A Dissertation on

A CLINICO PATHOLOGICAL STUDY AND MANAGEMENT OF NECROTIZING FASCIITIS

COIMBATORE MEDICAL COLLEGE HOSPITAL

Dissertation submitted to

THE TAMIL NADU DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI - 32, TAMIL NADU

With partial fulfilment of the regulations
For the award of the degree of

M.S. DEGREE EXAMINATION

BRANCH I – GENERAL SURGERY

COIMBATORE MEDICAL COLLEGE HOSPITAL

COIMBATORE

APRIL 2016

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation titled "**A CLINICO PATHOLOGICAL STUDY AND MANAGEMENT OF NECROTIZING FASCIITIS CASES IN CMCH "** is a bonafide and genuine research work carried out by me under the guidance of **Dr. V. ELANGO, M.S, FAIS., Professor and Head of the Department GENERAL SURGERY, Coimbatore Medical College and Hospital,** Tamil Nadu, India.

DATE: Signature of the Candidate

PLACE: Dr. G.NIRUBAN CHAKRAVARTHY

CERTIFICATE

This is to certify that dissertation entitled**, "A CLINICO PATHOLOGICAL STUDY AND MANAGEMENT OF NECROTIZING FASCIITIS CASES IN CMCH"** Submitted by Dr. G. NIRUBAN CHAKRAVARTHY in partial fulfilment for the award of the degree of master of surgery in GENERAL SURGERY by The Tamil Nadu Dr .M.G.R. Medical University, Chennai, is a bonafide record of the work done by him in the Department of general surgery, Coimbatore Medical College and Hospital, during the academic year 2013-2016.

Guide & Professor **Professor & HOD**

Department of general surgery **Department of general surgery**

Dean

Coimbatore Medical College Hospital

Coimbatore Medical College

COIMBATORE, TAMILNADU, INDIA - 641 014

(Affiliated to The Tamilnadu Dr. MGR Medical University, Chennai)

ETHICS COMMITTEE

CERTIFICATE

Name of the Candidate : Dr. G. Niruban chakravarthy

Course : Ms General surgery

Period of Study : 2013 - 2016

College : coimbatore Medical college

Dissertation Topic : A clinico pathological study and management of necrotising fascitis.

The Ethics Committee, Coimbatore Medical College has decided to inform that your Dissertation Proposal is accepted / ~~Not accepted~~ and you are permitted / ~~Not permitted~~ to proceed with the above Study.

DEAN
Coimbatore Medical College & Hospital,
Coimbatore

10.9.2014

turnitin

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:	221311308.m S General Surgery. G...
Assignment title:	TNMGRMU EXAMINATIONS
Submission title:	A CLINICO PATHOLOGICAL STUDY..
File name:	niruban_thesis_plagiarism.docx
File size:	770.07K
Page count:	80
Word count:	6,874
Character count:	39,802
Submission date:	03-Oct-2015 08:11PM
Submission ID:	572736253

ACKNOWLEDGEMENT

I am extremely thankful to **DEAN, Prof. Dr. A. EDWIN JOE M.D.,** Coimbatore Medical College and hospital, for his kind permission to carry out this study.

I am immensely grateful to **Professor Dr. V. ELANGO, M.S , PROFESSOR and Head of Department of GENRAL SURGERY,** Coimbatore Medical College and Hospital for her concern and support in conducting the study.

I am greatly indebted to my assistant professors **Dr. R. Narayanamoorthy M.S, Dr. P.Sumitra M.S., DGO., and Dr. B.Jayalakshmi M.S., Department of General Surgery,** Coimbatore Medical College and Hospital for his inspiration, guidance and comments at all stages of this study.

I am thankful to all our General Surgery unit chiefs for their support in conducting the study**.**

I am thankful to all assistant professors for their guidance and help. I am thankful to all my colleagues for the help rendered in carrying out this dissertation.

I thank all the patients for their support in this study.

ABSTRACT

A CLINICO PATHOLOGICAL STUDY AND MANAGEMENT OF NECROTIZING FASCIITIS

BACKGROUND

Necrotizing fasciitis is an infection occurring in the deep fascial layers. It is a progressive infection. The diagnosis of necrotizing fasciitis is usually clinical and is important to find it early as it is a fast spreading infection. Necrotizing fasciitis occurs as a result of necrosis of skin and subcutaneous layer. NECROTIZING FASCIITIS more frequent in elderly age group >50 years of age ,but it can occur in almost all age group including children ,and healthy adult can also get affected.

AIMS AND OBJECTIVES

To analyze

1. Presentation
2. Cause that predispose to necrotizing fasciitis
3. Comorbid condition associated with necrotizing fasciitis
4. Microbiology
5. Surgical and resuscitative management
6. Outcome

MATERIALS AND METHODS

STUDY DESIGN

Descriptional study

SOURCE OF DATA

60 patients of necrotizing fasciitis getting admitted in surgical ward

PLACE OF STUDY

Coimbatore Medical College and Hospital

STUDY PERIOD

September 2014 to august 2015

INCLUSION CRITERIA

All patients presenting with features of necrotizing fasciitis to Coimbatore medical college and hospital

EXCLUSION CRITERIA

Pregnant women

Age<13 yrs

METHODOLOGY

Patients presenting with features of necrotizing fasciitis were admitted in the general surgery ward in Coimbatore medical college and hospital were included in the study during the study period of September 2014-august 2015. Initial diagnosis were made by both clinical and anatomical findings. Details of the patient were noted. Detailed interview with the patient were made regarding history and other comorbid conditions.

Following complete history taking physical examination for the patient were done including blood pressure measurement and temperature and other clinical finding related to necrotizing fasciitis. Following clinical examination, routine investigations were investigated. Radiological investigations were done to note for gas formation in subcutaneous layer. Treatment were started as soon as diagnosis is suspected. It includes resuscitation of patient with intravenous fluids, antibiotics and wound debridement. And bacteriological culture is done for both aerobic and anaerobic bacteria.

The sample taken for culture is transported through proper transporation technique to culture laboratory. These samples were then cultured in blood agar and mc Conkey agar for aerobic bacteria and in Robertson’s cooked meat media

for anaerobic bacteria. The cultured organisms were tested for resistance pattern by disc diffusion method.

Following initial debridement the wound was inspected regularly and subsequent debridement were done periodically whenever necessary. And dressing were done using povidone iodine and saline guaze. After the wound is fit, patient undergone split skin graft surgery for raw area.

STATISTICAL ANALYSIS

STATISTICAL METHOD

In our study we used descriptive statistical analysis. Continuous measurement were represented on mean with or without standard deviation. Categorical measurementwere represented in number (%).

Confidence interval of 95% is used to find significancyofvalue. Confidence limit >50% is associated with statistical significance.

STATISTICAL SOFTWARE

Tables and charts were completed using Microsoft word and excel software.

RESULTS

This study was conducted during the period of September 2014- august 2015. About 60 patients were included in our study and their different aspects for predisposing factors, age of presentation, microbiological pattern and antibiotic pattern were analysed.

DISCUSSION

Most commonly the disease is poly microbial of about 62%. Most common isolated organism is E.coli 35% .almost all patients treated with broad spectrum antibiotics .most common antibiotic sensitive is ceftriaxone followed by aminoglycosides and meteronidazole.

Almost all patients underwent surgical debridement of about 95%and some may underwent procedures like primary suturing secondary suturing and ssg.

CONCLUSION :

NECROTIZING FASCITIS is most commonly seen in the elderly males. source of the infection is identifiable in most of the cases. diabetic mellitus is the most common comorbid factor . The disease is most commonly polymicrobical.

Most common bacterial includes gram positive cocci (streptococci and staphylococcal species). Gram negative (E.coli, actinobacter, pseudomonas).

Most common anaerobes is bacteroides .

INTRODUCTION

Necrotizing fasciitis is an infection occurring in the deep fascial layers. It is a progressive infection. The diagnosis of necrotizing fasciitis is usually clinical and is important to find it early as it is a fast spreading infection. Necrotizing fasciitis occurs as a result of necrosis of skin and subcutaneous layer. In some cases there will be necrosis of underlying muscles causing necrotizing myositis. Necrotizing fasciitis often spreads by direct spread. In severe cases, the microorganisms can spread via blood vessel and lymphatics resulting in sepsis and shock.

INCIDENCE:

NECROTIZING FASCIITIS more frequent in elderly age group >50 years of age ,but it can occur in almost all age group including children ,and healthy adult can also get affected.

0.4 to 0.53 cases per 1 lakh adults was reported .necrotizing fasciitis has higher pre ponderanceto male population .

Male to female ratio is 3:1 .

Necrotizing fasciitis mainly affects lower limb followed by perineum (Fournier s gangrene). Necrotizing fasciitis infection mainly seen in low economic status .

Incidence of Fournier s gangrene is about 1.6/1 lakh population .

Male to female ratio in Fournier s gangrene is 10:1, low incidence in female is mainly due to because of good drainage of genito urinary secretion .

Skin and soft tissue infection (SSTIs):

Divided in to three groups byinfection disease society of america

1.superficial infection

2.uncomplicated infection

3.necrotizing infection .

There has been recent increase in the prevelance of necrotizing fasciitis due to increased incidence of diabetes and other immunocompromised states like HIV. Necrotizing fascittis is common in adults, mostly in men. Its prevalence is about 0.4/1,00,000 population.

Necrotizing fasciitis occurs due to several predisposing factors. Immunodeficiency is the important risk factor for development of necrotizing fasciitis. The main immunodeficiency states are diabetes, AIDS, malignancy and drugs.

In our setup the most common causing being diabetes which occurs as result of microvascular and macrovascular complications. Along with these factors there is presenceof immunosuppression. As result of these

factors there will be formation of ulcer and infection and finally resulting in necrotizing fasciitis.

Diabetic neuropathy causes degeneration of nerve fibresi.e both sensory and motor nerve fibres and it also result in loss of autonomic function. As a result of these there will be formation of pressure ulcer, which results in secondary infection. It finally results in spread of uncontrolled infection and necrotizing fasciitis.

Any risk fator that results in disruption of mucosa or skin integrity it causes necrotizing fasciitis. Risk factors like skin trauma, iv drug abuse and needle prick/ thron prick injury results in necrotizing fasciitis. Sometimes mucosal injury in gastrointestinal or genitourinary tract can result in necrotizing fasciitis.

In our study, we are trying to identify the incidence of necrotizing fasciitis in our hospital. And also to find the predisposing factors, bacteriological profile and outcome of the patients.

AIMS AND OBJECTIVES

To analyze

1. Presentation
2. Cause that predispose to necrotizing fasciitis
3. Comorbid condition associated with necrotizing fasciitis
4. Microbiology
5. Surgical and resuscitative management
6. Outcome

In case of necrotizing fasciitis

HISTORY OF NECROTIZING FASCIITIS

History of necrotizing fasciitis goes back to 18 th century.

In the year 1871 army surgeon JOSEPH JONES during united states civil war first described the disease. JOSEPH JONES (1833-1896)medical professor and surgeon in us army . In ancient days it was thought that disease was restricted to military persons. NECROTIZING FASCIITIS rarely seen in civilian population .

In the year 1863 largest civilian out break in London almost ninety cases has been reported .

NECROTIZING FASCIITIS prevalent in many parts of the world. FOURNIER S GANGRENE is nothing but NECROTIZING FASCIITIS in the perianal and genital region .FOURNIER'S GANGRENE first described in the year 1764 by a physician named BAURIENNE. He described as idiopathic , rapidly spreading soft tissue infection .

In 1909 british surgeon FEDDE FEDDEN described cases of necrotizing fasciitis caused by streptococcus pyogenes.

American surgeon named FRANK MELENEY says “ streptococcus “” as a causative organism for gangrene.

In 1918 a Frenchvenerologist named FOURNIER documented NECROTIZING FASCIITIS in the perineal and genital region .fournier s gangrene was named after JEAN-ALFRED FOURNIER.the most important character of the disease is

1.rapidly spreading to the neighbouring structures,

2.no definitive cause ,

3.sudden onset.

In the year 1952 necrotizing fasciitis was named by WILSON .

NECROTIZING FASCIITIS is a rare life threatening disease .it is commonly known as

FLESH EATING BACTERIA.

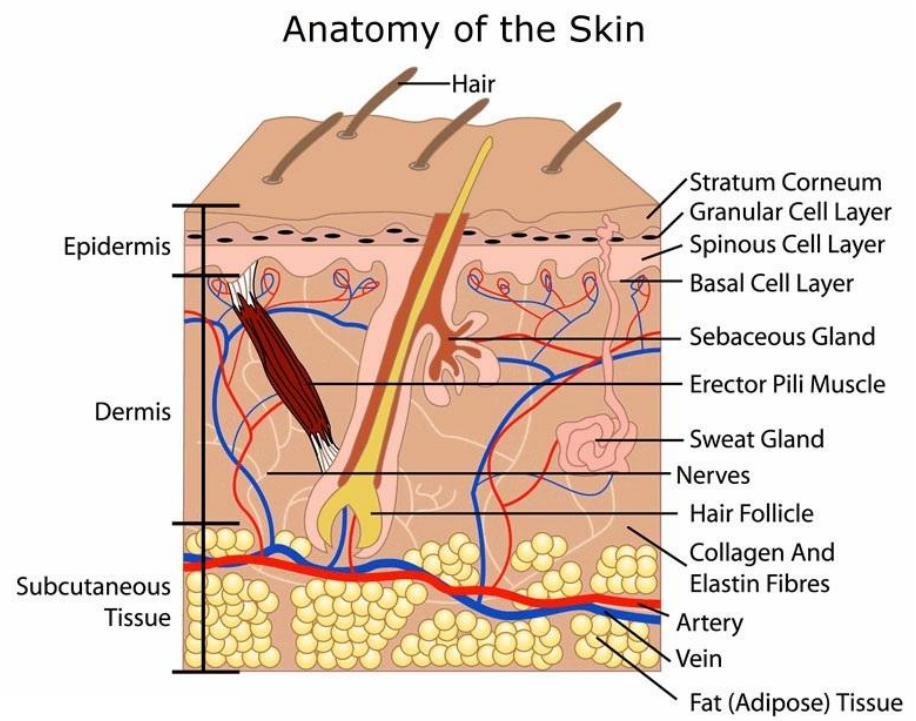
FLESH EATING DISEASE OR

FLESH EATING BACTERIA SYNDROME.

ANATOMY:

NECROTIZING FASCIITIS mainly affects skin and subcutaneous tissue.

Skin is the complex and largest organ in our body .it accounts about 15 to 20 % body weight.



EPIDERMIS:

1. Stratum corneum
2. Stratum granulosum
3. Stratum spinosum
4. Stratum basale

Stratum lucidum seen in betweens.corneum and s. granulosum ,this layer is seen mainly in the palmo-plantar region. Almost 80 t0 90 percent of epidermis is of ectodermal origin.

EPIDERMAL COMPONENTS:

1.keratinocytes - cytoskeleton

2.langerhans cells-antigenic presenting cells

3.melanocytes-melanin

4.merkel cells

5.toker cells

6.epidermal appendages

7.sweat gland

8. pilosebaceous follicles.

DERMIS:

It is of two types

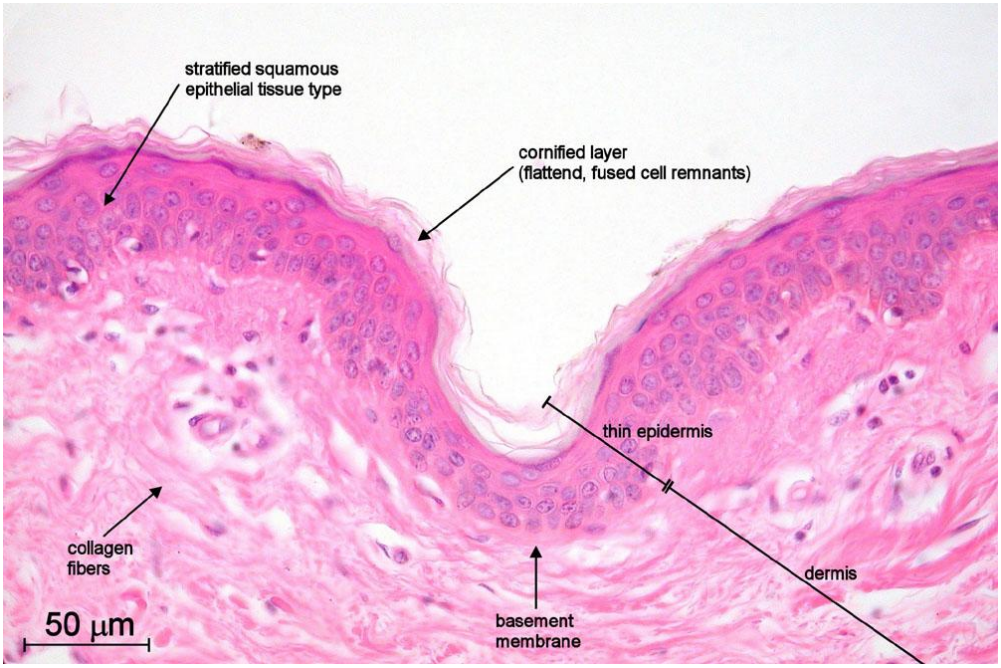
1.PAPILLARY DERMIS

It helps in good adhesion between epidermis and dermis , made up of loose collagen bundles and thin elastic fibre.

2.RETICULAR DERMIS.

It has thick elastic layer .& coarse collagen.

Dermal layer mainly contains type 1 and type3 collagen almost about 85 to 95% .it also contains type 4 and type 7 collagen. This layer act as a mechanical barrier .



Dermis contains 1.Fibroblast

2.dermaldendrocytes and

3.mast cells.

4.vessels and

5. nerve endings.

HYPODERMIS:

Very essential for storage of energy , thermoregulation, insulation, and protection from external injuries. It contains mainly adipocytes.

Necrotizing fasciitis mainly affects superficial fascia ,severe inflammation and edema of sub cutaneous tissue and dermis. In severe disease there will be necrosis of skin also present.

ANATOMY:

NECROTIZING FASCIITIS of scrotum and penis is said to be Fournier s gangrene.fournier s gangrene can affect superficial and deep planes. Subcutaneous layer divided in to

Outer fatty layer –camper ‘s fascia

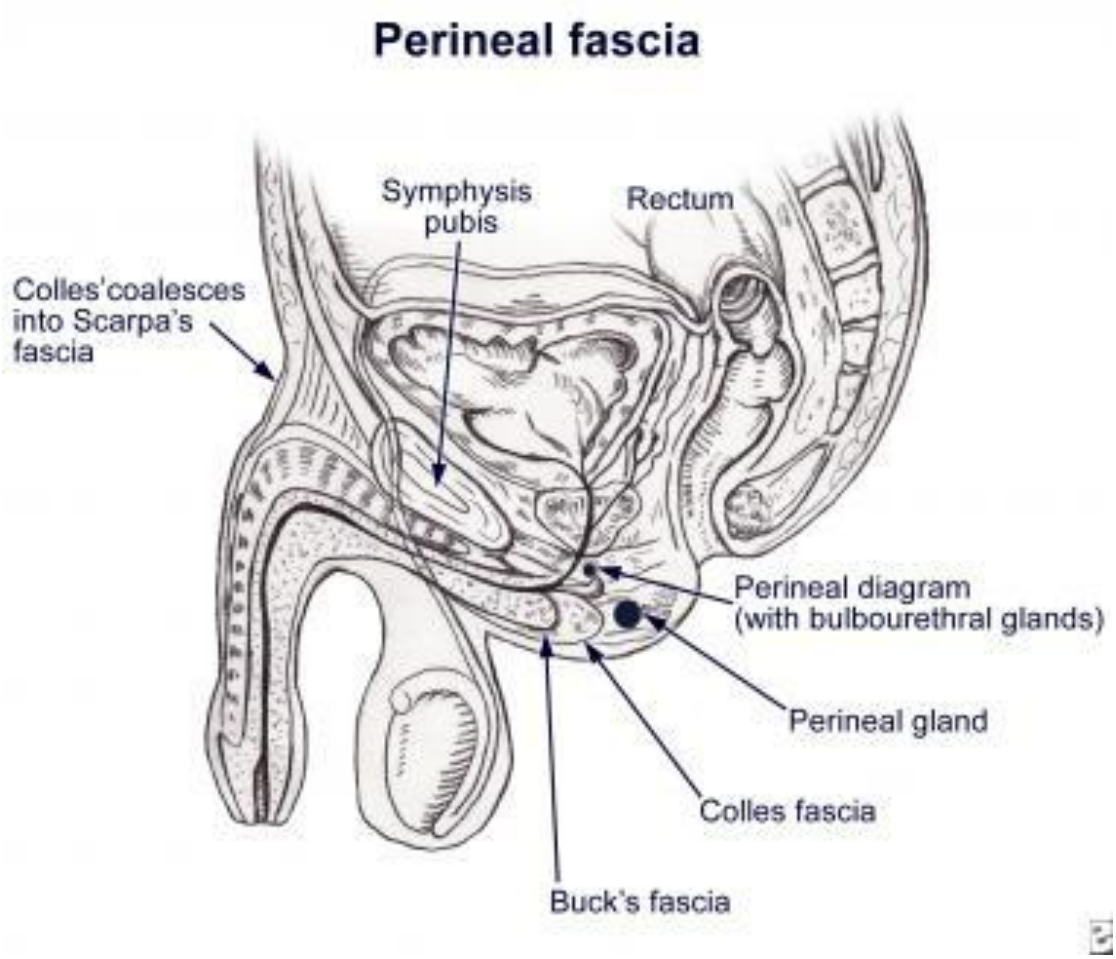
Depper membranous layer –scarpa’s fascia.

Fatty layer is absent in penis and scrotum ,only membranous layer and is in direct continuous with anterior abdominal wall through camper s fascia .it leads to rapid spread of infection from the perineal region to anterior abdominal wall.camper s and scarpa s fascia fuse together and attached to clavicle superiorly , so it prevents the spread of infection . inferior fascia of urogenital diaphragm is also known as perineal membrane. Perineal membrane with colles fascia said to be superficial

perinealspace .it includes membranous ,bulbar urethra and bulbourethral gland .superficial perineal membrane is in nearby to ischio rectal fossa and anterior part of anal wall.

Infection from rectum ,perineum,urethra and bulbourethral gland directlydrains in to superficialperineal space . infection can easily spreads in to scrotum and anterior abdominal wall.

Blood supply of anterior abdominal wall from branches of inferiorepigastric artery and deep circumflex iliac artery.



COLLES FASCIA:

Iaterallycolles fascia attached to the conjoint rami of ischium and pubis . anteriorly it attached to the anterior abdominal wall . posteriorly to pubic arch .

Layers of scrotum from superficial to deep .

1.skin

2.subcutaneous fascia- dartous muscle .

Dartous muscle helps in maintaining optimum temperature .

3.external spermatic fascia – aponeurosis of external oblique muscle .

4.cremaster

5.internal spermatic fascia – from transversalis fascia .

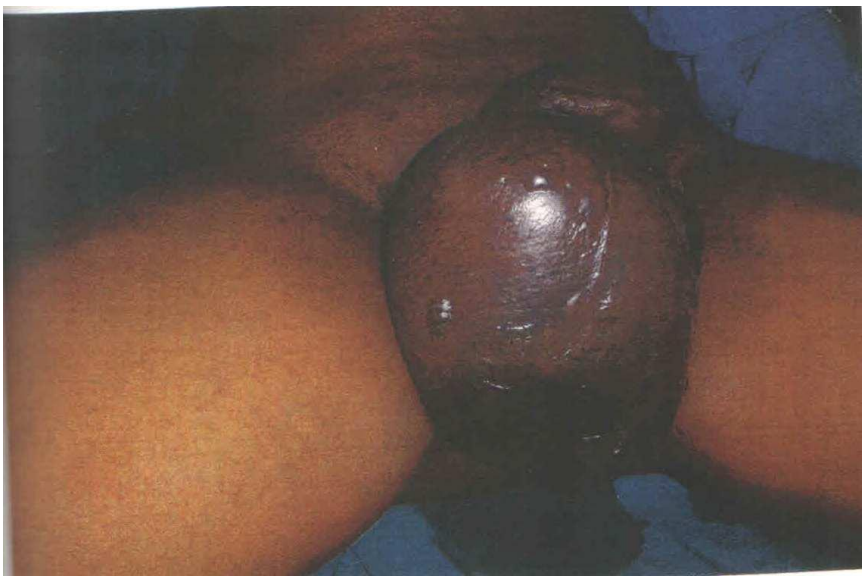
6.areolar tissue

7.testis.

External pudendal and internal pudendal arteries supplies blood to the scrotal wall .internal pudendal vessels supplies posterior aspect of scrotal wall. Because of infection and toxins released by bacterial organisms leads to thrombosis of artery travels in campers fascia ,except

internal pudendal artery so blood supply to the posterior scrotal wall will be intact so the skin in posterior aspect can be used for reconstruction .

Necrotizing fasciitis infection which is present in the perineal fascia can spread to scrotum through dartous fascia .sometimes it may spread to the anterior abdominal wall through scarpa s fascia .structures which is usually affected is skin, subcutaneous tissue ,and fascial layer.



In Fournier s gangrene the unaffected structures are

1.testis

2.urethra

3.cord structures

4.corporacavernosa .

REVIEW OF LITERATURE

Due to recent knowledge in the recent past and development in the field of medicine, there has been increase intresent in the area of necrotising fascitiis.

EPIDEMIOLOGY

Till now there is no apt definition for necrotizing fasciitis. It is also otherwise called as progressive synergistic gangrene, suppurative fasciitis, and acute dermal gangrene. When it occurs in genitals it is called as fournier's gangrene. when it occurs in the postoperative wounds it is called as progressive postoperative synergistic bacterial gangrene, in these cases it usually spreads cutaneously sparing deep fascia.

Necrotizing fasciitis has no sex or age predilection and is common in immunosuppressed states. But it can also occur in healthy young individuals also without any predisposing factors, the most common organism being group A hemolytic streptococci. And it usually present as toxic shock syndrome.

ETIOLOGY

Necrotizing fasciitis can occur anywhere in the body. But it is most commonly seen in lowerlimb, upperlimb, abdominal wall and genitals. Inoculation of organisms into the disrupted mucosa, skin by trauma, burns, or other modes of injury result in development of local infection

followed by necrotizing fasciitis. Though development of necrotizing fasciitis is usually due to direct local site infection it also results from infection in the distant site like pharyngitis.

Necrotizing fasciitis of abdominal wall is often fatal if not treated adequately. It occurs as complication of abdominal surgery. It occurs most commonly after surgery in the contaminated or clean contaminated environment. Necrotizing fasciitis of limbs are common when compared to abdominal wall fasciitis. In the perineal region it occurs as a result of neglected perianal abscess or trauma. Rarely it occurs in retroperitoneal region and it has highest mortality when compared to other necrotizing fasciitis of other areas.

Most of the cases of necrotizing fasciitis of vulva occur in diabetic patients. It begins as infection in the bartholin's gland which results in abscess and necrotizing fasciitis. There are also other causes which results in necrotizing fasciitis, these includes postoperative infection following hysterectomy, caesarean section or episotomy.

Necrotizing infection of genitals occur commonly due to perianal abscess or trauma. But it can also occurs following urinary tract infection with stricture urthera with or without extravasation of urine. It can also occur following traumatic instrumentation, uretheral calculi and prostatic massage.

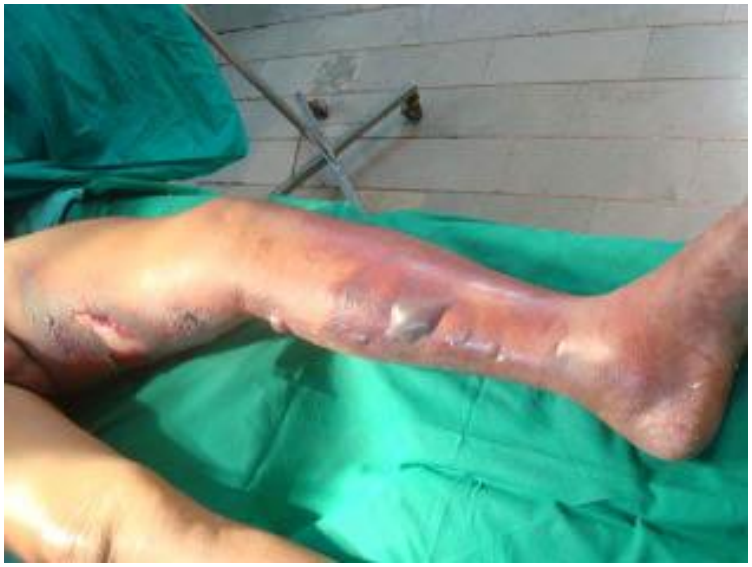
Necrotising fasciitis involving the head and neck region are relativellyrareand is sommon after blunt trauma, eyelid infection and pruritis.

Most of the cases of scalp were monomicrobial in origin and the commonest organism being Streptococcus pyogenes. Most of the cases of scalp necrotising fasciitis are benign.

Necrotizing fasciitis involving face and cervical region were common after dental infections, tonsillar abscess and cervical lymphadenitis. The most common complication being airway obstruction and mediastinitis. Most cases of culture showed polymicrobial growth.

In some instances necrotizing fasciitis occur in case of percutaneous catheter usage and following tube thoracostomy. In about 13 to 31% there has been no initiating factor for development of necrotizing fasciitis. It has been thought in these cases there has been undetected break in skin with inoculation of organism. Rarely the spread may be hematological in origin.

CLINICAL PRESNTATION AND PATHOPHYSIOLOGY



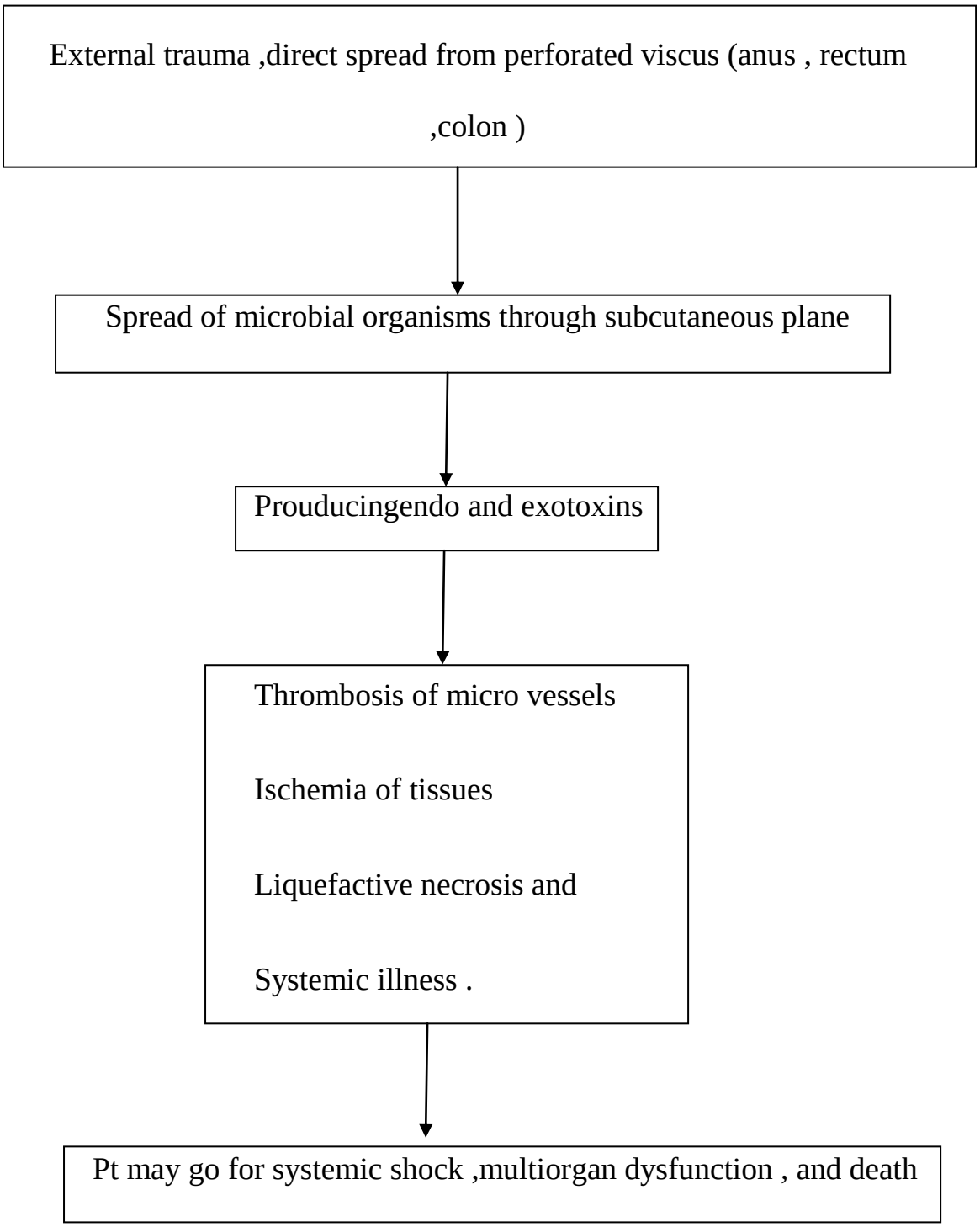
Necrotising fasciitis starts after 7 days of injurious event. It beings as mild skin change in the involved area as erythema. The involved areas will become swollen and red. It is usually accompanied by fever and local pain and is the most common physical sign. Pain occurring in these conditions are often out of proportion to the clinical signs.

There will not be clear line of demarcation between normal and areas involved in necrotizing fasciitis. Most of the patients present to us in toxic condition. The intial state of cellulitis is followed by development of patchy, blisters in the involved areas. Initially the blisters consists of clear fluid which on progression of disease the fluid becomes erythematous.

This stage is followed by necrosis of superificial fascia and fat in the underlying skin and it results in the formation foul smelling pus and is often called dishwater pus. Necrosis occurring in these layers are considered to be due to liquefactive necrosis or due to enzymes like hyaluronidase secreted by the bacteria causing necrotizing fasciitis. Involvement of fascial layer is usually more than that of the overlying skin in the affected region. After4-5 days , the overlying skin becomes completely gangrenous and about 2 weeks the overlying skin will completely slough off.

If necrostising fasciitis is not treated adequately in the early conditions it results in release of toxic contents into theblood stream and it results in sepsis and septic shock and death. Rarely some patients recover though untreated.

PATHOPHYSIOLOGY OF NECROTIZING FASCIITIS :



MICROBIOLOGY

No single organism is causative in case of necrotizing fasciitis. Necrotizing fasciitis is usually caused by polymicrobialinfection and is often due to both aerobic and anaerobic bacteria. Due to thecombined action of the bacteria this condition is often fatal.

Giuliano et al categorized the bacterial pathogens involve in the necrotizing fasciitis into three groups.

Type I – it occurs due to polymicrobial infection. It includes non groupA streptococci and anaerobes.

Type II – is usually caused bygroupA streptococci and anaerobic infection or along with staphylococci.

Type III- is caused by marine vibrio species and the most common species that causes necrotizing fasciitis is vibrio vulnifacies and is due to secretion of extracellular toxins due to these organisms. The other organisms that cause necrotizing fasciitis are group B streptococcus and pasturellamulticida.

Most common aerobes and anaerobes

STREPTOCOCCUS SPECIES AND E.COLI --AEROBES

BACTEROIDES ----- ANAEROBES

Other commonly isolated organism

1.pseudomonas

2.klebsiella

3.staphylococcus

4.proteus

5.enterococcus.

6.acinetobacter.

Rarely isolated necrotizing fasciitis are

1.Candidaalbicans

2.Clostridiumtetani

3.Clostridiumperferingens.

The type organism that causes necrotizing fasciitis often depend on the site involved. Type I is most commonly seen in case of abdominal and perianal infection and is oftendue to polymicrobial in origin. Themost common pathogens being in these conditions were enteric organisms and enterococci and anaerobic bacteria.

In case of extremities, types II is most common and are usually monomicrobial.

In case of necroting fasciitis the common area for culture is necrotingcentre of the lesion which is in contrast to cellulitis in which the culture is taken from the edges of the ulcer. Diabetics were the most common individual affected and is due to involvement of small vessel and decreased WBC function and low oxygen tension which are the substrate for bacterial growth.

The destructive effect in case of necrotizing fasciitis is due to the toxins secreted by the streptococcal species and also due to cytokine release. There aretwo main toxins released by the invasive virulent group A streptococcal bacteria, they are Exotoxin A and Exotoxin B.

Exotoxin A is seen in cases of invasive pathogen. Exotoxin B consists of cysteine proteases and is involved in necrotizing fasciitis and myositis by destructing the proteins. There has been recent increase in the

fatality following necrotizing fasciitis and is due to increased virulence of the organism and the development of resistance. Some authors suggest that increase in mortality rate may be due to increase in interest in this field.

RISK FACTORS :

Predisposing factor are

1. Diabetic mellitus
2. Obesity
3. Alcoholism
4. Renal disease
5. Liver failure
6. Cirrhosis
7. AIDS
8. Malignancy
9. Leukaemia
10. SLE
11. Crohn s disease
12. Immunosuppersion .

PATHOPHYSIOLOGY IN FOURNIER S GANGRENE:

Infection from perianal region ,

Rectum ,and

Urethral gland

↓

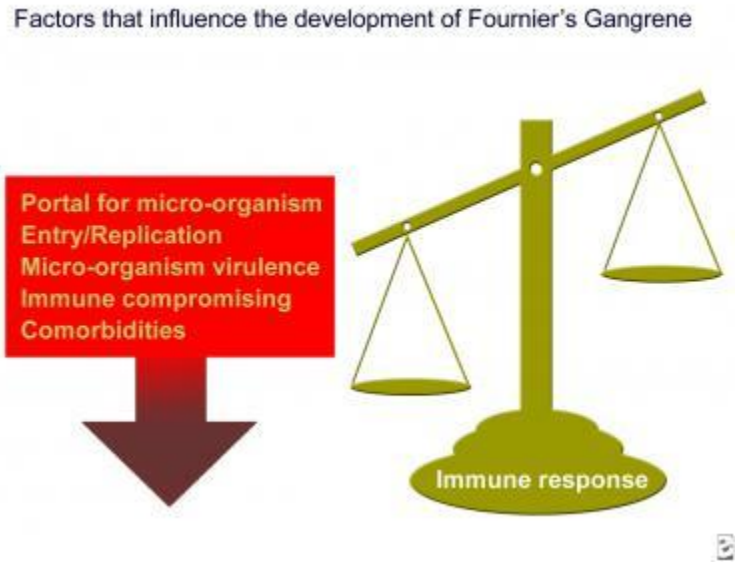
Bacteria releases various toxins , enzymes and activates proteins

↓

Activation of coagulation cascade,platelet aggregation and ischemic changes in the tissue.

↓

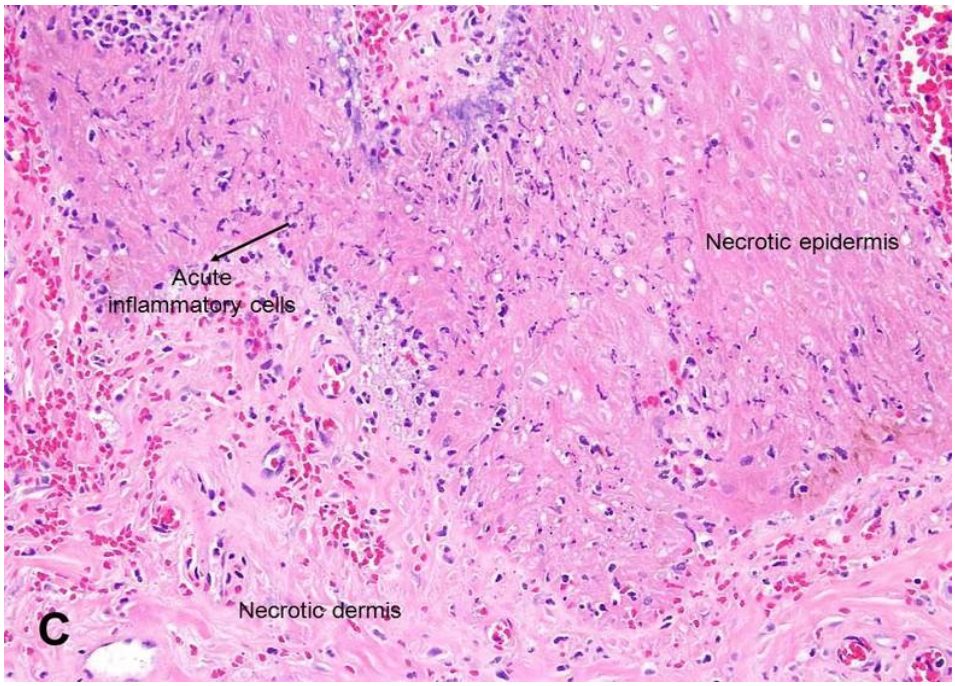
NECROTIZING FASCIITIS .



HISTOPATHOLOGY

The histopathological changes occurring in necrotizing fasciitis are

1. Superficial fascia necrosis and thrombosis of blood vessel
2. Suppuration



There are also other findings that are present in case of necrotizing fasciitis. They are fat necrosis in subcutaneous plane, end arteritis and local hemorrhage. Necrotizing fasciitis usually does not involve the epidermal layer. In the papillary dermis there will be dilatation of blood vessel in acute lesions. Plasma cells are seen in between the papillary and reticular dermal layer which arepredominantely perivascular.

In the deeper layers of skin i.e, reticular layer, edemaand inflammatory infiltrate are present.

In certain cases there is were necrosis of eccrine glands and ducts and it is due to thrombosis of blood vessel resulting in the infarction of the gland. Fascial layer may sometime be edematous and also has inflammatory infiltrate. In advanced cases there will be thrombosis of blood vessel.

Microorganisms are most commonly seen between the collagen and in between fat tissues. In severe cases there will be necrosis of underlying muscle fibres.

DIAGNOSIS

Diagnosing necrotizing fasciitis in early condition is often very difficult. And the diagnosis of necrotizing fasciitis is often clinically made. Important clinical features are pain, redness and toxic symptoms. It is highly important to identify these cases early to intervene at the right time. If diagnosis of necrotizing fasciitis is made, it is necessary to do appropriate debridement.

Decreased resistance in the fascial layer, while performing debridement is often a sign of necrotizing fasciitis. In some studies, it recommends use of frozen section biopsy in case where there is doubt in clinical diagnosis. In study conducted by Stamenkovic , he insisted the use of full thickness biopsy for diagnosis of necrotizing fasciitis.

The radiological conditions useful in the diagnosis of necrotizing fasciitis are

1. Plain radiograph
2. CT
3. Ultrasonography
4. MRI

The important finding in case of necrotizing fasciitis in plain radiography is presence of soft tissue gas. It is more important than physical finding in diagnosing this condition.

CT scan is useful in diagnosis in case of cervical necrotizing fasciitis. It is more accurate than plain x ray. It delineates the exact extension of the disease.

Ultrasonography is important in cases like fournier's gangrene. It is also helpful in differentiating it from other causes of acute conditions.

MRI plays a significant role in early diagnosis of necrotizing fasciitis. It has the ability to show the soft tissue fluid and has good tissue contrast. It is highly sensitive in delineating the pathology. In study conducted by Rahmouni, he used MRI in detecting the early cases of necrotising fasciitis and also differentiate between cellulitis. Though MRI helps to identify the early cases, due to high cost routine use of MRI in the diagnosis of necrotizing fasciitis is not used in our setup.

LRINEC:

Wong et al created a scoring system named “Laboratory Risk Indicator for Necrotizing Fasciitis” in which he compared six independent laboratory variables. With the help of the LRINEC Wong et al postulated a method in finding the difference between the Necrotizing soft tissue infection and non-necrotizing soft tissue infection.

Total score ranges from 0 to 13. Based on the score grouped into low, intermediate and high risk groups.

Value	LRINEC score, points
C-reactive protein, mg/L	
<150	0
>150	4
WBC count, cells/mm^3	
<15	0
15–25	1
>25	2
Hemoglobin level, g/dL	
>13.5	0
11–13.5	1
<11	2
Sodium level, mmol/L	
⩾135	0
<135	2
Creatinine level, mg/dL	
⩽1.6	0
>1.6	2
Glucose level, mg/dL	
⩽180	0
>180	1

Risk category	LRINEC score, points	Probability of NSTI, %
Low	≤5	<50
Intermediate	6–7	50–75
High	≥8	>75

LRINEC score thus helps in identifying the high risk group thus helpful in the early institution of the treatment.

Wall et al had made a similar retrospective study based on WBC count, and serum sodium. WBC count of more than 15,400 cells /mm^3 and serum sodium of less than 135 mmol/L. was more in favor of necrotizing soft tissue infection. His study had high sensitivity but low specificity.

Acute physiology and chronic health evaluation “APACHE” scoring can also be extended to Necrotizing fasciitis and Fournier s gangrene.

FGSI:

Fournier s gangrene severity index score is helpful in predicting the survival and mortality in Fournier's Gangrene. FGSI is calculated two times, first at the admission next at discharge / death.

Physiologic Variables	High Abnormal Values				Normal	Low Abnormal Values			
	+4	+3	+2	+1	0	+1	+2	+3	+4
Temperature (c)	> 41	39-40.9		38.5-39	36-38.4	34-35.9	32-33.9	30-31.9	< 29.9
Heart Rate	> 180	140-179	110-139		70-109		55-69	40-54	< 39
Respiratory Rate	> 50	35-49		25-34	12-24	10-11	6-9		< 5
Serum Sodium (mmol/L)	> 180	160-179	266-159	350-354	130-149		120-129	111-119	< 110
Serum Potassium (mmol/L)	> 7	6-6.9		5.5-5.9	3.5-5.4	3-3.4	2.5-2.9		< 3.5
Serum Creatinine (mg/100/ml*2 for acute renal failure)	> 3.5	2-3.4	1.5-1.9		0.6-1.4		< 0.6		
Hematocrit	> 60		50-59.9	46-49.9	30-45.9		20-29.9		< 20
WBC (Total/ mm*1000)	> 40		20-39.9	15-19.9	3-14.9		1-2.9		< 1
Serum Bicarbonate (Venous, mmol/l)	> 52	41-51.9		32-40.9	22-31.9		18-21.9	15-17.9	< 15

DIFFERENTIAL DIAGNOSIS:

1.cellulitis

2.gangrene

3.diabetic ulcer

4. abscess

5. hematoma

6.lymphangitis

7.deep vein thrombosis

TREATMENT

Necrotizing fasciitis suspected patient should be treated aggressively and promptly in order to reduce the morbidity and mortality .

1. surgeon opinion to be obtained immediately .
2. intravenous access in the healthy extremity .
3. intra venous fluids (NS /RL) .
4. central venous catheter to monitor cvp and central venous oxygen .
5. supplement oxygen , sometimes intubation require .

Different modes of treatment include

1. Broad spectrum antibiotics

2. Surgery

3. Supportive care

4. Hyperbaric oxygen

5. Vaccum assisted closure

1.Broad spectrum antibiotics:

The spectrum of antibiotics should include gram positive and gram negative bacteria. Initial antibiotic therapy includes combination of penicillin or Cephalosporin , an aminoglycoside and anaerobic coverage with Clindamycin or metronidazole.

Antibiotic therapy should be carried on according to culture and sensitivity. High dose Penicillin remains drug of choice for necrotizing fascitis.

Third generation cephalosporin is drug of choice in early stage of disease. It is active against gram negative and low efficacy to gram positive organisms.

Vancomycin can be used in penicillin and cephalosporin resistant individuals used to treat septicemia and skin infection.Metronidazole active against protozoa and anaerobes.

Thrombosis of superficial veins precludes effective antibiotic penetration into site of infection and tissue hypoxia impairs oxidative killing mechanism of leucocytes. Accumulation of bacteria and toxins occurs, which leads to development of sepsis. So early surgical intervention is crucial.

NON STEROIDAL ANTIINFLAMMATORY DRUGS AND NECROTIZING FASCIITIS

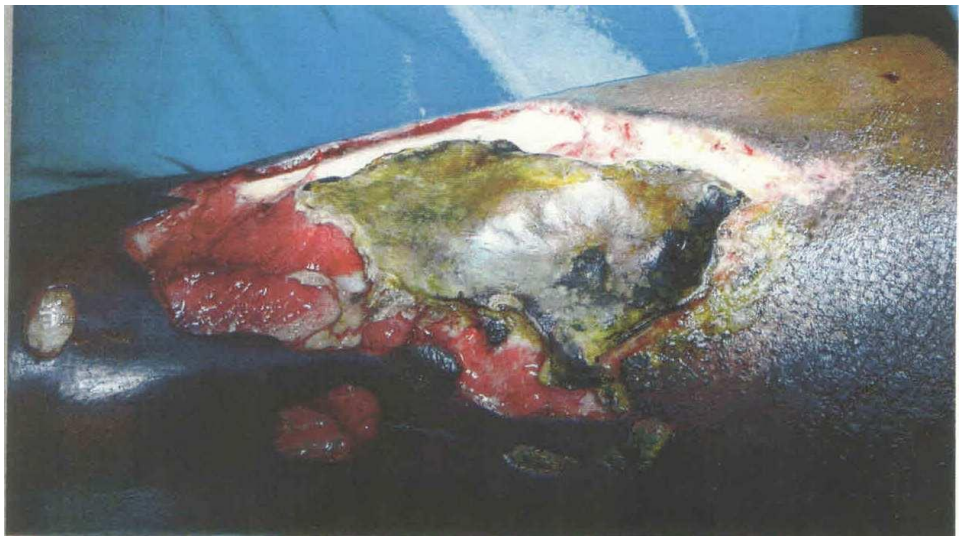
NSAID’s are the most commonly used drug as analgesic in case of minor injury and pain. In the recent studies there has been correlation between the use of NSAID’s and development of necrotizing fasciitis due to group A streptococcal infection in healthy individuals.

Since NSAID’s are most common drug ingested there has been simple correlation between development of necrotizing fasciitis, rather than cause and effect relationship. Some advised that NSAID should be used appropriately in case minor soft tissue inflammation, when infection cannot be ruled out.

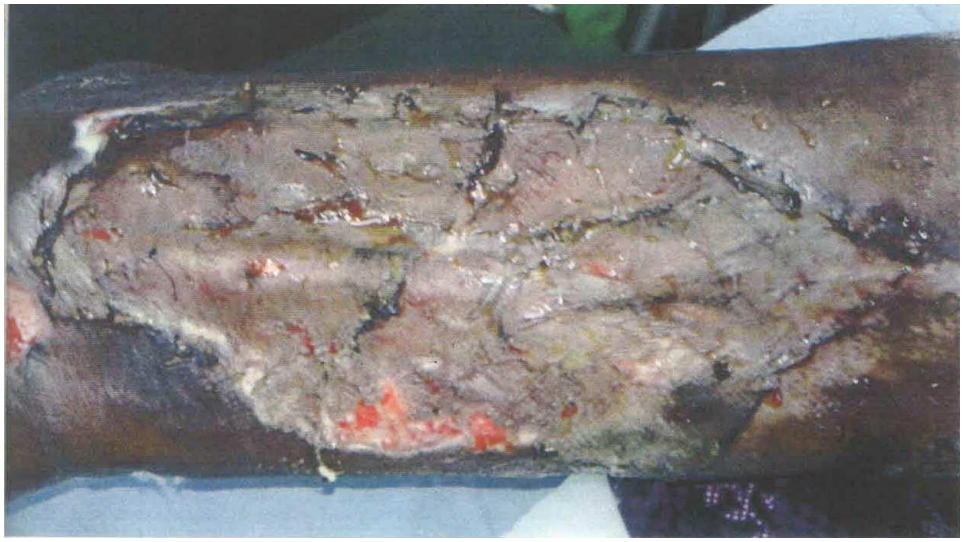
2.Surgery:

Treatment of necrotising include mainly surgical management. Adequate surgical management involves removal of necrotic tissueand drainage of fascialplanes via extensive fasciotomy till healthy fascia is encountered.early surgical treatment is associated with improved survival than delayed surgery.

Extension of fascial necrosis needs repeated fasciotomy and wound debridement. If extremities are involved amputation may be needed to control infection, particular vascular disease and /or diabetes.Inperinealnecrotising fasciitis diversion colostomy or urinary diversion needed to control infection.

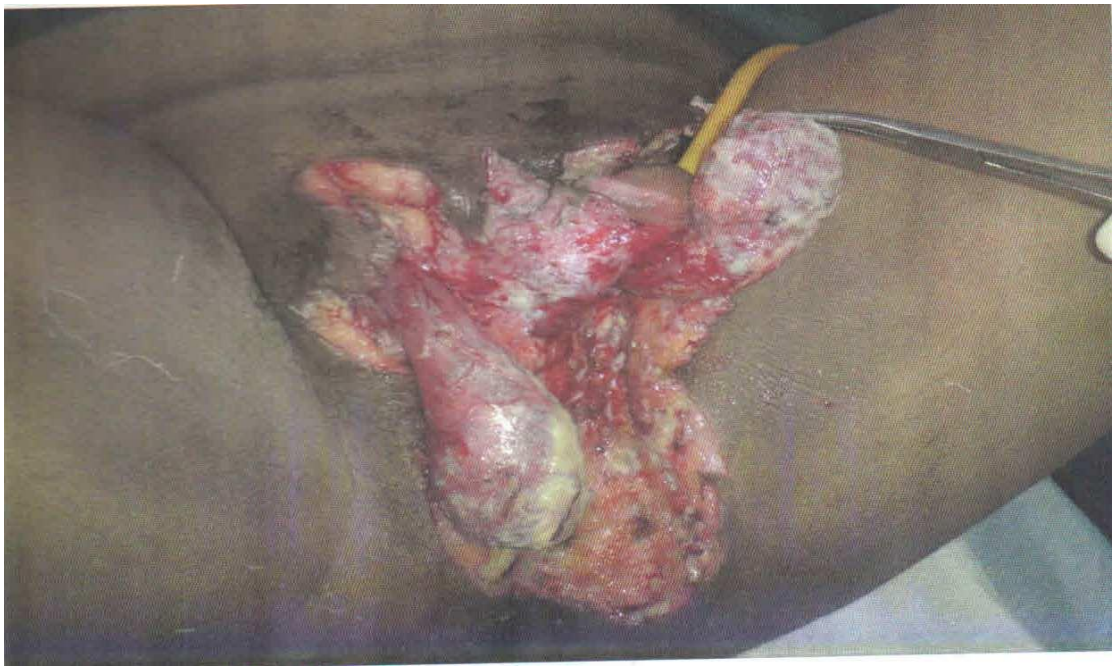


NECROTIZING FASCIITIS OF LOWER LIMB



POST SKIN GRAFTING

In fournier s gangrene when there is a extensive involvement in the perianal region then debridement along with fecal diversion procedures like colostomy to be done. This allows better wound healing and prevents spread of infection. Various studies shows patient needs colostomy in only 15 %. Diversion colostomy does not eliminate the need for multiple debridement .



FOURINER'S GANGRENE IN PERINEAL REGION

Surgical regimen :

1.Incision should be deep and it should be deepened until viable and healthy tissue s reached .

2.entire necrotic tissue must be excised .

3. through wound wash to be done .

4. hemostasis to be obtained .

5. daily dressing to be done .

DRESSINGS:

Dressings can be done with

1.silversulphadiazine

2.polysporin

3.bacitracin .

3.Supportive care:

It includes aggressive fluid management , analgesia and early intensive care. After initial debridement cooperation of multiple specialists needed for optimal patient treatment. Once patient general condition is stabilized and patient begins to recover plastic surgery evaluation is needed for reconstruction and skin grafting.

4. Hyperbaric oxygen:

It includes breathing oxygen at high atmospheric pressure. Hyperoxia at tissue level includes increased leucocyte killing, killing of anaerobes, Reduction of edema stimulation of fibroblasts and better collagen formation. It should be started as early as possible and it should not delay surgical treatment.

Delivering 100% oxygen through a pressure chamber ,which is greater than atmospheric pressure .

HBOT was given at the rate of 2.5 to 3.0 atmospheres for 90 minutes twice daily, to be given after surgical debridement. It improves the tissue oxygenation in both healthy and devitalized tissue also .

It can be used in

1. gas embolism

2.gas gangrene,

3.carbon mono oxide poisoning.

Indication of HBOT:

1.patient with necrotizing fasciitis fail to resolve inspite of adequate medical and surgical management.

2.patient with clostridial infection .

3.gangrene of muscle and deeper tissue involvement.

Advantages of HBOT:

1.by supplying 100% oxygen to the tissue HBOT inhibits the growth ofanaerobic organisms .

2.angiogenesis and fibroblast multiplication

3.improved phagocytic neutrophilic function

4.decreases edema by vasoconstriction andby increased delivery of intracellular antibiotics .

5.HBOT is beneficial for drugs to act .

Eg aminoglycosides acts via oxygen dependent pump .

6.it helps in production of collagen .

Disadvantage of HBOT :

Absolute contraindication :

1.untreated tension pneumothorax .

Relative contraindication :

1.barotrauma to middle ear cavity

2. cardiac disease

3.malignancy

4.URTI

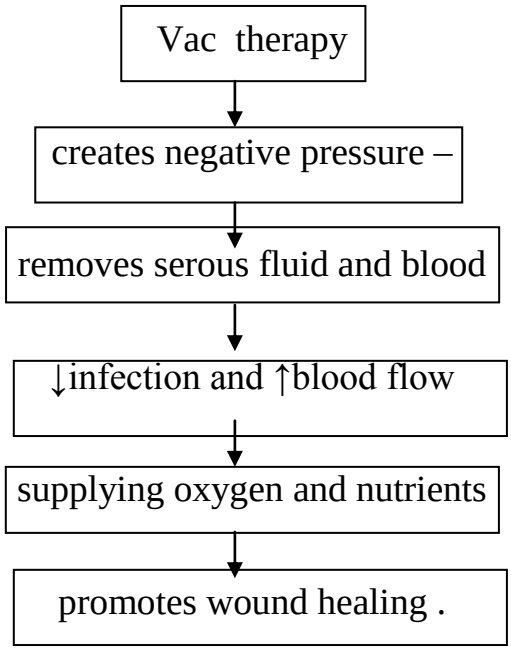
5 emphysema with carbondioxideretention .

But the use of HBOT is still controversial ,it can be used after failure of conventional medical and surgical management.

Six studies which conducted for effectiveness of HBOT in treating necroziting fasciitis ,out of which four reports HBOT improves survival of the patients ,and two of them not. No studies demonstrated the efficacy of hyperbaric oxygen therapy .

5.vaccum assisted closure :

Dr. Louis argenta and Dr Michael designed vaccum assisted closure .it acts through negative pressure , otherwise said to be topical negative pressure /sub atmospheric pressure .



Steps in vac :

1.prepration of the wound

2.placement of foam

3,sealing with drapes

4. application of negative pressure

Vac therapy can be used for

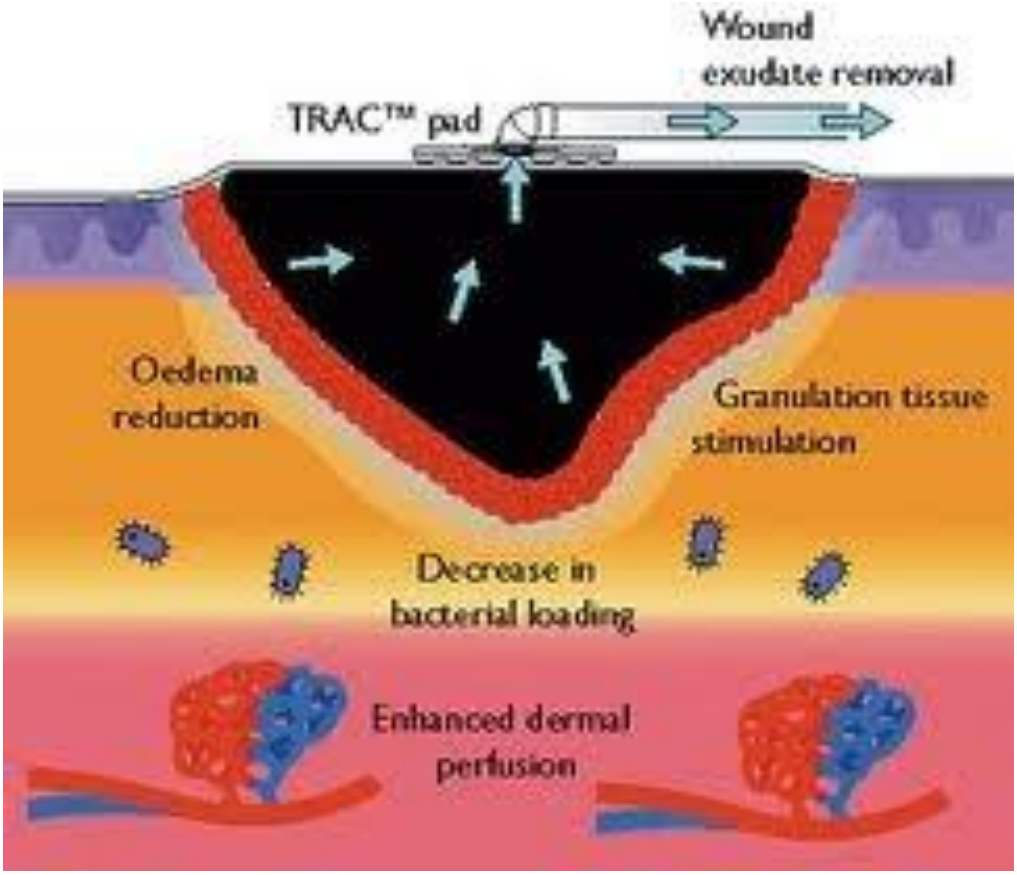
-diabetic ulcer

-pressure sore

-fournier's gangrene

-burns .

Its role is limited in necrotizing fasciitis but it can be used in fournier'sgangrene .in Fournier s gangrene these helps to promote wound healing faster .there is difficulty in placing vac in genitourinary wounds.



MORTALITY

Mortality due to necrotizing fasciitis in untreated condition is very high. Mortality rate has changed little since Meleney first recognized that early intervention needed in case of necrotizing fasciitis. Mortality rate ranges from 29% to 76%. There are other comorbid conditions that increases the mortality in case of necrotizing fasciitis they are diabetes mellitus, peripheral vascular disease and poor nutritional status.

The mortality in necrotizing fasciitis is due to sepsis or multiorgan dysfunction. Early cause of death were due to sepsis syndrome and late death were due to multiple organ dysfunction.

PREVENTION :

1.STREPTOCOCCUS infection can be easily prevented after good hand washing .

2. necrotizing fasciitis can be prevented by intact skin .

3.patient with rapidly spreading wound infection with toxic features should immediately require medical attention .

4. wound should be regularly dressed and through dressing should be done daily ,and patient should be look for any signs of infection like any erythema ,swelling ,tenderness and any discharge from wound .

5. patient with streptococcal throat infection should remain in home until 24 hrs after their last antibiotic dose .

MATERIALS AND METHODS

STUDY DESIGN

Descriptional study

SOURCE OF DATA

60 patients of necrotizing fasciitis getting admitted in surgical ward

PLACE OF STUDY

Coimbatore Medical College and Hospital

STUDY PERIOD

September 2014 to august 2015

INCLUSION CRITERIA

All patients presenting with features of necrotizing fasciitis to Coimbatore medical college and hospital

EXCLUSION CRITERIA

Pregnant women

Age<13 yrs

METHODOLOGY

Patients presenting with features of necrotizing fasciitis were admitted in the general surgery ward in Coimbatore medical college and hospital were included in the study during the study period of September 2014-august 2015. Initial diagnosis were made by both clinical and anatomical findings. Details of the patient were noted. Detailed interview with the patient were made regarding history and other comorbid conditions.

Following complete history taking physical examination for the patient were done including blood pressure measurement and temperature and other clinical finding related to necrotizing fasciitis. Following clinical examination, routine investigations were investigated. Radiological investigations were done to note for gas formation in subcutaneous layer. Treatment were started as soon as diagnosis is suspected. It includes resuscitation of patient with intravenous fluids, antibiotics and wound debridement. And bacteriological culture is done for both aerobic and anaerobic bacteria.

The sample taken for culture is transported through proper transporation technique to culture laboratory. These samples were then cultured in blood agar and mc Conkey agar for aerobic bacteria and in

Robertson’s cooked meat media for anaerobic bacteria. The cultured organisms were tested for resistance pattern by disc diffusion method.

Following initial debridement the wound was inspected regularly and subsequent debridement were done periodically whenever necessary. And dressing were done using povidone iodine and saline guaze. After the wound is fit, patient undergone split skin graft surgery for raw area.

STATISTICAL ANALYSIS

STATISTICAL METHOD

In our study we used descriptive statistical analysis. Continuous measurement were represented on mean with or without standard deviation. Categorical measurementwere represented in number (%).

Confidence interval of 95% is used to find significancyofvalue. Confidence limit >50% is associated with statistical significance.

STATISTICAL SOFTWARE

Tables and charts were completed using Microsoft word and excel software.

RESULTS

This study was conducted during the period of September 2014-august 2015. About 60 patients were included in our study and their different aspects for predisposing factors, age of presentation, microbiological pattern and antibiotic pattern were analysed.

In this study we noted that the patients affected with necrotising fasciitis ranges from 14-81yrs. Mean patients were in the age group of 50.42 ±17.31yrs. most of the patients wereinagegroup of above 50 yrs.

In our study most of the patients were male (49) when compared to female (11)and in ratio of 4.45:1. The most common occupation among the patients presenting to us were farmers (28.3%).

Among 60 patients we had studied, the most common part to be affected was lowerlimb (53.3%), next to it was perinealregion(30%) and involvement of trunk in 8.3% patients.

Regarding the clinical presentation, the commonest symptom being pain and is present in about 95% cases. Next to pain is fever and discharge in 61.7% and 58.3% respectively. Presence of swelling is seen inabout 45% . Blisters were seen in about 18.3%.

In this study we noticed there was delay in presentation of patients to healthcare setup in most cases. Only 55% of patients presented to us

within 1 week of clinical symptoms. 31.7% in 2nd week and 8% in >2 weeks group. The mean duration of presentation was 10.08±10.09 days.

Oedema and ulcer were the most common clinical signs and were seen in about 85% and 76.7% cases respectively.

We noticed in our study that the commonest cause for developing necrotizing fasciitis is trauma which is about 30% cases, in about 6.7% there was no defined etiological factor for development of necrotizing fasciitis.

Regarding the comorbid conditions causing necrotizing fasciitis, the commonestbeing diabetes mellitus in 38.3% cases. Next to it was hypertension in 30% cases.

By blood investigations from our study we notices that, 30% of patients were anemic and 23.3% had elevated blood sugar levels. 23.3% had elevated serum creatinine value and 15% had hyponatremia(<128meq/l).

The cultured organism in our study werepolymicrobial in 79.9% and mono microbial in case of 13.3%. there were no growth in about 6.6%. most of the cultured were aerobic organisms in our study and it constitutes around 86.7 % and anaerobic about 13.3%.

The most common organism cultured in our study were Escherichia coli in about 46.6%. The next most common species being streptococcus in 41.6% and klebsiella in about 36.6%. All the patients initially received broad spectrum antibiotic and it consists of cephalosporin with aminoglycoside with metronidazole.

In about 95% patients we performed wound debridement. In about 5% we had done fasciotomy and secondary suturing in about 28.3%.

Most of the patient discharged after 25 days of hospital stay. And there mortality in our study was 21.7%.

RESULTS ANDOBSERVATIONS

Table 1: AGE & GENDER DISTRIBUTION OF NECROTIZING FASCIITIS						
Age in years	**Male**		**Female**		**Total**	
	No	**%**	**No**	**%**	**No**	**%**
<20	2	4.1	2	18.2	4	6.7
21-30	1	2.1	1	9.1	2	3.3
31-40	10	20.4	2	18.2	12	20
41-50	7	14.3	1	9.1	8	13.3
51-60	14	28.6	3	27.3	17	28.3
>60	15	30.6	2	18.2	17	28.3
Total	49	100	11	100	60	100
Mean SD	52.27+17.1		42.18+16.71		50.42+17.31	

TABLE 2 : INCIDENCE AMONG MALES

Age in years	Male	
	No	%
<20	2	4.1
21-30	1	2.1
31-40	10	20.4
41-50	7	14.3
51-60	14	28.6
>60	15	30.6
Total	49	100

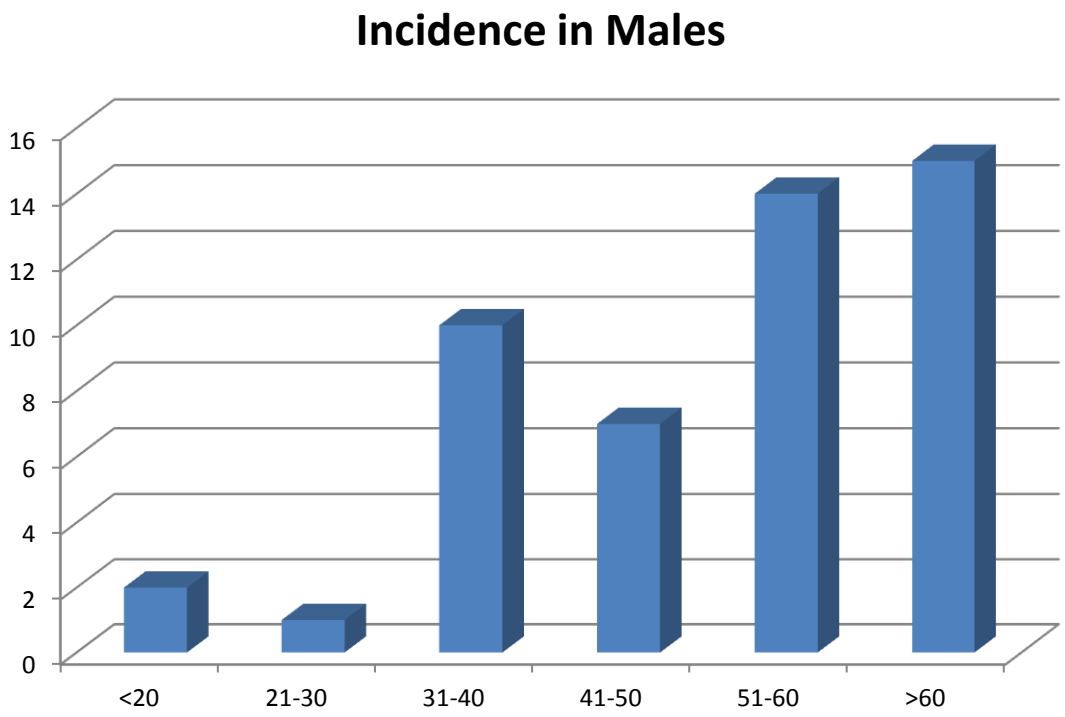


TABLE 3 : INCIDENCE AMONG FEMALES

Age in years	Female	
	No	%
<20	2	18.2
21-30	1	9.1
31-40	2	18.2
41-50	1	9.1
51-60	3	27.3
>60	2	18.2
Total	11	100

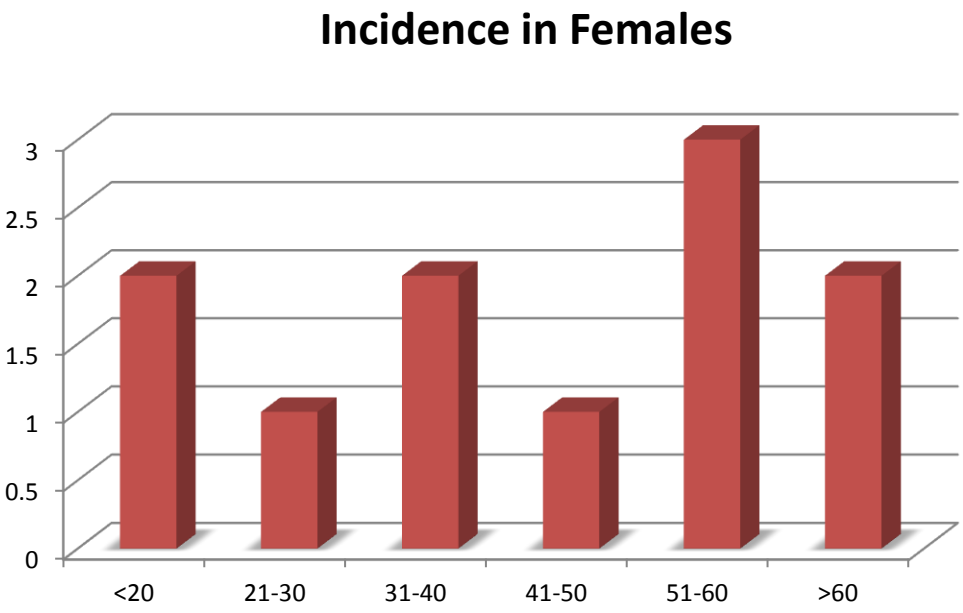


TABLE 4 : INCIDENCE

Age in years	Male		Female		Total	
	No	%	No	%	No	%
<20	2	4.1	2	18.2	4	6.7
21-30	1	2.1	1	9.1	2	3.3
31-40	10	20.4	2	18.2	12	20
41-50	7	14.3	1	9.1	8	13.3
51-60	14	28.6	3	27.3	17	28.3
>60	15	30.6	2	18.2	17	28.3
Total	49	100	11	100	60	100
Mean SD	52.27+17.1		42.18+16.71		50.42+17.31	

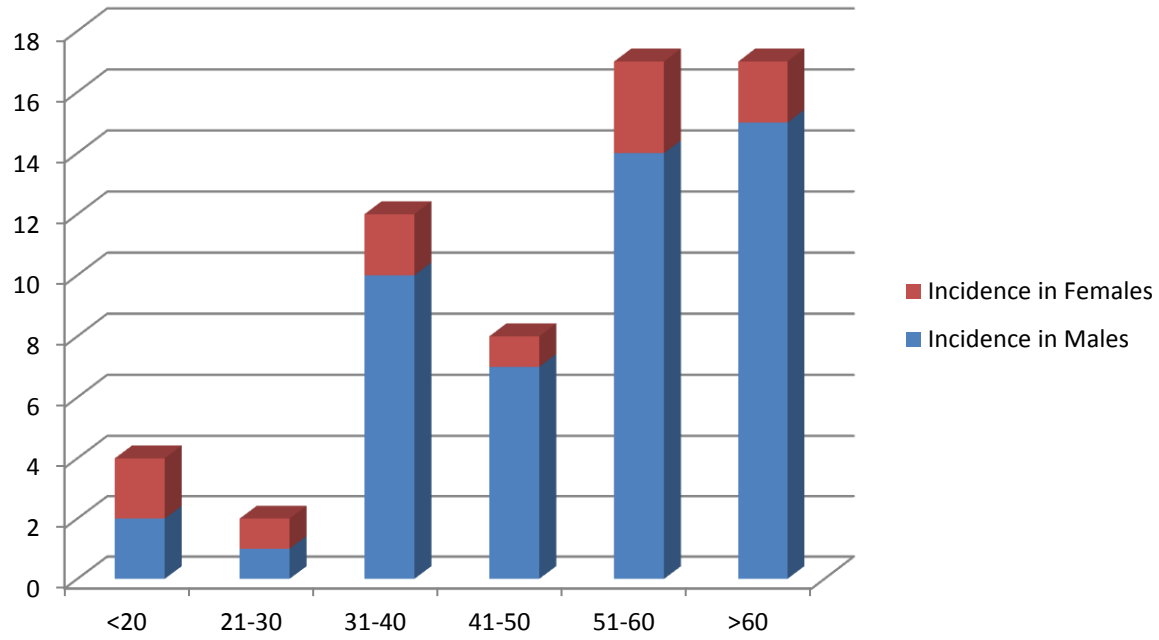
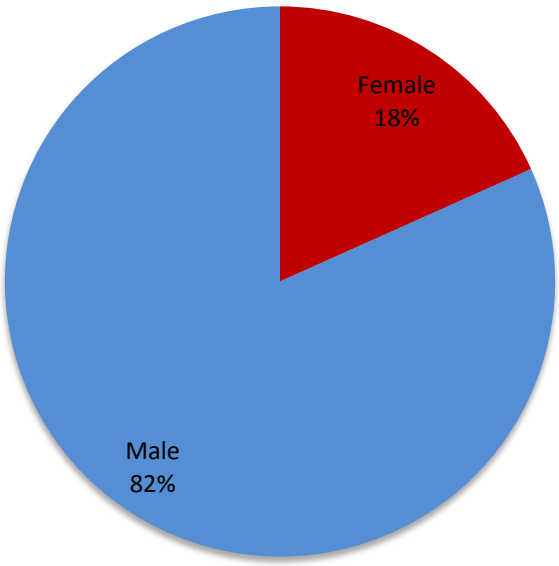


TABLE 5 : GENDER DISTRIBUTION

Age in years	Male	Female
<20	4.1	18.2
21-30	2.1	9.1
31-40	20.4	18.2
41-50	14.3	9.1
51-60	28.6	27.3
>60	30.6	18.2



GENDER DISTRIBUTION OF THE PATIENTS STUDIED

TABLE 6: OCCUPATION OF PATIENTS

Occupation	Number of Patients	%
Farmer	17	28.3
Housewife	10	16.7
Unskilled	12	20
Service	9	15
Skilled	10	16.7
Not applicable	2	3.3
Total	60	100

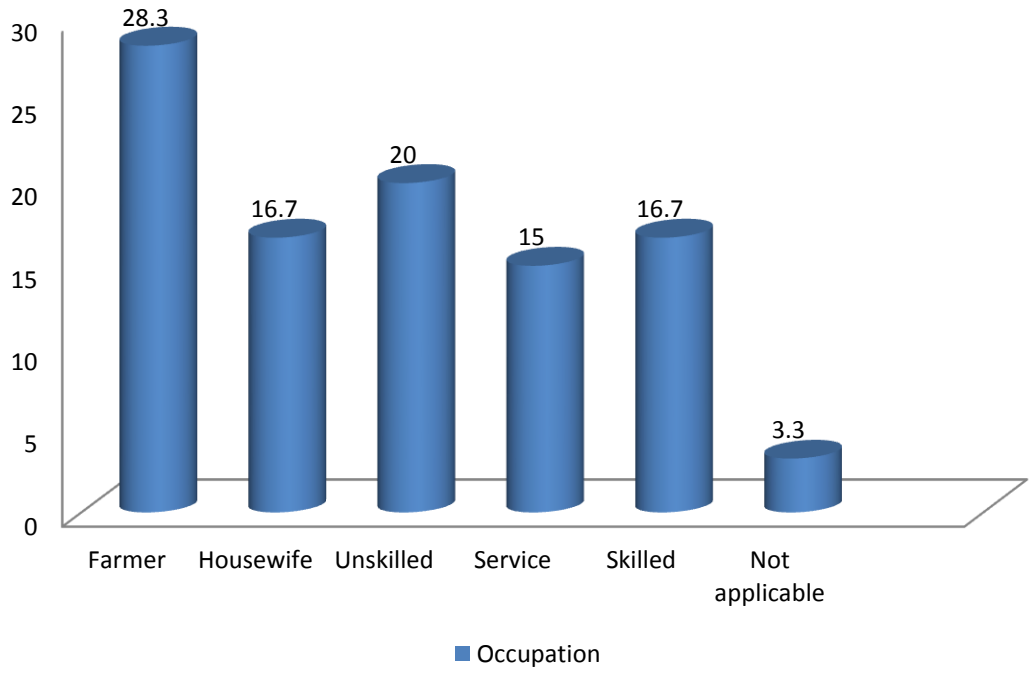
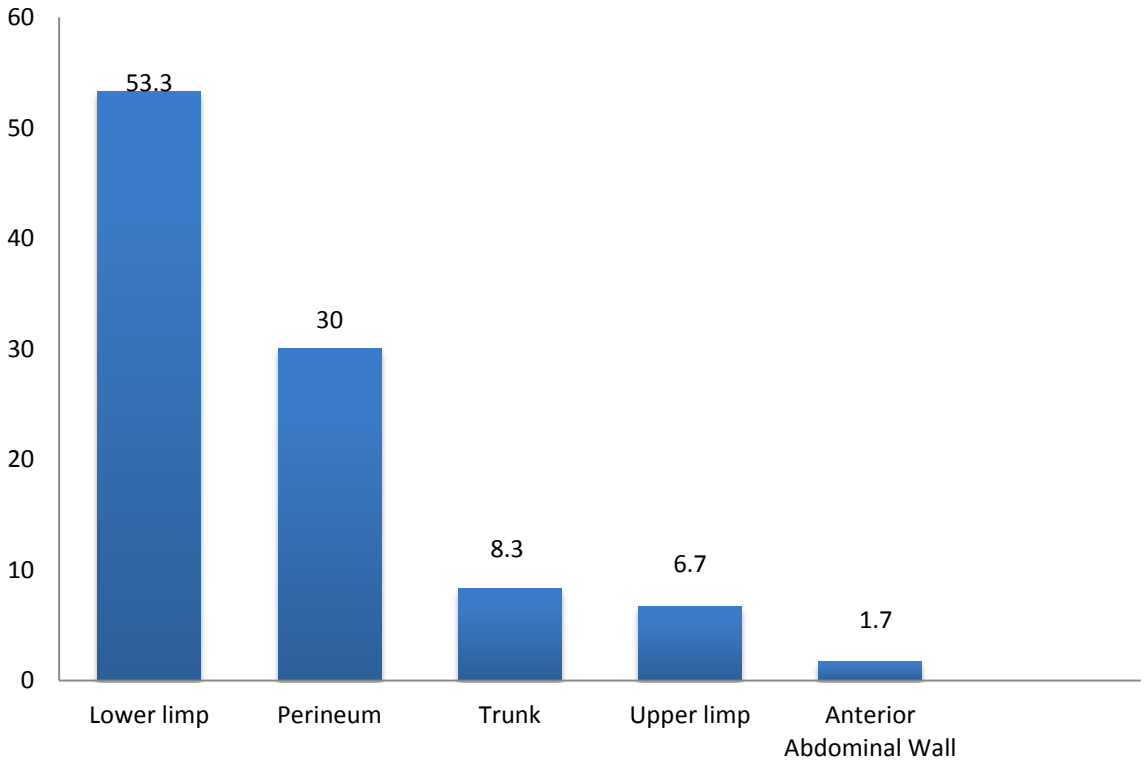


Table 7 : SITE

Site	Number of Patients	%	95% CI
Lower limb	32	53.3	40.89-65.37
Perineum	18	30	19.90-42.61
Trunk	5	8.3	3.61-18.07
Upper limb	4	6.7	2.62-15.93
Anterior Abdominal Wall	1	1.7	0.3-8.96
Total	60	100	-



Site of Necrotizing Fasciitis

Table 8 : SYMPTOMS AT PRESENTATION

Symptoms	Number of patients (n=60)	%	955CI
Pain	57	95	86.30-98.29
Fever	37	61.7	49.62-72.91
Discharge	35	58.3	45.73-69.94
Swelling	27	45	33.09-57.51
Blisters/Skin Vesicles	11	18.3	10.56-29.52
Hyper pigmentation/ Hard Texture of skin	8	13.3	6.91-24.17

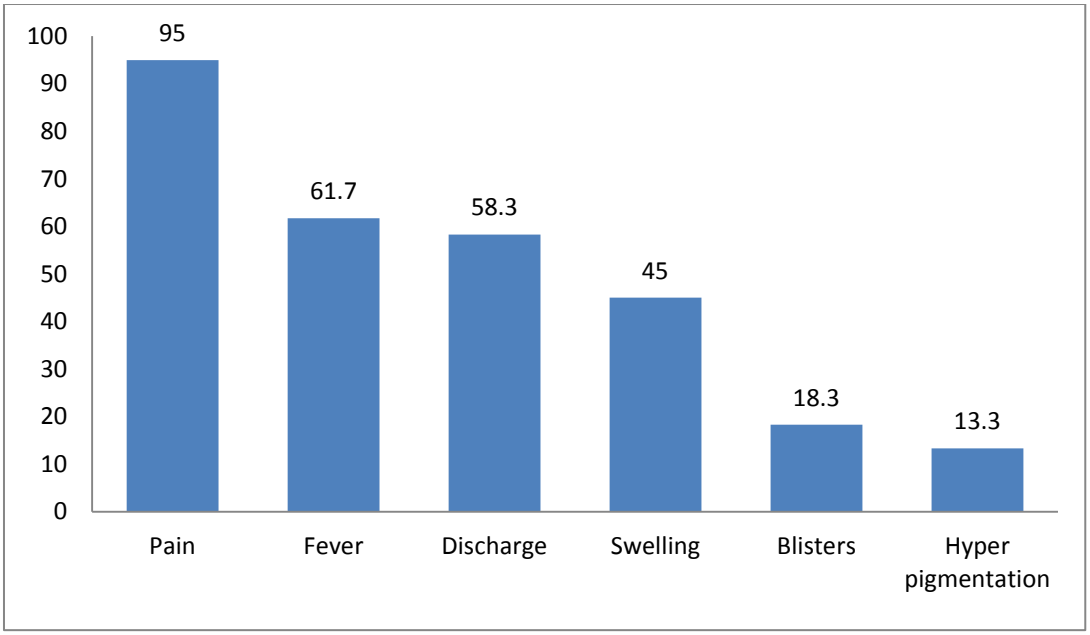


TABLE 9 : DURATION OF SYMPTOMS

Duration of Symptoms	Number of patients (n=60)	%
1-7 days	33	55
8-14 days	19	31.7
>14 days	8	13.3

Mean + SD: 10.08 + 10.09

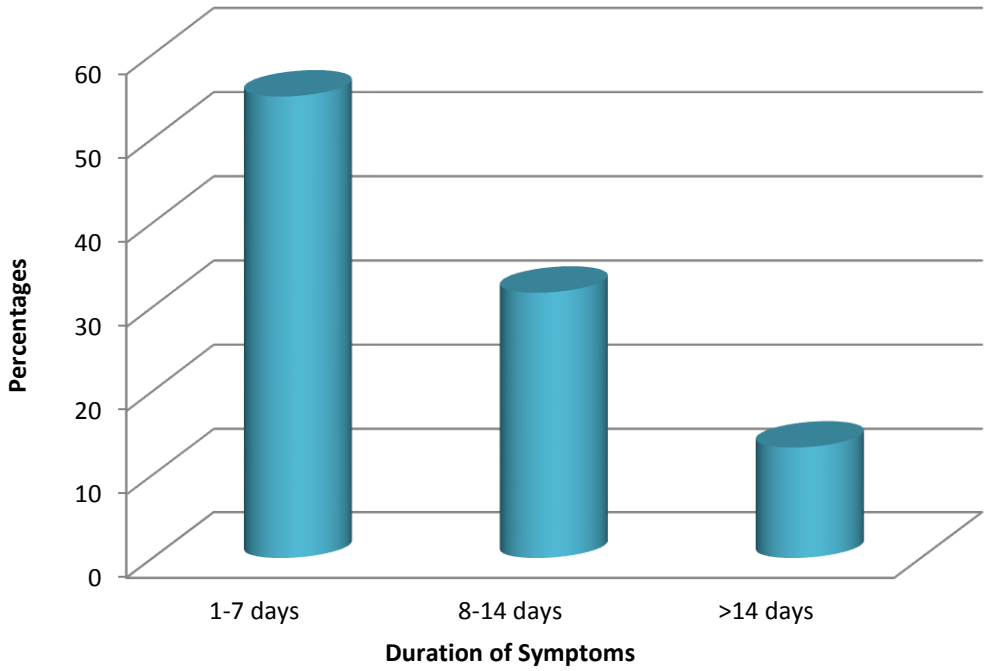
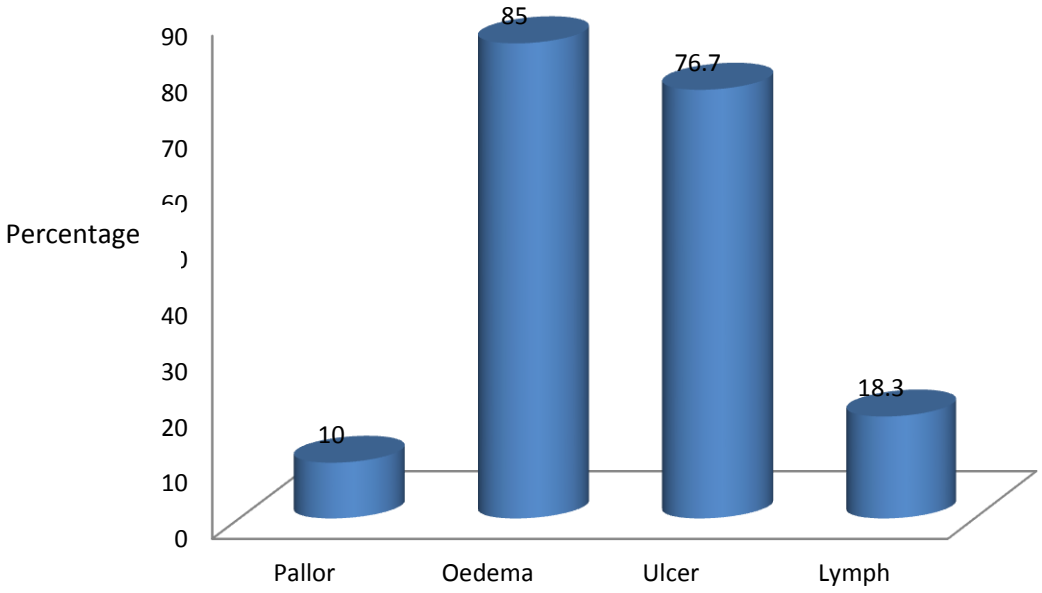


Table 10 : SIGNS

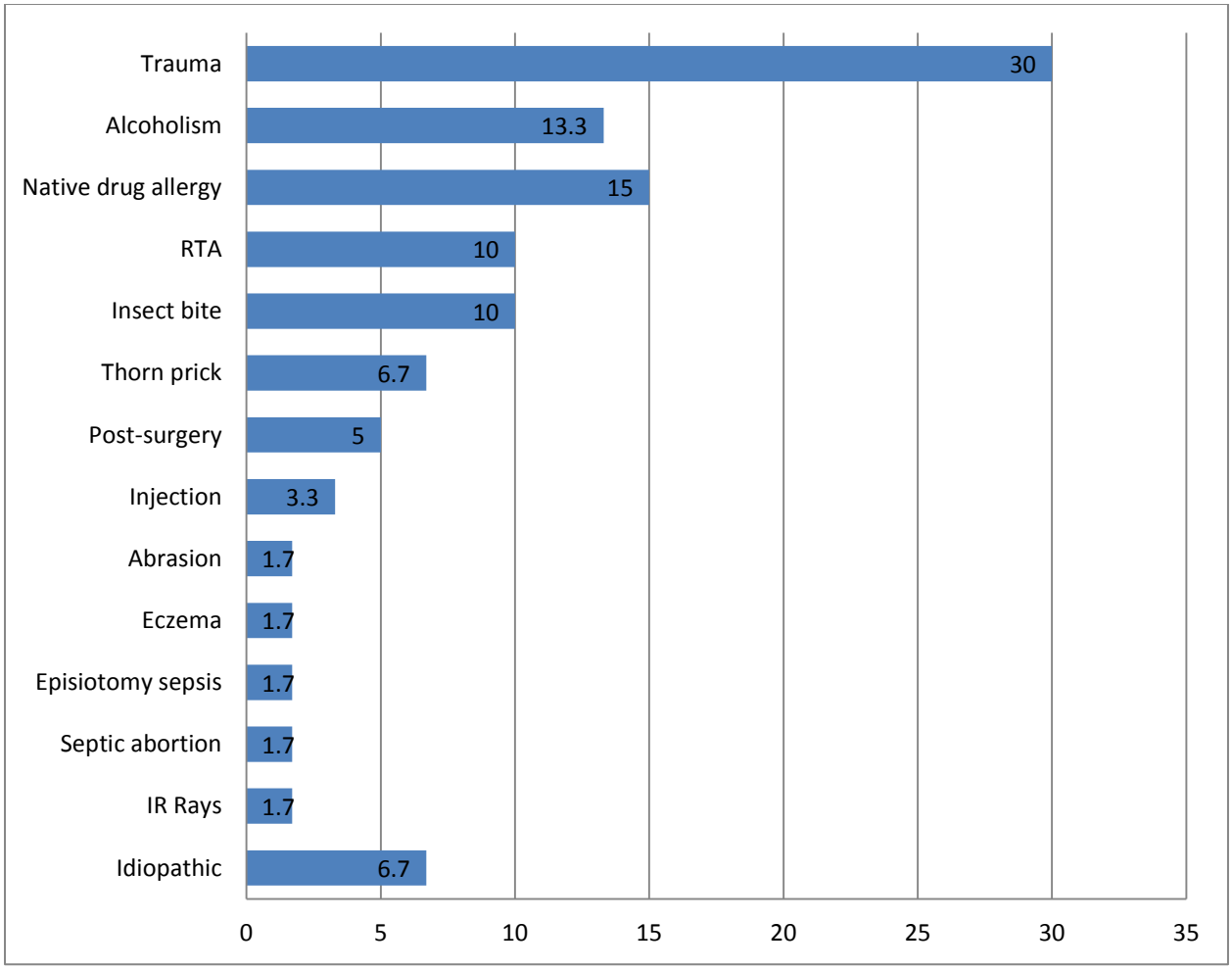
Signs	Number of patients (n=60)	%	95% CI
Pallor	6	10	3.61-18.07
Oedema	51	85	73.89-91.90
Ulcer	46	76.7	64.56-85.56
Lymph	11	18.3	10.56-29.92



Signs

Table 11 : ETIOLOGY

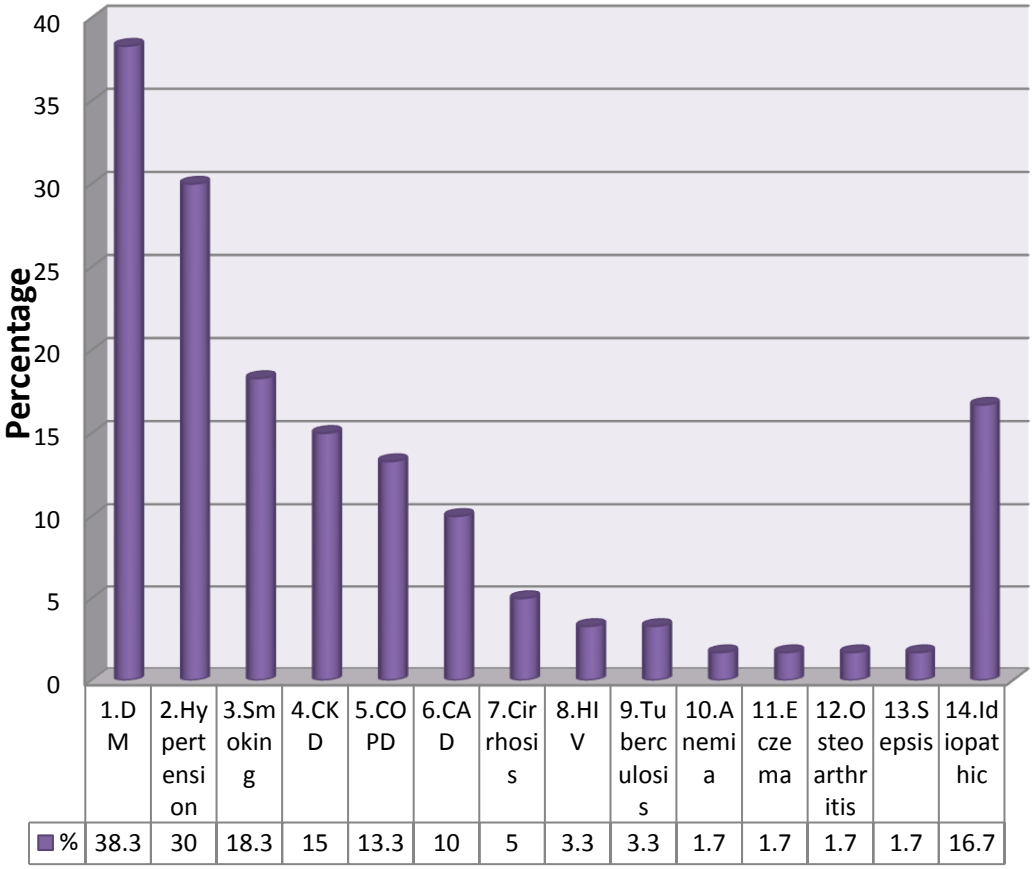
Etiology	Number of patients (n=60)	%	95 % CI
Trauma	18	30	19.90-42.61
Alcoholism	8	13.3	6.91-24.17
Native drug allergy	9	15	8.10-26.11
RTA	6	10	4.86-20.15
Insect bite	6	10	4.86-20.15
Thorn prick	4	6.7	2.62-15.93
Post-surgery	3	5	1.71-13.70
Injection	2	3.3	0.9-11.36
Abrasion	1	1.7	0.3-8.86
Eczema	1	1.7	0.30-8.86
Episiotomy sepsis	1	1.7	0.30-8.86
Septic abortion	1	1.7	0.30-8.86
IR Rays	1	1.7	0.30-8.86
Idiopathic	4	6.7	2.62-15.93



Etiology of Necrotizing Fasciitis

TABLE 12: ASSOCIATED CO MORBID CONDITIONS

Associated Co Morbid conditions	Number of patients (n=60)	%
1.DM	23	38.3
2.Hypertension	18	30
3.Smoking	11	18.3
4.CKD	9	15
5.COPD	8	13.3
6.CAD	6	10
7.Cirrhosis	3	5
8.HIV	2	3.3
9.Tuberculosis	2	3.3
10.Anemia	1	1.7
11.Eczema	1	1.7
12.Osteo arthritis	1	1.7
13.Sepsis	1	1.7
14.Idiopathic	10	16.7



Associated Co morbid conditions

Table 13 : COMPOSITION OF ISOLATED ORGANISMS

Isolated organisms	Number of cases	Percentage of cases
E coli	28	46.6
Streptococcus	25	41.6
Klebsiella	22	36.6
Pseudomonas	16	26.6
Staphylococcus	10	16.6
Enterococcus	8	11.6
Proteus	8	11.6
Bacteroides	4	6.6
Acinetobacter	3	5

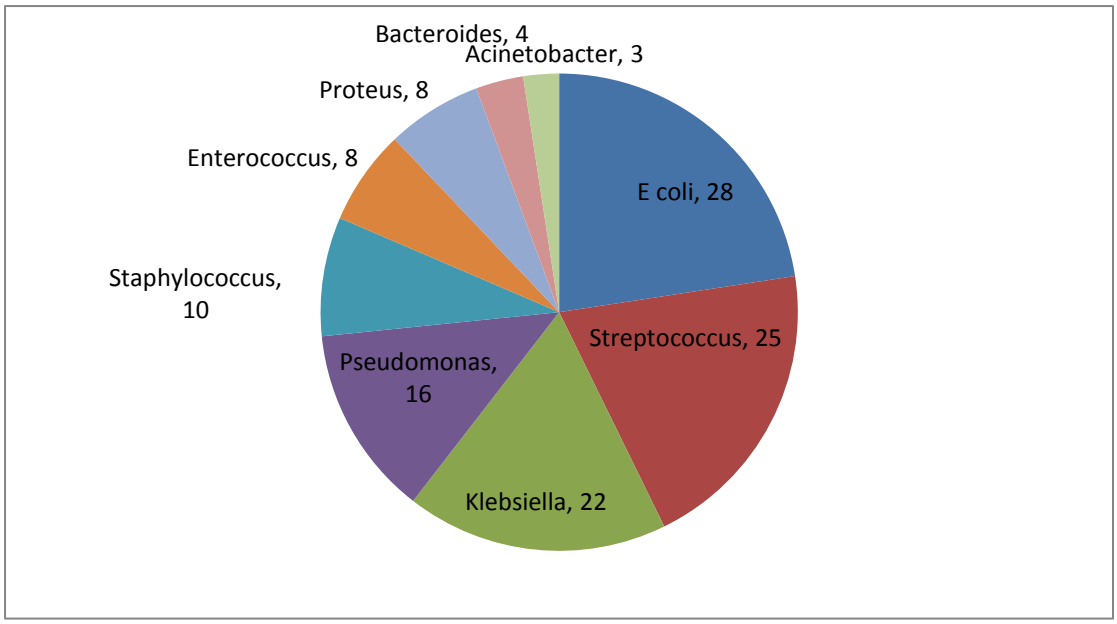


Table 14 : MICROBES ISOLATED

Number of microbes isolated	Number of cases	Percentage of cases
Nil	4	6.6
One	8	13.3
Two	35	58.3
Three	8	13.3
Four	5	8.3

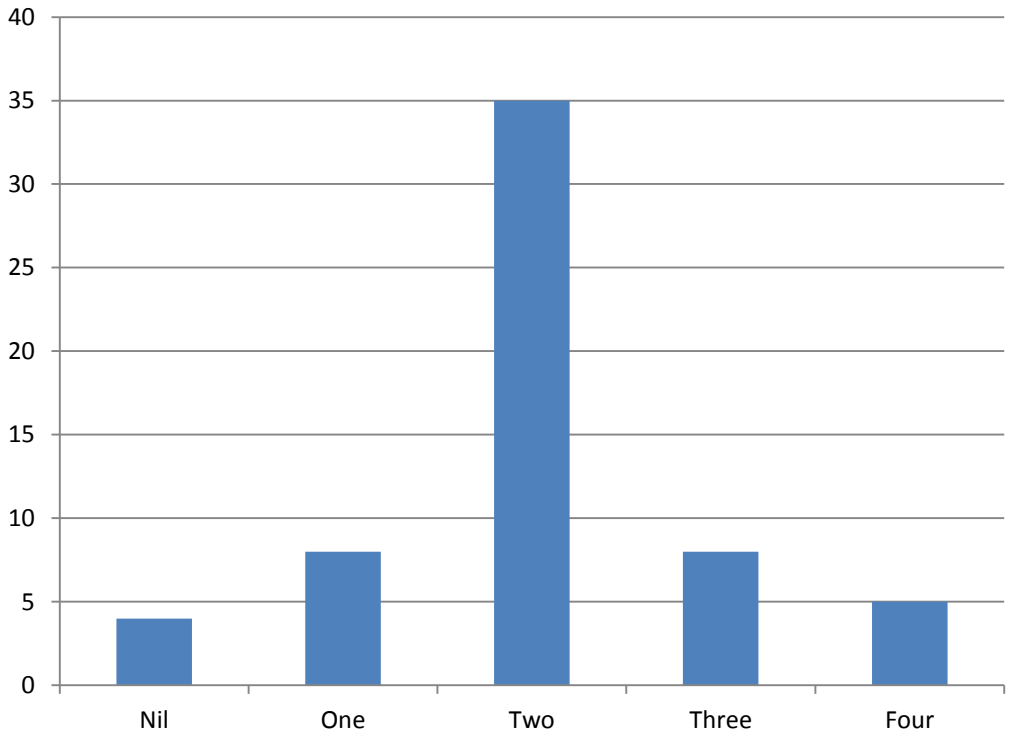
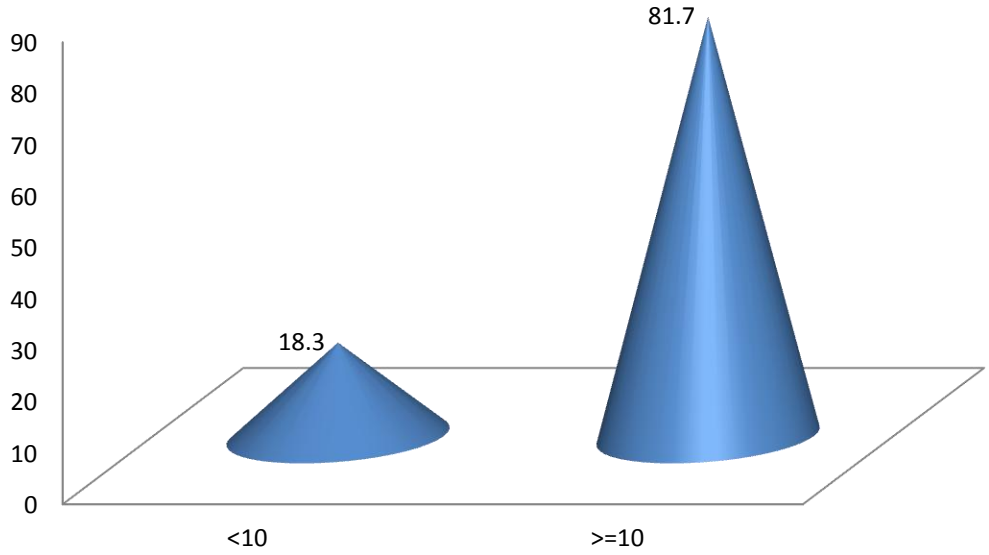
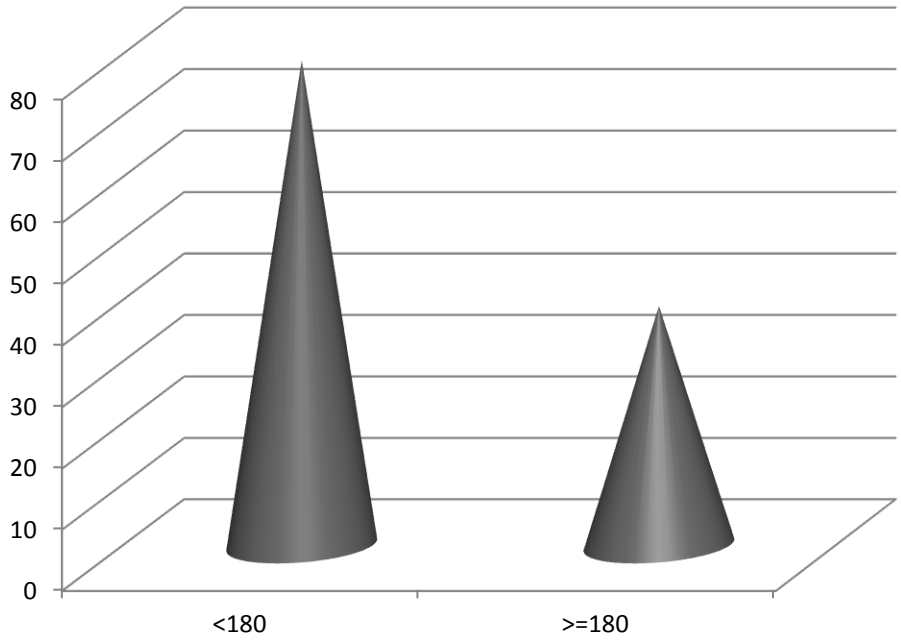


Table 14 : LABORATORY INVESTIGATIONS ON ADMISSION

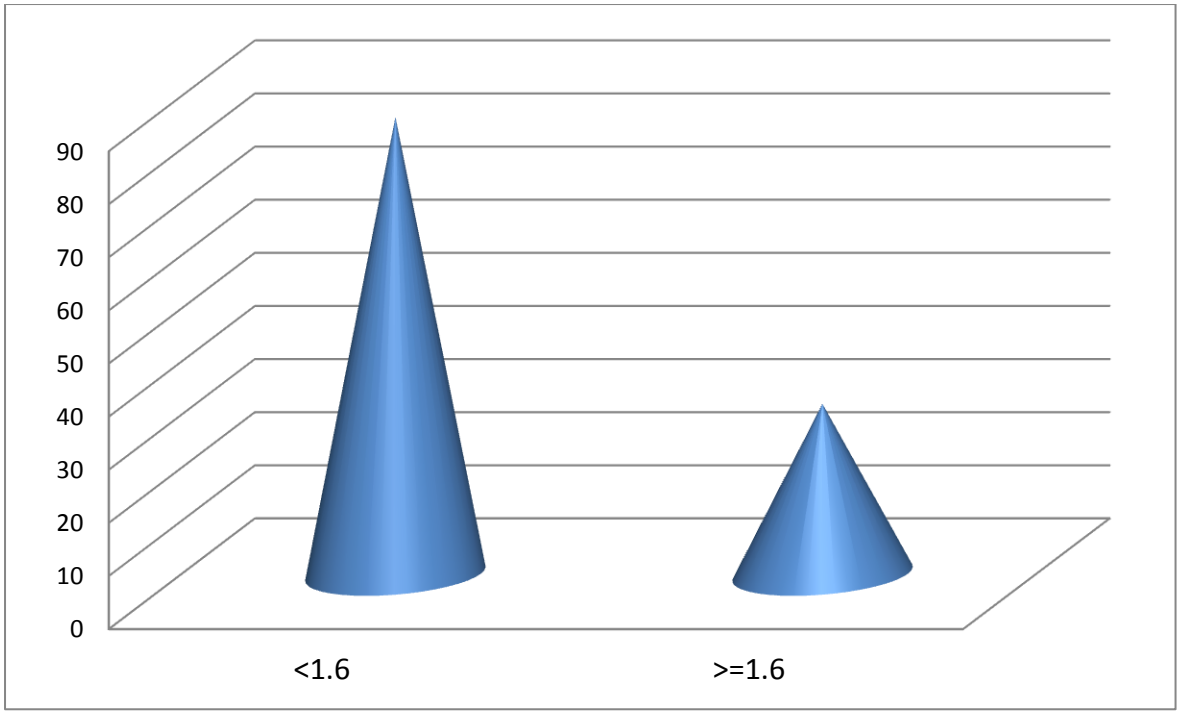
Blood Parameters	Number of patients (n=60)	%	95% CI
Hemoglobin gm%			
<10	11	18.3	10.66-29.92
>=10	49	81.7	70.08-89.44
RBS mg/dl			
<180	42	70	57.49-80.10
>=180	18	30	19.90-42.51
Serum Creatinine mg/dl			
<1.6	46	76.7	64.56-85.66
>=1.6	14	23.3	14.44-35.44
Sodium mEq/L			
<128	9	15	8.10-26.11
>=128	51	85	73.89-91.90



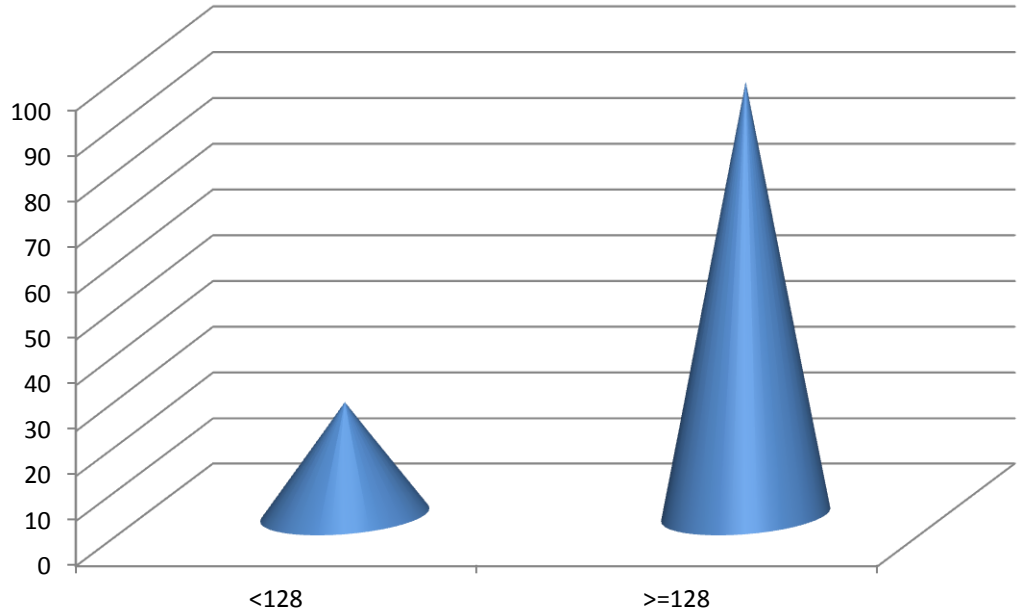
Hemoglobin gm%



RBS mg/dl



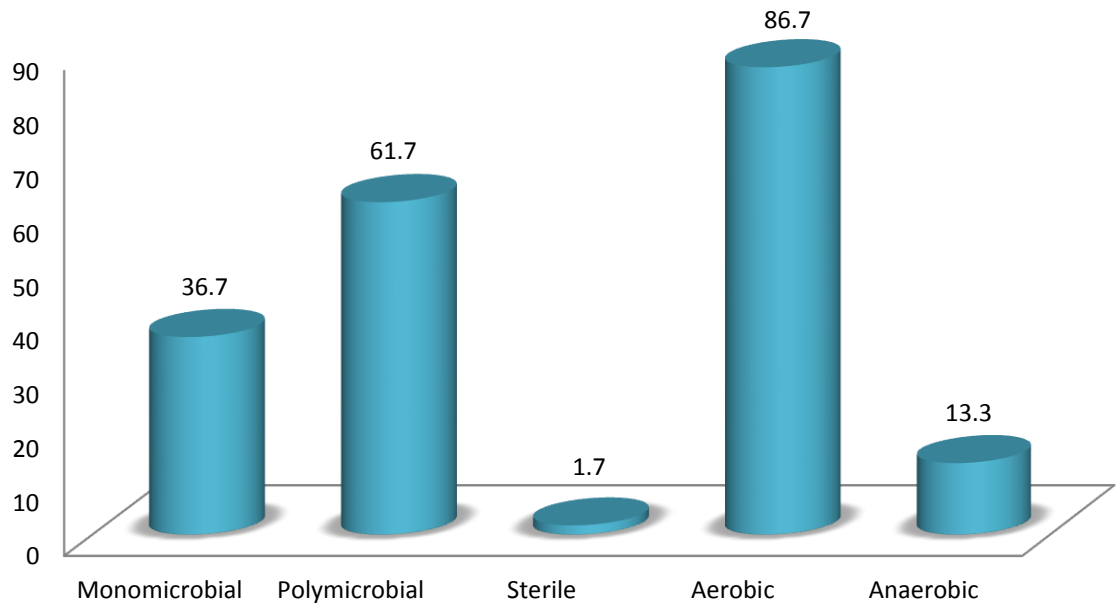
Serum Creatinine mg/dl



Sodium mEq/L

Table 15 : BACTERIOLOGICAL STUDY

Bacteriological Study	Number of patients (n=60)	%	95% CI
Monomicrobial	22	36.7	25.62-49.32
Polymicrobial	37	61.7	49.02-72.91
Sterile	1	1.7	0.3-8.86
Aerobic	52	86.7	75.83-93.09
Anaerobic	8	13.3	6.91-24.17



Bacteriological Study

Table 16: COMMONLY IDENTIFIED BACTERIA

Isolated organisms	Numberof cases	Percentage of cases
E coli	28	46.6
Streptococcus	25	41.6
Klebsiella	22	36.6
Pseudomonas	16	26.6
Staphylococcus	10	16.6
Enterococcus	8	11.6
Proteus	8	11.6
Bacteroides	4	6.6
Acinetobacter	3	5

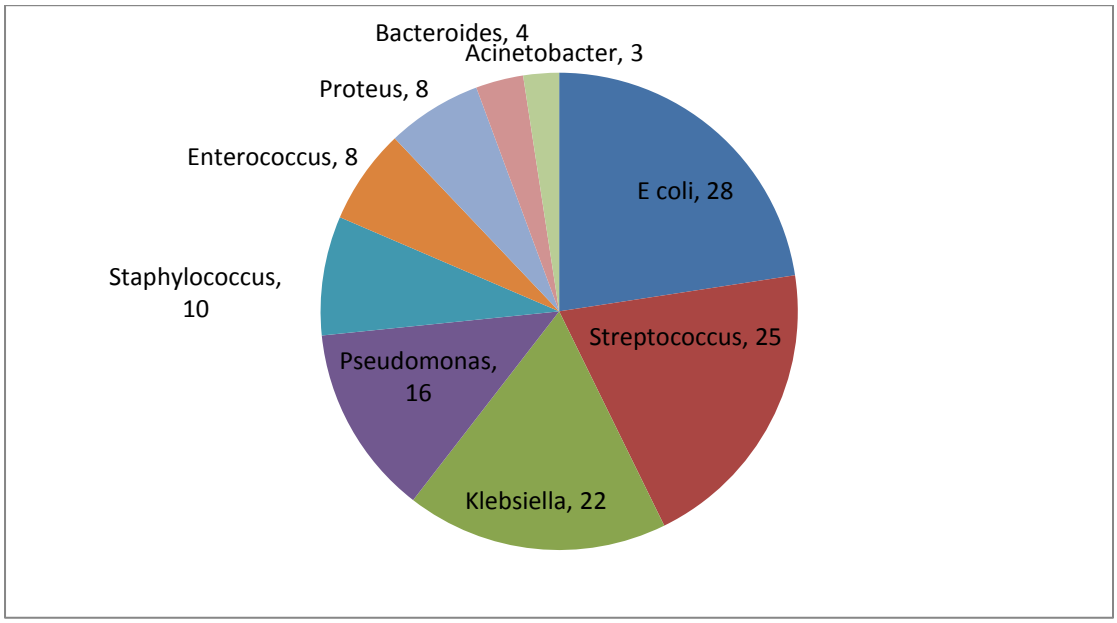
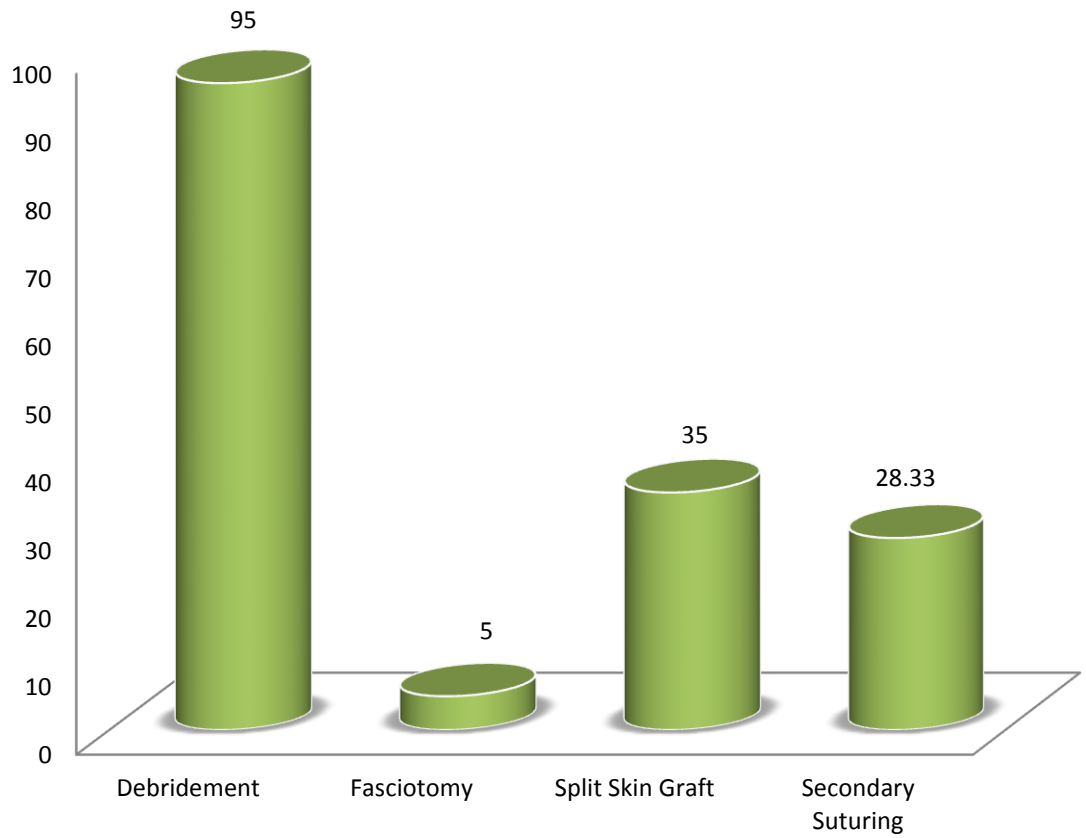


Table 17: TREATMENT

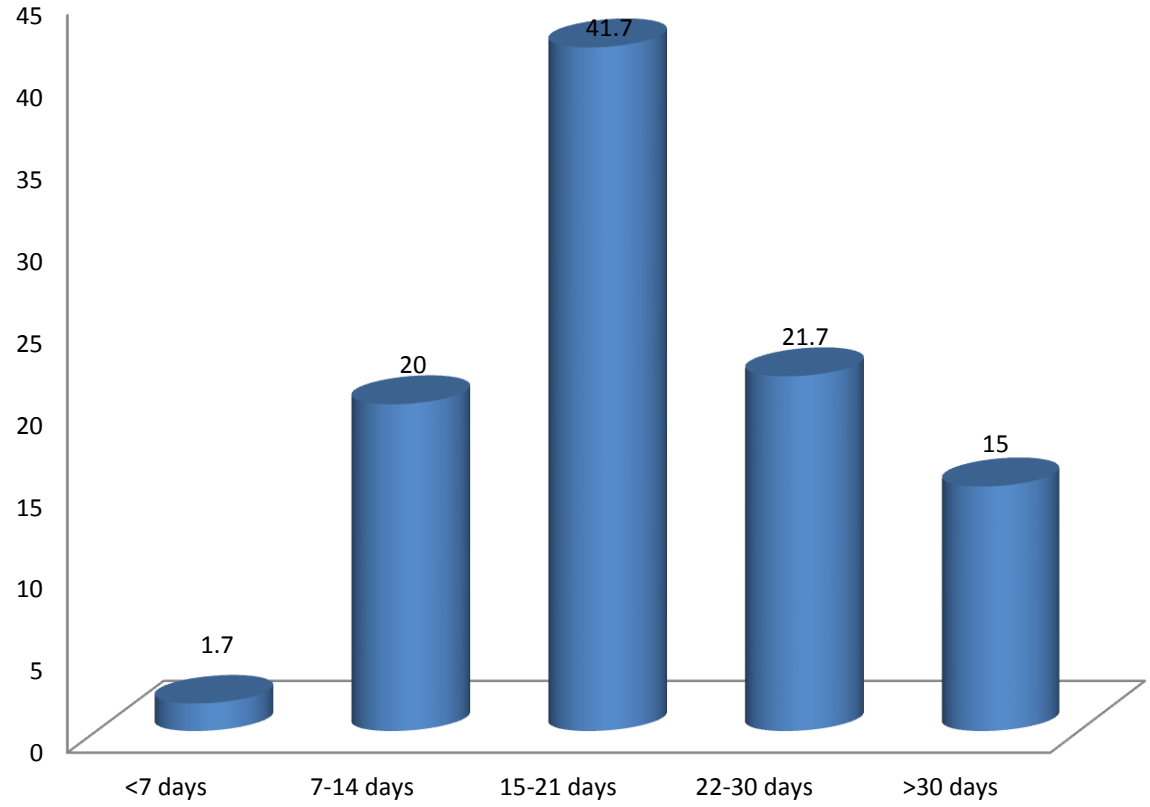
Treatment	Number of Patients	%
Debridement	57	95
Fasciotomy	3	5
Split Skin Graft	21	35
Secondary Suturing	17	28.33



TREATMENT

Table 18 : HOSPITAL STAY IN DAYS

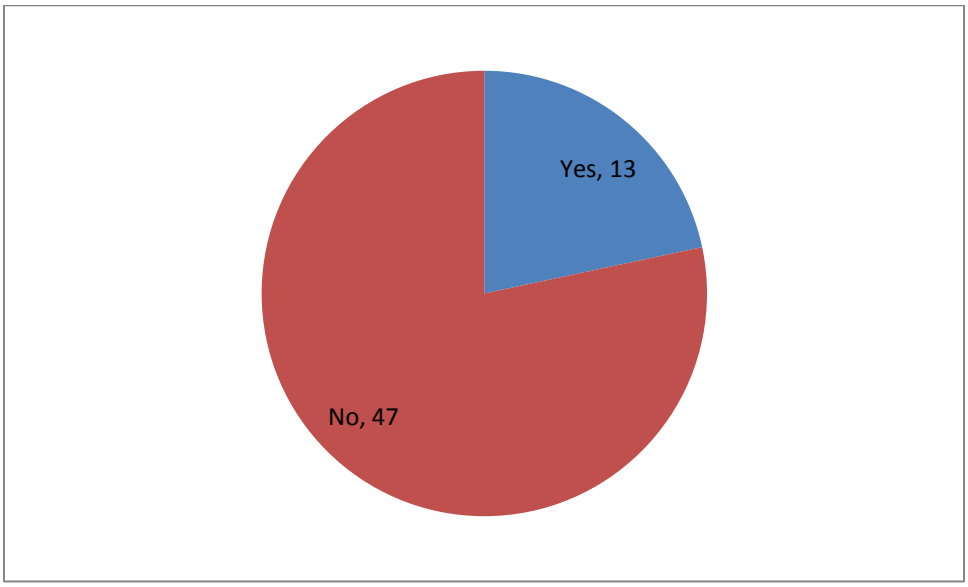
Hospital stay in days	Number of patients (n=60)	%
<7 days	1	1.7
7-14 days	12	20
15-21 days	25	41.7
22-30 days	13	21.7
>30 days	9	15



HOSPITAL STAY IN DAYS

Table 19 : MORTALITY

Mortality	Number of cases	Percentage of cases
Yes	13	21.6
No	47	78.3



DISCUSSION

Incidence of necrotizing fasciitis was high in the males. The incidence of necrotizing fasciitis increases with increase in age with the highest incidence noted in above 60 yrs. age. On the contrary the incidence of necrotizing fasciitis in women has more even distribution with the highest in the 50-60 yrs. age group. Majority of the cases of necrotizing fasciitis occurs in the males with more than 80% occurrence in male. The overall incidence was higher in the older age group with the highest in the 50 – 60 yrs.

This increase in the incidence with age might be due to higher occurrence of the risk factors in the older age group.Male population have higher incidence of traumatic injury, alsohigher workplace hazards and alcoholism among male population are the predisposing factors for higher incidence of necrotizing fasciitis in male population.

Work place forms the one of the most common place for the origin of infection. Minor injuries are common in workplaces that requires physical labor. Lack of proper safety precautions and bad hygiene in the work place forms it a perfect combination for the origin of infection. The incidence was highest among the farmers. Unskilled labor and housewife's form the major bulk along with the farmers. This pattern shows that with improving the hygiene, safety and the working

environment and through proper training of manual labor the incidence of necrotizing fasciitis can be reduced.

Majority of the cases of necrotizing fasciitis follow minor injury due to trauma, RTA, insect bite, thorn prick, etc.,.poor care for the wound following the trivial trauma is the major cause for necrotizing fasciitis. The incidence of necrotizing fasciitis was highest following the trauma. The foreign body that might get lodged or the deep inoculation that occurs with trauma, thorn prick and other cause forms a perfect incubator foe the organisms to flourish this complemented by lowered host defense due to alcoholism, diabetes leads to fulminant local infection leading to necrotizing fasciitis.

Lower limb is the most common site for necrotizing fasciitis. This is followed by perineum and upperlimb. The least common site for necrotizing fasciitis is the anterior abdominal wall. Similar studies conducted in other parts of the world show the perineum as the most common site of necrotizing fasciitis. This difference might due to difference in work pattern, higher safety precautions in the west and difference in the hygiene among the population.

Most of the patients came with the presenting complain of pain, swelling and discharge of the affected area. Most of them had high grade fever. Most common sign in necrotizing fasciitis is edema followed by

ulcer .farmer s has highest incidence of necrotizing fasciitis ,that is because due to minor trauma duringoccupation which was unnoticed at the earliest and rapid spread of infection .trauma is the most common etiology in this study .

Host defense lost due to severe systemic illness. of the patient with diabetic mellitus 20 to 40 percent were found to be diabetic followed by hypertension .

Most commonly the disease is poly microbial of about 62%

Most common isolated organism is E.coli 35% .almost all patients treated with broad spectrum antibiotics .most common antibiotic sensitive is ceftriaxone followed by aminoglycosides and meteronidazole.

Almost all patients underwent surgical debridement of about 95%and some may underwent procedures like primary suturing secondary suturing and ssg .

CONCLUSION :

NECROTIZING FASCITIS is most commonly seen in the elderly males. source of the infection is identifiable in most of the cases.

diabetic mellitus is the most common comorbid factor . The disease is most commonly polymicrobical.

Most common bacterial includes gram positive cocci (streptococci and staphylococcal species).

Gram negative (E.coli, actinobacter ,pseudomonas).

Most common anaerobes is bacteroides .

Necrotizing fasciitis is a rare life threatening condition , early recognition of disease , and through wound debridement and broad spectrum antibiotics is essential extensive raw area due to debridement can be managed by reconstructive procedures .

Inspite of early diagnosis and aggressive management of disease also leads to significant mortality and morbidity . surgical management must be aggressive and standard procedures to be followed . new modalities of treatment like HBOT and VAC may be considered .

Diagnosis of necrotizing fasciitis is mainly through by means of clinical examination ,but diagnostic adjuvant such as LRINEC scoring system can be used for early diagnosis .

Multidisciplinary team approach is required for necrotizing fasciitis.

BIBILOGRAPHY

1. Bailey and love's TEXT BOOK OF SURGERY,25th edition, pg no 29-30
2. SRB manual of surgery, 4th edition, pg no 65-67
3. Sabiston text book of surgery, 19th edition , pg no 2011-2012
4. Schwartz principles of surgery, 10thedition,pg no 152-154
5. Learning surgery, the surgery clerkship manual, pgno 111-112
6. S das text book of clinical surgery, pg no 618, 632
7. T S Ranganathan, 9th edition, pg no 140-143
8. Baek JH et al surgical management of Fournier's gangrene. J Korean Soccolproctol 19:349-53
9. Basoglu M, Ozbey et al, management of fournier's gangrene, surg today 37: 558-563
10. Wilson B Necrotisingfasciitis,Amsurg 1952 Apr;18(4): 416-31

11. Loudon I. necrotizing fasciitis, hospital gangrene and phagedema, Lancet1994: 344: 1416-1419
12. Nallathambi MN, Ivatury et al craniocervicalnecrotising fasciitis critical factor in management. Can J surg 1987; 30:61-3
13. Rea WJ , necrotizing fasciitis. Ann surg 1970; 172;957-64

PROFORMA

Name : Age: Sex:

IP.No:

Address : D.O.Admission :

D.O.Discharge/Death :

Duration of illness:

History:

1. Fever
2. Lethargy
3. Pain
4. Pruritis
5. Redness of skin
6. Other skinchanges
7. Purulent discharge
8. Obvious gangrene
9. Anorectal symptoms
10. Urogenital symptoms
11. History of skin infections
12. History of comorbid illness
13. History of recent invasive procedures
14. History of alcohol intake

GENERAL EXAMINATION

Pulse

BP

Temperature

Respiratory rate

Nutrition

Hydrational status

LOCAL EXAMINATION

Fulctuations

Localizing tenderness

Crepitations

Occult wounds

Skin changes

Foul smelling odour

Subcutaneous crepitations

Digital examination of the rectum

Testicular involvement

Extent of the disease

INVESTIGATIONS

Complete hemogram

B.Urea

B.Sugar

S.creatinine

S.Electrolytes

Blood Culture

Pus Culture

Urine Culture

Chest X-Ray

ECG

VCTC

LRIHEC

MANAGEMENT

Debridement details :

Number of debridements :

Recovery period :

Culture and sensitivity reports :

Reconstructive procedures:

Postoperative period :

Outcome :

Follow up :

Sl.NO	Name	Age	Sex	IP.No	OCCUPATION	region affected
1	chellapan	63	m	13492	FARMER	LOWER LIMB RT ANKLE
2	Arusamy	58	m	34642	COOLIE	LEFT TIBIA LOWER LIMB
3	Ranganathan	52	m	38518	ELECTRICIAN	LT ANKLE LOWER LIMB
4	rajan	60	m	40111	SHOP KEEPER	SCROTUM PERINEUM
5	aruna	23	f	47155	HOUSE WIFE	EXTERNAL GENITALIA PERINEUM
6	mariammal	50	f	52258	HOUSE WIFE	LOW BACK TRUNK
7	Subramani	58	m	56215	FIREMAN	RT THIGH LOWER LIMB
8	Badrinath	45	m	21800	FARMER	LT THIGH LOWER LIMB
9	Annadurai	72	m	41264	STUDENT	SCROTUM PERINEUM
10	Sulaiman	57	m	61275	FARMER	LT INGUINAL REGION TRUNK
11	Madhu	37	m	52955	CARPENTER	LEFT FOOT DORSUM LOWER LIM
12	rani	35	f	43777	HOUSE WIFE	LT LOWER LIMB
13	Veeran	37	m	56188	SECURITY	RT THIGH LOWER LIMB AND ANI
14	vellaiammal	40	f	58597	HOUSE WIFE	LT HAND UPPER LIMB
15	Paramasivam	81	m	69374	FARMER	LT MEDIAL MALLEOLUS LOWER
16	vigneshwari	16	f	148	SALES GIRL	RT LEG LOWER LIMB
17	Jeya raman	70	m	954	PENSIONER	RT THIGHLOWER LIMB
18	murugeshwari	65	f	1542	HOUSE WIFE	LOW BACK TRUNK
19	Rajkumar	78	m	47963	FARMER	RT KNEE LOWER LIMB
20	Raghupathy	60	m	69180	FARMER	LT FOOT LOWER LIMB
21	Subramanian	37	m	74205	FARMER	RT FOOT AND LAT MALLEOLUS L LIMB

22	Thiyagarajan	76	m	45204	PENSIONER	LT ANKLE LOWER LIMB
23	Rangammal	61	f	32102	HOUSE WIFE	LT THIGH LOWER LIMB
24	devandran	58	m	57270	COOLIE	RT FOOT AND LAT MALLEOLUS L LIMB
25	rangan	41	m	43777	FARMER	RT ENTIRE LEG
26	Santhanam	58	m	33850	FARMER	LT ANKLE LOWER LIMB
27	Poongothai	51	f	59895	HOUSE WIFE	LT THIGH LOWER LIMB
28	Gnanasekaran	62	m	48253	FARMER	LT THIGH LOWER LIMB
29	Rashid	58	m	1347	FARMER	RT THIGH LOWER LIMB
30	Natraj	22	m	7008	ELECTRICIAN	LOW BACK TRUNK
31	Aruchamy	65	m	30819	FARMER	LT THIGH LOWER LIMB
32	mahalakshmi	51	f	10219	HOUSE WIFE	RT HAND UPPER LIMB
33	Kandhasamy	65	m	10354	COOLIE	RT FOOT AND LAT MALLEOLUS L LIMB
34	Peter	77	m	9658	PENSIONER	LT THIGH LOWER LIMB
35	Velu	40	m	32457	DRIVER	LT FOOT LOWER LIMB
36	Poonmudi	40	m	65492	TEACHER	LT THIGH AND CALF LOWER LIM
37	durairaj	50	m	55233	COOLIE	RT THIGH MEDIAL LOWER LIMB
38	manokaran	48	m	62703	SALES MAN	LT HAND UPPER LIMB
39	karuppasamy	58	m	15603	TEACHER	RT THIGH LOWER LIMB
40	mallika	20	f	67880	HOUSE WIFE	ANT ABDOMINAL WALL
41	sureshbabu	40	m	65003	CARPENTER	LT ANKLE LOWER LIMB
42	palinisamy	40	m	69318	FARMER	LOW BACK TRUNK
43	rajendran	45	m	40347	CLERK	LT THIGH LOWER LIMB

44	manjula	52	f	45512	HOUSE WIFE	RT HAND UPPER LIMB
45	Mariyappan	51	m	20899	DRIVER	RT THIGH LOWER LIMB
46	radha krishnan	70	m	8452	FARMER	FOURNIER GANGRENE PERINEUI
47	velusamy	80	m	59595	PENSIONER	FOURNIER GANGRENE PERINEUI
48	mayilsamy	60	m	73255	FARMER	FOURNIER GANGRENE PERINEUI
49	Kandhasamy	63	m	26397	DRIVER	FOURNIER GANGRENE PERINEUI
50	marimuthu	61	m	39366	COOLIE	FOURNIER GANGRENE PERINEUI
51	kittan	63	m	36823	CARPENTER	FOURNIER GANGRENE PERINEUI
52	raja	37	m	20379	DRIVER	FOURNIER GANGRENE PERINEUI
53	Natraj	32	m	57464	MECHANIC	FOURNIER GANGRENE PERINEUI
54	Paramasivam	45	m	4656	COOLIE	FOURNIER GANGRENE PERINEUI
55	adalarasu	37	m	67161	CLERK	FOURNIER GANGRENE PERINEUI
56	Veeran	63	m	47163	FARMER	FOURNIER GANGRENE PERINEUI
57	simon	57	m	23748	SALES MAN	FOURNIER GANGRENE PERINEUI
58	bhuvaneshwaran	41	m	39938	COOLIE	FOURNIER GANGRENE PERINEUI
59	palanisamy	14	m	10132	NA	FOURNIER GANGRENE PERINEUI
60	mani	35	m	10219	FARMER	FOURNIER GANGRENE PERINEUI