streptococci*, Staphylococci*, Peptostreptococci, Clostridia, & Pseudomonas * Frequently isolated organisms

Most authorities believe the Polymicrobial nature of this disease is necessary to create the synergy of enzyme production that promotes rapid multiplication and spread of the infection.

For example, one microorganism might produce the enzymes necessary to cause coagulation of the nutrient vessels. Thrombosis of these nutrient vessels reduces local blood supply; thus, tissue oxygen tension falls.

The resultant tissue hypoxia allows growth of facultative anaerobes and microaerophilic organisms. These latter microorganisms, in turn, may produce enzymes (e.g., lecithinase, collagenase), which lead to digestion of fascial barriers, thus fueling the rapid extension of the infection. The fascial necrosis and digestion are hallmarks of this disease process; this is important to appreciate because it provides the surgeon with a clinical marker of the extent of tissue involvement.

Specifically, if the fascial plane can be separated easily from the surrounding tissue by blunt dissection, it is quite likely to be involved with the ischemic-infectious process; therefore, any such dissected tissue should be excised. Far-advanced or fulminate disease can spread from the fascial envelopment of the genitalia throughout the perineum, along the torso, and, occasionally, into the thighs.

CLINICAL FEATURES:

Clinical features

· TRIAD: severe pain + swelling + fever

· Bullae; Crepitus (50 to 62%)

· Gangrene

· "Dirty dishwater fluid"

Cutaneous signs >> Tip of an iceberg

The hallmark of Fournier gangrene is intense pain and tenderness in the genitalia.

The clinical course usually progresses through the following phases:

1. Prodromal symptoms of fever and lethargy, which may be present for 2-7 days
2. Intense genital pain and tenderness that is usually associated with edema of the overlying skin
3. Increasing genital pain and tenderness with progressive erythema of the overlying skin
4. Dusky appearance of the overlying skin; subcutaneous crepitations
5. Obvious gangrene of a portion of the genitalia; purulent drainage from wounds

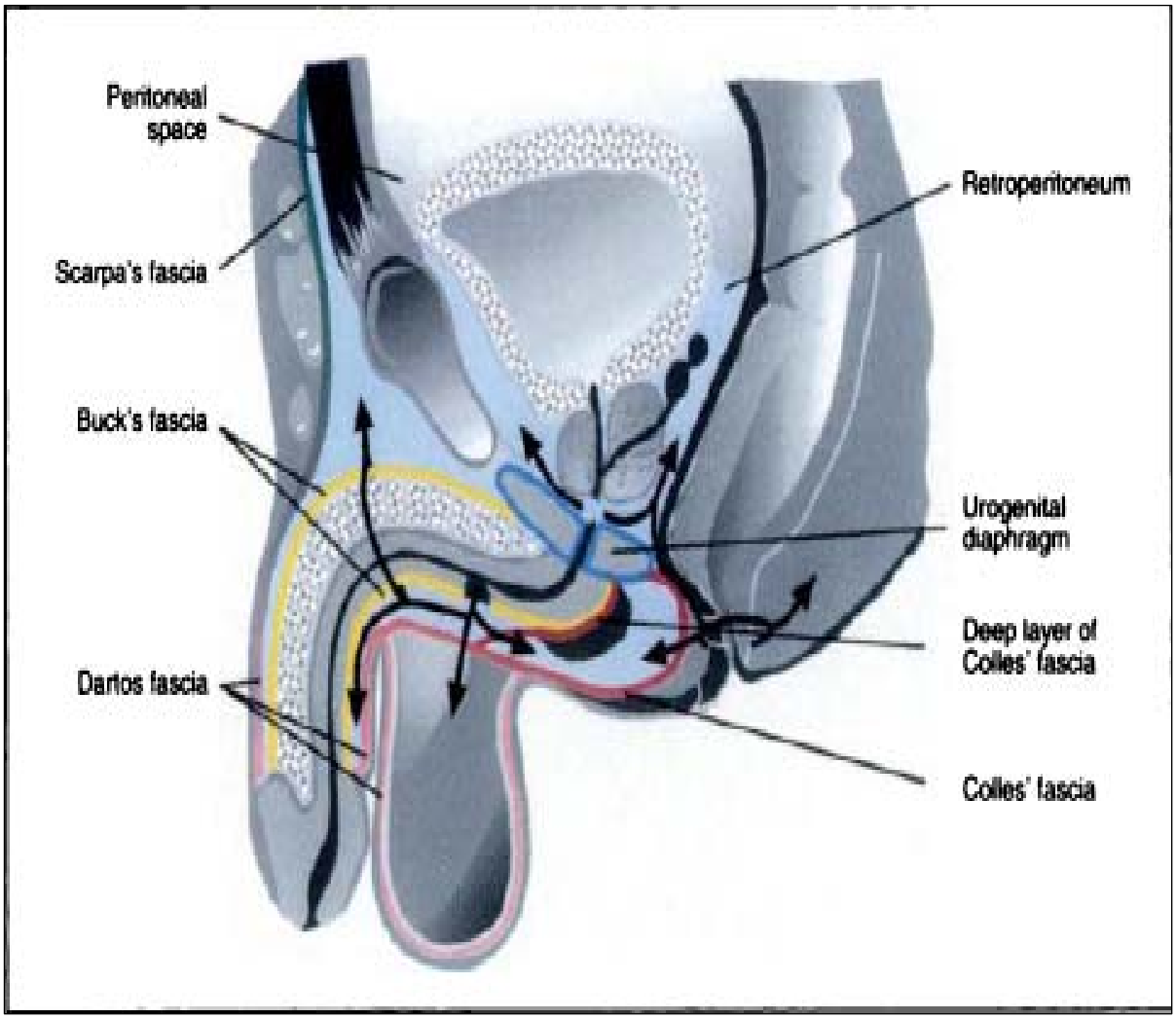
The systemic effects of this process vary from local tenderness with no toxicity to florid septic shock. In general, the greater the degree of necrosis, the more profound the systemic effects. The typical patient is an elderly male in his sixth or seventh decade of life with co morbid diseases; females are not immune to this disease but are affected much less frequently.

RELEVANT ANATOMY:

The complex anatomy of the male external genitalia influences the initiation and progression of Fournier gangrene. This infectious process involves the superficial and deep fascial planes of the genitalia. As the microorganisms responsible for this infection multiply, infection spreads along the anatomical fascial planes, often sparing the deep muscular structures and, to variable degrees, the overlying skin. This phenomenon has implications for both initial debridement and subsequent reconstruction. Therefore, a working knowledge of the anatomy of the male lower urinary tract and external genitalia is critical for the clinician treating a man with Fournier gangrene.

Skin and superficial fascia

Because Fournier gangrene is predominately an infectious process of the superficial and deep fascial planes, understanding the anatomic relationship of the skin and subcutaneous structures of the perineum and abdominal wall is important.


Peritoneal space
Scarpa's fascia
Buck's fascia
Dartos fascia
Retroperitoneum
Urogenital diaphragm
Deep layer of Colles' fascia
Colles' fascia

The skin cephalad to the inguinal ligament is backed by Camper fascia, which is a layer of fat-containing tissue of varying thickness and the superficial vessels to the skin that run through it. Scarpa fascia forms another distinct layer deep to Camper fascia. In the perineum, Scarpa fascia blends into Colles fascia (also known as the superficial perineal fascia), while it is continuous with Dartos fascia of the penis and scrotum.

Several important anatomic relationships should be considered. A potential space between the Scarpa fascia and the deep fascia of the anterior wall (external abdominal oblique) allows for the extension of a perineal infection onto the anterior abdominal wall. Superiorly, Scarpa and Camper fascia coalesce and attach to the clavicles, ultimately limiting the cephalad extension of an infection that may have originated in the perineum. Colles fascia is attached to the pubic arch and the base of the perineal membrane, and it is continuous with the superficial Dartos fascia of the scrotal wall.

The perineal membrane is also known as the inferior fascia of the urogenital diaphragm and, together with Colles fascia, defines the superficial perineal space. This space contains the membranous urethra, bulbar urethra, and bulbourethral glands. In addition, this space is adjacent to the anterior

anal wall and ischiorectal fossae. Infectious disease of the male urethra, bulbourethral glands, perineal structures, or rectum can drain into the superficial perineal space and can extend into the scrotum or onto the anterior abdominal wall up to the level of the clavicles.

Vascular supply to the skin of the lower abdomen and genitalia

Branches from the inferior epigastric and deep circumflex iliac arteries supply the lower aspect of the anterior abdominal wall. Branches of the external and internal pudendal arteries supply the scrotal wall. With the exception of the internal pudendal artery, each of these vessels travels within Camper fascia and, therefore, can become thrombosed in the progression of Fournier gangrene.

If thrombosed, the viability of the skin of the anterior scrotum and perineum is jeopardized. Often, the posterior aspect of the scrotal wall supplied by the internal pudendal artery remains viable and can be used in the reconstruction following resolution of the infection.

Penis and scrotum

The contents of the scrotum, namely the testicles, epididymides, and cord structures, are invested by several fascial layers distinct from the Dartos fascia of the scrotal wall. Again, several important anatomic relationships should be considered.

The most superficial layer of the testis and cord is the external spermatic fascia, which is continuous with the external aponeurosis of the superficial inguinal ring (external abdominal oblique). The next deeper layer is the internal spermatic fascia, which is continuous with the transversalis fascia. A deep fascia termed Buck fascia covers the erectile bodies of the penis, the corpora cavernosa, and the anterior urethra. Buck fascia fuses to the dense tunica albuginea of the corpora cavernosa, deep in the pelvis. The fascial layers described in this section do not become involved with an infection of the superficial perineal space and can limit the depth of tissue destruction in a necrotizing infection of the genitalia. The corpora cavernosa, urethra, testes, and cord structures are usually spared in Fournier gangrene, while the superficial and deep fascia and the skin are destroyed.

INVESTIGATION:

Diagnosis
CLINICAL DIAGNOSIS
"Finger test" / "Frozen section"
Plain X-ray: Air in soft tissues
US: Scrotal wall thickening
Subcutaneous Air / Peri-testicular fluid
CT: Delineates the extent of necrosis accurately
Defines the cause

Lab Studies:

- Complete history and physical examination
    - Direct particular attention to palpation of the genitalia and perineum and to the digital rectal examination.
    - Fluctuance, soft tissue crepitations, localizing tenderness, or occult wounds in any of these sites should alert the examiner to possible Fournier disease.

- Chemistry panel: Perform these tests to evaluate possible electrolyte disturbances, to look for laboratory evidence of dehydration (elevated BUN/creatinine ratio), and to determine the presence of glucose intolerance (either because of preexisting diabetes or sepsis-induced metabolic disturbance).

- Blood tests

    - Obtain a complete blood cell count to assess the immunologic stress induced by the infectious process, check the adequacy of the red blood cell mass, and evaluate the potential for sepsis-induced thrombocytopenia.

    - Blood samples should be drawn for culture to assess the presence of septicemia.

    - A coagulation profile (prothrombin time, activated partial thromboplastin time, platelet count, and fibrinogen level) is helpful to look for sepsis-induced coagulopathy.

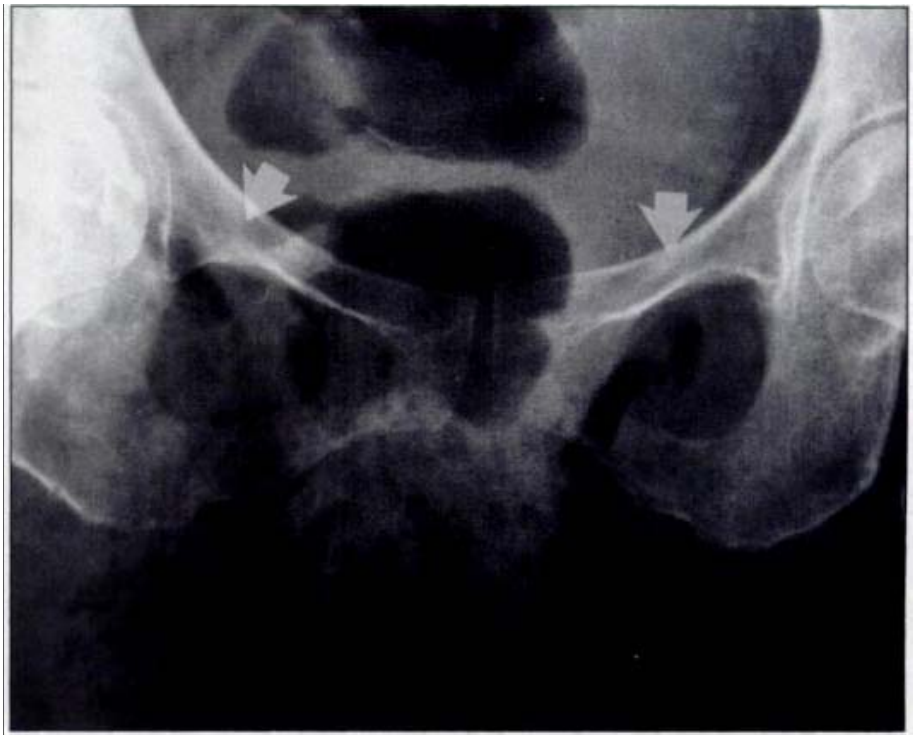
    - Consider type and screen if surgical exploration is undertaken.

- Other: Any test deemed necessary to assess exacerbation of a co morbid condition (e.g., ECG and cardiac enzyme evaluation in patients with coronary artery disease) is warranted.

Imaging Studies:

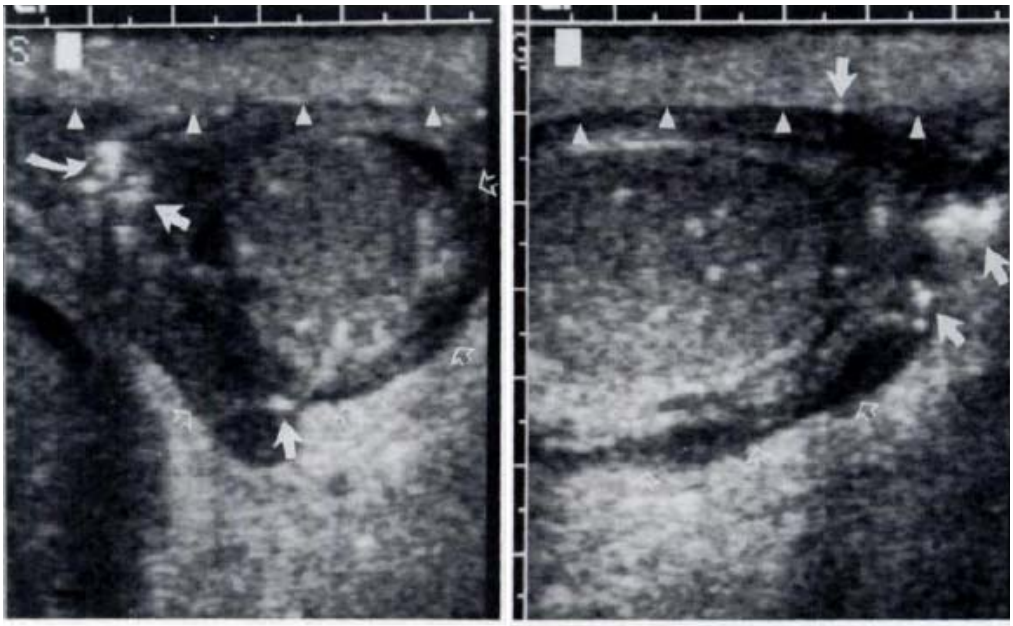
- Radiography

  Consider whether the clinical examination findings are inconclusive. The presence of gas within the soft tissues is detected more sensitively by imaging modalities than physical examination.



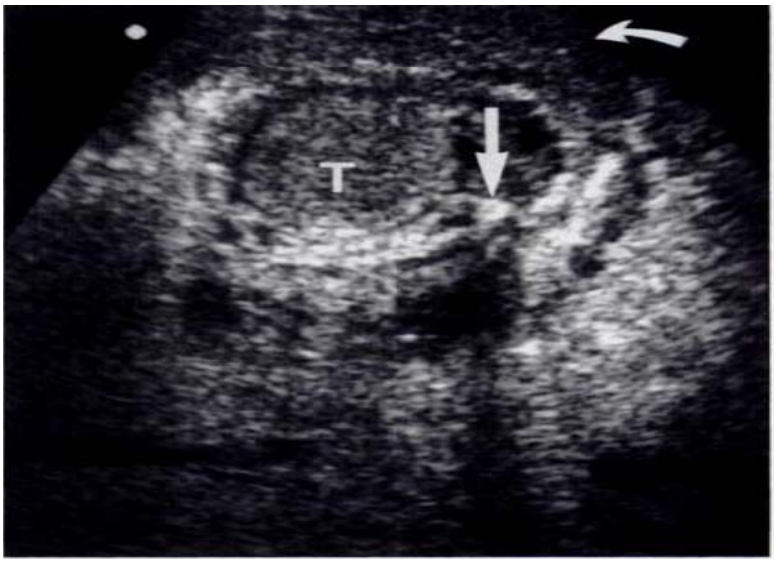
- Demonstration of soft tissue gas or detection of subcutaneous crepitations is an absolute indication for surgical exploration.
- Plain film radiography should be the initial imaging study. It may reveal moderate-to-large amounts of soft tissue gas or foreign bodies.

- Ultrasonography
  - This can be used to detect fluid or gas within the soft tissues. In addition, ultrasonography can assess the blood flow to the testes if testicular torsion is in the differential diagnosis.
  - The drawback of ultrasonography is the need for direct pressure on the involved tissue; patients with Fournier gangrene probably will not tolerate this procedure.



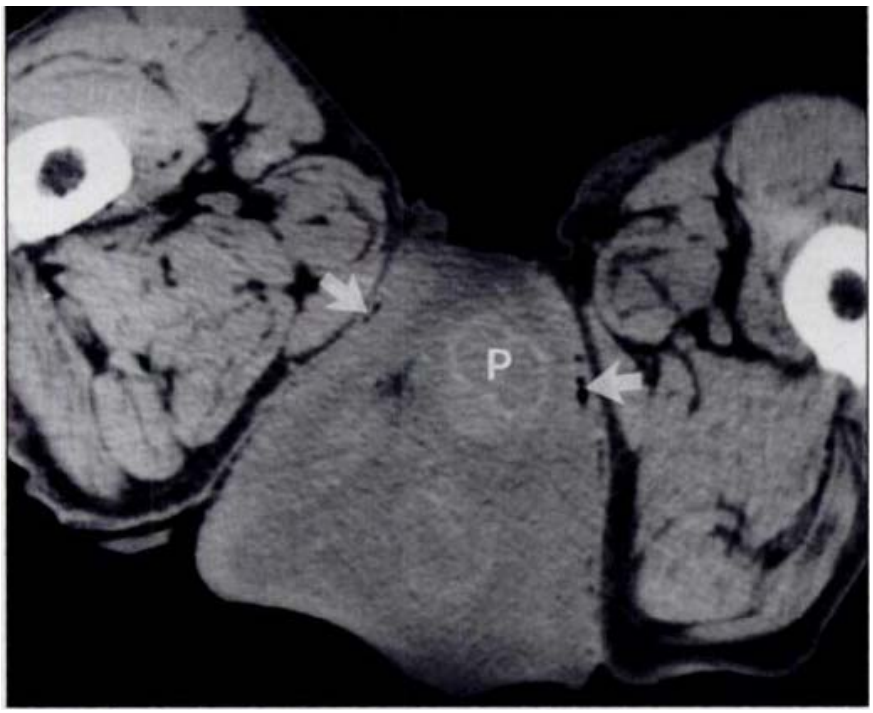
a. b.

Transverse (a) and longitudinal (b) USG scrotum demonstrates skin thickening (arrow heads), peritesticular fluid (open arrow), subcutaneous air (straight solid arrow) and air in median raphe (curved solid arrow).



USG Scrotum showing scrotal soft tissue thickening (curved arrow) and Cutaneous air (straight arrow).T-testicle.

- CT scanning
    - This study can detect smaller amounts of soft tissue gas than plain radiography and can demonstrate fluid collections that track along the deep fascial planes.
    - CT scanning is readily available in most hospitals and should be considered the diagnostic tool of choice.



CT scan shows extensive inflammatory soft tissue swelling of scrotum and limited amount of soft tissue air (arrow). Inflammatory changes also involves penis.

- MRI: This study yields greater soft tissue detail than a CT scanning but creates greater logistical challenges, especially in patients with critical illness.

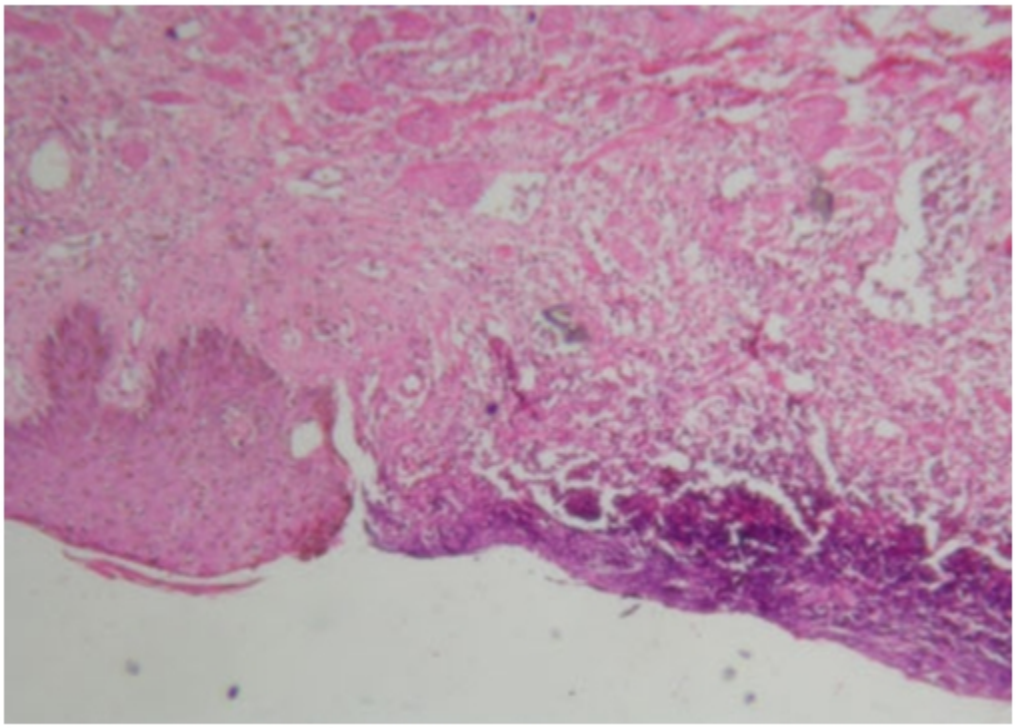
Histological Findings: The pathognomonic findings upon pathologic evaluation of the involved tissue include

(1) Necrosis of the superficial and deep fascial planes,

(2) Fibrinoid coagulation of the nutrient arterioles,

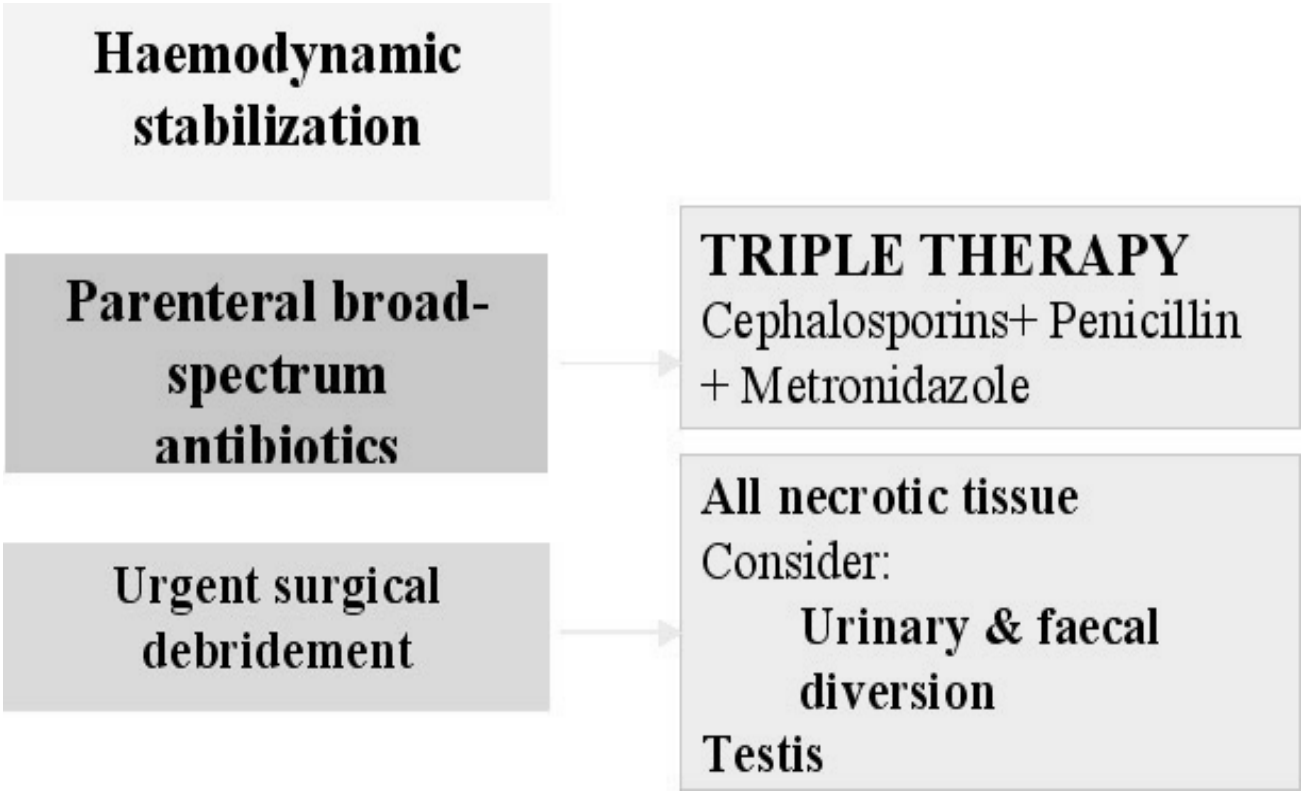
(3) Polymorphonuclear cell infiltration, and

(4) Microorganisms identified within the involved tissues.



The characteristic finding that most commonly indicates Fournier disease is Fibrinoid thrombosis of the nutrient vessels that supply the superficial and deep fascia. A frequent occurrence is widespread necrosis of the fascia with acute inflammatory cell infiltration, necrotic debris, and frequent demonstration of causative microorganisms within the tissues. This extensive inflammatory process is frequently present deep to intact skin, which is often minimally involved with the inflammatory process until late in the disease.

TREATMENT:



MEDICAL THERAPY:

Treatment of Fournier gangrene involves several modalities, including restoration of normal organ perfusion. In patients who present with systemic toxicity manifesting as hypo perfusion or organ failure, aggressive resuscitation to return normal organ perfusion and function must take

precedence over diagnostic maneuvers, especially if these diagnostic studies could compromise the resuscitative interventions.

Treatment also involves the institution of broad-spectrum antibiotic therapy. The antibiotic spectrum should cover staphylococci, streptococci, the Enterobacteriaceae family of organisms, and anaerobes. A reasonable empiric regimen might consist of ciprofloxacin and clindamycin. Clindamycin is particularly useful in the treatment of necrotizing soft tissue infections due to its gram-positive and anaerobic spectrum of activity. Clindamycin has been shown in animal models of streptococcal infection to have superior response rates compared with penicillin or erythromycin, even if treatment was delayed (Stevens, 1998).

If initial tissue stains (i.e., KOH stain) show fungi, add an empiric antifungal agent such as amphotericin B or caspofungin. In cases associated with sepsis syndrome, therapy with intravenous immunoglobulin (IVIG), which is thought to neutralize super antigens such as the streptotoxins (A, B) believed to mitigate the exaggerated cytokine response, has been shown to be a good adjuvant to appropriate antibiotic coverage and complete surgical debridement (Cawley, 1999).

Hyperbaric oxygen, if available, has shown some promising results. One needs to balance this therapy with the stability of the patient, and surgical therapy must not be delayed.

SURGICAL THERAPY:

- Establishing the diagnosis
    - In the event of a presumptive diagnosis based on a clinical examination or diagnostic study, the definitive diagnosis is established by examination with the patient under anesthesia followed by incision into the area of greatest clinical concern.
    - If frankly gangrenous tissue is found or purulence is drained , the diagnosis is established.
    - Occasionally, early-stage Fournier disease manifests as severe cellulitis. If an incision is made, the fascia may appear edematous rather than the gray-black appearance of well-established Fournier gangrene. In this instance, obtain an excisional biopsy sample of the deep fascia for frozen section evaluation to eliminate the potential for early necrotizing disease.

- Excising necrotic tissue
    - Once the diagnosis is established, all necrotic tissue must be excised. The skin should be opened widely to expose the full extent of the underlying fascial and subcutaneous tissue necrosis.
    - Send tissue for aerobic and anaerobic cultures and a histologic assessment.
    - Given the characteristic thrombosis of the nutrient vessels, the overlying skin has impaired blood supply and should be excised if significantly undermined. The authors strongly recommend radical excisional debridement with electrocautery in order to reduce the considerable operative blood loss if the area of involvement is extensive.
    - Given the potential fulminant nature of this necrotizing process, consider repeated operative debridement procedures to ensure complete eradication of the infection.
    - Once the results of the tissue cultures are known, alter the antibiotic regimen to cover the causative organisms. Continue antibiotics for 10-14 days or until reconstruction are accomplished.

- If the perineal involvement is extensive, fecal diversion should be considered at subsequent operative explorations to eliminate the potential for fecal contamination of the wounds. Fecal diversion is not usually necessary when the necrosis is limited to the genitalia but is mandatory when the perianal area is extensively involved.
- Urinary diversion is accomplished with a urethral catheter in most instances. Suprapubic cystostomy is used when urethral drainage of the bladder is not possible because of pathology (e.g., stricture disease, Prostatic hypertrophy).
- The testicles are often spared in the necrotizing process. If uninvolved, place the exposed testicle in a subcutaneous pocket to prevent desiccation. If a testicle is involved in the necrotic process or its viability is questioned, perform Orchidectomy.
- Once the infection is eradicated, healthy granulation tissue develops; this signifies the time to proceed to reconstruction.
- Vacuum-assisted closure (VAC) has shown great promise in the hastening wound healing in these patients (Kovacs, 2001). Application after initial debridement may shorten hospital stay and speed up the grafting and flap placement process.

- Options for reconstruction
    - Primary closure of the skin, if possible
    - Local skin flap coverage
    - Split-thickness skin grafts
    - Medial thigh flap

COMPLICATIONS:

Complications

Major systemic:
· Acute renal failure
· ARDS / Pneumonia
· Gastrointestinal bleeding
· Heart failure
Hypocalcaemia

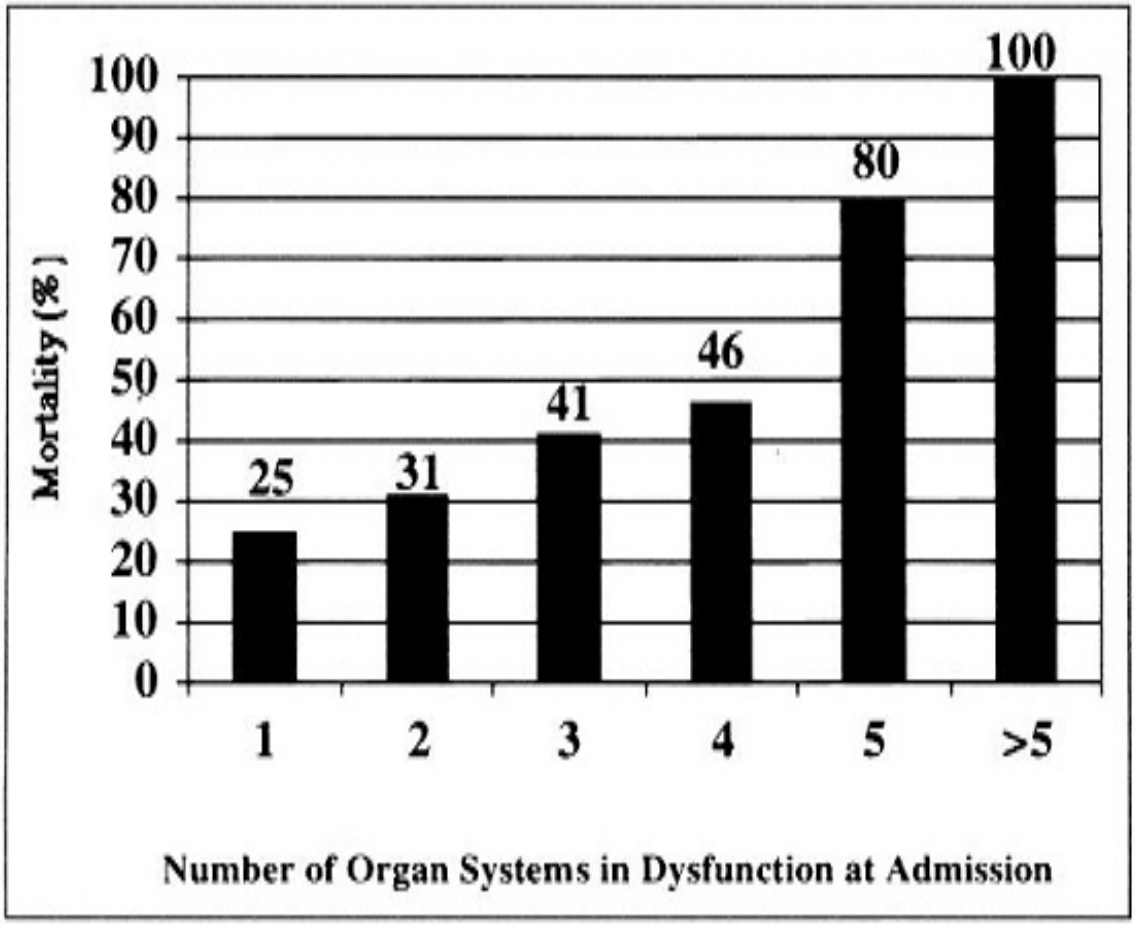
The main complication associated with Fournier disease is unresolved sepsis, often caused by one of the following:

- Unrecognized cause of the infection (e.g., perforated peptic ulcer disease, appendicitis, diverticulitis) or extension of the necrotizing process outside the obvious wound (CT scanning is helpful for evaluating these 2 possibilities.)
- Complication of severe acute illness (e.g., line sepsis, bacterial endocarditis, pneumonia)
- The plethora of co morbid conditions (e.g., acute myocardial infarction, respiratory failure, pressure ulcerations, delirium) or the bed-rest conditions imposed on patients who are acutely ill (eg, pulmonary embolus, deep venous thrombosis, atelectasis, pneumonia).

OUTCOME & PROGNOSIS:

- Mortality rates: 3 -40%
- Poor prognostic factors
  - Age
  - Female gender
  - Anorectal causes*
  - Number of organ failure @ admission*
  - Delay in presentation & treatment*
  - ? Diabetes* / HIV

*** Most important prognostic factors.**



Despite modern supportive measures, the reported mortality rate still is high and this is due in part to the aggressive nature of the infection and in part to the underlying co-morbid diseases.

Conclusion:

Fournier gangrene is a notorious disorder which requires early diagnosis and treatment. Minor infection should be given due attention as negligence may lead to this life threatening complication.

An early diagnosis, stabilization of hemodynamic status and debridement of whole necrotic tissues with antibiotic cover will certainly reduce the risk of morbidity.

BIBLIOGRAPHY:

1. Eke N. Fournier's gangrene: a review of 1726 cases. BJS 2000; 87(6): 718-728
2. Ayumba BR, Magoha GA: Management of Fournier's gangrene at the Kenyatta National Hospital, Nairobi. East Afr Med J 1998 Jun; 75(6): 370-3
3. Basoglu M, Gul O, Yildirgan I, et al: Fournier's gangrene: review of fifteen cases. Am Surg 1997 Nov; 63(11): 1019-21
4. Ben-Aharon U, Borenstein A, Eisenkraft S, et al: Extensive necrotizing soft tissue infection of the perineum. Isr J Med Sci 1996 Sep; 32(9): 745-
5. Benchekroun A, Lachkar A, Bjijou Y, et al: [Gangrene of the external genital organs. Apropos of 55 cases]. J Urol (Paris) 1997; 103(1-2): 27-31
6. Benizri E, Fabiani P, Migliori G, et al: Gangrene of the perineum. Urology 1996 Jun; 47(6): 935-9
7. Yu, P, Sanger JR, Matloub HS, Gosain A, Larson D. Anterolateral Thigh Fasciocutaneous Island Flaps in Perineoscrotal Reconstruction. Plastic & reconstructive surgery 2002; (2): 610-616.
8. Laucks SS. Fournier's gangrene. Surg Clin N Am 1994; 74: 1339-52

9. . Dunford C, Cooper R, Molan P, White R. Nursing standard 2000; 15(11): 63-68.
10. Effem SEE. Recent advances in the management of Fournier' gangrene: preliminary observations. Surgery 1993; 13:2000-4.
11. Kaul R, McGear A, Low DE, Green K, Scwartz B. Population-based surveillance for group a streptococcal necrotizing fasciitis: clinical features, prognostic indicators, and microbiologic analysis of seventy-seven cases. Is J Med 1997; 103:18-24.
12. Brings HA, Matthews R, Brinkman J, Rotolo J: Crohn's disease presenting with Fournier's gangrene and enterovesical fistula. Am Surg 1997 May; 63(5): 401-5
13. Chen CS, Liu KL, Chen HW, et al: Prognostic factors and strategy of treatment in Fournier's gangrene: a 12-year retrospective study. Changgeng Yi Xue Za Zhi 1999 Mar; 22(1): 31-6
14. 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 Jan; 170(1): 49-55
15. Corman JM: Classic articles in colonic and rectal surgery. Dis Colon Rectum 1988; 31: 984-8.

16. Corman JM, Moody JA, Aronson WJ: Fournier's gangrene in a modern surgical setting: improved survival with aggressive management. BJU Int 1999 Jul; 84(1):
17. Ekelius L, Björkman H, Kalin M, Fohlman J: Fournier's gangrene after genital piercing. Scand J Infect Dis 2004; 36(8):
18. El Khader K, el Fassi J, Nouri M, et al: [Fournier's gangrene. Analysis of 32 cases]. J Urol (Paris) 1997; 103(1-2):
19. Faber HJ, Girbes AR, Daenen S: Fournier's gangrene as first presentation of promyelocytic leukemia. Leuk Res 1998 May; 22(5):
20. Fan CM, Whitman GJ, and Chew FS: Radiologic-Pathologic Conferences of the Massachusetts General Hospital. Necrotizing fasciitis of the scrotum (Fournier's gangrene). AJR Am J Roentgenol 1996 May; 166(5)
21. Gamagami RA, Mostafavi M, and Gamagami A, Lazorthes F: Fournier's gangrene: an unusual presentation for rectal carcinoma. Am J Gastroenterol 1998 Apr; 93(4):
22. Church et al; dec 2000: analysis of outcome of fournier gangrene in different studies.
23. Gould SW, Banwell P, and Glazer G: Perforated colonic carcinoma presenting as epididymo-orchitis and Fournier's gangrene. Eur J Surg Oncol 1997 Aug; 23(4):

24. Goyette M: Group A streptococcal necrotizing fasciitis Fournier's gangrene--Quebec. Can Commun Dis Rep 1997 Jul 1; 23(13): 101-
25. Hejase MJ, Simonin JE, Bihrle R, Coogan CL: Genital Fournier's gangrene: experience with 38 patients. Urology 1996 May; 47(5): 734-
26. Hollabaugh RS Jr, Dmochowski RR, Hickerson WL, Cox CE: Fournier's gangrene: therapeutic impact of hyperbaric oxygen. Plast Reconstr Surg 1998 Jan; 101(1):
27. Kane CJ, Nash P, McAninch JW: Ultrasonographic appearance of necrotizing gangrene: aid in early diagnosis. Urology 1996 Jul; 48(1):
28. Kohagura K, Sesoko S, Tozawa M, et al: [A female case of Fournier's gangrene in a patient with lupus nephritis]. Nippon Jinzo Gakkai Shi 1998 Jul; 40(5):
29. Korhonen K, Hirn M, Niinikoski J: Hyperbaric oxygen in the treatment of Fournier's gangrene. Eur J Surg 1998 Apr; 164(4):
30. Kovacs LH, Kloeppel M, Papadopulos NA, et al: Necrotizing fasciitis. Ann Plast Surg 2001 Dec; 47(6
31. Martinelli G, Alessandrino EP, Bernasconi P, et al: Fournier's gangrene: a clinical presentation of necrotizing fasciitis after bone marrow transplantation. Bone Marrow Transplant 1998 Nov; 22(10): 1023

32. Meleney FL: Hemolytic Streptococcus gangrene. Arch Surg 1924; 9: 317-21.
33. Mergenhagen SE, Thonard JC, Scherp HW: Studies on synergistic infections. I. Experimental infections with anaerobic streptococci. J Infect Dis 1958 Jul-Aug; 103(1):
34. Mindrup SR, Kealey GP, Fallon B: Hyperbaric oxygen for the treatment of fournier's gangrene. J Urol 2005 Jun; 173(6):
35. Moses AE: Necrotizing fasciitis: flesh-eating microbes. Isr J Med Sci 1996 Sep; 32(9):
36. Mouraviev VB, Pautler SE, Hayman WP: Fournier's gangrene following penile self-injection with cocaine. Scand J Urol Nephrol 2002; 36(4):
37. Nomikos IN: Necrotizing perineal infections (Fournier's disease): old remedies for an old disease. Int J Colorectal Dis 1998; 13(1): 48-
38. Norton KS, Johnson LW, Perry T, et al: Management of Fournier's gangrene: an eleven year retrospective analysis of early recognition, diagnosis, and treatment. Am Surg 2002 Aug; 68(8): 709-13[Medline].
39. Papachristodoulou AJ, Zografos GN, Papastratis G, et al: Fournier's gangrene: still highly lethal. Langenbecks Arch Chir 1997; 382(1): 15-
40. Paty R, Smith AD: Gangrene and Fournier's gangrene. Urol Clin North Am 1992 Feb; 19(1): .

41. Pizzorno R, Bonini F, Donelli A, et al: Hyperbaric oxygen therapy in the. J Urol 1997 Sep; 158(3 Pt 1):
42. Rajan DK, Scharer KA: Radiology of Fournier's gangrene. AJR Am J Roentgenol 1998 Jan; 170(1):
43. Rajbhandari SM, Wilson RM: Unusual infections in diabetes. Diabetes Res Clin Pract 1998 Feb; 39(2):
44. Roca B, Cunat E, Simon E: HIV infection presenting with Fournier's gangrene. Neth J Med 1998 Oct; 53(4):.
45. Sherman J, Solliday M, Paraiso E, et al: Early CT findings of Fournier's gangrene in a healthy male. Clin Imaging 1998 Nov-Dec; 22(6): 425-
46. Smith GL, Bunker CB, Dinneen MD: Fournier's gangrene. Br J Urol 1998 Mar; 81(3):
47. Stevens DL, Gibbons AE, Bergstrom R, Winn V: The Eagle effect revisited: efficacy of clindamycin, erythromycin, and penicillin in the treatment of streptococcal myositis. J Infect Dis 1988 Jul; 158(1):
48. Wilson B: Necrotizing fasciitis. Am Surg 1952 Apr; 18(4): 41
49. Yu, P, Sanger JR, Matloub HS, Gosain A, Larson D. Anterolateral Thigh Fasciocutaneous Island Flaps in Perineoscrotal Reconstruction. Plastic & reconstructive surgery 2002; (2): 610-616

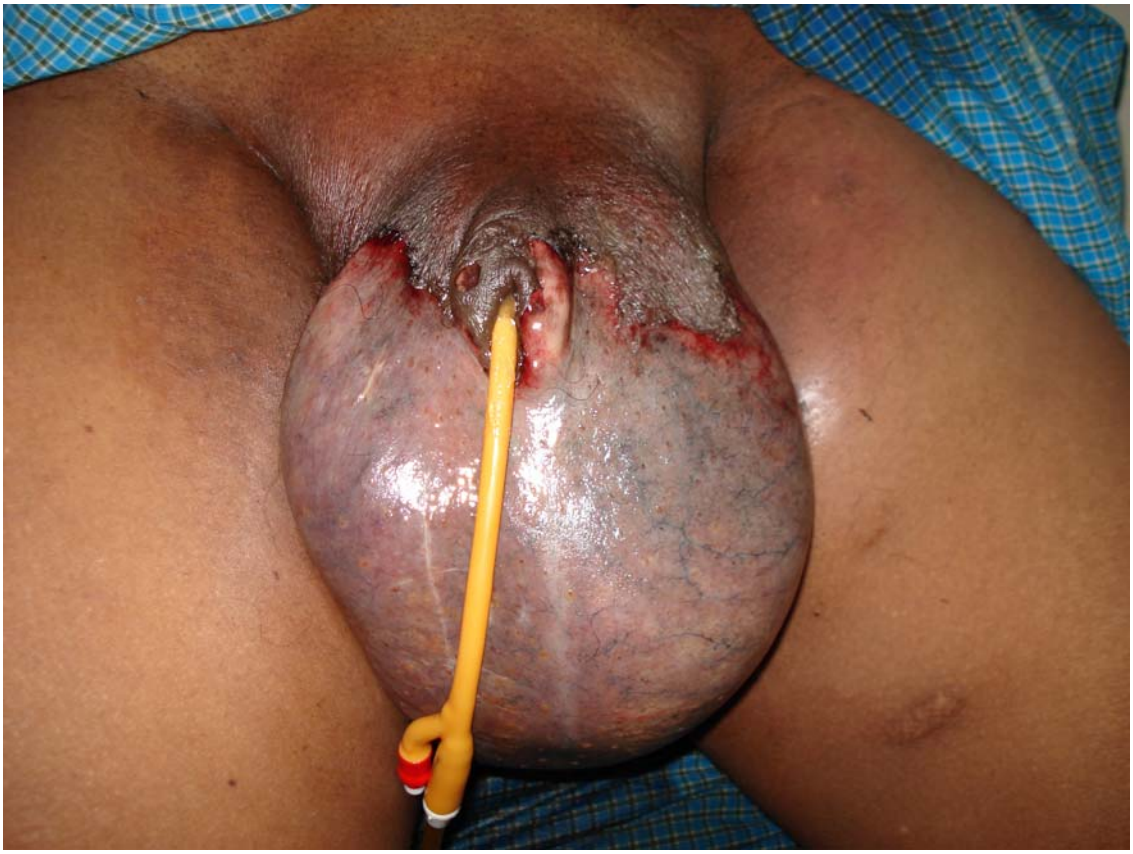


Figure 1: On day of admission (case 2)

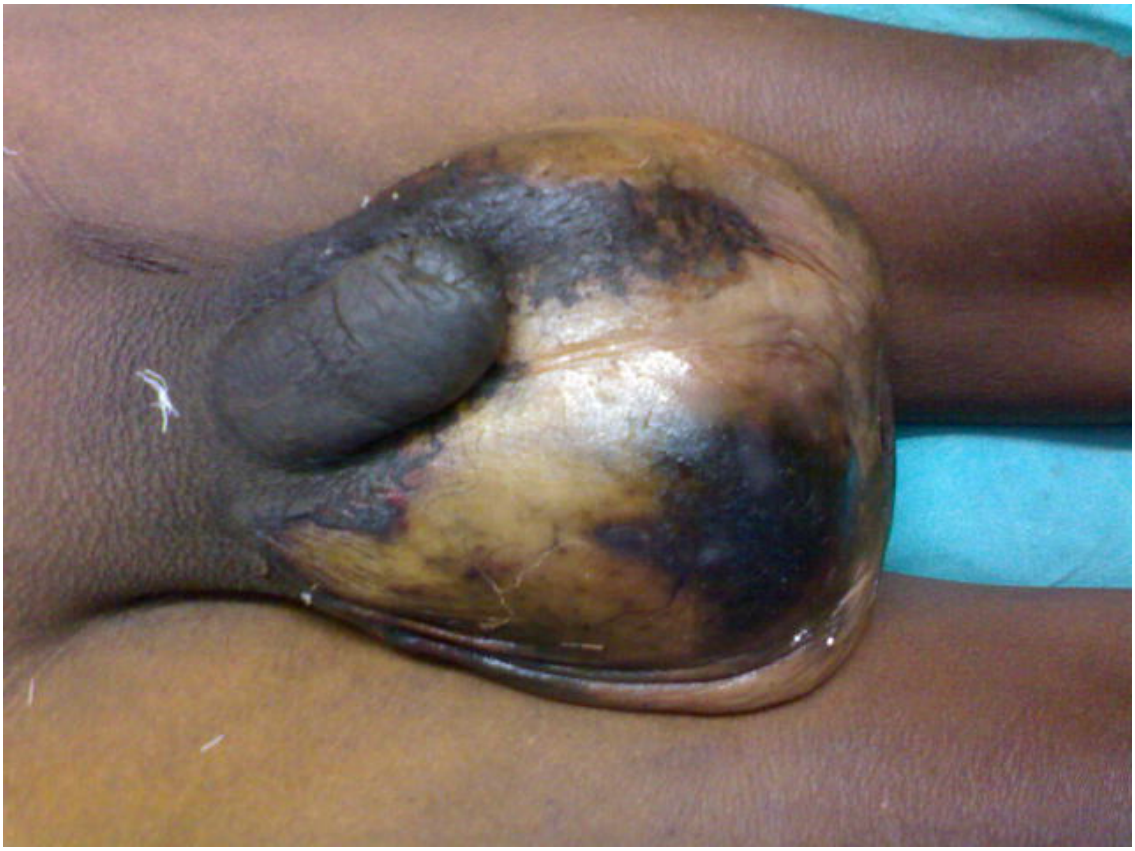


Figure 2: on day of admission (case 3)

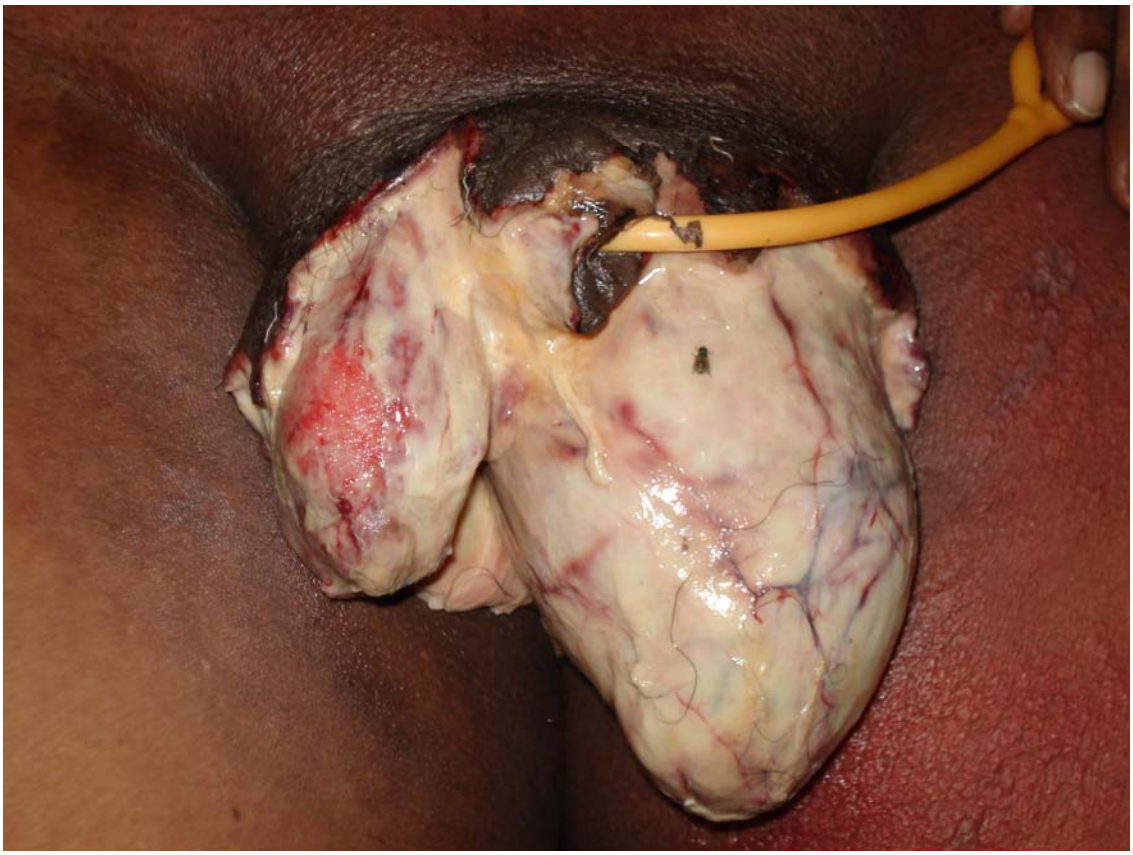


Figure 3: 12 hours after debridement (case 2)

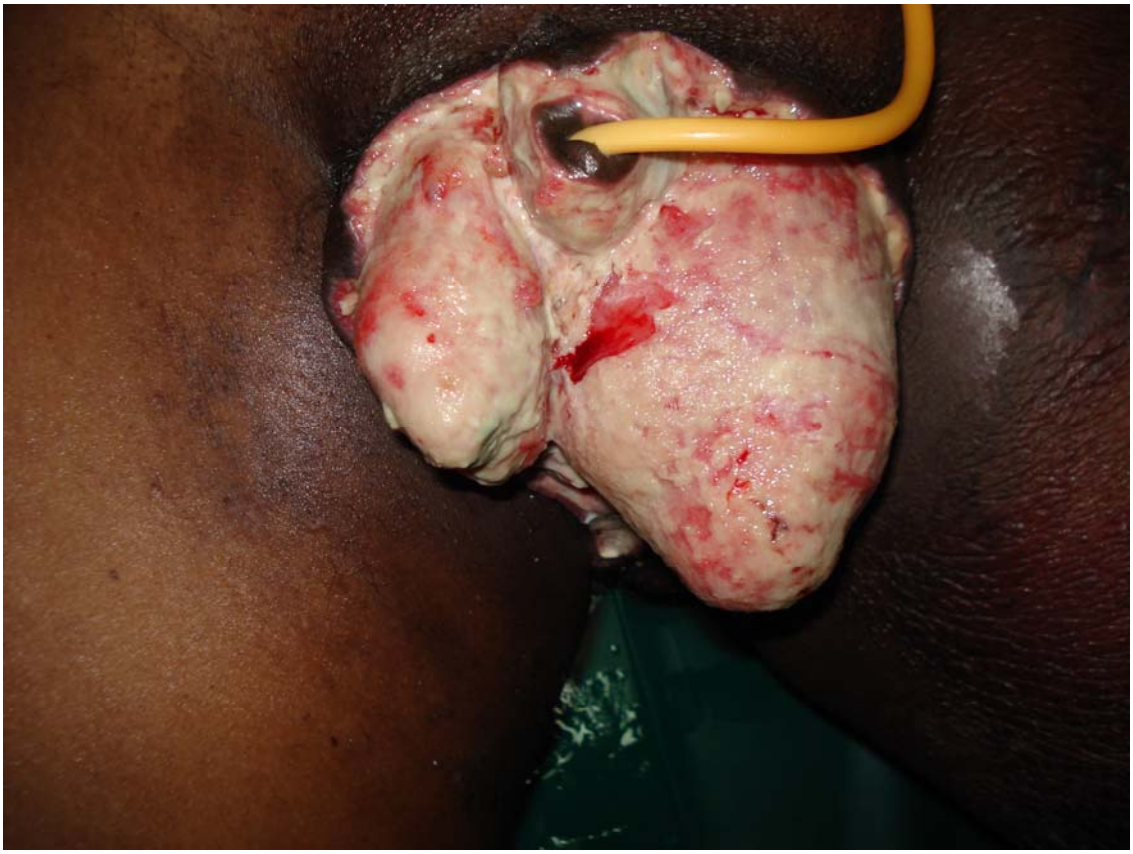


Figure 4: After Redebridement (case 2)

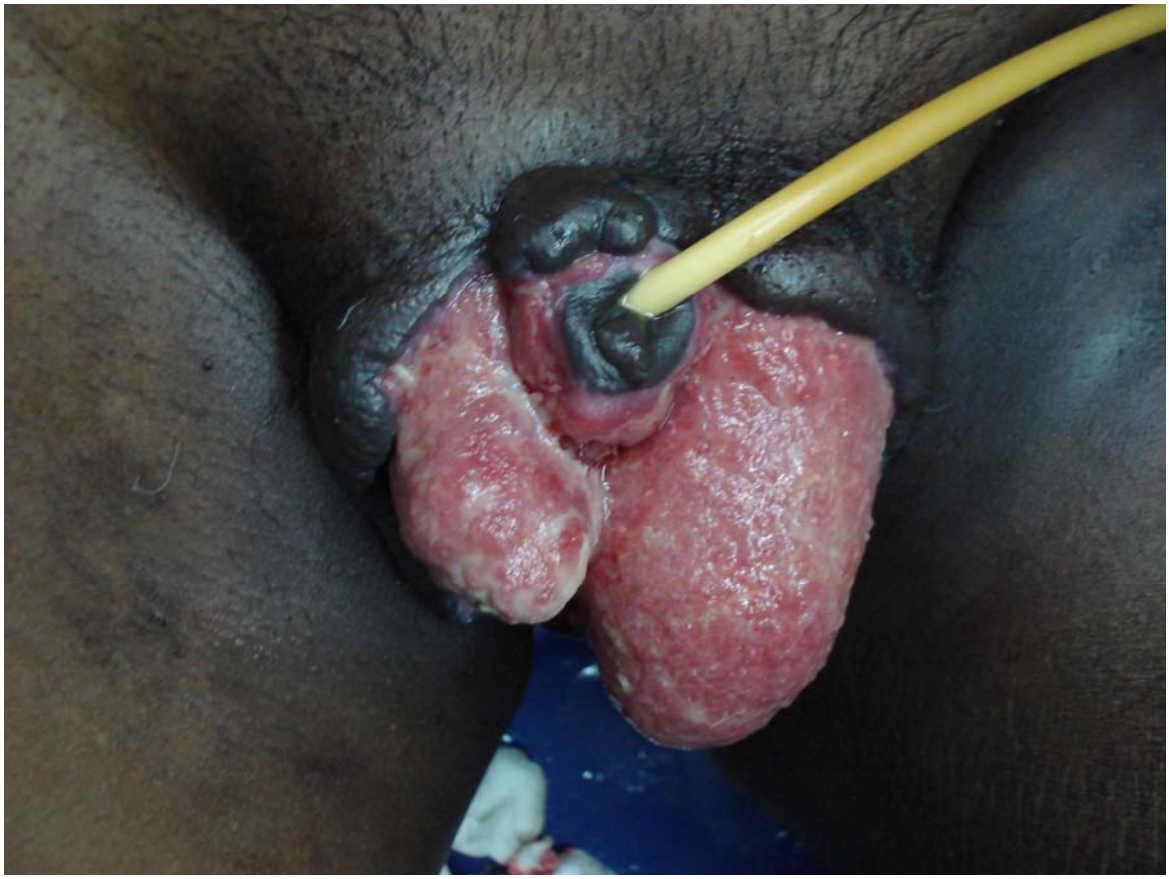


Figure 5: 10 days after cleaning and dressing (case 2)

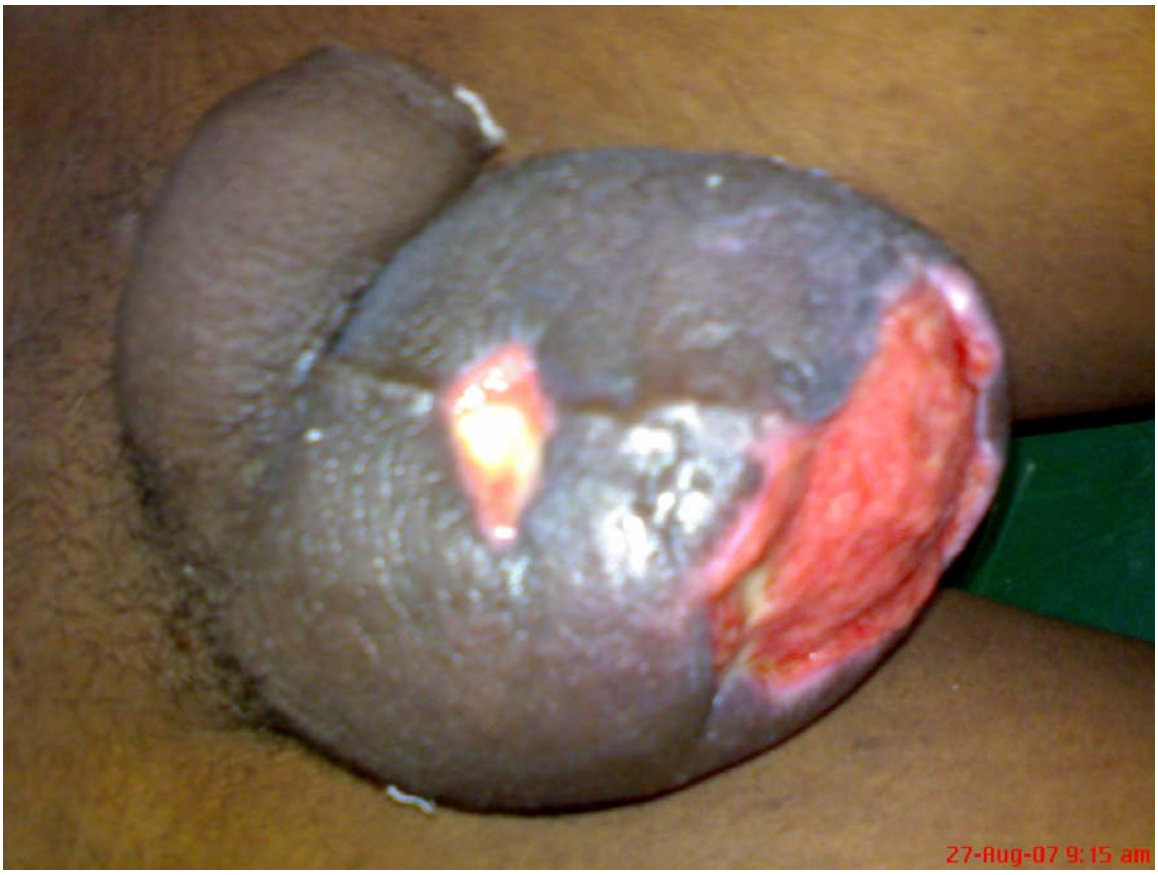


Figure 6: healing by secondary intention(case 12)

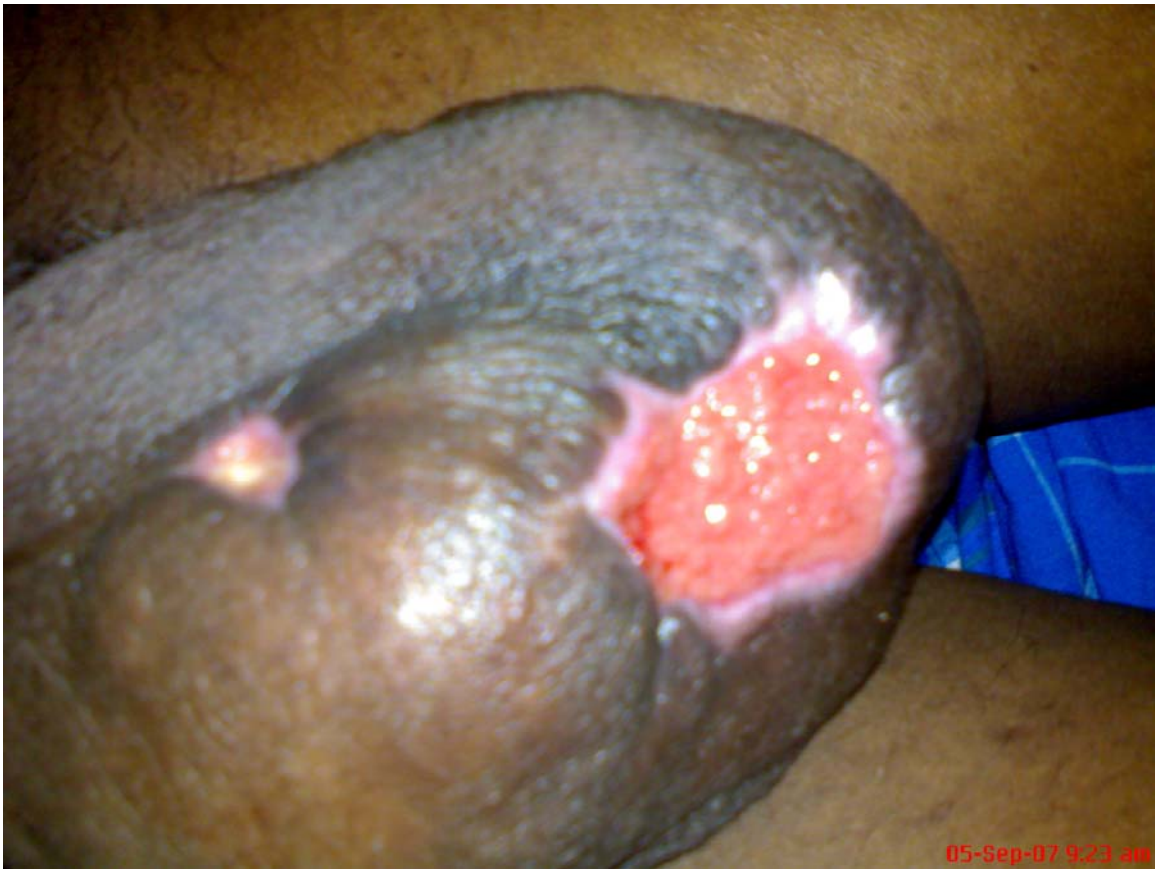


Figure 7: healing by secondary intention

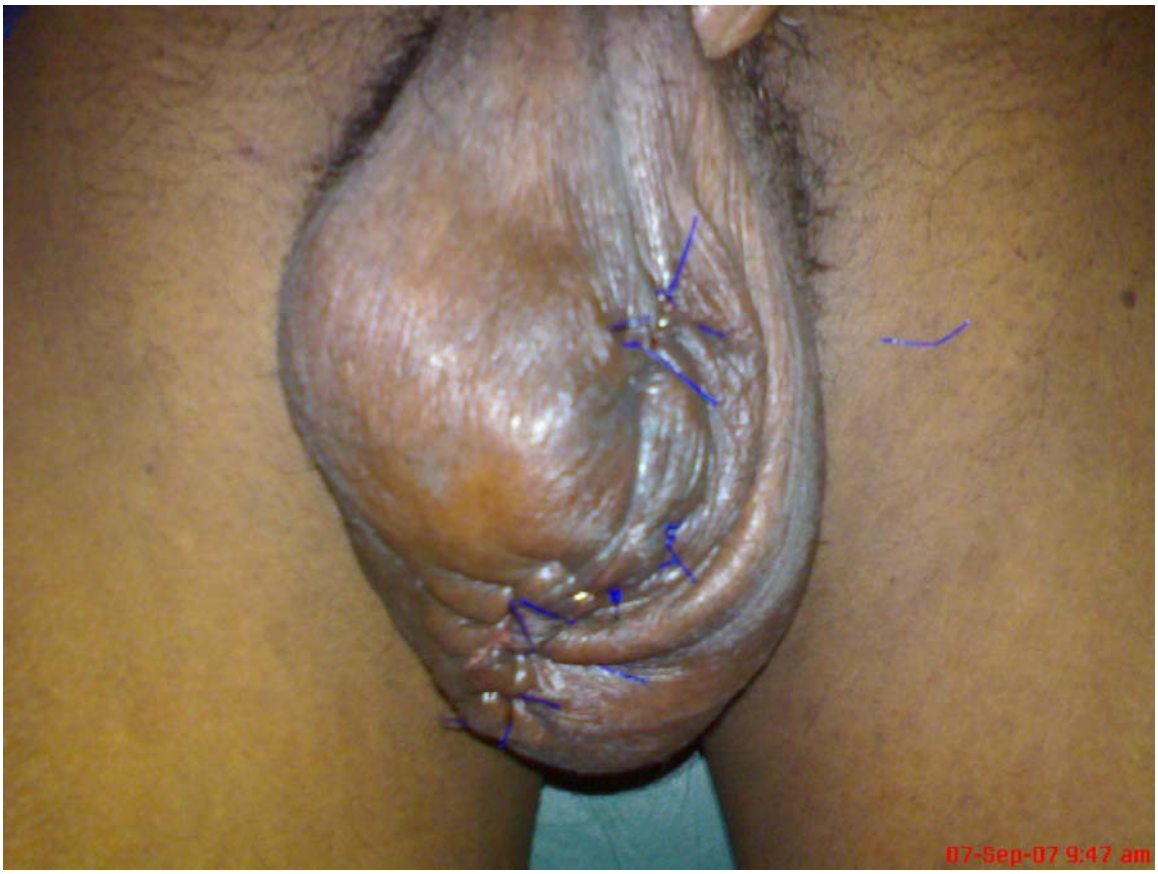


Figure 8: delayed suturing (case 5)

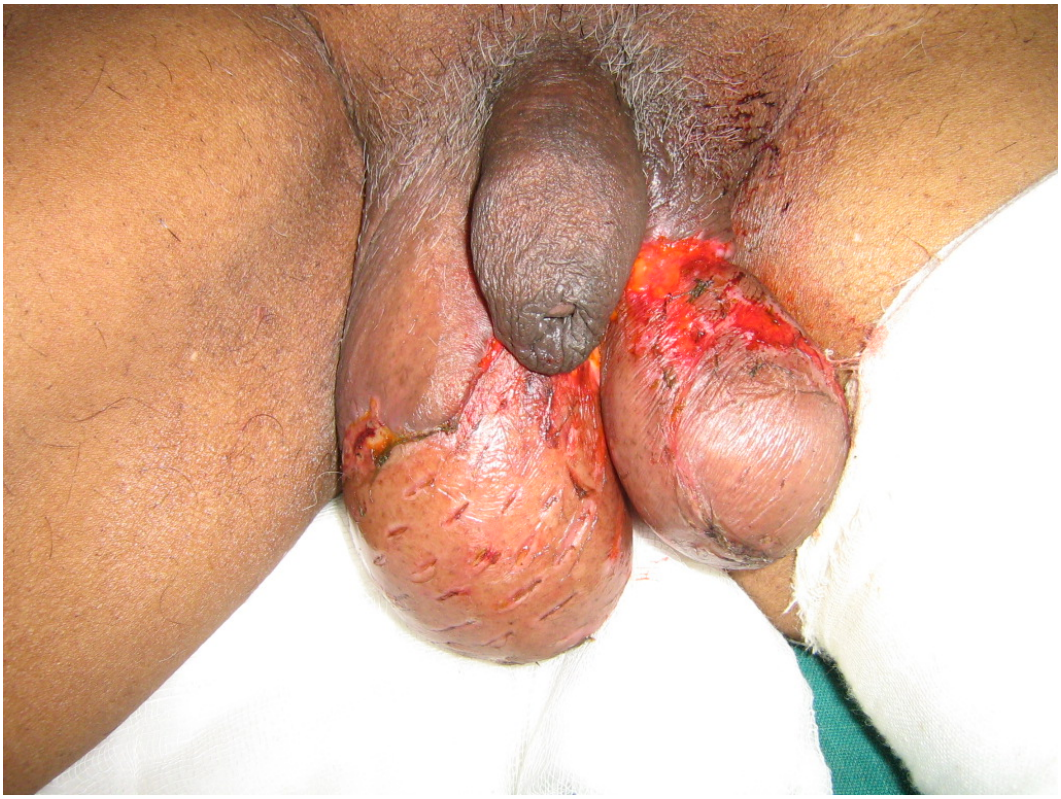


Fig 9: Split skin grafting done (case 23)

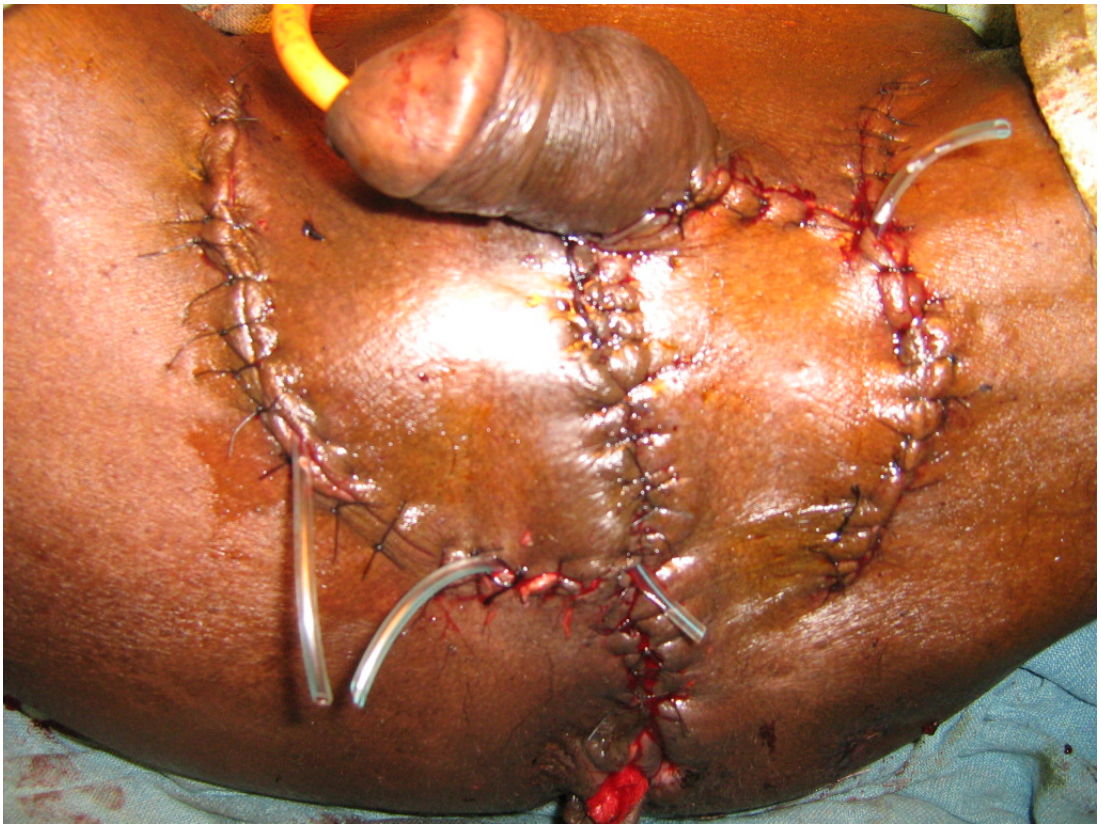


Figure 10: Medial thigh flap (case 37)

MASTER CHART

S.No	NAME	AGE	IP NO	Time for Consultation (Days)	Extent Of Involvement	Etiology	Predisposing factor	TC, Hb%	Blood sugar,urea,Creatinine Mg%	Pus C/S	Surgical treatment	Hospital stay (days)
1.	Sambandam	80/M	867423	4	Scrotum	-	-	7400 9.8	96,36 1.3	Streptococcus	SSG	51
2.	Logidoss	60/M	871890	7	Scrotum	Trauma	DM	7800 9.8	92,30 1.2	-	Orchidectomy	13
3.	Mohan	45/M	875988	10	Scrotum, penis	-	Liver disease	9200 9.6	104,65, 2.0	-	X	
4.	Ponnusamy	58/M	876883	3	Scrotum	Perianal abscess	DM	8600 9.6	170,67, 1.6	-	Debridement C&D	35
5.	Ramarao	63/M	877546	2	Scrotum	Trauma	-	8900 10.6	92,28, 0.9	Pseudo-monas	Delayed suturing	26
6.	Srinivasan	60/M	879603	4	Scrotum, penis	Trauma	Filarial	8600 10.0	78,56, 1.4	Pseudo-monas	Delayed suturing, circumcision	55
7.	Mohan	35/M	883782	5	Scrotum	-	-	9600 10.6	80,28, 0.9	-	Delayed suturing	22
8.	Kapali	35/M	883539	4	Scrotum	-	Liver disease	9800 9.6	114,30, 1.2	Klebsiella	Delayed suturing	45
9.	Arumugam	60/M	880964	4	Scrotum	Perianal abscess	Alcoholic	10,900 9.8	70,77, 1.6	-	Delayed suturing	35
10.	Mani	50/M	844996	6	Scrotum,penis	-	Obese /CAD/ DM	14,100 8.4	126,28, 0.9	-	Debridement	X
11.	Subramani	65/M	844996	3	Scrotum	-	Alcoholic	7400 9.8	96,36, 1.3	-	SSG	42
12.	Suryan	28/M	843352	1	Scrotum	Trauma	-	10,400 10.6	92,28, 0.9	-	C&D	12
13.	Panner	75/M	851956	7	Scrotum	-	DM	9600 9.4	160,60, 1.4	=	X	=
14.	Mohanraj	56/M	861173	3	Scrotum	-	Alcoholic	8400 10.2	100,32, 1.2	Klebsiella	Delayed suturing	24
15.	Mohamed	35/M	850328	2	Scrotum	Trauma	Alcoholic	9200 10.0	104,28,0.8	Pseudo-monas	C&D	21

S.No	Name	Age	IP NO	Time for Consultation (days)	Extent Of Involvement	Etiology	Predisposing factor	TC, Hb%	Blood sugar,urea,Creatinine Mg%	Pus C/S	Surgical treatment	Hospital stay (days)
16.	Raju	55/M	855121	2	Scrotum	-	DM	9600 10.6	132,24, 0.8	E.coli	Debridement, C&D	40
17.	Parthasarathy	35/M	850328	2	Scrotum	-	Alcoholic	11200 10.6	64,24, 0.9	E.coli	Debridement, C&D	28
18.	Munusamy	65/M	850596	3	Scrotum	Trauma	-	10,400 9.8	100,30, 1.2	-	Orchidectomy	20
19.	Mani	55/M	847792	2	Scrotum	-	-	9800 10.2	70,28, 0.9	-	Thigh implantation	33
20.	Palani	41/M	850535	3	Scrotum	-	DM	10,800 10.4	138,26, 0.9	-	Thigh implantation	40
21.	Shankar	35/M	881851	2	Scrotum	Trauma	Alcoholic	8200 10.2	96,24, 0.9	-	C&D	20
22.	Anadan	54/M	853877	4	Scrotum	-	Alcoholic	9600 9.8	100,30, 0.9	-	Delayed suturing	25
23.	Immanuel	65/M	884396	8	Scrotum, penis	-	DM	6800 9.6	144,28, 1.0	Klebsiella	SSG	52
24..	John	82/M	853090	2	Scrotum	Trauma	Alcoholic	10,400 19.4	96,30, 1.2	-	Orchidectomy	13
25.	Sankar	32/M	855560	2	Scrotum	-	Alcoholic	8600 10.2	84,26, 0.9	-	Delayed suturing	16
26.	Aparanji	74/M	858777	5	Scrotum	-	-	7400 10.2	96,36, 1.2	Pseudo-monas	SSG	46
27.	Dharman	40/M	859410	5	Scrotum,penis, perineum	-	DM	9800 9.6	140,38, 1.2	Ecoli	SSG	58
28.	Govindasamy	65/M	859452	4	Scrotum	-	DM	8400 9.8	140,40, 1.4	Ecoli	Delayed suturing	52
29.	Sudhakar	30/M	859964	1	Scrotum	Trauma	-	9600 10.2	90,30, 9	-	C&D	13
30.	Shanmugam	38/M	852299	2	Scrotum	Trauma	-	8400 10.6	84,24, 0.9	-	C&D	16

S.No	Name	Age	IP NO	Time for Consultation (days)	Extent Of Involvement	Etiology	Predisposing factor	TC, Hb%	Blood sugar,urea,Creatinine Mg%	Pus C/S	Surgical treatment	Hospital stay (days)
31.	Mohanraj	56/M	861173	2	Scrotum	-	DM	9600 9.8	136,36, 1.2	Ecoli	Delayed suturing	48
32.	Johnson	30/M	876381	2	Scrotum	Trauma	Alcoholic	9100 10.2	84,30 1.0	-	C&D	20
33.	Chokkalingam	55/M	836851	2	Scrotum	-	-	10,400 10.2	90,28, 0.9	streptococcus	SSG	33
34.	Rajendran	45/M	821391	2	Scrotum	-	Alcoholic	10,400 10.2	96,28, 1.0	-	Thigh implantation	31
35.	Mohan	42/M	823403	3	Scrotum	-	DM	9600, 10.4	100,30 0.9	Pseudo-monas	Delayed suturing	26
36.	Haridoss	50/M	826363	2	Scrotum	-	Alcoholic	9600 10.0	90,26, 0.9	-	Thigh implantation	35
37	Rajan	33/M	827820	2	Scrotum	-	-	9200 10.2	88,26, 0.9	Streptococcus	Thigh flap	28
38.	Anjaneyar	65/M	828308	2	Scrotum	-	DM	8400 9.8	130,26, 1.0	-	C&D	30
39.	Krishnamoorthy	70/M	829162	3	Scrotum	-	DM	8400 10.0	140,30, 1.0	-	C&D	35
40.	Pushparaj	31/M	829799	1	Scrotum	Trauma	-	9200 10.4	84,24, 0.9	-	C&D	13
41.	Sampath	55/M	826058	4	Scrotum	-	DM	8600 10.0	151,48, 1.2	Pseudo-monas	Delayed suturing	45
42.	Subban	45/M	822169	2	Scrotum	Trauma	Alcoholic	9400 10.2	114,42, 0.9	-	C&D	18
43.	Tirunavukarasu	80/M	819313	6	Scrotum, penis, perineum	-	DM ,CAD	7400 9.0	164,65, 2.1	-	C & D	X
44.	Gunasekar	32/M	819122	1	Scrotum	Trauma	-	8800 10.6	92,24, 0.9	-	C&D	15

DM-Diabetes Mellitus; CAD-Coronary Artery Disease; SSG-Split Skin Grafting; C & D- Cleaning and Dressing