DISSERTATION ON

A STUDY ON FOURNIER'S GANGRENE

Dissertation submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

In partial fulfillment of the regulations
for the award of the degree of

M.S.-GENERAL SURGERY- BRANCH – I

THANJAVUR MEDICAL COLLEGE,
THANJAVUR - 613 004.

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY
CHENNAI - 600 032.

APRIL – 2017

CERTIFICATE

This is to certify that the dissertation titled "A **STUDY ON FOURNIER'S GANGRENE"** is a bonafide research work done by **Dr. ARUN T SEN** under the guidance of **Dr. ANTONY PRABAKAR M.S.,** (Professor, Department of General Surgery) Thanjavur Government Medical College Hospital, Thanjavur.

Dr. M. VANITHAMANI M.S. MCh
The Dean,
Thanjavur Medical College,
Thanjavur – 613004.

Dr.M.ELANGOVAN M.S.,
The Professor and HOD,
Department of General surgery,
Thanjavur Medical College,
Thanjavur – 613004.

CERTIFICATE BY THE GUIDE

This is to certify that this dissertation titled **" A STUDY ON FOURNIER'S GANGRENE"** is a bonafide research work done by **Dr. ARUN T SEN,** in partial fulfilment of the requirement for the degree of **M.S. GENERAL SURGERY – BRANCH I.**

Dr. ANTONY PRABAKAR M.S.,
Professor
Dept. of General Surgery
Thanjavur Medical College and Hospital

Date :

Place :

DECLARATION BY THE CANDIDATE

I solemnly declare that this Dissertation "A STUDY ON FOURNIER'S GANGRENE **"**was done by me in the Department of General Surgery, Thanjavur Medical College, and Hospital, Thanjavur. Under the Guidance and Supervision of my Professor Dr. ANTONY PRABAKAR M.S. Department of General Surgery,Thanjavur Medical College, Thanjavur

between 2015 and 2016.

This Dissertation is submitted to The TAMILNADU Dr. M.G.R Medical University, Chennai in partial fulfilment of University requirements for the award of M.S Degree (GENERAL SURGERY).

DR. ARUN T SEN
Postgraduate Student,
Thanjavur Medical College.
Thanjavur.

Thanjavur Medical College

THANJAVUR, TAMILNADU, INDIA - 613001

(Affiliated to the T.N.Dr.MGR Medical University, Chennai)

INSTITUTIONAL ETHICAL COMMITTEE

CERTIFICATE

Approval No. : 270

This is to certify that The Research Proposal / Project titled

A STUDY ON FOURNIER'S GANGRENE

submitted by Dr. ARUN.T.SEN **of**

Dept. of GENERAL SURGERY **Thanjavur Medical College, Thanjavur**

was approved by the Ethical Committee.

Thanjavur

Dated : 21.9.16

Secretary
Ethical Committee
TMC, Thanjavur.

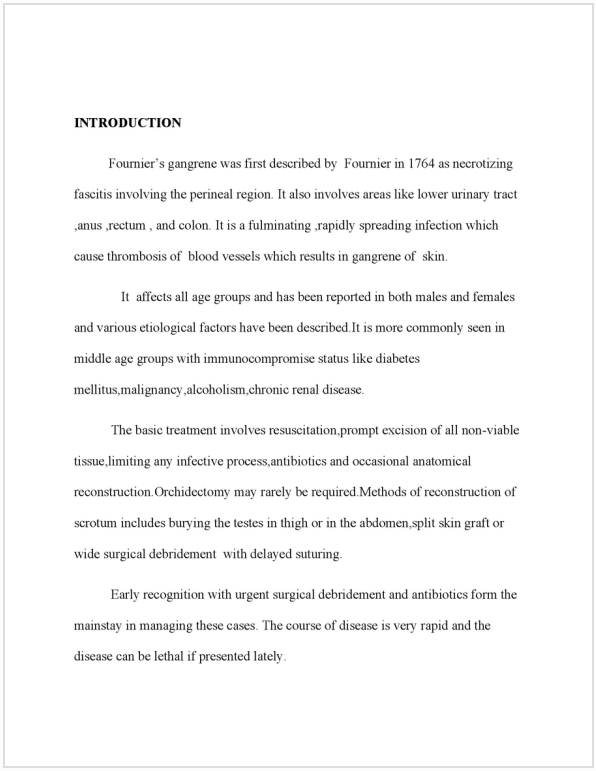
THE SECRETARY
INSTITUTIONAL ETHICAL COMMITTEE
THANJAVUR MEDICAL COLLEGE,
THANJAVUR.

Digital Receipt

This receipt acknowledges that Turnitin received your paper. Below you will find the receipt information regarding your submission.

The first page of your submissions is displayed below.

Submission author:	221411201 Ms Gensur ARUN T SEN
Assignment title:	2015-2015 plagiarism
Submission title:	A STUDY ON FOURNIER'S GANGR..
File name:	A_STUDY_ON_FOURNIER_S_GAN..
File size:	6.89M
Page count:	75
Word count:	4,576
Character count:	26,590
Submission date:	27-Sep-2016 01:07AM
Submission ID:	710240794



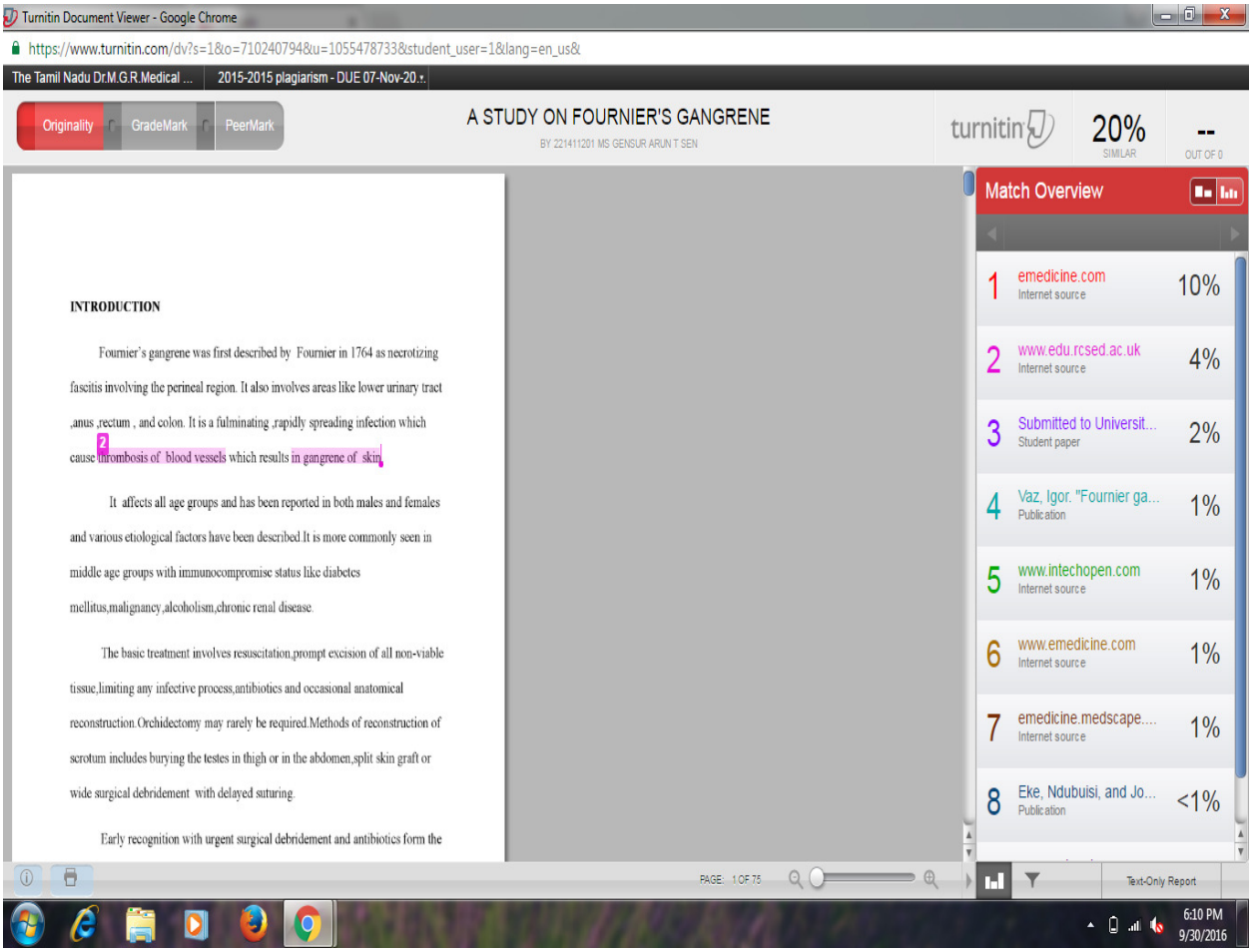
INTRODUCTION

Fournier's gangrene was first described by Fournier in 1764 as necrotizing fascitis involving the perineal region. It also involves areas like lower urinary tract ,anus ,rectum , and colon. It is a fulminating ,rapidly spreading infection which cause thrombosis of blood vessels which results in gangrene of skin.

It affects all age groups and has been reported in both males and females and various etiological factors have been described.It is more commonly seen in middle age groups with immunocompromise status like diabetes mellitus,malignancy,alcoholism,chronic renal disease.

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

Early recognition with urgent surgical debridement and antibiotics form the mainstay in managing these cases. The course of disease is very rapid and the disease can be lethal if presented lately.


Turnitin Document Viewer - Google Chrome
https://www.turnitin.com/dv?s=1&o=710240794&u=1055478733&student_user=1&lang=en_us&
The Tamil Nadu Dr.M.G.R.Medical ...
2015-2015 plagiarism - DUE 07-Nov-20.
Originality
GradeMark
PeerMark
A STUDY ON FOURNIER'S GANGRENE
BY 221411201 MS GENSUR ARUN T SEN
turnitin
20%
SIMILAR
--
OUT OF 0
INTRODUCTION
Fournier's gangrene was first described by Fournier in 1764 as necrotizing fasciitis involving the perineal region. It also involves areas like lower urinary tract ,anus ,rectum , and colon. It is a fulminating ,rapidly spreading infection which cause thrombosis of blood vessels which results in gangrene of skin.
It affects all age groups and has been reported in both males and females and various etiological factors have been described.It is more commonly seen in middle age groups with immunocompromise status like diabetes mellitus,malignancy,alcoholism,chronic renal disease.
The basic treatment involves resuscitation,prompt excision of all non-viable tissue,limiting any infective process,antibiotics and occasional anatomical reconstruction.Orchidectomy may rarely be required.Methods of reconstruction of scrotum includes burying the testes in thigh or in the abdomen,split skin graft or wide surgical debridement with delayed suturing.
Early recognition with urgent surgical debridement and antibiotics form the
PAGE: 1 OF 75
Match Overview
1 emedicine.com Internet source 10%
2 www.edu.rcsed.ac.uk Internet source 4%
3 Submitted to Universit... Student paper 2%
4 Vaz, Igor. "Fournier ga... Publication 1%
5 www.intechopen.com Internet source 1%
6 www.emedicine.com Internet source 1%
7 emedicine.medscape.... Internet source 1%
8 Eke, Ndubuisi, and Jo... Publication <1%
Text-Only Report
6:10 PM
9/30/2016

ACKNOWLEDGEMENT

I am grateful to **Dr**. **M. VANITHAMANI M.S. MCh,** Dean P.G. for giving me permission and opportunity to conduct study and data collection at Thanjavur Medical College and Hospital.

I am deeply grateful to my professor and Head of the Department of General Surgery , **Prof. Dr. ELANGOVAN.M. M.S.**,for his encouragement and suggestions in preparing this work.

I owe my sincere and grateful acknowledgement to my beloved chief, teacher and guide **Prof. ANTONY PRABAKAR M.S.,** Professor of General Surgery who inspired me to take this topic of "**A STUDY ON FOURNIER'S GANGRENE".** I extend my grateful acknowledgement to my teachers,**Dr.G. KARTHIKEYAN M.S.,** Asst. Prof. of General Surgery and **Dr. R. SYED RAJ M.S.,** Asst. Prof. of General Surgery.

I wish to thank Dr. Anand V, Dr. Venkatesh, and all my Junior Residents in the Department of GENERAL SURGERY for having helped me in compiling data and for extending their fullest cooperation during the study period.

I wish to express my whole hearted thanks to all the patients who participated in the study. Completion of this work would not have been possible without their co-operation. And most of all I would like to thank my family for their wholehearted support, and patience in helping me to finish the study.

And last but not the least I thank the Almighty for guiding me to complete the study.

Dr. Arun T Sen

INTRODUCTION

Fournier's gangrene was first described by Fournier in 1764 as necrotizing fascitis involving the perineal region. It also involves areas like lower urinary tract ,anus ,rectum , and colon. It is a fulminating ,rapidly spreading infection which cause thrombosis of blood vessels which results in gangrene of skin.

It affects all age groups and has been reported in both males and females and various etiological factors have been described.It is more commonly seen in middle age groups with immunocompromise status like diabetes mellitus,malignancy,alcoholism,chronic renal disease.

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

Early recognition with urgent surgical debridement and antibiotics form the mainstay in managing these cases. The course of disease is very rapid and the disease can be lethal if presented lately.

AIM OF THE STUDY

1) To study the age distribution and risk factors of fournier's gangrene
2) To study the most common organisms associated with fournier's gangrene
3) To study outcome in management of fournier's gangrene

MATERIALS AND METHODS

Study was conducted in DEPARTMENT OF GENERAL SURGERY, GOVERNMENT MEDICAL COLLEGE ,THANJAVUR over a period of 1 yr.

35 cases of fournier's gangrene were analyzed. Patient's history and clinical examination was done to arrive at a diagnosis. Age, etiology, predisposing factors, extent of involvement, Pus culture and sensitivity , lab investigations and surgical outcome were evaluated.

OBSERVATIONS

35 male patients admitted with diagnosis of Fournier's gangrene were included in the study. The observations were

Age distribution

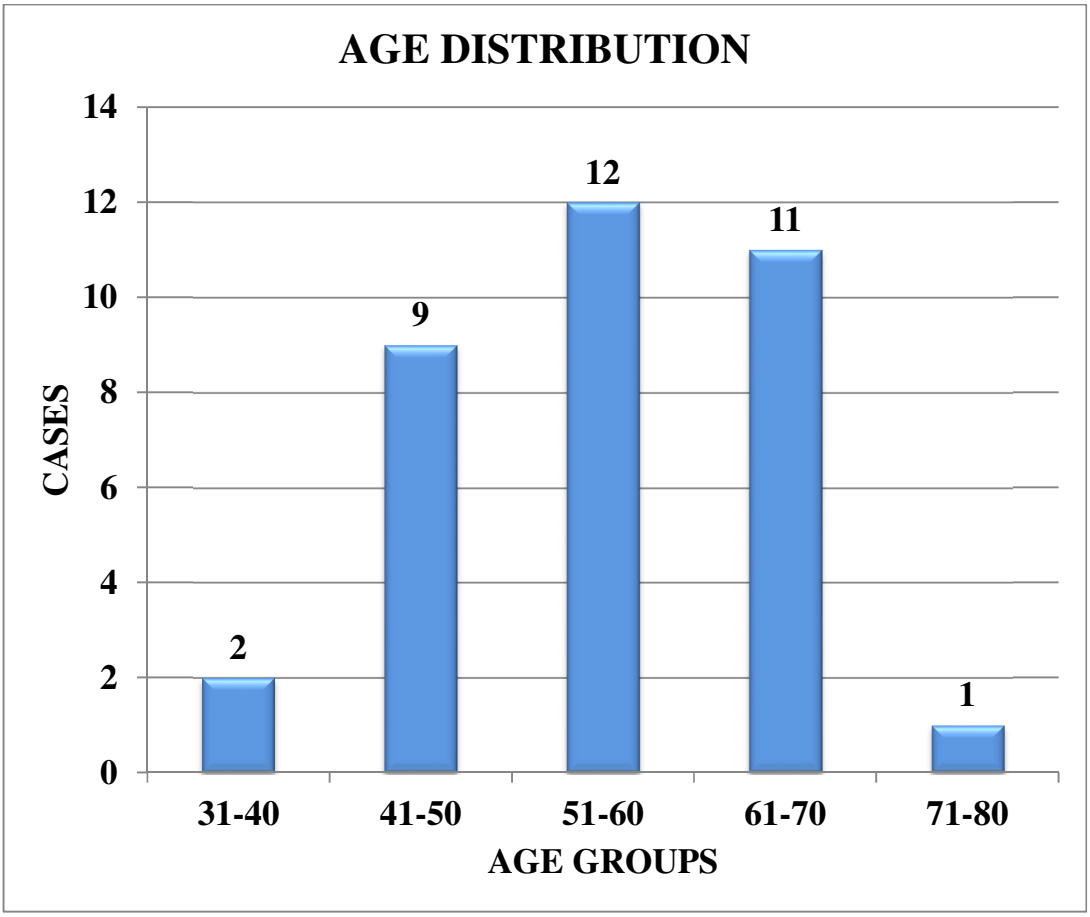
The age of patients varied from 36 to 72 yrs with majority of patients in 61 to 70 yrs age group. This goes along with the reports that the mean age of patients appear to be increased from 40yrs previously to more than 50yrs in recent studies.

The mean age in this study is 55.3yrs.

AGE GROUP	No. OF CASES	PERCENTAGE
31- 40	2	5.7
41-50	9	25.7
51-60	12	34.3
61-70	11	31.4
71-80	1	2.9

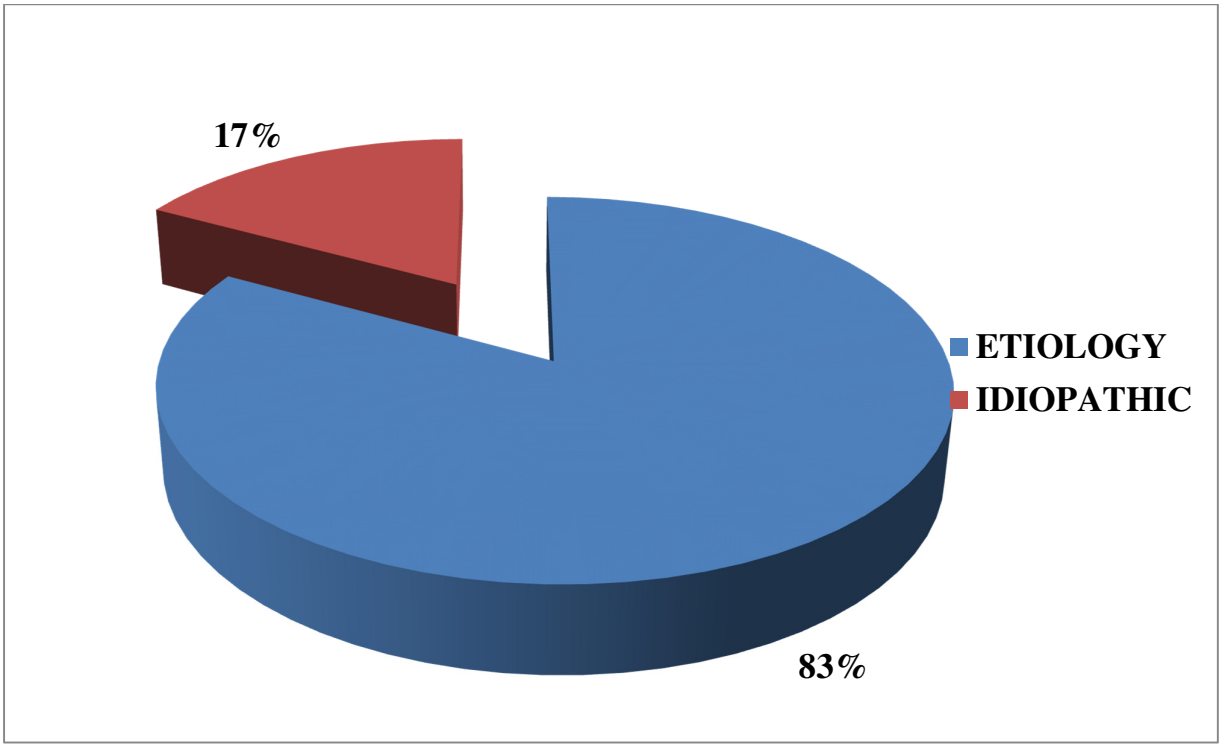
About 31.4% cases are in age group 61-70years. 34.3% were in age group 51-60years. 25.7% cases were in age group of 41-50 and only 5.7% were in age group of 31-40. There were no patients below 30years of age.

AGE DISTRIBUTION



ETIOLOGY AND PREDISPOSING FACTORS

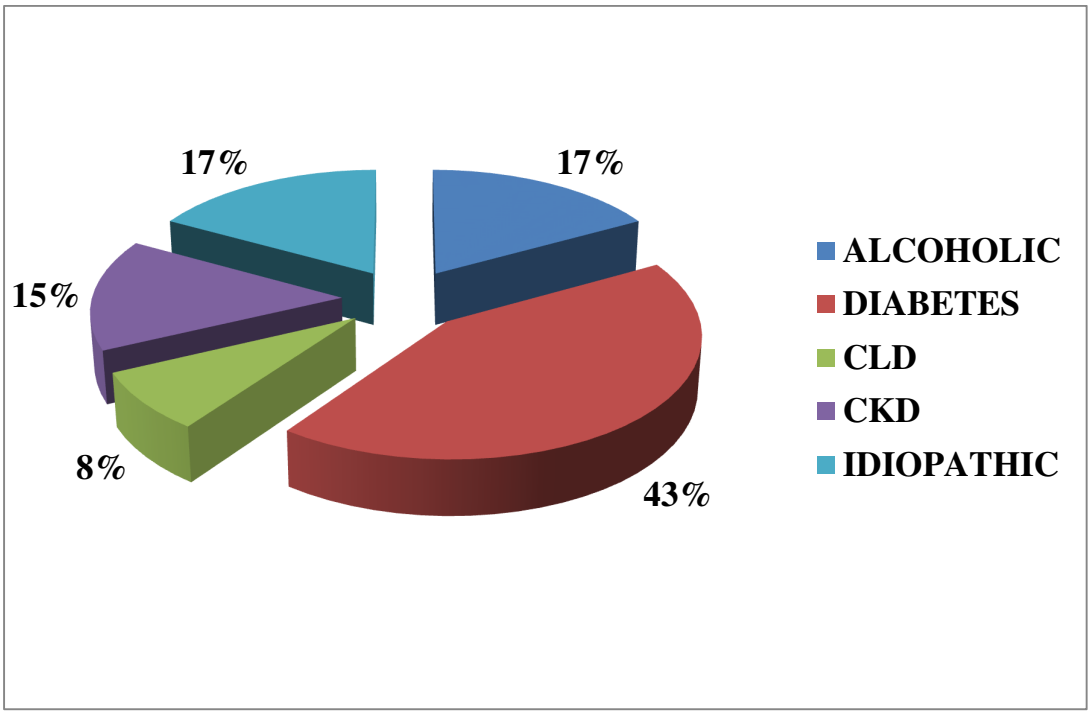
In our study , out of 35 patients , 28 patients had predisposing factors and among the 28 patients 15 were diabetic and 6 were alcoholic. There were 6 cases of idiopathic fournier's gangrene as no causes or predisposing factor could be made out.



CAUSES

14 patients gave history of trauma as intiating factor, the most striking features over recent years is that patients now always found to have underlying systemic disorder.

In this study 15 patients are diagnosis diabetes mellitus that is about 43% and 6 patients gave history of chronic alcoholism , 17% Idiopathic.



Fournier's gangrene has an identifiable cause in approximate 82.8% of cases and the co morbid disease that compromises the immune system are:

1) Diabetes mellitus
2) Alcoholism
3) Chronic kidney disease
4) Liver disease

CLINICAL

In this study patients were admitted with history of fever and malaise for about 1 week .There was history of trauma in14 patients and 2 patients had history of perianal abscess. The patients complain of sever perineal pain with progressive erythema of skin. Obvious gangrene of portion of genitalia and purulent foul smelling discharge was noted in majority of patients.

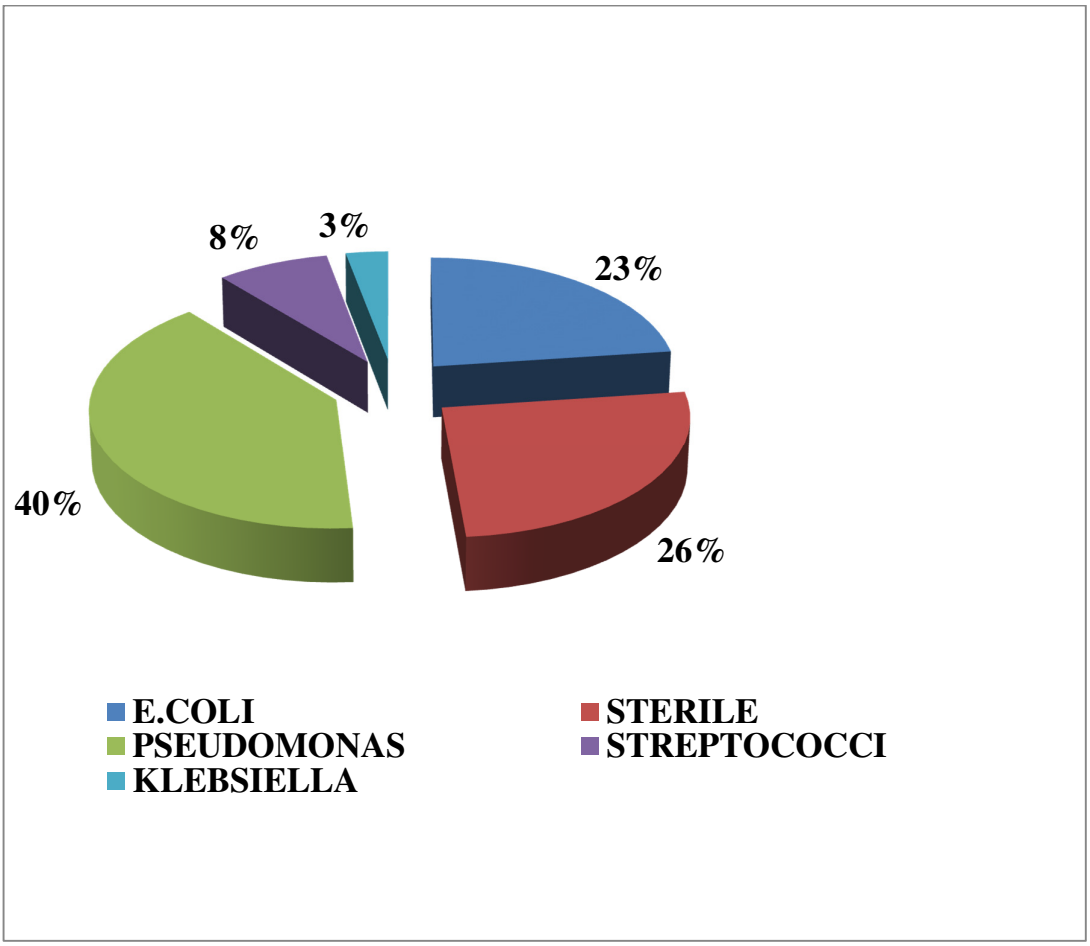
The systemic effect varied from local tenderness with no toxicity to florid septic shock. Toxic features were mainly noted in elderly patients with delayed consultation and co morbid disease. Being a spreading infective process, gangrene was either involving the part or whole of scrotum and some with extention to penis perineum and thigh. Out of 35 patients gangrene was confirmed to scrotum in 26 patients with extension to penis in 9.

Soft tissue crepitations, tenderness, foul smelling discharge was noted in whole patients.

LAB STUDIES

ECG and chest x ray where taken routinely and basic investigation were done. There were 15 patients who where diabetic with uncontrolled blood sugar values.

Wound swab and pus culture sensitivity were sent for all patients. In this study the culture was sterile in 9 cases which may be due to inadequate antibiotic therapy received from outside before coming to our hospital. The organism isolated were, 14 cases were positive for pseudomonas, 8 cases for E coli, 3 cases for streptococci, and one case for klebsiella,


8%
3%
23%
40%
26%
E.COLI
STERILE
PSEUDOMONAS
STREPTOCOCCI
KLEBSIELLA

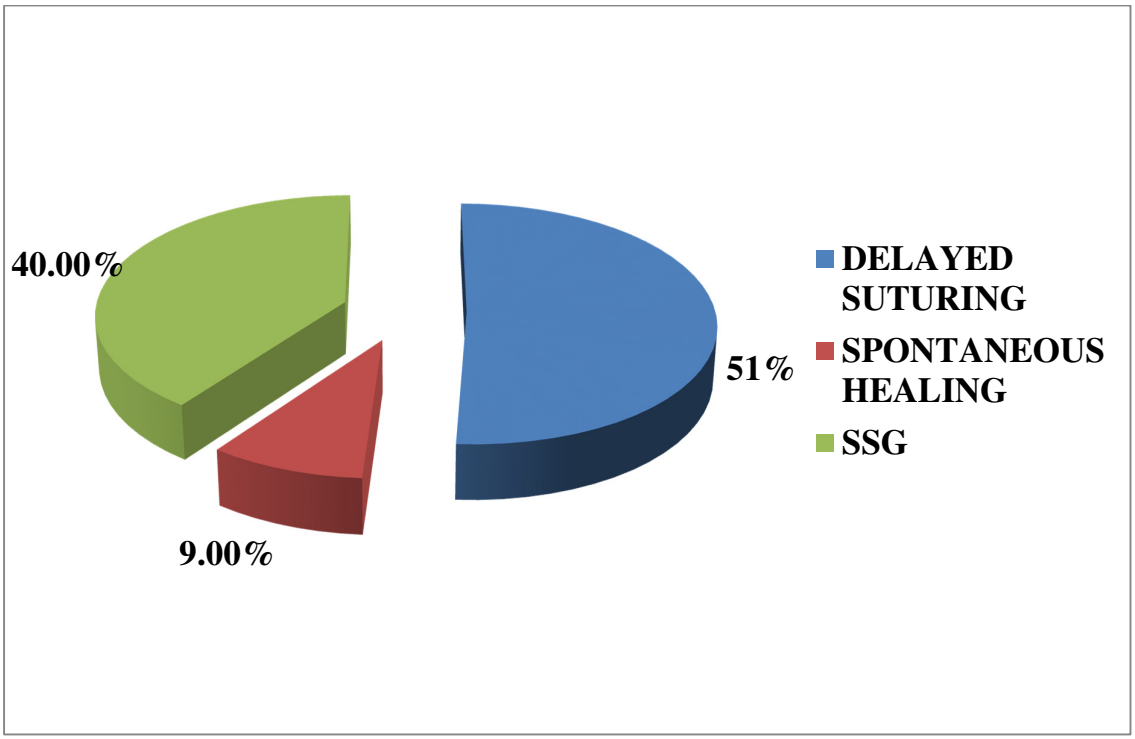
PUS CULTURE

TREATMENT

Is mainly 3 fold. Firstly the patient should be resuscitated with fluids and blood transfusion should be given if needed and higher antibiotic should be started, after that patient should be taken for emergency wound debridement which is done under iv sedation or spinal Anaesthesia.Wound debridment done was extensive and all necrotic tissues where removed. Wound debridement was done until fresh oozing of blood was noted. Patient was initially started on ceftriaxone and metronidazole and later antibiotics were changed according to culture sensitivity. For all patients urinary catheterization was done, blood culture and urine culture was done routinely. Blood culture was sterile in all patients

Regular cleaning and dressing was done with hydrogen peroxide, betadine and normal saline. Once granulation tissue develop normal saline was alone used.

Healing and spontaneous closure was achieved in 3 patients, 18 patients needed delayed suturing and 14 patients needed Split thickness skin graft.



COMPLICATION

Complications like pneumonia bed sore and raised renal parameters were observed and treated accordingly.

OUT COME

Mortality nil, following are the poor prognostic factors

1) Age
2) Diabetis mellitus
3) CKD
4) Extent of involvement

STUDY	COUNTRY	No. OF CASES	MORTALITY RATE(%)	MEAN DISTRIBUTION 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 **TMC**	Thanjavur	35	-	26	60

TMC-THANJAVUR MEDICAL COLLEGE

RESULTS

The following are the results of my study of fournier's gangere

1) The mean age in this study is 55.3yrs
2) The major predisposing factors are diabetics mellitus and alcoholism
3) Chronic kidney disease was found in 15%
4) There were only 17% of cases for which no causes could be found out
5) Predominant microbial agents are pseudomonas (40%) and e coli (23%)
6) In 26% of cases first culture was sterile
7) All patients are treated with antibiotics and early debridement
8) Spontaneous healing was achieved in 3 patients
9) 15 patients (51%) needed delayed suturing and 14 patients needed skin grafting
10) Adequate resuscitation, transfusion of blood and blood products, early administration of broad spectrum antibiotics with early extensive thorough wound debridement improves the out come.

DISCUSSION

Fournier gangrene is necrotizing infection affecting the soft issues of external genitalia.Nowadays Fournier gangrene is a form of necrotizing fascitis, a general term introduced in 1951 by Wilson, describing infection of soft tissue involving deep and superficial fascia, regardless of location.

Even though term Fournier gangrene was used to describe idiopathic gangrene of external genitalia; it also has been used to describe soft tissue necrotizing infection of perineum, whatever is the cause. Now the term Fournier gangrene is restricted to describe infections that primarily involve the external genitalia. The indiscriminate use of the word has made it tough to understand occurrence of the disease.

HISTORICAL BACKGROUND:

Jean Alfred Fournier, a Parisian dermatologist& venereologist first described about this, based on 5 young men's scrotal gangrene. He described it as

1) Sudden onset in a healthy young man
2) Rapid progression to gangrene
3) No 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, before that in 1764 Bauriene was the first one to describe Fournier’s gangrene. In 1924 in China Meleny described a generalized form of the disease and he called it as “streptococcal gangrene”. In the 1990s dramatization of entire process of the necrotizing fasciitis was done by sensational medical journalists by associating it with flesh–eating microorganisms.

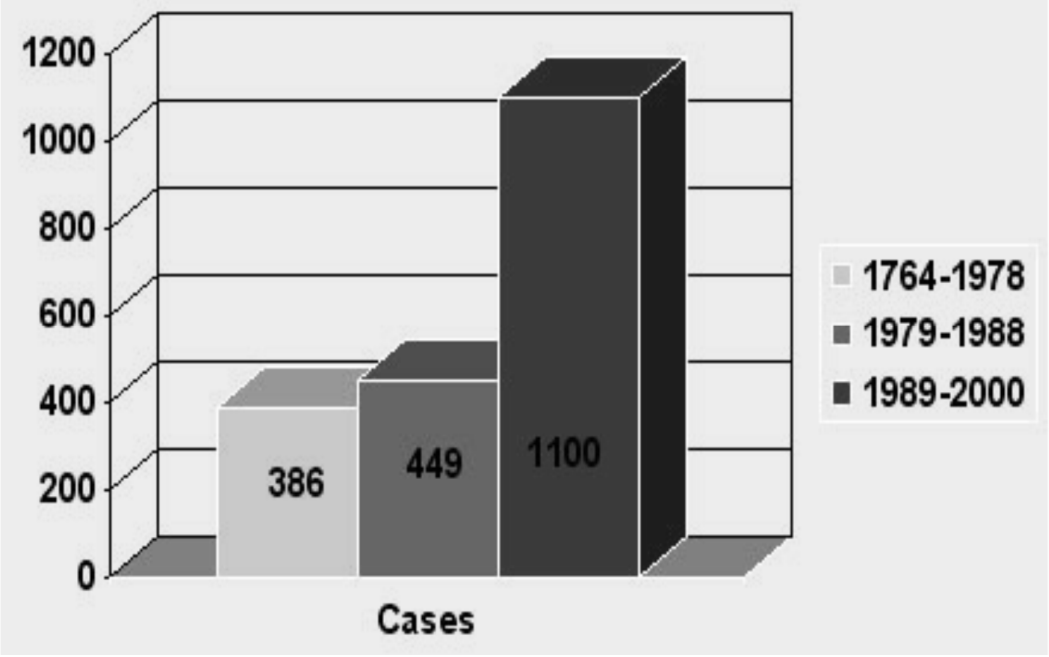
EPIDEMIOLOGY

Sex

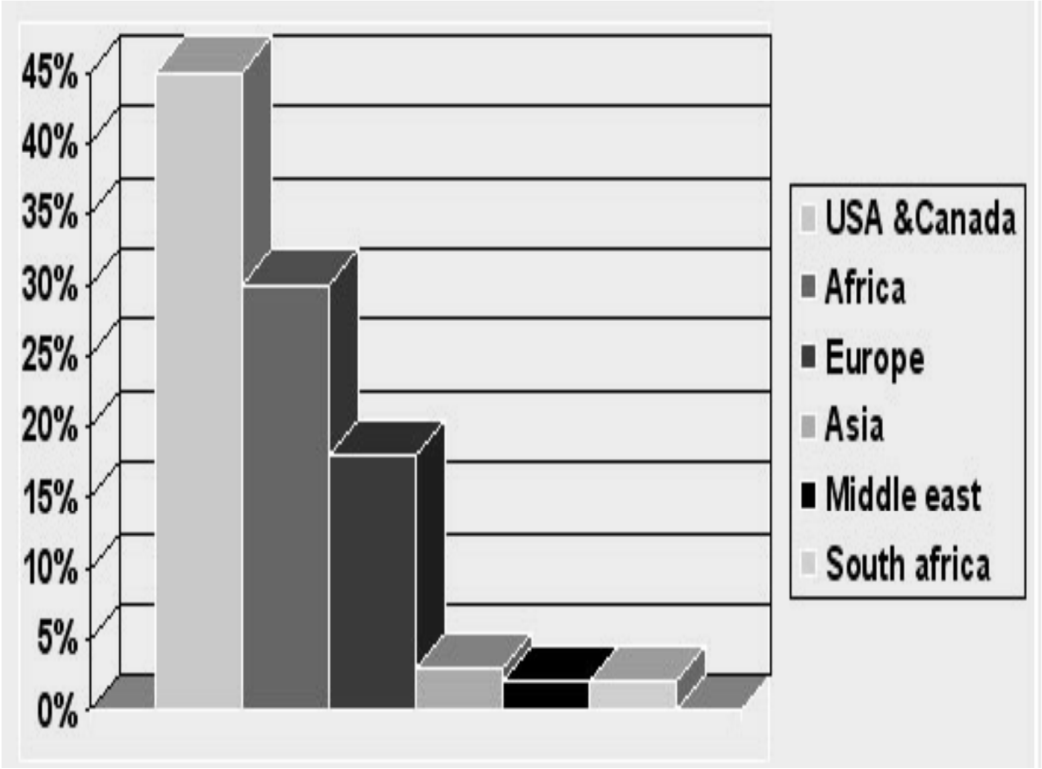
Male : Female = 10:1

There occurred a change in the epidemiology that the disease is not only seen in young men but also affected all ranges from neonates to the very old. There are many case reported in pediatrics where two-third of it belongs to age group less than 3 months.

The mean age of occurrence of the disease is in between 40-50 year group. It is seen both in males and females, but more common in male.



There is an increase in the incidence of the cases due to better recognition and reporting of the condition. A suggestion has been come out that the poor socioeconomic conditions are more prone to develop Fournier's gangrene. Fournier's gangrene is demonstrated among the well as well as the poor.



It has been observed that this condition is more prevalent in Africa and Asia, at the same time its pattern is different is in these areas when compared to those in Europe and America. In the developing world the source of sepsis is the skin and rate of death is lower. It was noticed that there may be strong resistance is shown by Negro race against the infection or the organism in Africa does not have enough virulence.

Disorders which are associated

Chronic alcoholism: 25-50%

Diabetes: 40-60%

Immunosuppressant

One of the most noticeable change is that disease is always accompanied by an underlying systemic disorder. The major disorders which are associated with the Fournier's disease are chronic alcoholism, diabetics and immunosuppression.

HIV infection has resulted in the formation of a new group of patients which are prone for developing Fournier's in Africa and other developing nations. Fourniers gangrene has been noticed as a presenting feature in case of undiagnosed HIV infections.

ETIOLOGY

It is been described as an unknown gangrene of external genitalia, It posess an identifiable reason in almost 93% of cases. It starts as a necrotizing infection of urogenital tract, anorectum, or the genitalia's skin.

- Perianal infections, manifesting due to colorectal injuries or colorectal carcinoma or appendicitis are included as Anorectal causes

- Bulbourethral gland infections, iatrogenic injuries secondary to urethral injury, or lower urinary tract infections are been included under urogenital tract causes

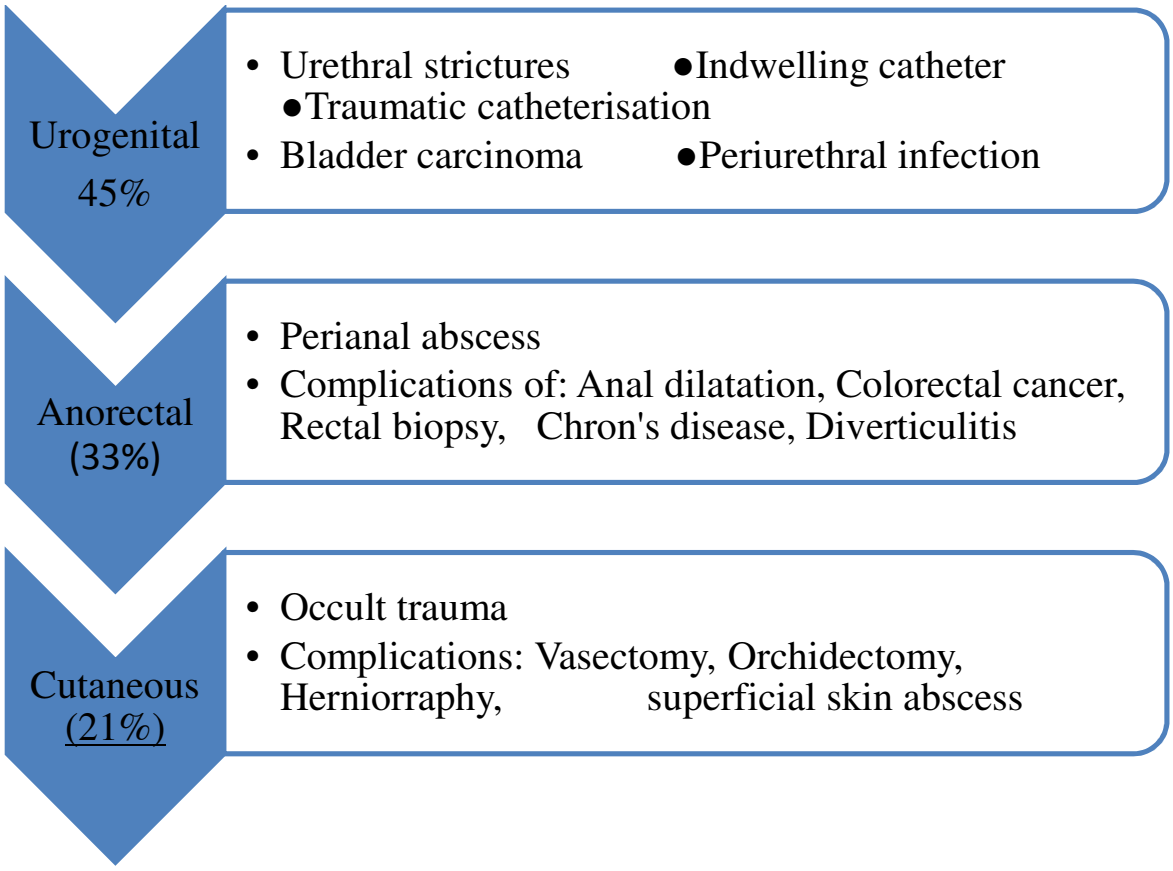
- Dermatologic causes are ulceration from scrotal pressure hidradenitis suppurativa, trauma, complication from surgery.

- Other causes, even though is not common, Fournier's gangrene can develop as a consequence of bone marrow malignancy, SLE, HIV infection, Crohn's disease. It may also result from iatrogenic injuries in the perineum.

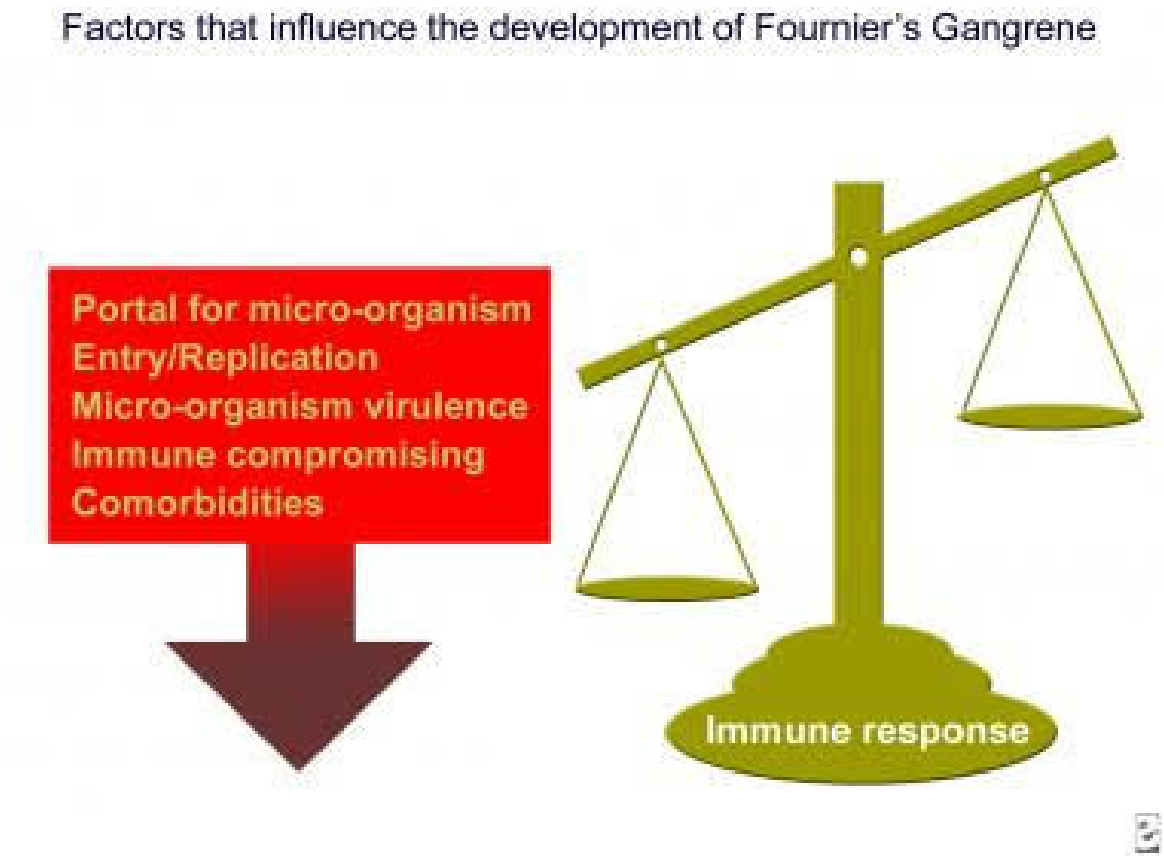
Co morbid diseases which are predisposing for the Fournier's disease are;

1) Diabetes mellitus (most common)
2) Cirrhosis
3) Morbid obesity
4) Malignancies
5) Vascular disease of pelvis
6) Immune suppression from systemic sisease
7) Due to steroid administration

PORTAL OF ENTRY

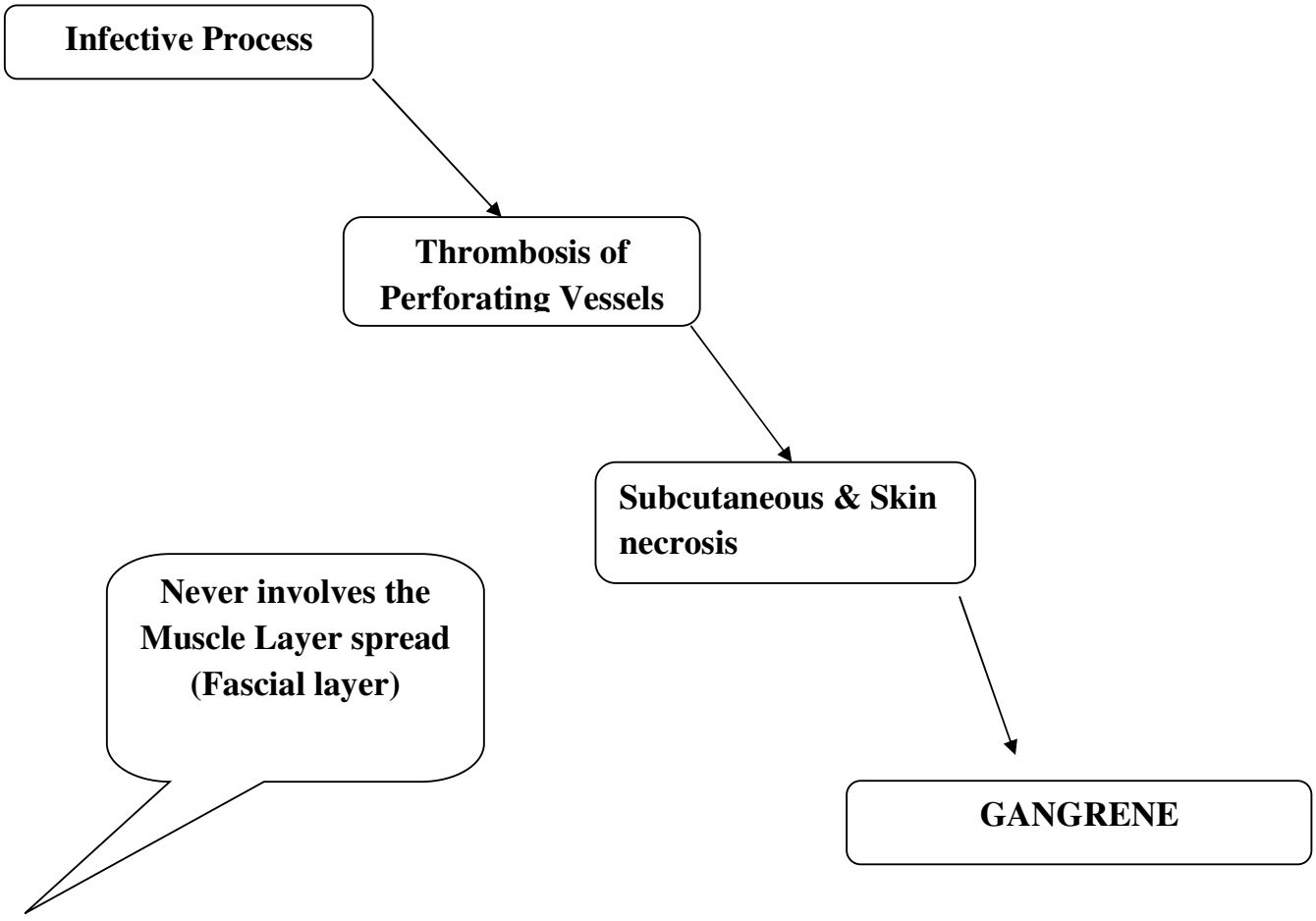


BALANCING FACTORS IN FOURNER'S GANGRENE



PATHOPHYSIOLOGY

PATHOLOGY



The following are the pathognomomic finding on evaluating the tissue involved:

- Superficial and deep planes undergone necrosis
- Nutrient arteriole undergone fibrinoid coagulation
- Infiltration of polymorphonuclear cells
- Presence of organisms in the affected tissue
- Presence of air in perineal area

Infection indicates a delicate balance between:

1) patient immunity, which is frequently compromised by co morbid systemic illness

2) Virulance of infective agent.

The etiologic agents which make the entry of the micro organism into the perineum, this will lead to initiation of infection which is been favoured by the compromised immune state more the virulance of organism faster and wider will be its spread.

The producton of toxins or enzymes as the result of virulence will result in the formation of an environment which favours the rapid multiplication of organism

In 1924 Meleney reported that a series of patients he saw in china with necrotizing infection were predominantly affected by streptococcal species. According to him necrotizing infections where solely due to streptococcal species, but subsequent studies conducted world wide has proven the multiorganism involment in necrotizing infections including Fournier's Gangrene. now adays a culture which purely showing streptococci are unusual, in culture it is found to be mixed with other 5 organisms.

Common causative organisms includes;

1) Streptococcal species

2) Staphylococcal species

3) enterobacteriaceae species

4) Anaerobic organisms

5) Fungi

MICROBIOLOGY

Common organisms:	
E Coli*	
P. Mirabilis	Polymicrobial
K. Pneumoniae	(Aerobic+ Anerobic)
Streptococci*	
Bacterioides*	Type 1 infection
Staphylococci*	Type 2 infection
Peptostreptococci,	
Clostridia,	
Pseudomonas	
*Most common	

Most of the studies concluded that the multiorganism involment of this disease is needed to form the synergic production of enzymes that result in the fast multiplication and wider spread of infection.

For example, among the group of organisms one may be responsible for the production of enzyme which result in the coagulation of nutrient vessels. As a result thrombosis of the nutrient vessel will take place which will eventually reduce the blood supply making the oxygen tension in the tissue to fall

Thus created tissue hypoxia will facilitate the growth of facultative anaerobes and microaerophilic organism. Later these micro organisms reduces enzymes like collagenase and lecithinase which causes the facial barriers destruction, this will eventually result in the rapid spread of infection. The necrosis and digestion of the fascia is the pathognomomic feature of this disease.

Particularly, if the plane of fascia can be dissected out carefully by means of blunt dissection, it is likely to have involvement of ischemic process of infection. Therefore any such tissue if present should be removed. The fulminant or late stage of the disease can result in spreading of disease from the fascial layers of external genitalia through the perineum, some times into the thigh region and also into torso region.

CLINICAL FEATURES:

- TRIAD: Severe pain+ swelling +fever
- Bullae; crepitus (50-62%)
- Gangrene
- “Dirty dishwater fluid”
- Cutaneous signs >> Tip of an iceberg

The distinctive feature of Fournier gangrene is intense pain and tenderness in the genitalia

The clinical course of the disease is as follows:

1. Prodromal symptoms: fever & lethargy, 2-7 days
2. Intense genital pain and tenderness associated with oedema, erythema and dusky appearance of the overlying skin, subcutaneous crepitations.
3. Macroscopic gangrene of portion of the genitalia; pus discharge from wounds.

There also occurs systemic effect due to fournier's gangrene which includes localized tenderness with features of inflammation to profound shock with multiorgan failure . Generally greater the degree of necrosis, greater the systemic effects. It usually affects males of 60-70yrs with co-morbid diseases females are affected less frequently.

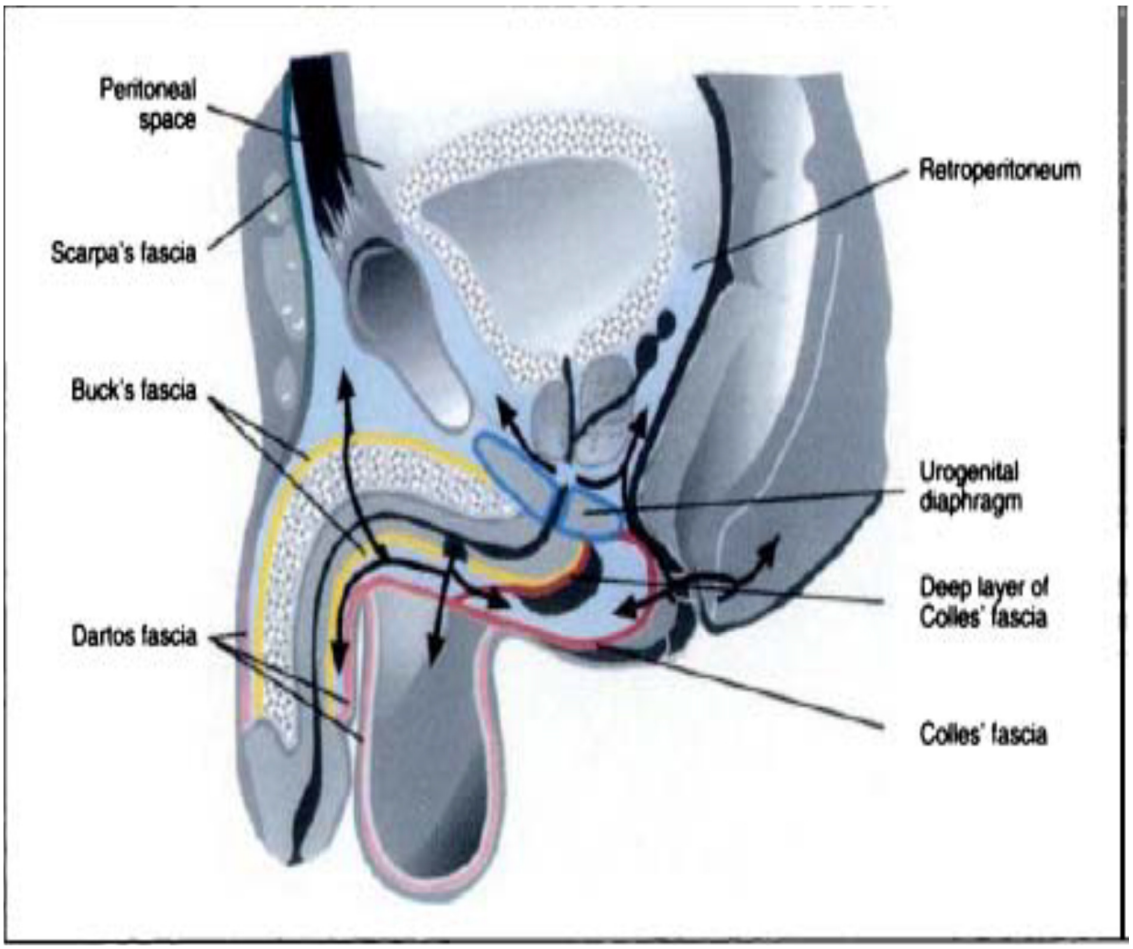
RELEVANT ANATOMY:

Progression of the Fournier gangrene is influenced by the complicated anatomy of the male external genitalia. The bacteria responsible spreads along the fascial planes not involving the deep structures and the overlying skin to varying degrees. This process influences both initial wound debridement and subsequent management. The clinician treating Fournier gangrene requires a detailed knowledge about the anatomy of the lower urinary tract and external genitalia.

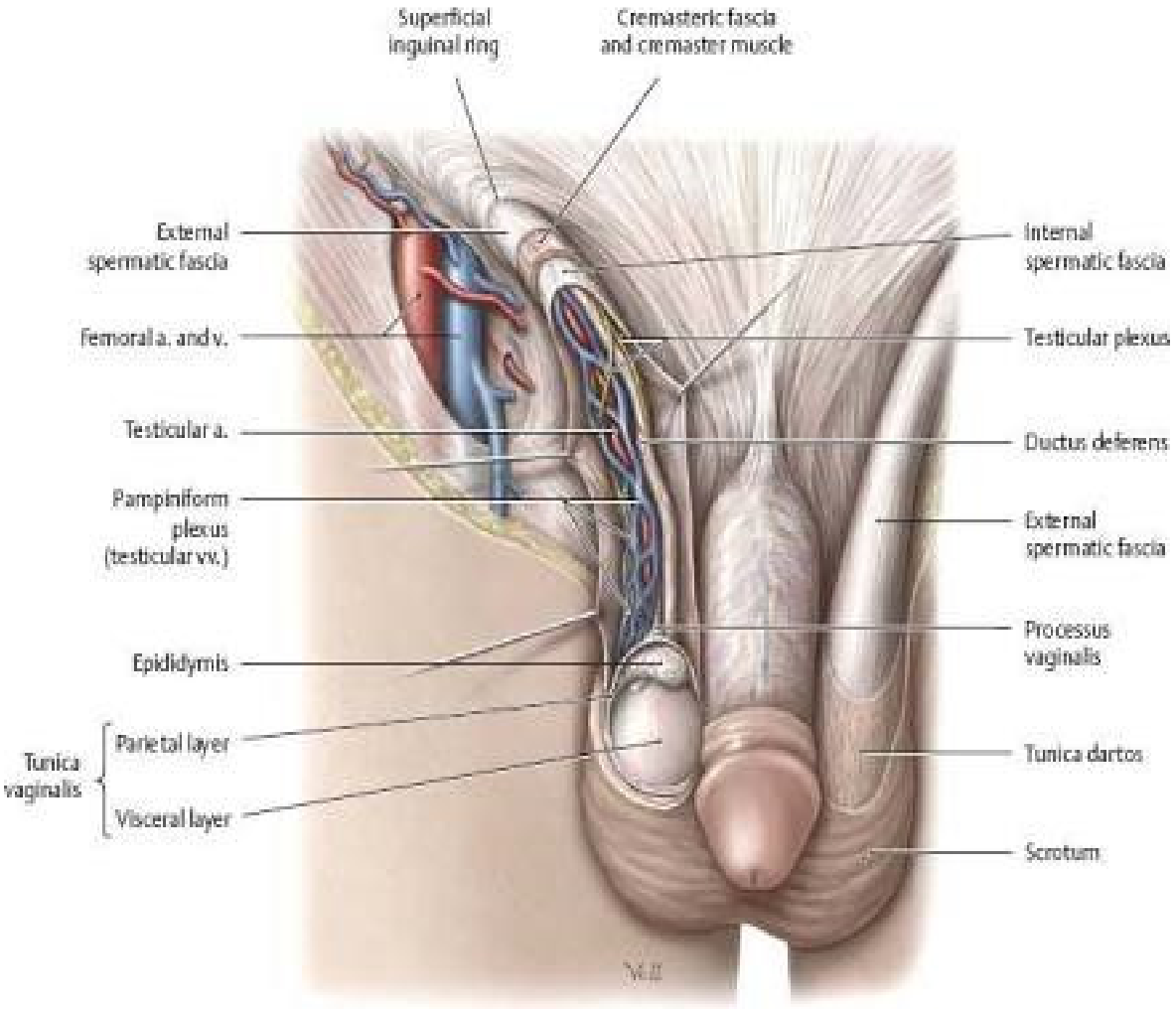
SKIN AND SUPERFICIAL FASCIA

Since the fascial planes are predominantly affected by the gangrene, the appreciation of the anatomy of the skin, subcutaneous layers and anterior abdominal wall is very important.

SKIN AND FASCIA



INGUINAL AREA AND EXTERNAL GENITALIA



The skin craniad to the inguinal ligament is strenghthened by the Camper's fascia, which contains fat tissue of variable thickness and superficial vessels to the skin also passes through it. Deep to it is the Scarpa fascia, in the perineum it merges with the Colles fascia while it is continuous with the Dartos fascia.

Space between the Scarpa fascia and the external oblique allows the spread of perineal infection onto the anterior abdominal wall. Superiorly the Scarpa and Camper fasciae amalgamate and get affixed to the clavicles, thus limiting the infection that may have commenced in the perineum. Colles fascia runs uninterupted with the superficial Dartos fascia of the scrotum.

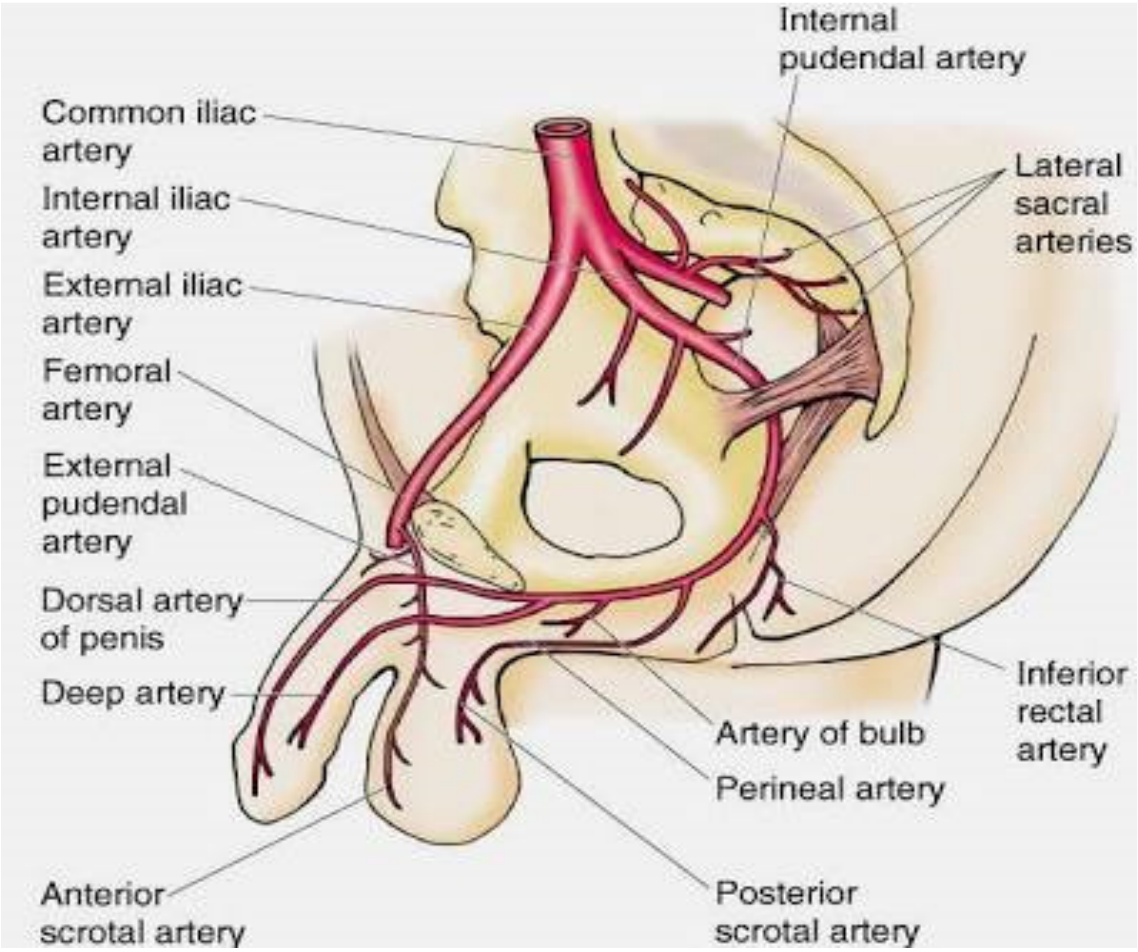
The perineal membrane along with Colles fascia delineates the superficial perineal space. This space encompasses the membranous & bulbar urethra and the bulbourethral glands. Infectious diseases of the male urethra, bulbourethral glands, perineal structures or rectum can spread onto the superficial perineal space and can extend into the scrotum or onto the anterior abdominal wall ultimately till the level of the clavicles.

BLOOD SUPPLY TO THE SKIN OF THE LOWER ABDOMEN AND GENITALIA

The lower aspect of the anterior abdominal wall is supplied by branches from the inferior epigastric and the deep circumflex iliac arteries. The scrotal wall receives supply from the external and internal pudendal artery branches. Each of these vessels except the internal pudendal is enclosed by the Camper's fascia and hence may undergo thrombosis during progression of Fournier gangrene.

The blood supply of the skin of the anterior scrotal and abdominal wall is put at risk if thrombosis occurs. As the posterior aspect of the scrotal wall is supplied by the internal pudendal it is spared and can be used for reconstruction following the cessation of the disease.

BLOOD SUPPLY

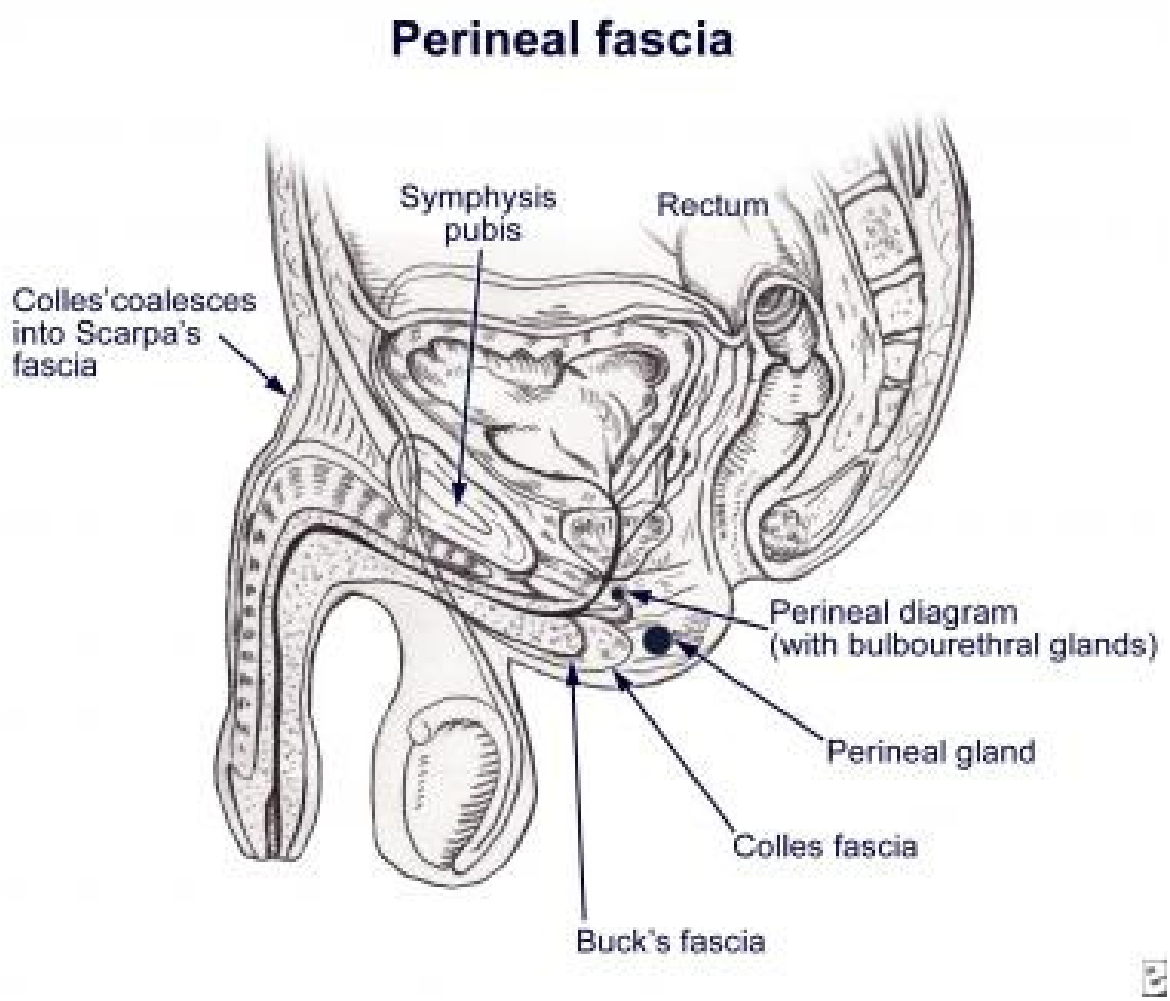


PENIS AND SCROTUM

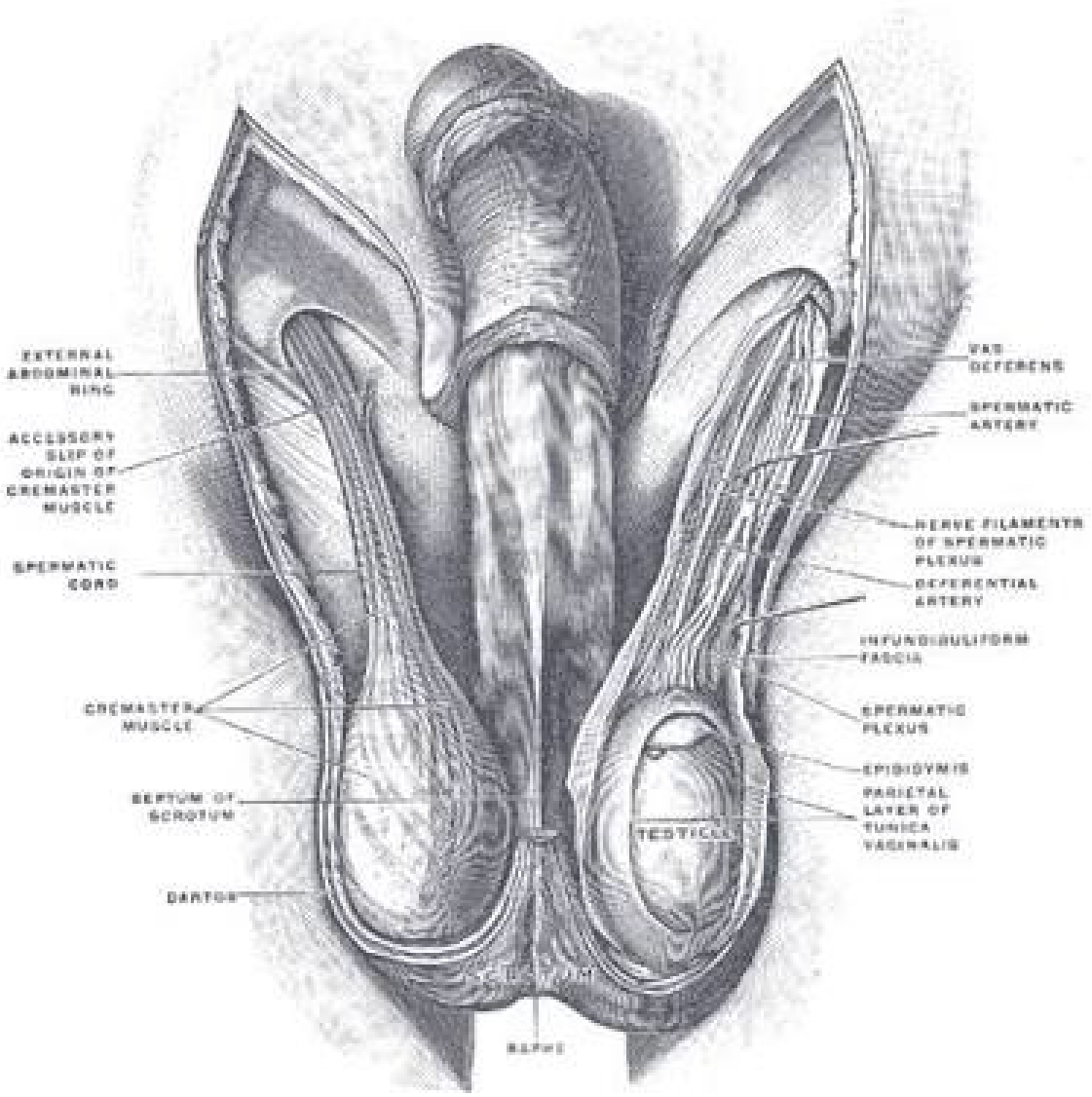
Several fascial layers distinct from the Dartos fascia invest the contents of the scrotum namely the testicles, epididymis and the cord structures.

The external spermatic fascia which is continuous with the external aponeurosis of the superficial inguinal ring [present in the external oblique] forms the superficial most layer of the testis and the cord. Deep to it is the internal spermatic fascia which is continuous with the transversalis fascia. The erectile bodies of the penis, the corpora cavernosa and the anterior urethra is enclosed by the deep "Buck" fascia which merges to the tunica albuginea of the corpora cavernosa deep in the pelvis. These fascial layers do not get involved in case of an infection of the superior perineal space and hence limits the extent of tissue destruction. The corpora cavernosa, urethra, teatis and cord structures are unharmed in Fournier gangrene while the deep and superficial fascial planes along with the overlying skin are destroyed.

PERINEAL FASCIA



Anatomy of external genitalia



INVESTIGATIONS:

CLINICAL DIAGNOSIS

- "Finger test"/ "frozen section"
- Plain X-ray: Air in soft tissues
- USG: scrotal wall thickening & subcutaneous air/ peri-testicular air
- CT:
    - Accurately defines the extent of necrosis
    - Elucidates the cause

LAB STUDIES:

- Complete history and physical examination
    - Palpation of the genitalia and perineum and also the digital rectal examination must be under direct scrutiny
    - The examiner should be alerted by the presence of any fluctulance, soft tissue crepitations, localised tenderness or occult wounds in any of the above mentioned sites as they are indications of Fournier disease.

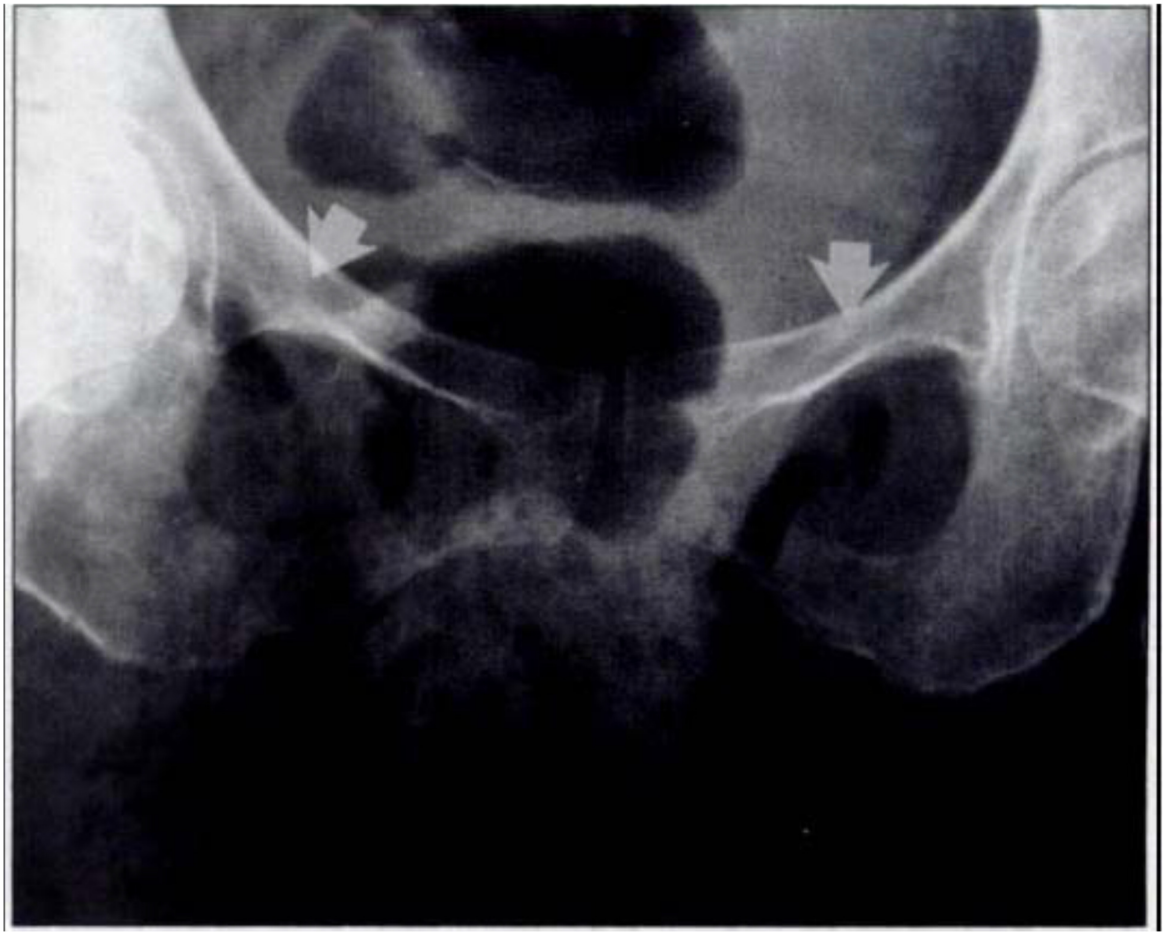
- Biochemistry workup:
    - Electrolyte disturbances
    - Laboratory evidence of dehydration [elevated BUN/creatinine ratio]
    - Glucose intolerance [in case of pre-existing diabetes or any sepsis induced metabolic disturbance].
- Blood tests:
    - Complete blood count: look for any infection induced immunologic stress, the adequacy of the red cell mass, also check for any possibility for sepsis induced thrombocytopenia.
    - Blood culture: check for septicaemia
    - Coagulation profile: Prothrombin time, activated partial thromboplastin time, platelet count and fibrinogen level → for sepsis induced coagulopathy
    - If surgical exploration is undertaken consider type and screen.

- **Other**: any investigations needed to study exacerbation of a co morbid condition is warranted. It may include : ECG and Cardiac enzyme evaluation in patients with coronary artery disease

IMAGING STUDIES:

- RADIOGRAPHY

Considered If the findings from the clinical examinations are not conclusive. Imaging techniques helps in easy identifiaction of air with in the tissues than by physical examination



If gas inside soft tissue or crepitations of subcutaneous tissue is demonstrated it can be considered as an absolute indication for surgical exploration.

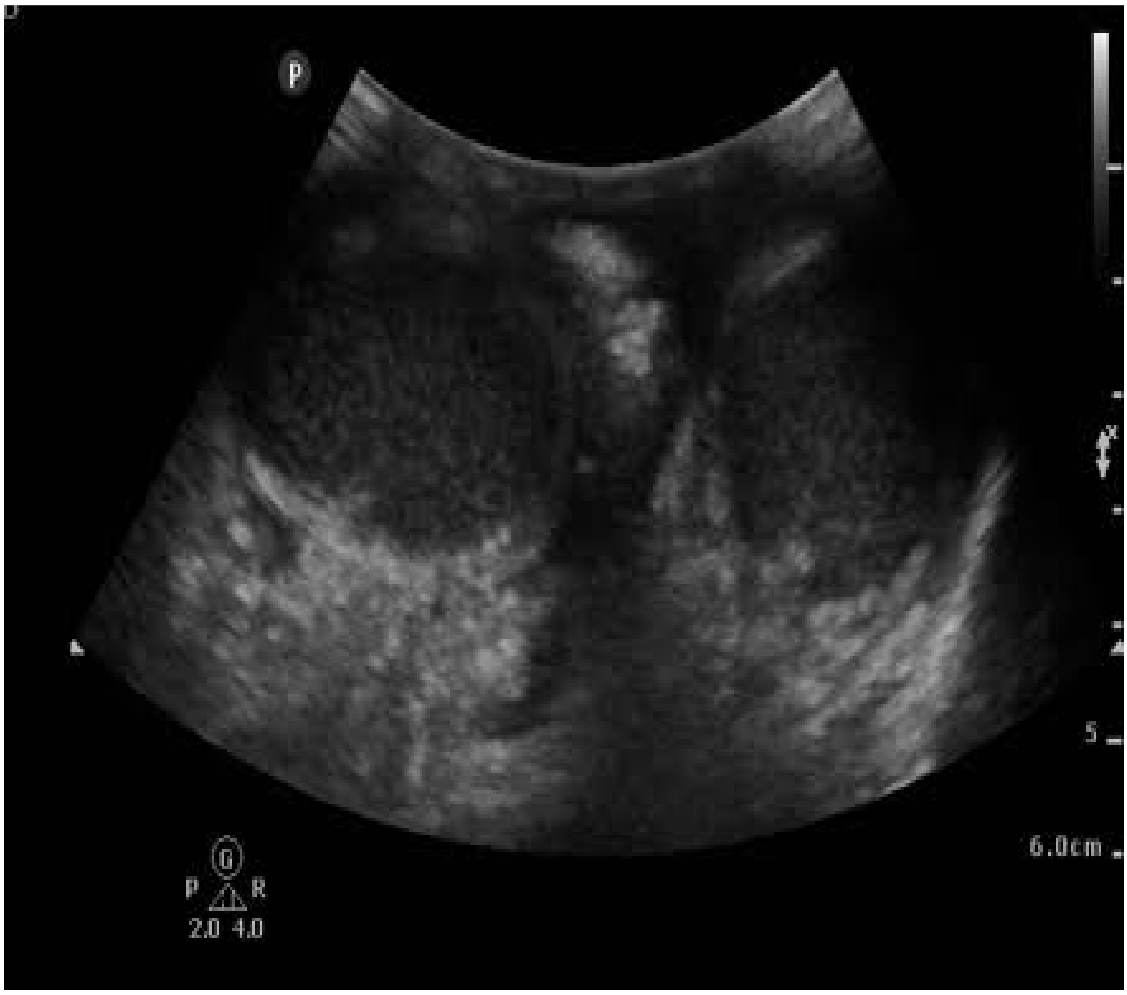
Initially imaging studies should start from plain film radiography. It is helpful in revealing moderate to large amount of gas filled area of soft tissue or foreign bodies.

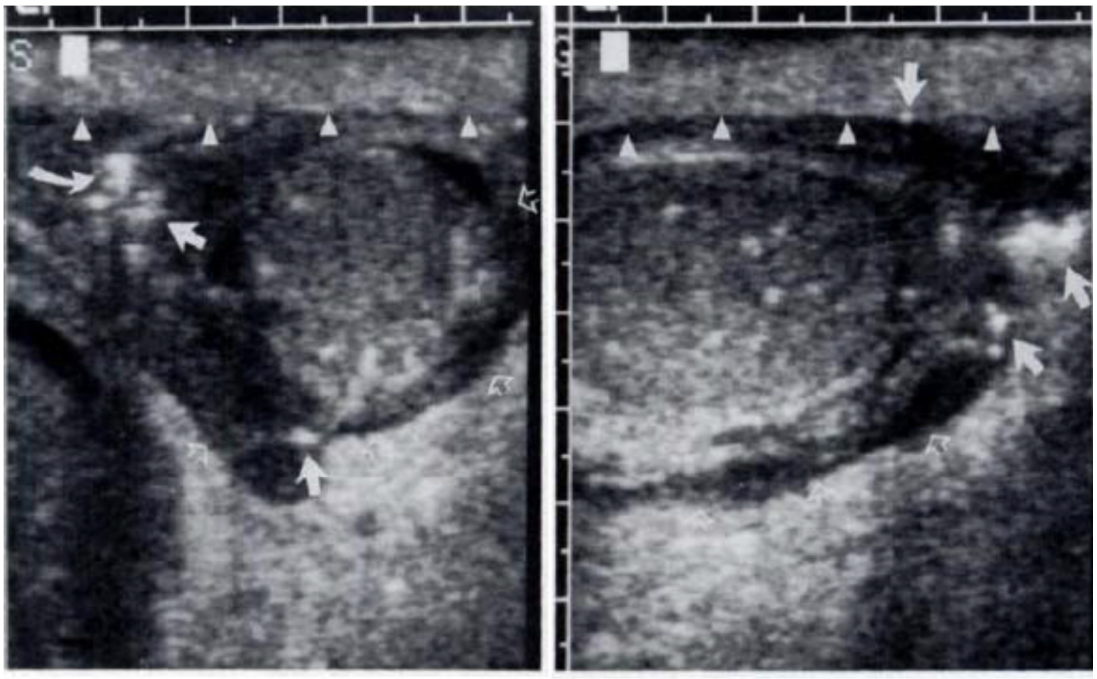
● ULTRASONOGRAPHY

Ultrasonography is useful in detecting fluid or air within the tissue. Other than that it can also be used to assess the blood flow to the testes, in the condition where testicural torsion is considered as adifferential diagnosis.

Major disadvantage caused by this modality is that it will result in application of direct pressure over the affected tissue. Patients may not be able to tolerate the pain generated do to this.

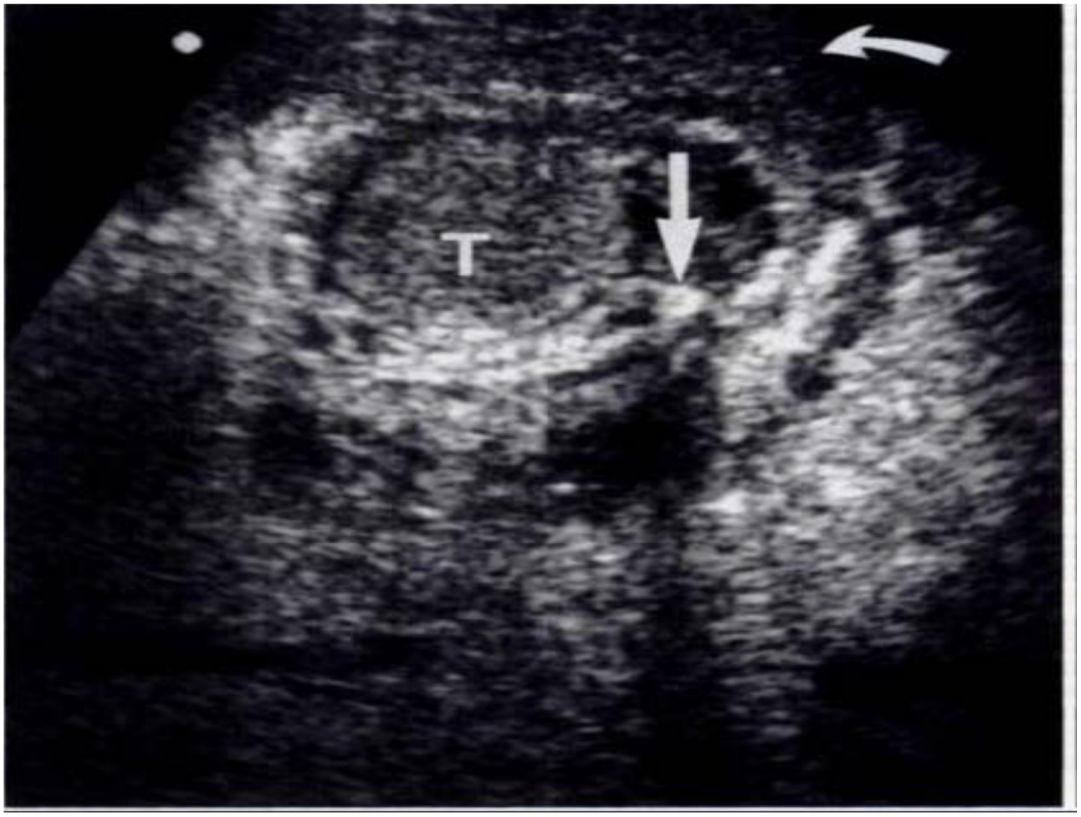
USG SCROTUM SHOWING GANGRENE





a. b.

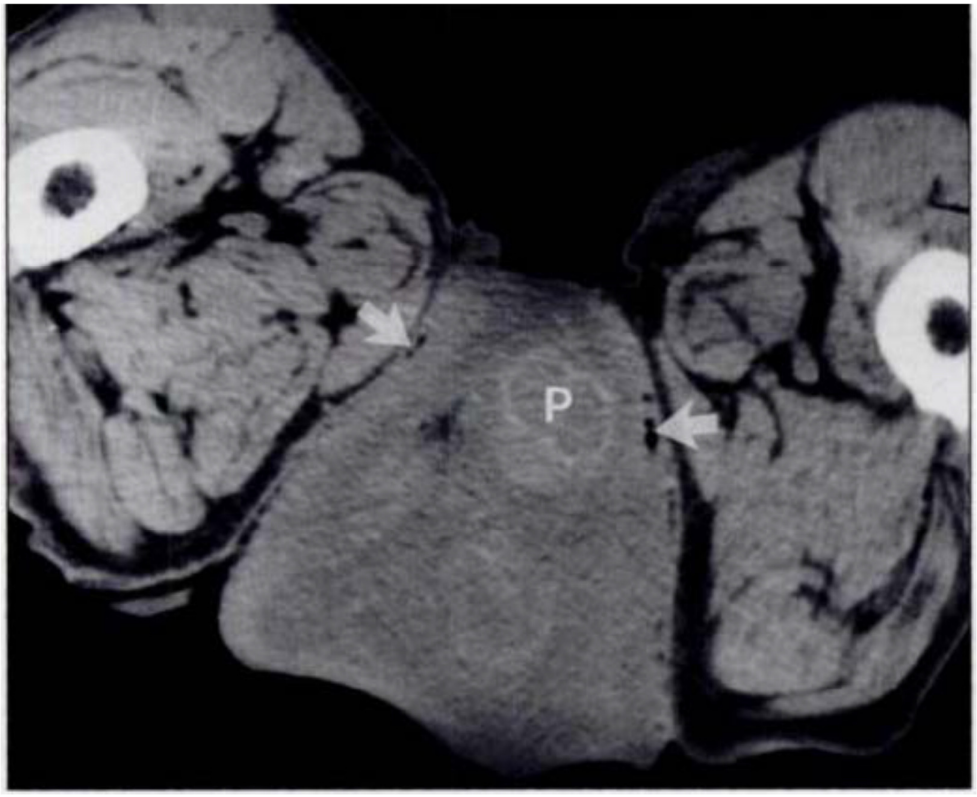
(a)Transverse and (b) longitudinal USG scrotum showing skin thickening (marked arrow head), Periitesticular fluid (open arrow), subcutaneous air (straight solid arrow) and air filled median raphe(curved solid arrow)



USG scrotum showing soft tissue thickening (curved arrow) and cutaneous air(straight arrow. T- testicle.

● **CT SCANNING**

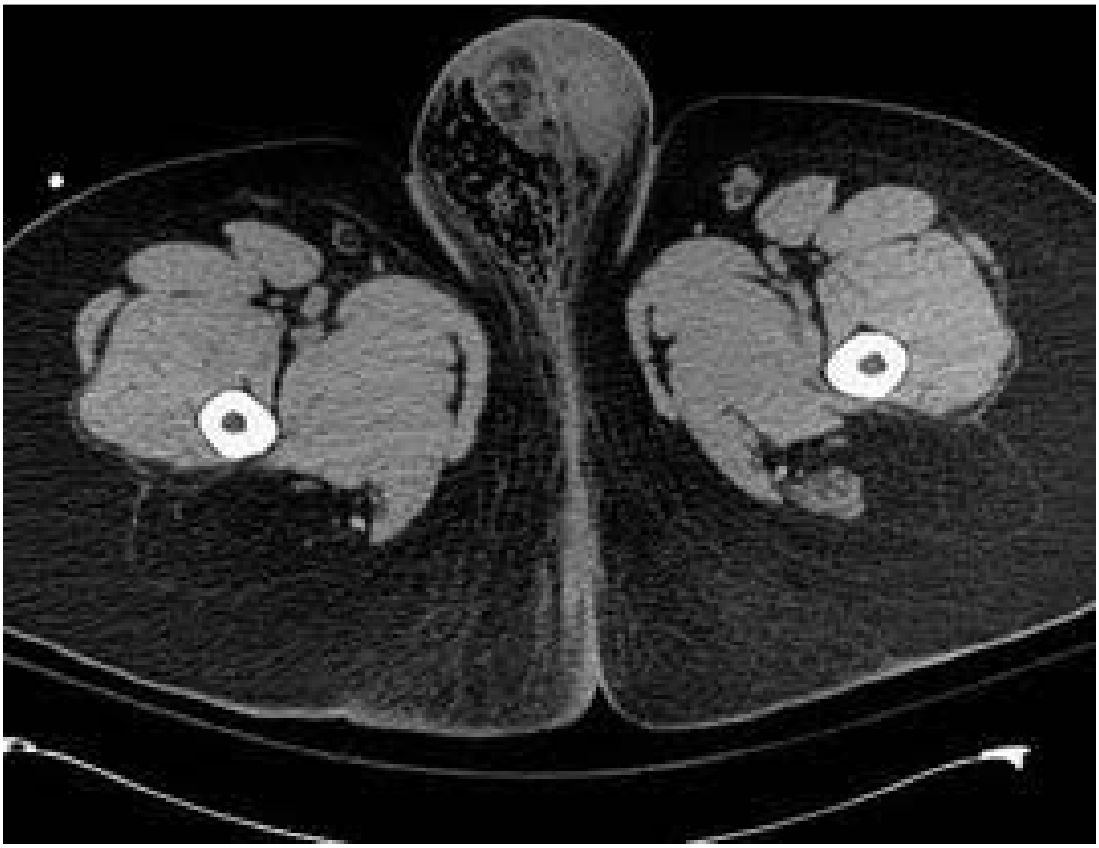
- CT scanning is helpful in detecting small amount of gas than in case of plain radiography. It can be also used to track fluid collection along the deep fascial planes.
- CT scanning is common in most of the hospitals and is considered as the diagnostic tool of choice



CT scan showing extensive inflammatory soft tissue swelling of scrotum and minimal amount of air in soft tissue(arrow).Inflammatory can also involves the penis.

●**MRI**

MRI studies yields more details about soft tissue than in CT scanning, but it causes more logistical challenges, especially in critically ill ones

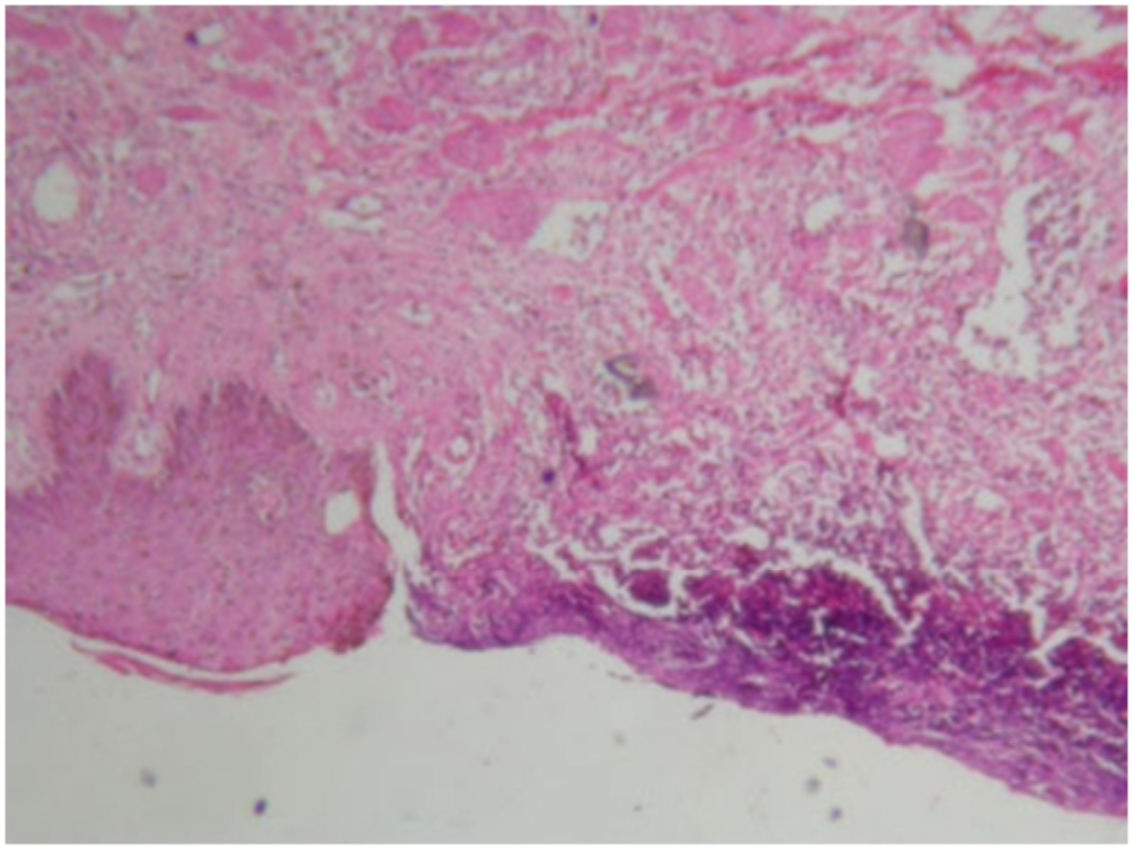


MRI

●HISTOLOGICAL FINDINGS

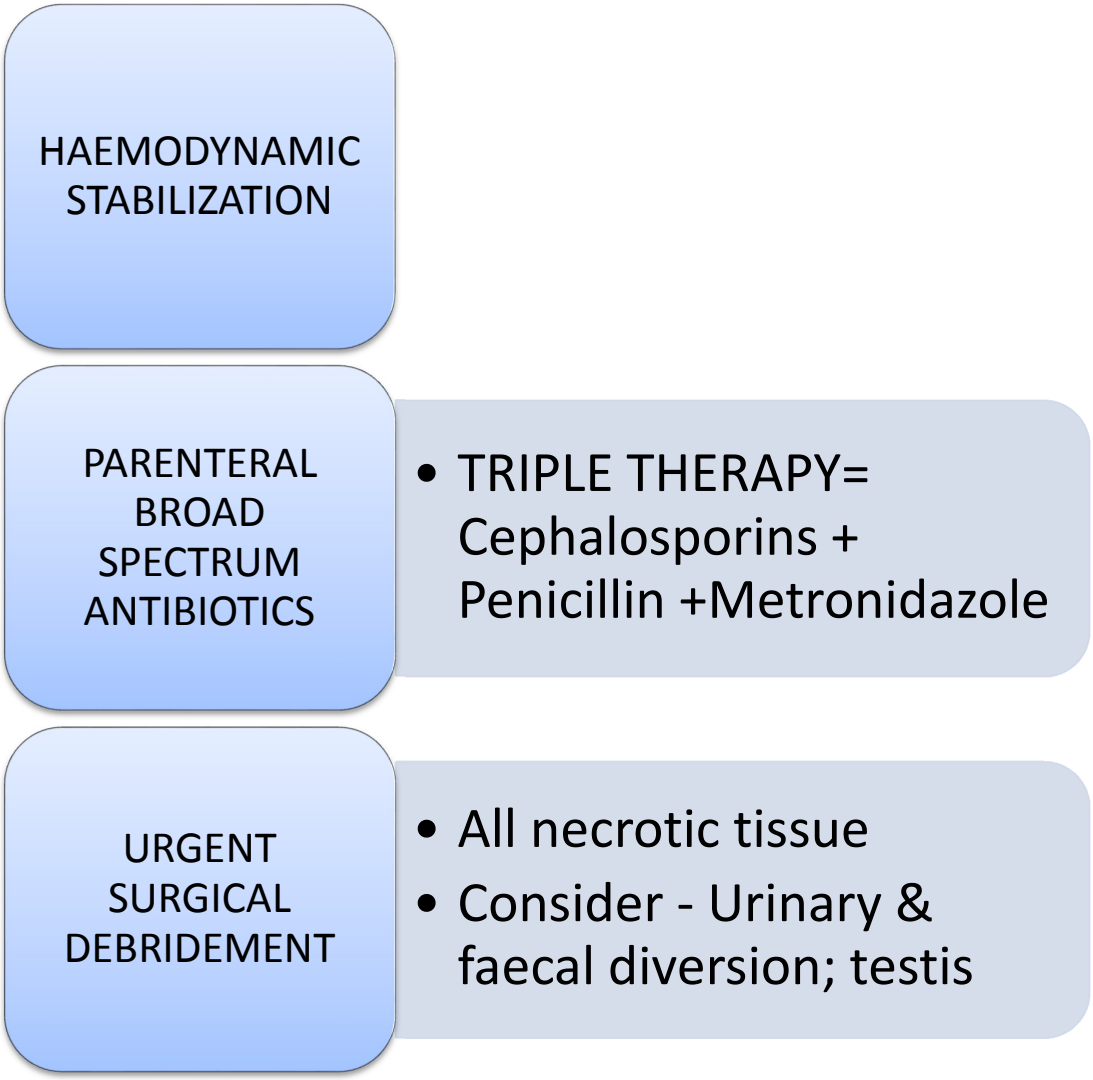
The pathological evaluations reveal the following finding

- Superficial and deep fascial planes undergone necrosis
- Nutrient arterioles undergone fibrinoid coagulation
- Infiltration of polymorphonuclear cells
- Presence of organisms in the affected tissue



The main finding that indicates Fournier disease is Fibrinoid thrombosis of nutrient vessels that supply the deep as well as superficial fascia. Within the tissue , there is widespread necrosis of fascia with infiltration of inflammatory cells necrotic debris and demonstration of causative microbes. Skin is often involved with inflammatory process in the late course of the disease, at the beginning deep tissues are involved with intact skin.

TREATMENT:


HAEMODYNAMIC STABILIZATION
PARENTERAL BROAD SPECTRUM ANTIBIOTICS
• TRIPLE THERAPY= Cephalosporins + Penicillin +Metronidazole
URGENT SURGICAL DEBRIDEMENT
• All necrotic tissue
• Consider - Urinary & faecal diversion; testis

MEDICAL THERAPY:

Fournier gangrene can be treated by several techniques, one of them being restoring the normal organ perfusion. Resuscitation to normal organ function and perfusion should be a priority over the diagnostic investigations in conditions where the patient presents with systemic toxicity manifesting as hypo perfusion or organ failure, as the investigations may compromise the resuscitative arbitrations.

Broad spectrum antibiotic therapy is also used. The spectrums covered are: Staphylococci, Streptococci, Enterobacteriaceae family and anaerobes. Ciprofloxacin and Clindamycin are considered as an empiric regimen for therapy. Due to its gram positive and anaerobic spectrum activity, Clindamycin is used in the treatment of necrotizing soft tissue infections, also it has higher response rates compared with penicillin and erythromycin in experimental animal specimen of streptococcal infection even in circumstances where the treatment has been delayed (Stevens 1998).

Anti-fungal agents [Amphotericin-B or Caspofungin] can be used if the initial tissue stains [i.e. KOH stain] shows fungi. In cases associated with sepsis syndrome there is a heightened cytokine response due to the super antigens like streptotoxins [A,B] . These antigens can be counterbalanced by intravenous immunoglobulin therapy [IVIG]. Along with proper antibiotic coverage and a complete surgical debridement it has been proved to be a good adjuvant (Cawley, 1999).

If obtainable hyperbaric oxygen can be administered as it has shown to have some favorable results. This therapy has to be balanced with the stability of the patient and the surgical therapy should not be put off for long.

SURGICAL THERAPY:

1) Authenticating the Diagnosis

   i) The tentative diagnosis based on clinical examinations or investigations is verified by incision into the area of greatest clinical concern with the patient under anesthesia.

   ii) The diagnosis can be established if obvious gangrenous tissue present or if any purulence is drained.

   iii) At times severe cellulitis can be seen as a manifestation of early stage Fournier disease. The fascia will appear edematous in comparison with the grey black appearance of well-established Fournier gangrene. During such situations a frozen section evaluation has to be carried out to rule out the potential for early necrotizing disease.

2) Excision of the necrotic tissue

   i) Once the diagnosis is validated all necrotic material needs to be excised. The skin should be incised and retracted to expose the full extent of the underlying fascial and subcutaneous necrosis.

ii) Tissue specimens are collected for aerobic & anaerobic cultures and also for histological assessment.

iii) If the overlying skin has been markedly debilitated due to impaired blood supply, because of characteristic thrombosis of the nutrient vessels it also should be excised. Authors recommend radical excisional debridement with electrocautery as it reduces blood loss intra-operatively if the area of involvement is extensive.

iv) Repeated operative debridement procedures should be carried out for complete eradication of the infection taking into consideration the fulminant nature of this necrotizing process.

v) The appropriate antibiotic regimen against the causative organisms can be administered to the patient once the results of the tissue cultures are known. The antibiotic therapy should be continued for about 10-14 or until the reconstruction has been accomplished.

vi) Fecal diversion is considered in subsequent operative procedures if the perineal involvement is extensive; this helps in eliminating the potential for fecal contamination of the wounds. It is mandatory when the perianal area is extensively involved but not done if necrosis is confined to the genitalia.

vii) Urinary diversion is usually done using a urethral catheter, suprapubic cystostomy is used when it's impossible to drain the bladder through the urethra because of some underlying pathology (e.g. stricture disease, prostatic hypertrophy).

viii) Most of the times the testicles are spared from the necrotizing process. If they are not affected, the exposed testicle is placed in a subcutaneous pocket so that desiccation can be avoided. If they are affected in the necrotic process or their viability is challenged orchidectomy is performed.

ix) Healthy granulation tissue develops once the infection has been completely eradicated; this signifies the apt time to advance to reconstruction.

x) Wound healing in these patients has been shown to improve through the usage of Vacuum-assisted closure [VAC] (Kovacs 2001). Implementation of this procedure can considerably shorten the hospital stay and accelerate the grafting and flap placement process.

3) Options for reconstruction

i) Primary closure of skin [if possible].

ii) Local skin flap coverage.

iii) Split-thickness skin grafts.

iv) Medial thigh flap.

COMPLICATIONS:

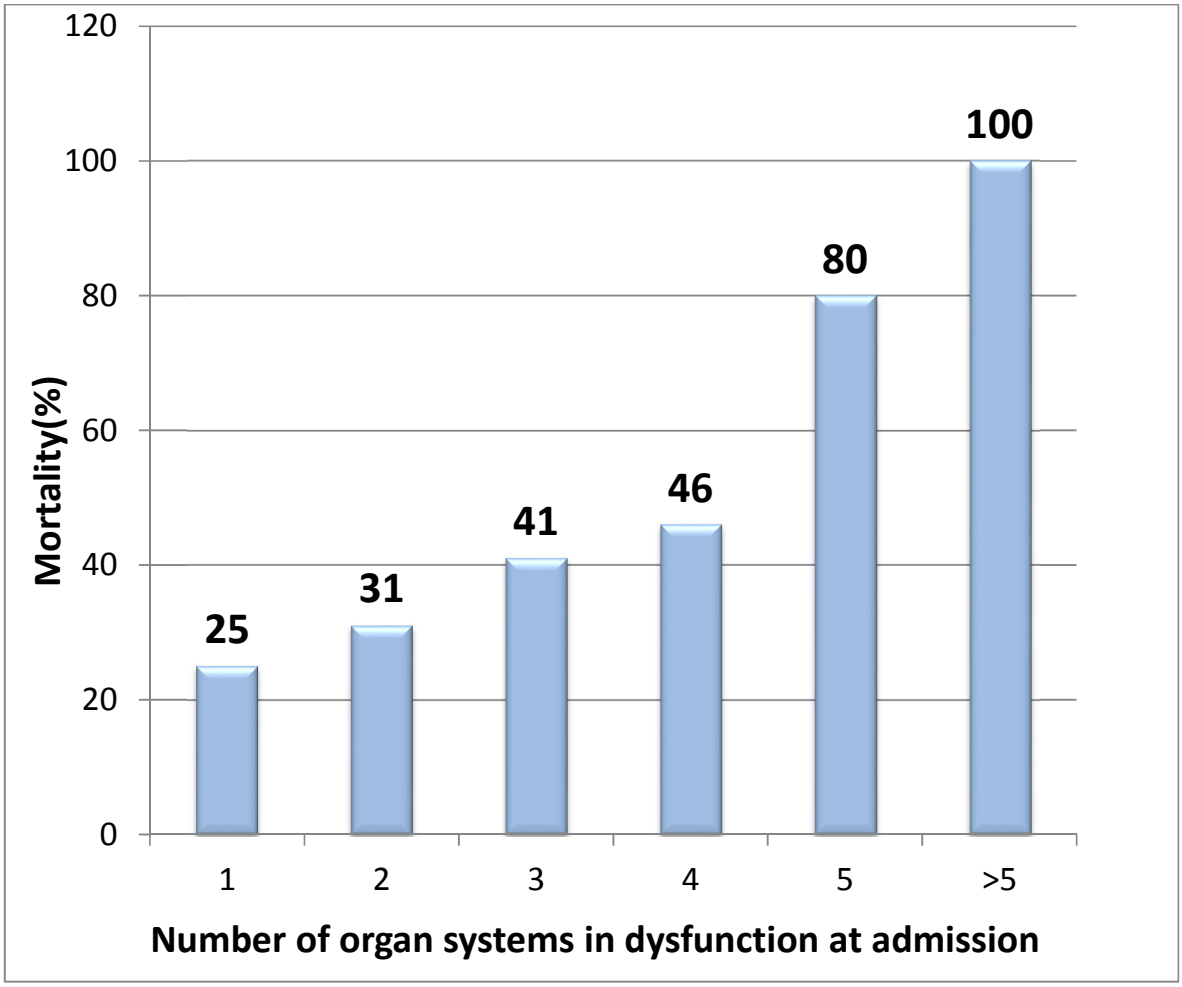
- Acute Renal failure
- ARDS/Pneumonia
- Gastrointestinal Bleeding
- Heart failure
- Hypocalcaemia

The chief complication of Fournier disease is unresolved sepsis, which is often caused by either

- Unestablished cause of infection [e.g. peptic ulcer perforation, appendicitis, diverticulitis] or invasion of the necrotizing process outside the obvious wound [CT scanning can be used to evaluate these 2].
- Severe acute illness complications [e.g. line sepsis, bacterial endocarditis, pneumonia].
- The abundance of co-morbid conditions (e.g. Acute myocardial infarction, Respiratory failure, Pressure ulcerations, delirium) or the bed rest conditions affecting acutely ill patients (e.g. pulmonary embolus, deep venous thrombosis, atelectasis, pneumonia)

OUTCOME & PROGNOSIS:

- Mortality rates: 3-40%
- Poor prognostic factors: [*- Most important prognostic factors]
    - Age
    - Female gender
    - Anorectal causes*
    - Number of organs in failure at the time of admission*
    - Delay in presentation and treatment*
    - Diabetes or HIV*



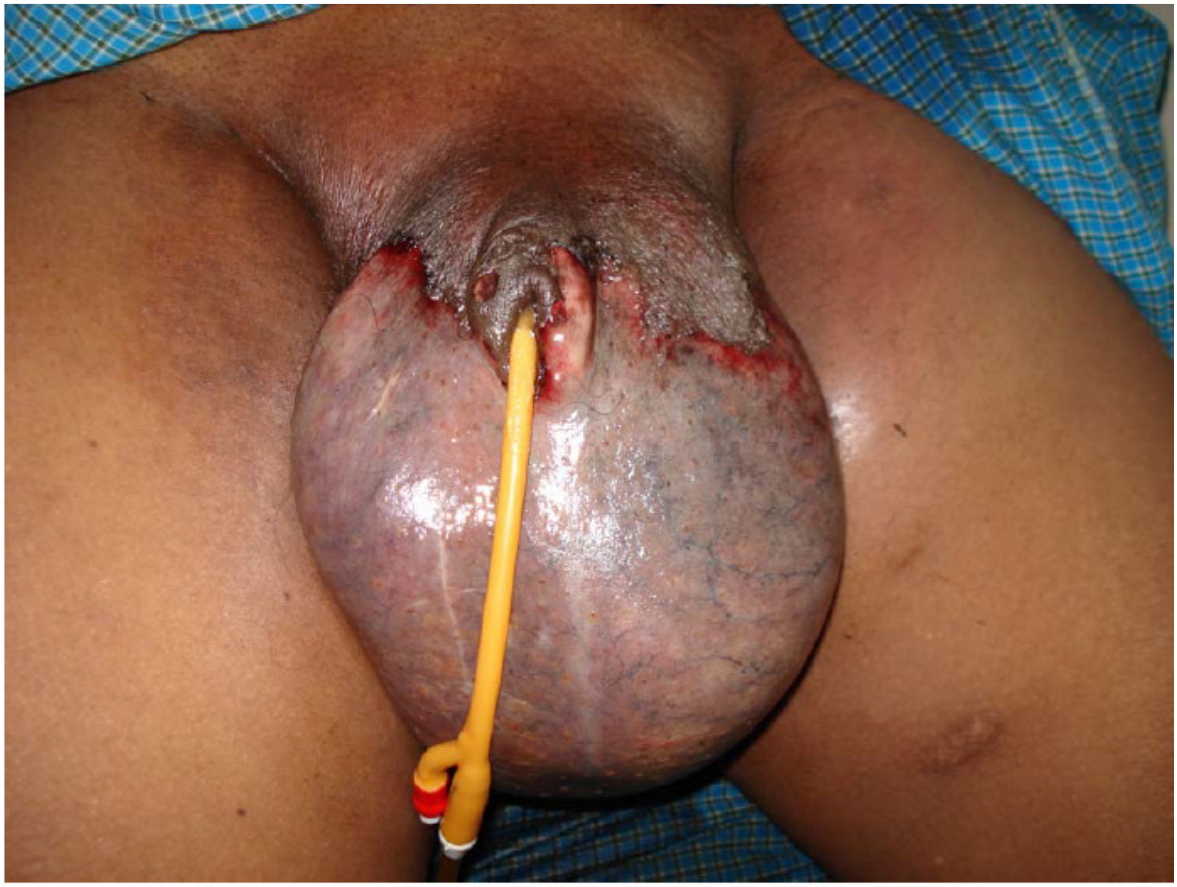
Even with the presence of modern supportive measures, the reported mortality rate is still high and this is due to the aggressive nature of the infection and the underlying co-morbid conditions.

CONCLUSION:

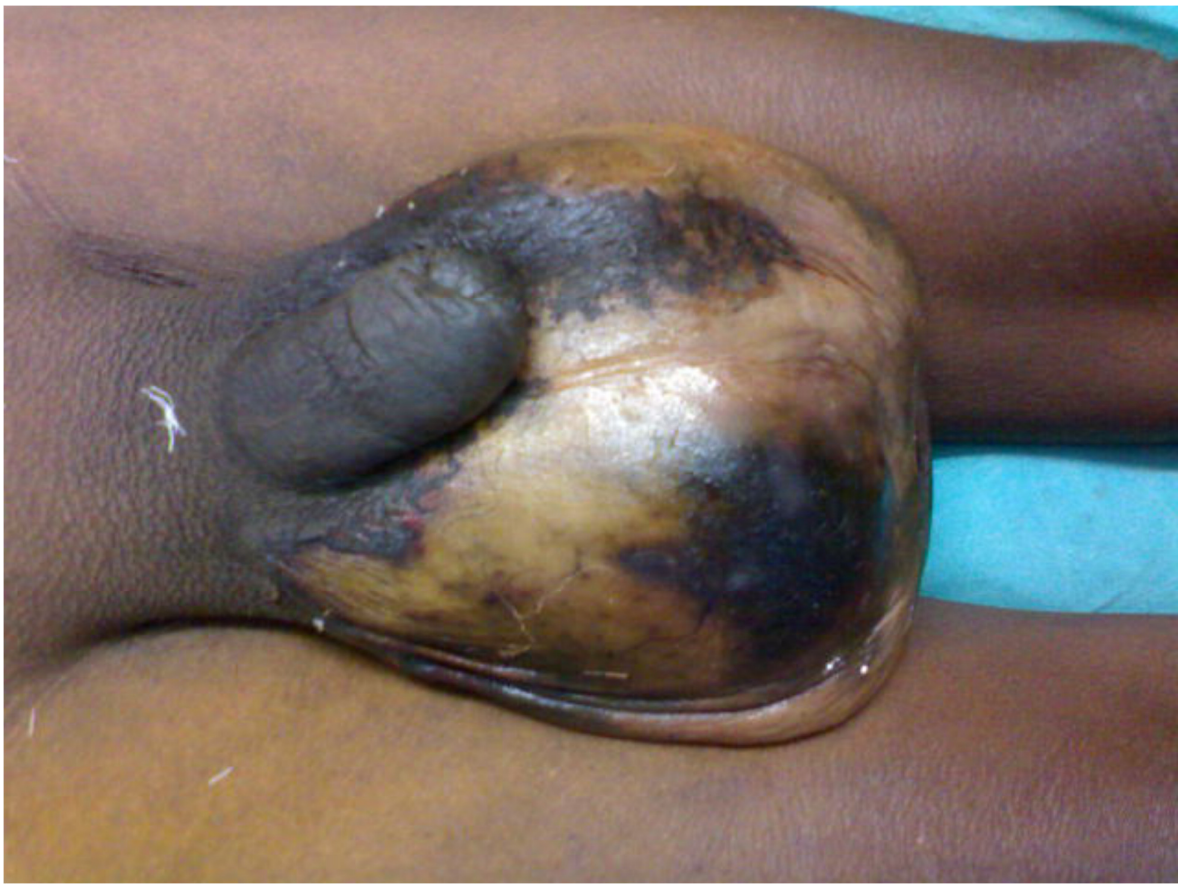
Fournier's gangrene is a dare emergency disorder that needs early diagnosis and treatment. So even minor infection to perineal region should be give due attentions negligence may lead to life threatening complication

So in Fournier's gangrene early resuscitation of vitals with transfusion of blood and blood products if needed, the adequate antibiotic care and early extensive thorough wound debridment will certainly improve the outcome.

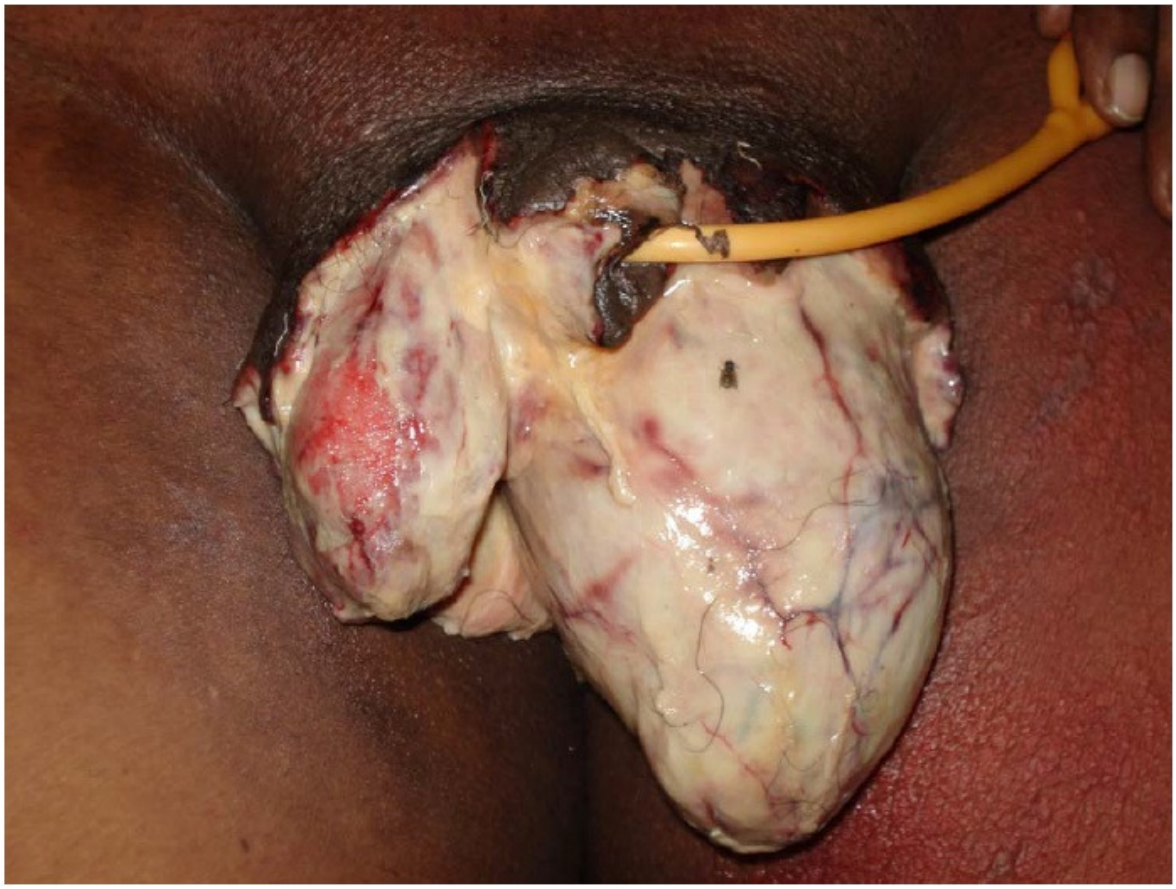
Case 1



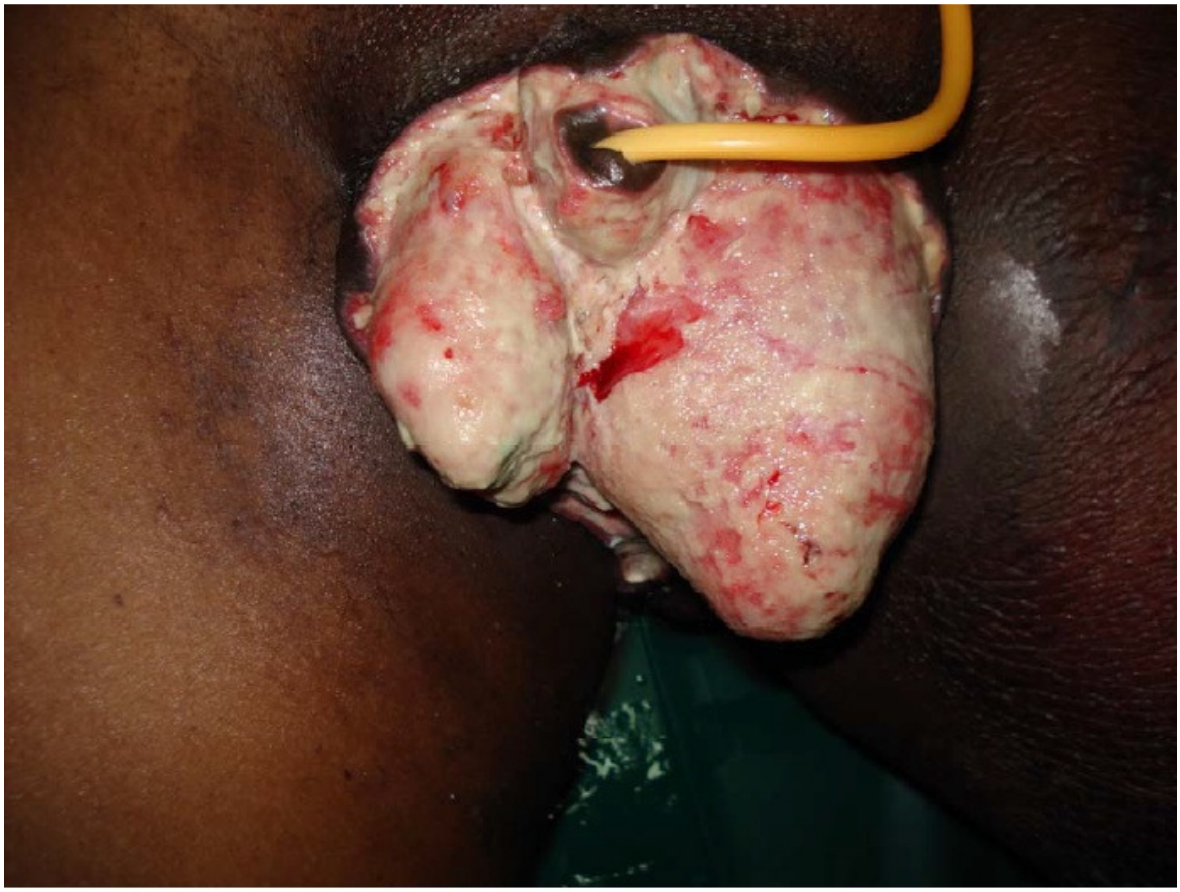
Case 3



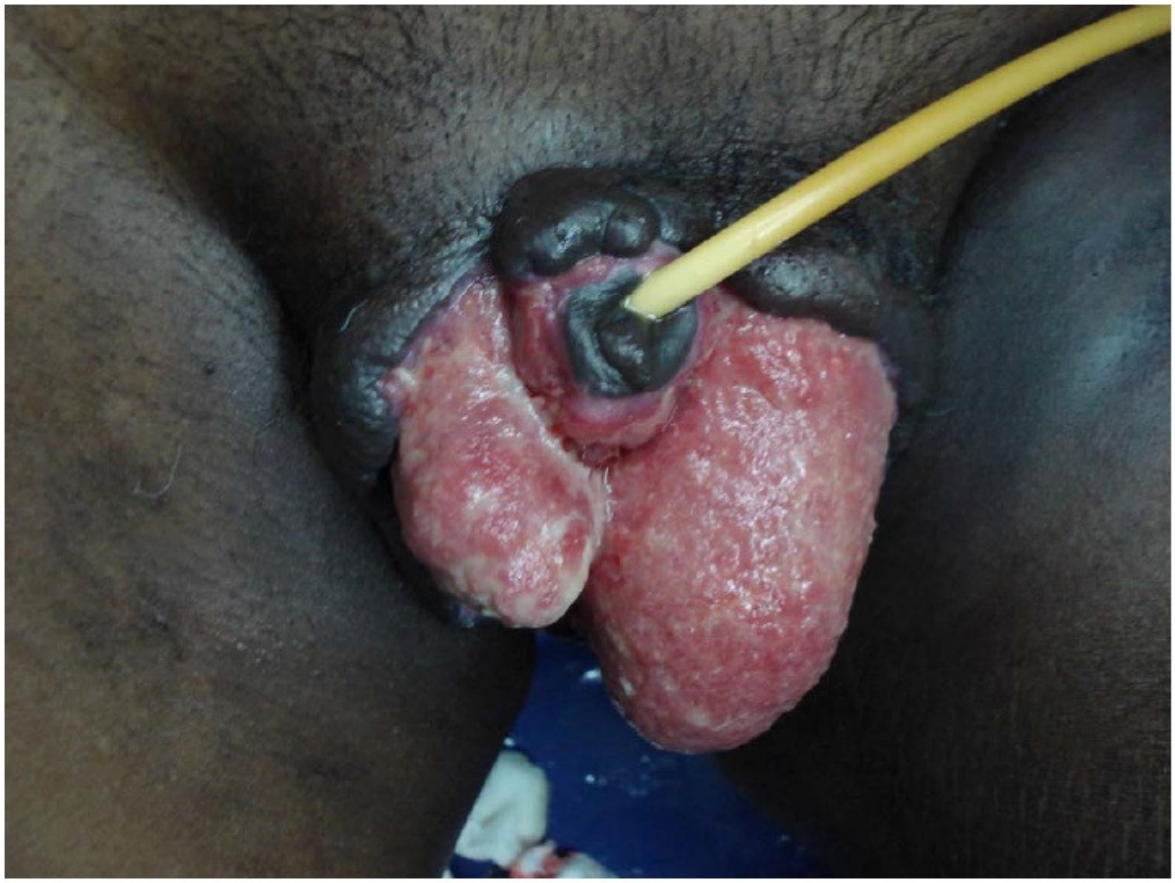
Case 5



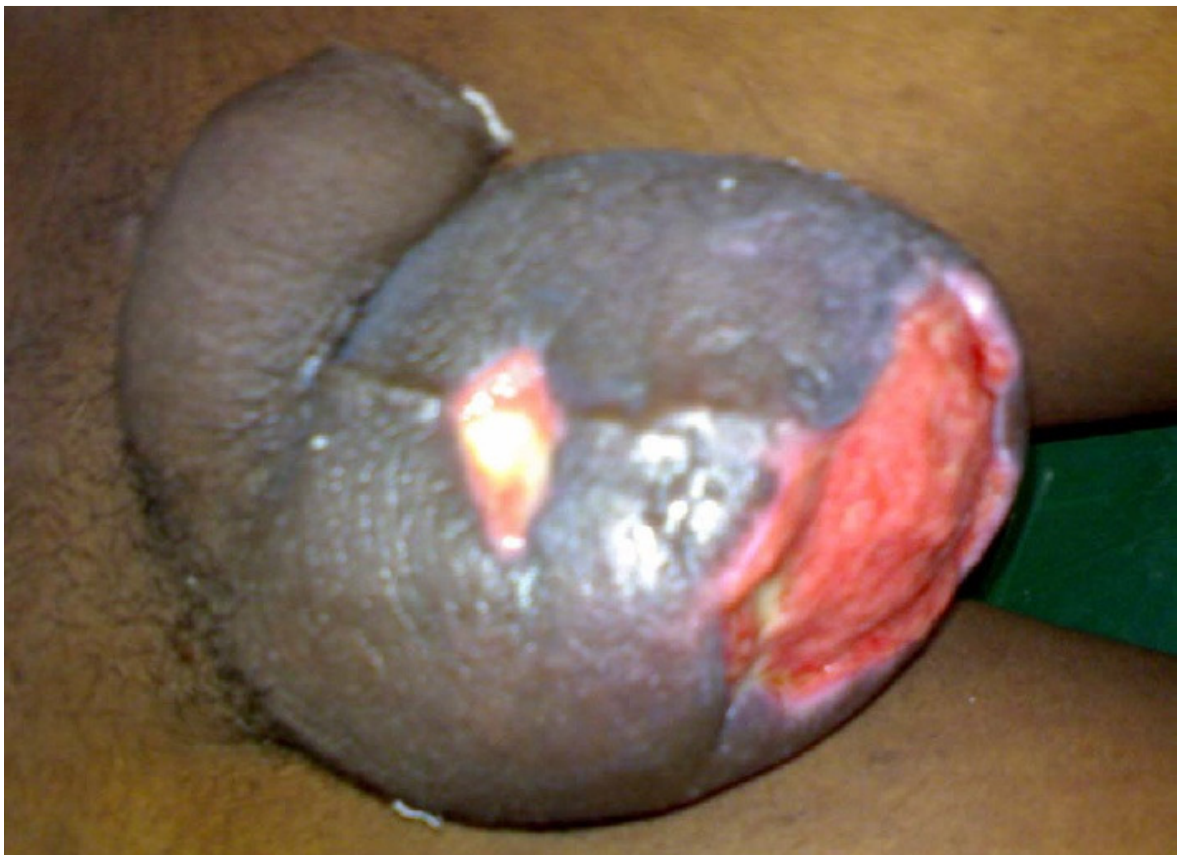
Case 5 after debridement



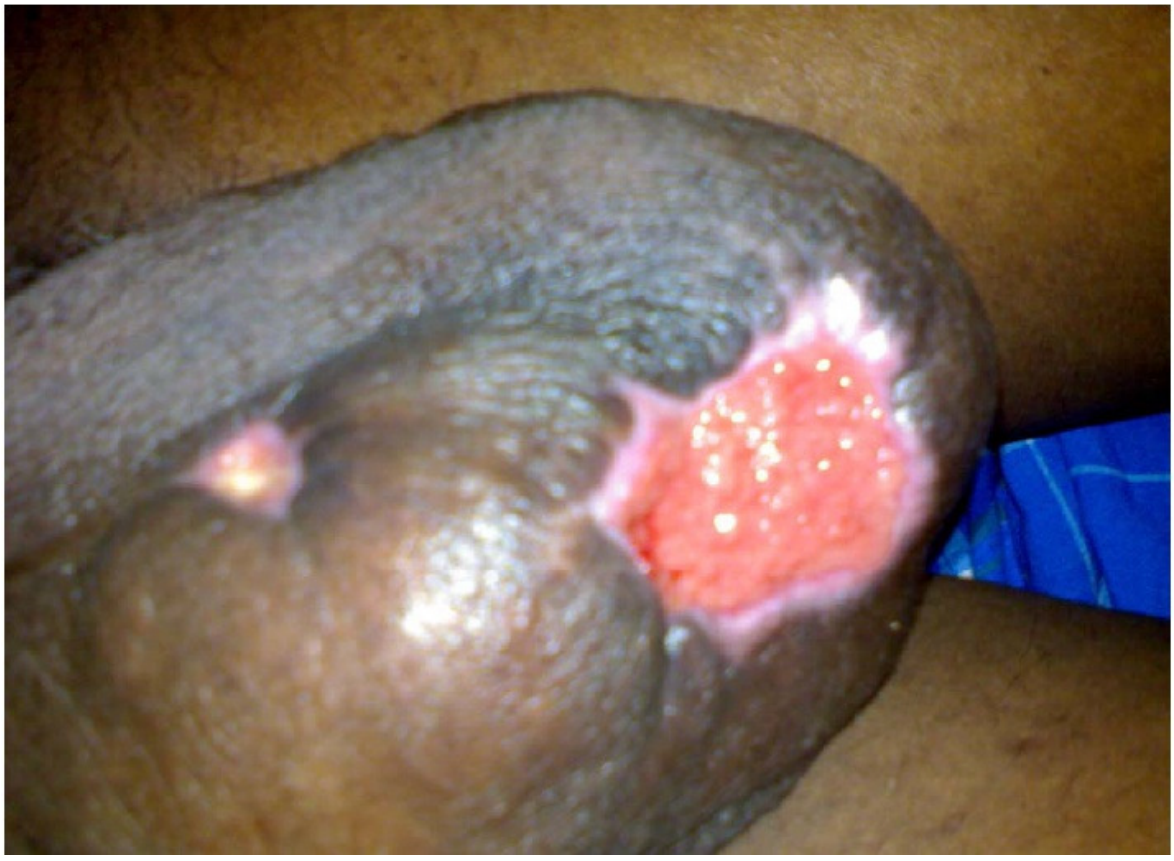
Case after 1 week



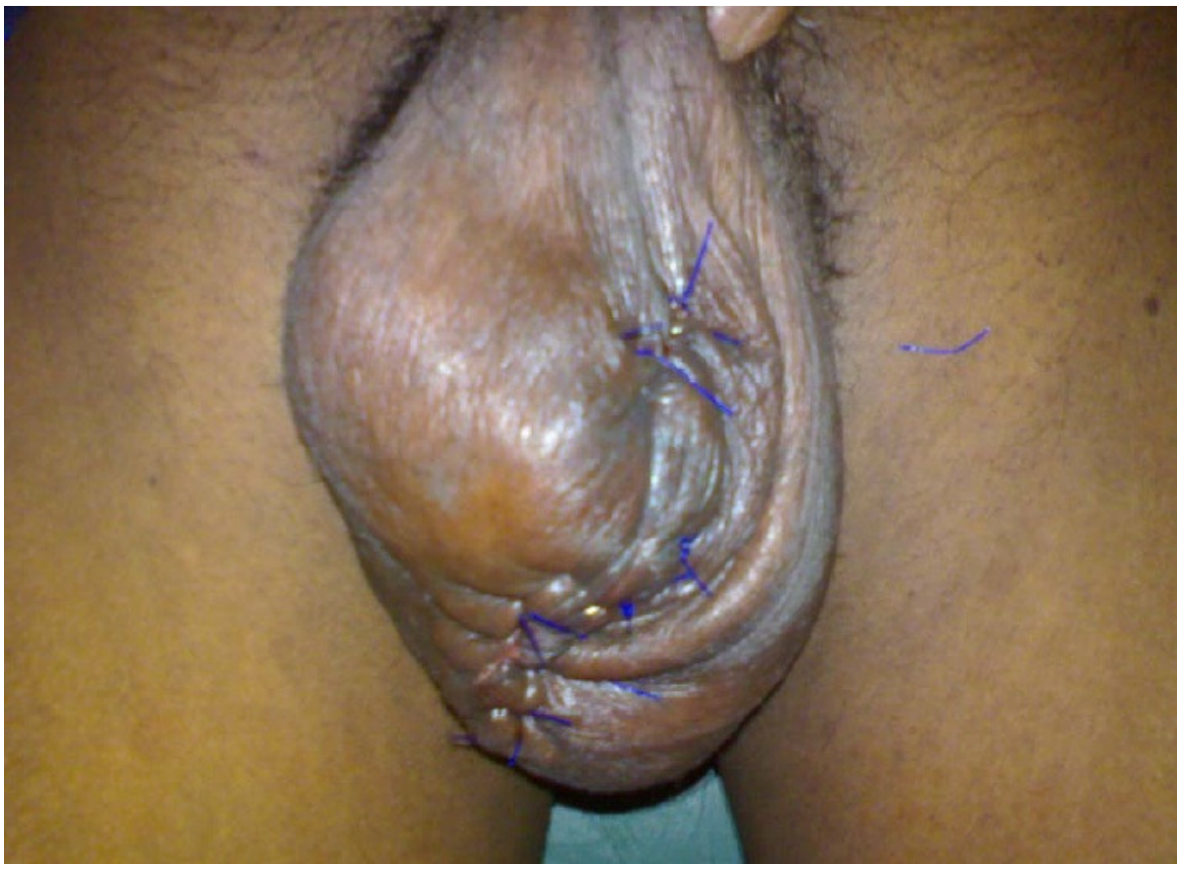
Case 7



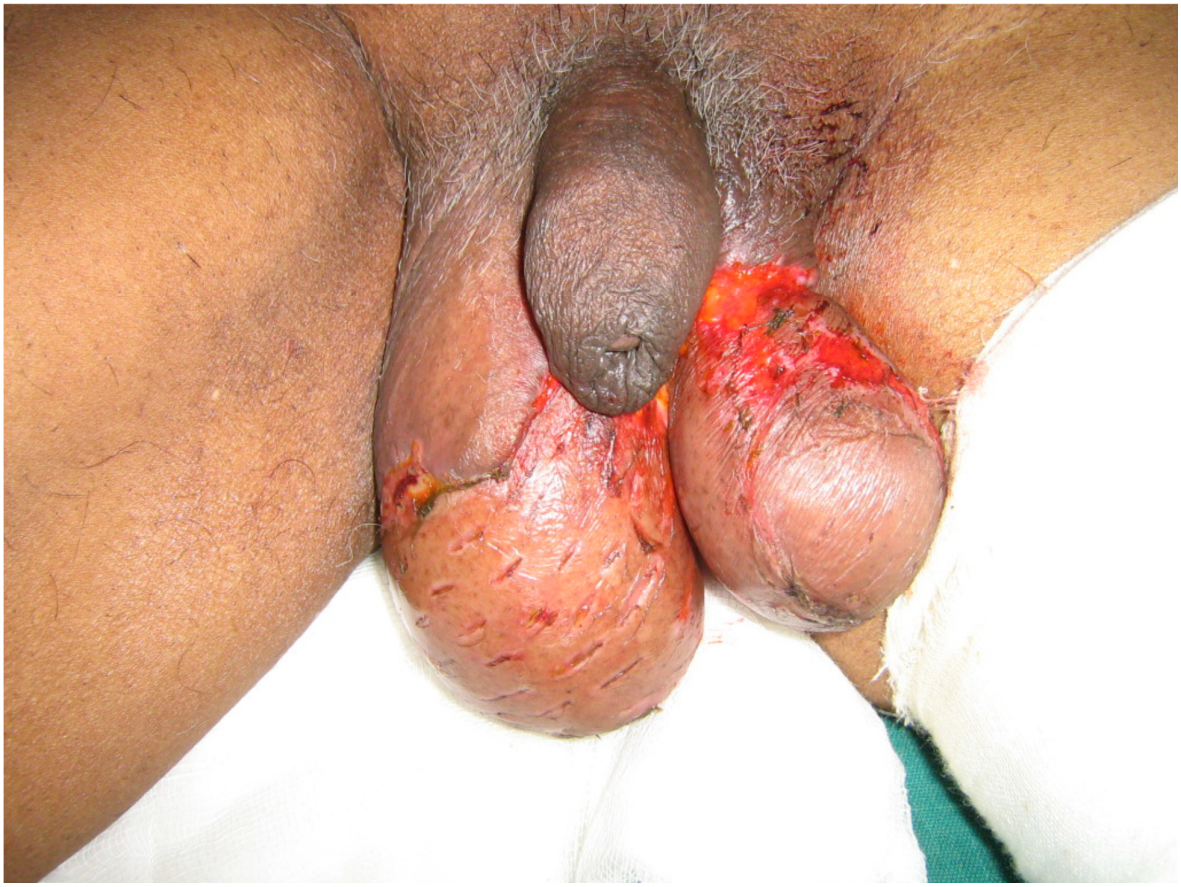
Case 7 after 4 days



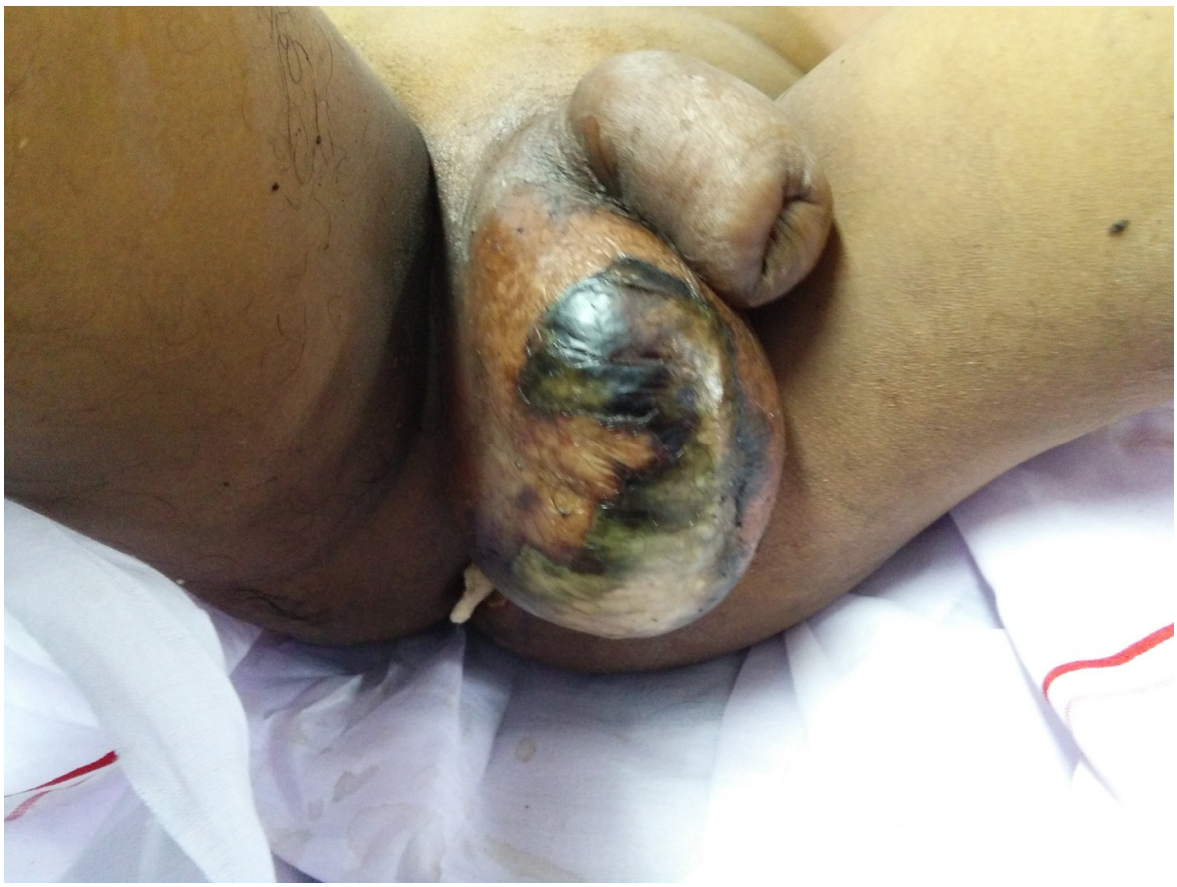
Delayed suturing



Case 9



Case 15



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 seventyseven 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

MASTER CHART

S.No	NAME	AGE	IP NO	Extent Of Involvement	Etiology	Predisposing factor	TC, Hb%	Blood sugar,urea, Creatinine Mg%	Pus C/S	Surgical treatment
1	Muthusami	52	51036	Scrotum	idiopathic	-	9.8 10000	104 28 0.6	E coli	Delayed Suturing
2	Rajendran	56	52129	Scrotum &penis	idiopathic	Alcoholic	10.8 8600	96 30 1	Pseudo monas	Delayed Suturing
3	Parthipan	68	54642	Scrotum &penis	idiopathic	Alcoholic	11.2 9600	100 30 1.2		Delayed Suturing
4	Kumar	64	55941	Scrotum &penis	Trauma	Diabetes mellitus	7.8 12000	360 50 1.2	Pseudo monas	SSG
5	Sundharava divel	66	57152	Scrotum	idiopathic	Diabetes mellitus	10.8 11000	280 46 1.2	Pseudo monas	Delayed Suturing
6	Dhanavel	62	57971	Scrotum	Trauma	CKD	10.2 8800	120 86 4.3	Strepto cocci	Wound Debridement
7	Raj	46	58082	Scrotum &penis	idiopathic	CLD	9.2 8200	96 30 1	Klebsi ella	Wound Debridement
8	Ganeshan	62	60954	Scrotum	idiopathic	Alcoholic	9 8600	84 28 0.8	E coli	SSG
9	Anthonisam i	50	62132	Scrotum	idiopathic		9.6 8000	88 28 0.8	E coli	Delayed Suturing
10	Nadeshan	48	63206	Scrotum &penis	idiopathic	Diabetes mellitus	10 7000	174 30 0.8	Pseudo monas	Delayed Suturing

11	Baskar	54	1473	Scrotum	idiopathic		9.6 7200	100 28 0.6		Delayed Suturing
12	ayyadurai	46	2501	Scrotum	idiopathic	Alcoholic, Diabetes mellitus	8.8 7600	120 30 0.8	Pseudo monas	Delayed Suturing
13	saminadhan	64	3446	Scrotum	Trauma	Alcoholic	8.4 6800	240 30 0.8	Pseudo monas	SSG
14	ilangovan	68	3776	Scrotum	idiopathic	CKD	11.2 6000	96 72 3.2		Delayed Suturing
15	Murugan	66	6513	Scrotum	Trauma	Diabetes mellitus	8.6 7800	198 32 0.8	Pseudo monas	SSG
16	Palaniswami	72	6538	Scrotum	Trauma	Diabetes mellitus	8.8 7800	202 40 1.2		Delayed Suturing
17	Raman	64	33394	Scrotum	Trauma	CKD	8.6 6800	134 84 3.2		Delayed Suturing
18	Govindharaj	66	17813	Scrotum	Trauma	Diabetes mellitus	8.4 6000	198 30 0.2		Delayed Suturing
19	Shekalavudeen	42	35164	Scrotum	idiopathic	Alcoholic	7.6 8000	100 30 0.6	Pseudo monas	SSG
20	Chinnayi	36	40653	Scrotum &penis	idiopathic		10.8 6200	108 30 0.8	E coli	SSG
21	Ramalingam	46	56823	Scrotum	Trauma		8.8 6200	178 30 0.8	Pseudo monas	Delayed Suturing
22	Kalayamani	52	46537	Scrotum	Trauma	Alcoholic, CLD	9.2 12400	108 40 1.2	Strepto cocci	SSG

23	Mahalingham	54	4041	Scrotum	perianal abcess	Diabetes mellitus	10 9600	392 30 0.8	Pseudomonas	SSG
24	Ramasami	58	46982	Scrotum	idiopathic	Alcoholic	8 16400	108 30 0.6		Delayed Suturing
25	Velamkanni	44	53642	Scrotum	perianal abcess	Diabetes mellitus	9.6 9600	362 40 1.5	E coli	Delayed Suturing
26	Selvam	38	43685	Scrotum	idiopathic		8.4 9200	126 38 0.8	Pseudomonas	SSG
27	Iyyakannu	48	36852	Scrotum	idiopathic	CKD	9.6 8400	138 60 3.2	Pseudomonas	Delayed Suturing
28	Nachimuthu	54	46357	Scrotum	idiopathic	Diabetes mellitus	8.8 8800	196 40 0.8	Pseudomonas	SSG
29	kaviselvan	56	65394	Scrotum	Trauma	Diabetes mellitus	11.6 7200	260 27 0.8		SSG
30	gobibagya	60	62344	Scrotum	Trauma		9.2 6800	84 30 0.6		SSG
31	Rajapan	54	5463	Scrotum	idiopathic	Diabetes mellitus	7.8 6200	84 28 0.8	E coli	SSG
32	Manikandan	52	58463	Scrotum & penis	Trauma		8.2 5600	102 26 1	E coli	Delayed Suturing
33	Gobi	58	46865	Scrotum	Trauma	Diabetes mellitus	7.6 6200	108 30 0.9		Delayed Suturing
34	Palanivel	64	46929	Scrotum	idiopathic	Diabetes mellitus	8 9200	112 36 1.2	Pseudomonas	Delayed Suturing
35	Marimuthu	46	45803	Scrotum	Trauma		6.4 8400	110 28 0.9	E coli	Wound Debridement