A CROSS SECTIONAL STUDY TO ASSESS THE OUTCOME OF NECROTIZING FASCIITIS AMONG DIABETIC PATIENTS IN CHENGALPATTU MEDICAL COLLEGE

A 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

DEPARTMENT OF GENERAL SURGERY

CHENGALPET MEDICAL COLLEGE

CHENGALPATTU - 603001

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DECLARATION

I hereby declare that the dissertation entitled "**A CROSS SECTIONAL STUDY TO ASSESS THE OUTCOME OF NECROTIZING FASCIITIS AMONG DIABETIC PATIENTS IN CHENGALPATTU MEDICAL COLLEGE"** was done by me in the Department of General surgery, Chengalpattu Medical College during the tenure of my course in M.S. General Surgery from April-2018 to April – 2019 under the guidance and supervision of **Dr. J. Selvaraj M.S,** Professor and HOD, Department of General Surgery, Chengalpattu Medical College.

This dissertation is submitted to The Tamilnadu Dr. MGR Medical University, Chennai-32 towards the partial fulfilment of the requirement for the award of **M.S. DEGREE IN GENERAL SURGERY – BRANCH I**.

I have not submitted this dissertation on any previous occasion to any University for the award of any degree.

Place: Chengalpattu **DR. G. SATHISHKUMAR**

Date: **Reg No: .221711262**

CERTIFICATE

This is to certify that the dissertation entitled **"A CROSS SECTIONAL STUDY TO ASSESS THE OUTCOME OF NECROTIZING FASCIITIS AMONG DIABETIC PATIENTS IN CHENGALPATTU MEDICAL COLLEGE CERTIFICATE"** is the bonafide work done by **DR. G. SATHISHKUMAR,** Post Graduate student (2017 – 2020) in the Department of General Surgery, Government Chengalpattu Medical College and Hospital under my direct guidance and supervision, in partial fulfilment of the regulations of The Tamil Nadu Dr. M.G.R Medical University, Chennai for the award of M.S., Degree (General Surgery) Branch - I,

Examination to be held in May 2020.

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CERTIFICATE FROM THE GUIDE

This is to certify that the dissertation entitled **"A CROSS SECTIONAL STUDY TO ASSESS THE OUTCOME OF NECROTIZING FASCIITIS AMONG DIABETIC PATIENTS IN CHENGALPATTU MEDICAL COLLEGE "** submitted by the candidate **Dr. G. SATHISHKUMAR,** in partial fulfilment for the award of the degree of M.S. GENERAL SURGERY – BRANCH I by The Tamilnadu Dr. M.G.R. Medical University , Chennai-32 is a record of original and bonafide work done by him under my guidance and supervision in the Department of General surgery, Chengalpattu Medical College, Chengalpattu during the tenure of his course in M.S. General Surgery from April – 2018 to April – 2019, submitted in partial fulfilment of the requirements for the award of M.S. DEGREE IN GENERAL SURGERY – BRANCH I by The Tamilnadu Dr. MGR Medical University, Chennai-32.

Signature of the Guide **Dr. G. SATHISHKUMAR**

INSTITUTIONAL ETHICAL COMMITTEE

CHENGALPATTU MEDICAL COLLEGE, CHENGALPATTU

Title of Work	:	A cross sectional study to assess the outcome of Necrotizing Fascitis among Diabetic Patients in Chengalpattu Medical College.
Principal Investigator	:	Dr.Sathish Kumar.G
Designation	:	1st yr PG
Co-Investigators	:	Dr.T.Selvaraj,M.S., Professor and HOD, Department of General Surgery
Department	:	General Surgery

The request for an approval From the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 27.03.2018 at the Medical Education Unit, Government Chengalpattu Medical College, Chengalpattu at 11.00 AM.

The Members of the committee, the Secretary and the Chairman are pleased to inform you that your proposed project mentioned above is approved.

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MEMBER SECRETARY,
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PLAGARISM CERTIFICATE

This is to certify that this dissertation work titled **"A CROSS SECTIONAL STUDY TO ASSESS THE OUTCOME OF NECROTIZING FASCIITIS AMONG DIABETIC PATIENTS IN CHENGALPATTU MEDICAL COLLEGE"** of the candidate Dr .G. SATHISHKUMAR for the award of degree of M.S.GENERAL SURGERY – BRANCH I . I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows **6%** of plagiarism in the dissertation.

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I was able to finish my thesis to my satisfaction, thanks to guidance, support, motivation and constant input given to me, by **My Guide Prof. Dr. J. Selvaraj M.S, Professor and HOD of Department of General Surgery**, **Chengalpattu Medical College and Hospital**

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I sincerely thank those **patients** who in spite of their sufferings extended their kind co-operation to complete my study

I am fortunate to have co PG's of my Unit and other units and their invaluable inputs, and tireless help.

Finally, I thank my family members especially my parents and my wife who always encouraged me whenever I needed help and support.

CONTENT

INTRODUCTION

Necrotizing fasciitis is a rapidly spreading soft tissue infection that travels along subcutaneous fascial planes and obliterates perforating skin vessels, but spares the underlying muscle. The rapid pace of advancement coupled with high mortality rates has resulted in a heightened awareness by surgeons with a low threshold for intervention.

Predisposing factors include intravenous illicit drugs, immunosuppression, diabetes, malignancy, chronic kidney disease, vascular pathology, burns, insect bites, needle stick injury, and trauma. A rapid diagnosing of necrotizing fasciitis reduces morbidity and mortality. However, the diagnosis is notoriously difficult and often missed until very late in the hospital course.

Since there is no definitive test to diagnose necrotizing fasciitis, it is mandatory that the overall clinical picture be carefully considered. When there is doubt, early surgical treatment is preferred as the infection can progress to sepsis and death within hours.

Broad spectrum antibiotics, aggressive surgical debridement and intensive care unit support are essential. Here we studied the outcome of necrotising fascitis among the diabetic patients in our hospital with respect to age, sex, duration of hospital stay, common site of involvement and predisposing factors. And to study the available surgical and treatment modalities available in our institution for the management of necrotizing fascitis.

AIM OF STUDY

To assess the outcome of necrotizing fasciitis in diabetic patients with respect to

- Age
- Sex
- Common site of involvement,
- Duration of hospital stay
- Outcome of the patient
- Predisposing factors
- And to study the available surgical and treatment modalities available in our institution for the management of necrotizing fasciitis.

MATERIALS AND METHODS

- **Type of study**

  Cross sectional study.

- **Place of study**

  Chengalpattu medical college and hospital, chengalpattu.

- **Period of study**

  One year duration from April 2018 to April 2019

- **Sampling method**

  Convenience sampling method.

- **Approval for the study**

  Before starting this study approval was given by ethical committee of Chengalpattu medical college.

- **SAMPLE SIZE**

  100

- **INCLUSION CRITERIA**

  Diabetic patients admitted to Chengalpattu medical college and hospital with clinical diagnosis of necrotising fascitis.

- **AGE GROUP**

  20 to 75 years.

➢ EXCLUSION CRITERIA:

- Those with ischemic heart disease, peripheral vascular disorders, CVA.
- Those who had treatment in some other hospital like wound debridement.
- Patients who are not interested to participate in study
- Patient less than 20 years age and more than 75 years of age.

➢ ETHICAL CONSIDERATION

- Explanation about the study and about the investigation and intervention procedures with their merits and demerits, expected results, and possible complications are explained to all the patients and their local guardians
- After getting consent the case had been selected for this study.
- The study did not involve any additional investigation or any significant risk.
- No economic burden will be given to the patients.
- The study was approved by the institutional review board prior to commencement of data collection.
- Informed consent was taken from each patient/guardian.
- Data were collected by approved data collection form.

➢ Data collection

- pre-prepared structured questionnaire used to collect information.
- After getting informed written consent from them or from their legal guardian. Data were collected from all the respondents by direct interview.

➢ Study procedure

- The data for the study was obtained from diabetic patients (hospitalized patients) with a provisional diagnosis of necrotizing fasciitis on clinical evaluation and who are admitted at Government chengalpattu medical college Hospital.
- Patients presenting with signs and symptoms of Necrotizing Fasciitis admitted during April 2018 to April 2019 at chengalpattu medical college counselled for investigation and treatment of Necrotizing Fasciitis and its complication.
- Of those patients admitted with necrotizing fasciitis, 100 patients were randomly choosen for the study group.
- All the patients were studied and clinical findings were recorded as per proforma case sheet.
- Necessary investigations were done and analyzed for predisposing factors, precipitating factors, complications.

- And also studied, analyzed and discussed about the treatment and sequel.
- Name, age, occupation, socioeconomic status, residence were recorded in the proforma case sheet.
- The presenting complaints and details were recorded in chronological order.
- Detailed physical examination including nutritional status, built, status of vascular system and neurological system were recorded.
- Detailed local examination of involved part done

➢ **INVESTIGATIONS DONE INCLUDES**

- Routine blood investigations: Hemoglobin, total leucocyte count, differential count, ESR.
- FBS, PPBS, HBA1C and corresponding urine sugar and urine acetone on regular basis
- Blood urea and serum creatinine
- Lipid profile
- Radiograph of affected part (lower limbs)
- Wound discharge for culture and sensitivity
- Biopsy of the affected part
- Arterial and venous Doppler study(optional)

- Common mode of presentation was with swelling of the affected part with blebs and blisters, erythema and pain.
- On admission, general and medical treatment of necrotizing fasciitis was done and followed by wound debridement as the definitive procedure.
- The patients were later managed by regular wound dressings, antibiotics, and supportive therapy for maintenance of blood pressure and renal status and in few cases vacuum assisted dressings were tried for faster healing.
- Once the wound was healthy split skin grafting and secondary suturing was done in most cases. Some cases healed by secondary intention.
- Some cases had to undergo major amputations for control of infection and its spread.
- Diabetic patients were managed by diabetic treatment like diabetic diet, sugar restriction and anti diabetic treatment was given with oral hypoglycaeic drugs and insulin.
- Patients who developed renal complications were managed by salt restrictions, dialysis and supportive renal treatment.
- Supportive treatment was given for patients who had bed sores as a complication of NF by regular dressings and water beds.
- Patients who went into septicemia were managed in intensive care units on ventilators under guidance of anesthetists and physicians.

- Post discharge patients were followed up to one month regularly on outpatient basis for dressings, further management of diabetes and hypertension and also to review liver and renal parameters.
- Major amputation patients were advised for clutches and artificial prosthesis 4 weeks after surgery.
- In this study, we try to assess the outcome of necrotising fascitis among diabetic patients in our college

REVIEW OF LITERATURE

Introduction

- The term necrotizing fasciitis (NF) describes a group of relatively uncommon, but life-threatening infections of the skin, soft tissues, and muscles.
- which tend to progress rapidly through the fascia planes, causing gradual destruction of the fascia at a rate reaching 2–3 cm/h.
- Developing in the lower or upper extremities, the perineum and genital area (Fournier's gangrene) and in the abdominal wall, its swift clinical course is correlated with polymicrobial infection and synergy, which usually co-exists .
- The majority of cases present anaerobic bacteria that proliferate in a hypoxic environment and produce gas, which accumulates in the soft tissue spaces, giving the characteristic image of gas gangrene on plain X-rays and computed tomography (CT)
- Early diagnosis of NF is mandatory. Any delay could prove fatal, given its association with more extensive surgery, higher rates of amputation, and higher mortality rates.
- If patients with necrotizing fasciitis are not treated early it will lead to systemic complications.

EPIDEMIOLOGY

- The incidence of NF per year is estimated at 500–1,000 cases, and its prevalence is found to be 0.40 cases per one lakh population.
- It is seen more common among men, with a male-to-female ratio of 3:1; this ratio is associated with the increased incidence of Fournier's gangrene in men.
- All age groups are commonly affected, even though middle-aged and elderly patients (over 50 years of age) are more likely to be infected.
- As per the literature, concluded that the median mortality ratio was 21.5%. And its range in the literature is extensive, from 8.7 to 76%.
- The mortality rate is slightly lower for necrotizing fasciitis in extremities than that recorded for abdominal and perineal infections.
- Fournier's gangrene that has not spread to the abdominal wall tend to have a survival better.
- When necrotizing fasciitis is not treated, the mortality rate approaches 100%.

ETIOLOGY

- Most common etiology is trauma. The most of patients have a history of trauma minor or major, may be external injuries or surgical wounds.
- Perforated appendicitis, necrotic cholecystitis , infection following the repair of an incarcerated hernia, diverticulitis, hollow viscous perforation, and obstructed malignancies with perforation are the most common causes of complicated intra-abdominal infections that can cause cause necrotizing fasciitis. .
- The incidence of NF resulting from a chest wall surgical wound is greater than that recorded from analogous wounds in the lower abdominal wall. Such cases have a high risk of osteomyelitis, which substantially increases the mortality of these patients.
- Surgical wounds, skin abscess drainage, and pressure sores are the cause of necrotising fasciitis. A Anorectal infection, colon perforations, ischiorectal abscesses can be complicated to necrotizing fasciitis.
- In women, NF due to Bartholin abscesses or vulval skin infection are seen. Other causes may be a possible urethral stricture and traumatic urinary catheterization.
- Bacteria such as *Vibrio* spp., *Aeromonas* spp., and *Shewanella* spp. are commonly involved and are called as marine bacteria. consumption of raw seafood or trauma by fish fins can lead to NF.

COMORBIDITIES AND RISK FACTOR

- The co-morbidity commonly associated in patients with NF is diabetes mellitus.
- The prevalence of diabetes mellitus in any type of NF patients ranges between 40 to 60 percentage
- Other common co-morbidities include systemic lupus erythematosus, liver cirrhosis, alcohol abuse, peripheral vascular disease chronic heart failure, obesity, immunodeficiency, Addison's disease, systemic hypertension,
- Presence of sepsis and hypotension on admission are significant predicting factors for mortality and outcome in NF patients.
- Another predisposing factor for higher mortality in patients with NF is chronic renal failure. Elevated serum creatinine and blood urea, are strongly associated with higher mortality rates.
- Non-steroidal anti-inflammatory drugs or steroid drugs usage can suppress fever, thereby leading to the diagnosis of NF.
- Elevated serum creatine kinase and lactate parameters, lowered serum antithrombin III, proved by a low INR, are significant parameters for a poor prognosis of Fournier's gangrene.
- Other parameters are systemic acidosis, low PCV, and protein levels, are also strongly linked with a high mortality.

- Older age is another risk factor for increased mortality but only when associated with other risk factors such as CKD, or delayed surgical debridement.

PATHOPHYSIOLOGY

- Infection starts in the superficial fascia or sub dermal region.
- More superficial layers (dermis and epidermis) are not affected at the beginning .
- The combined action of the virulence factors of organisms and the specific host factors are seen in the development of NF.
- The limit of the infection and necrosis deopends on the synergy between different bacteria and toxins and enzymes they produce .
- Hypoxic environment promotes growth of anaerobic bacteria.
- Bacterial invasion cause thrombosis of the nutrient vessels of hypodermis, leading to tissue ischemia aggravated by the presence of edema.
- Intense pain phenomena are usually observed when the nerve branches are affected. Such cases also display signs of regional hypoesthesia/anesthesia.
- Necrotizing fasciitis is associated with high mortality in diabetic patients its management is a highly challenged.

MICROBIOLOGY

- NF can be divided into 4 sub types

TYPE I

- Also known as the *polymicrobial type*. Accounting for 70–90% of cases
- patients with several co-morbidities are commonly affected
- More than two organisms are associated. Trunk and perineum are commonly affected.

TYPE 2

- Monomicrobial type, Beta-hemolytic Streptococcus A (*Streptococcus pyogenes*) is most commonly implicated with this type.
- Some cases can be associated with *Staphylococcus aureus* infection *S. aureus* secretes toxins, which cause tissue destruction.
- Associated with fulminant forms of NF and *S.* aureus is difficult to manage, especially when it is the methicillin-resistant *S. Aureus*.
- Can occur in patients without serious co-morbidities and most often found in the limbs. The risk of toxic shock syndrome is increased in such cases, and the outcome is unfavorable.

TYPE 3

- Associated with single pathogen Clostridium species or Gram-negative bacteria.
- *Clostridium* species are associated with external injuries or surgical wounds (intestinal and obstetric).

- *Vibrios* spp. infections can also lead to type III NF. *Vibrio vulnificus* bacterium frequently isolated in Asia.

TYPE 4

- Associated with fungal infections, *Candida* species or Zygomycetes.
- Commonly found in immunocompromised host. Predisposing factor is trauma.
- Clinically aggressive and extensive difficult to control.

DIABETES MELLITUS

- Group of metabolic disorders characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both.
- Long-term damage, dysfunction of different organs, especially the eyes, kidney vascular system, are associated with chronic hyperglycemia.
- Deficient insulin action results from inadequate insulin secretion and/or diminished tissue responses to insulin at one or more points of complex action of hormone pathways.
- Polyuria, polydipsia, weight loss, sometimes with polyphagia, and blurred vision are the symptoms of diabetes mellitus.

- Long-term complications of diabetes include
  - Retinopathy with diminishion of vision
  - Nephropathy to renal compromise and failure.
  - Pheripheral neuropathy associated with foot ulcers, amputations.
  - Autonomic neuropathy causing gastrointestinal, genitourinary, and cardiovascular symptoms and sexual dysfunction.
  - Increased incidence of atherosclerotic cardiovascular, cerebrovascular disease, peripheral arterial disease can be seen.
- **Type 1 diabetes,** the cause is a complete absence of insulin secretion. This type of diabetes is often associated with autoimmune pathologic process occurring in the pancreatic islets and by genetic markers.
- **Type 2 diabetes**, resistance to insulin action is usually present. Hyperglycemia enough to cause pathologic changes in different target tissues, in absence of clinical symptoms, can be present for a prolonged period of time before detection can be present.
- In asymptomatic period, it is possible to demonstrate an abnormality in carbohydrate metabolism by measurement of plasma glucose in the fasting state or after a challenge with an oral glucose load or by HBA1C**.**

Type 1 Diabetes

- Accounts for only 5–10% of people with diabetes, due to a cellular mediated autoimmune destruction of the b-cells of the pancreas.
- Markers include islet cell autoantibodies, autoantibodies to the tyrosinephosphatases.
- Fasting hyperglycemia is an important factor for association of type 1 diabetes.
- Strong HLA associations, linkage to the DQA and DQB genes, present.
- The rate of b-cell destruction is quite variable, rapid in infants and children and slow in adults.
- ketoacidosis can be the first manifestation of the disease in infants and children.
- Adults have modest fasting hyperglycemia that can rapidly change to severe hyperglycemia and/or ketoacidosis.
- At this latter stage of the disease, there is little or no insulin secretion, as manifested by low or undetectable levels of plasma C-peptide.
- Type1 diabetes commonly occurs in childhood but it can occur at any age, even in the 8th and 9th decades of life.

Type 2 Diabetes

- Associated predominantly with Insulin Resistance, Insulin Deficiency to mostly an Insulin Secretory defect.
- Constitutes 90–95% of those with diabetes, have insulin resistance and usually have relative insulin deficiency.
- Mostly throughout their life, these patients do not need insulin replacement to survive.
- Patients with this form of diabetes are obese since it causes some degree of insulin resistance.
- Patients who are having increased amount of body fat distributed predominantly in the abdominal region are at risk for type 2 diabetes.
- Ketoacidosis seldom occurs spontaneously in this type of diabetes; when seen, it usually arises in association with the stress of another illness such as infection.
- This form of diabetes the hyperglycemia develops gradually hence frequently goes undiagnosed for many years at earlier stages.
- patients are associated with increased risk of developing macrovascular and microvascular complications.
- Whereas patients with this form of diabetes may have insulin levels that appear normal or elevated, the higher blood glucose levels in these diabetic patients would be expected to result in even higher insulin values had their b-cell function been normal.

- For insulin resistance the amount of insulin secretion is defective in these patients and insufficient to compensate for insulin resistance.
- Weight reduction and pharmacological treatment of hyperglycemia are usually followed but is seldom restored to normal.
- Age, obesity, and lack of physical activity leads to the risk of developing this form of diabetes increases
- Genetic predisposition is more complexly associated with type 2 diabetes more than with type 1.

DIAGNOSTIC CRITERIA FOR DIABETES MELLITUS

- FBG or the 75-g OGTT are used for diagnosing diabetes mellitus.
- Then the criteria changed to FBG and retinopathy
- The retinopathy with fundus photography or direct ophthalmoscopy and measured glycaemia as FPG, 2-h PG, and A1C.
- Diagnostic 2-h PG value of 200 mg/dL (11.1 mmol/L).
- FBG diagnostic cut point of 140 mg/dL (7.8 mmol/L) identified only a little number of patients with diabetes hence cut of point was reduced to 126mg/dL(7.0mmol/L).
- A1C is a widely used marker of chronic glycemia, reflecting average blood glucose levels over a 2-to 3-month period of time.
- A1c is used to assess the micro and macro vascular complications of diabetes

DIAGNOSIS

CLINICAL SIGNS AND SYMPTOMS

- Classical three symptoms are seen in necrotizing fasciitis patients
    1. Erythema, local pain, swelling is the common triad.
    2. Abnormal signs
    3. Fever and tachycardia are the most common abnormal sign.
    4. Hypotension systolic blood pressure < 100 mmHg and respiratory rate >20/min.
- Skin erythema and variations in pulse, BP, temperature are useful in diagnosing NF from other soft tissue infections.
- Tenderness, skin necrosis, and hemorrhagic bullae are the points helpful in clinching diagnosis of NF.
- **Early signs** includes redness, local increased temperature, and edema.
- But in the severe form of disease, the patient is severly ill with signs and symptoms of acute septic shock and MODS, with extensive necrosis of skin and subcutaneous tissue.
- Patient worsens quickly in a few hours; pain is the first symptom usually manifests before the skin signs.pain seems to be more when compared to the clinical findings.
- The **subacute** form of the NF has a relatively long clinical course for days or weeks.

- **Late signs** the pain increases in intensity. The clinical picture is associated with symptoms of sepsis including elevated temperature, dehydration, confusion, giddiness, diarrhea, nausea, vomiting, weakness.
- Not diagnosing NF at this stage will lead the clinical status to deteriorate rapidly. The circumscribed necrosis of the skin will occur.
- Bullae : Contain serous fluid initially but they may become hemorrhagic at late stages.Anaerobic infection with *C. Perfringens* lead to crepitus in the overlying skin indicating gas formation. Five days or later only this cutaneous manifestation occurs.
- Sepsis and MODS occurs in later stage As a result, the patient displays decreased blood pressure, increased wBC level, metabolic acidosis, bleeding manifestation, altered sensorium, and lethargy.

SYMPTOMS

- The symptoms of necrotizing fasciitis are often misdiagnosed as cellulitis or abscess.
- The most consistent symptom of initial NF is pain, and it is out of proportion to the swelling or redness of skin. Pain on palpation usually beyond the area of visual involvement.
- The margins of involvement are usually not clearly distinguishable, when the infection involves deep fascia more than the skin lymphangitis may be present.
- NF with upper limb involvement usually have little systemic involvement.
- other causes that makes diagnosis difficult is the absence of raised temperature in many of the cases. Several drugs can mask fever. Hence absence of fever does not necessarily make diagnosis of NF unlikely.
- Pain and itching of the scrotal skin and perineum is usually the initial symptom of necrotizing fasciitis.
- The pathogenic organisms spread through the
    - Buck's fascia in penis
    - Darto's fascia of the scrotum and penis.
    - Colle's fascia of the perineum.
    - Scarpa's fascia of the lower abdominal wall .
- Infective fluid and crepitus are usually produced due to necrosis of superficial fascia and fat of anterior abdominal wall.

- when patient goes for septic shock they present with elevated temperature,altered sensorial,elevated WBC,and increased respiratory rate.
- When the NF reaches the late phase, the visible bruising, bullae formation and skin necrosis occurs.

LABORATORY INVESTIGATIONS

LRINEC (Laboratory Risk Indicator for Necrotising Fasciitis) scoring system is designed to assess the severity.

- Parameters used are
  - C- reactive protein (less or more than 150 mg/L)
  - Total leukocyte count
  - (less than 15,000; 15,000 to 25,000; more than 25,000).
  - Hb% (> 13.5 g, 11 to 13.5, < 11)
  - serum sodium (> 135 or less than 135)
  - serum creatinine (less or more than 1.6).
  - Blood sugar (less or more than 180 mg).

Score > 8 is severe

Score 6–8 is moderate

Score < 6 is mild.

BEDSIDE TESTS

- **The finger test and frozen section** biopsy are used as complementary diagnostic modalities in patients with an equivocal diagnosis
- The *finger test* is a bedside procedure performed under local anesthesia by which, a 2-cm incision is made down to the deep fascia, at which level gentle probing of the index finger is applied.
- The presence of characteristic dishwater like discharge along with the lack of bleeding and lack of tissue resistance to blunt finger dissection are positive findings correlated with NF.
- Another useful bedside test is an incisional biopsy down to the fascial level with an immediate frozen section, culture, and Gram stain

IMAGING TESTS

- Plain XRAY of local part
    - low sensitivity and specificity,
    - Shows gas formation in the soft tissue seen in all patients with clostridium infection.
- A CT scan delineates the extent of tissue infection, fascial swelling, inflammation, and gas formation.
- An MRI scan provides better accuracy than CT, though not widely used, due to cost.
- Ultrasonography shows details concerning the nature and extent of infection.

- Most classical finding is the hyperechoic foci with artifact and dirty shadowing at the subcutaneous region showing subcutaneous gas.

TREATMENT

ANTIBIOTICS

- Conservative treatment is of little value Since ischemia and hypoxia decrease the adequate delivery of antibiotics to site of infectio.
- Patients should be immediately treated with broad-spectrum antibiotics, when NF is suspected. The empirical usage of antibiotics is based on the microbiological classification of NF.
- Antibiotic treatment of a polymicrobial infection should be based on history, Gram stain, and culture. Initial treatment includes ampicillin or ampicillin–sulbactam combined with metronidazole or clindamycin.
- Anaerobic coverage is quite important for type 1 infection; metronidazole, clindamycin, or carbapenems (imipenem) are effective antimicrobials.
- Type 2 disease is treated with antibiotics against S. pyogenes and S. aureus, which usually coexist with the former. Hence, first or second generation of cephalosporins are used for the coverage of methicillin-sensitive Staphylococcus aureus (MSSA). MRSA tends to be covered by vancomycin, or daptomycin and linezolid in cases where S. aureus is resistant to vancomycin.
- Type 3 NF should be managed with clindamycin and penicillin, which cover the *Clostridium* species.

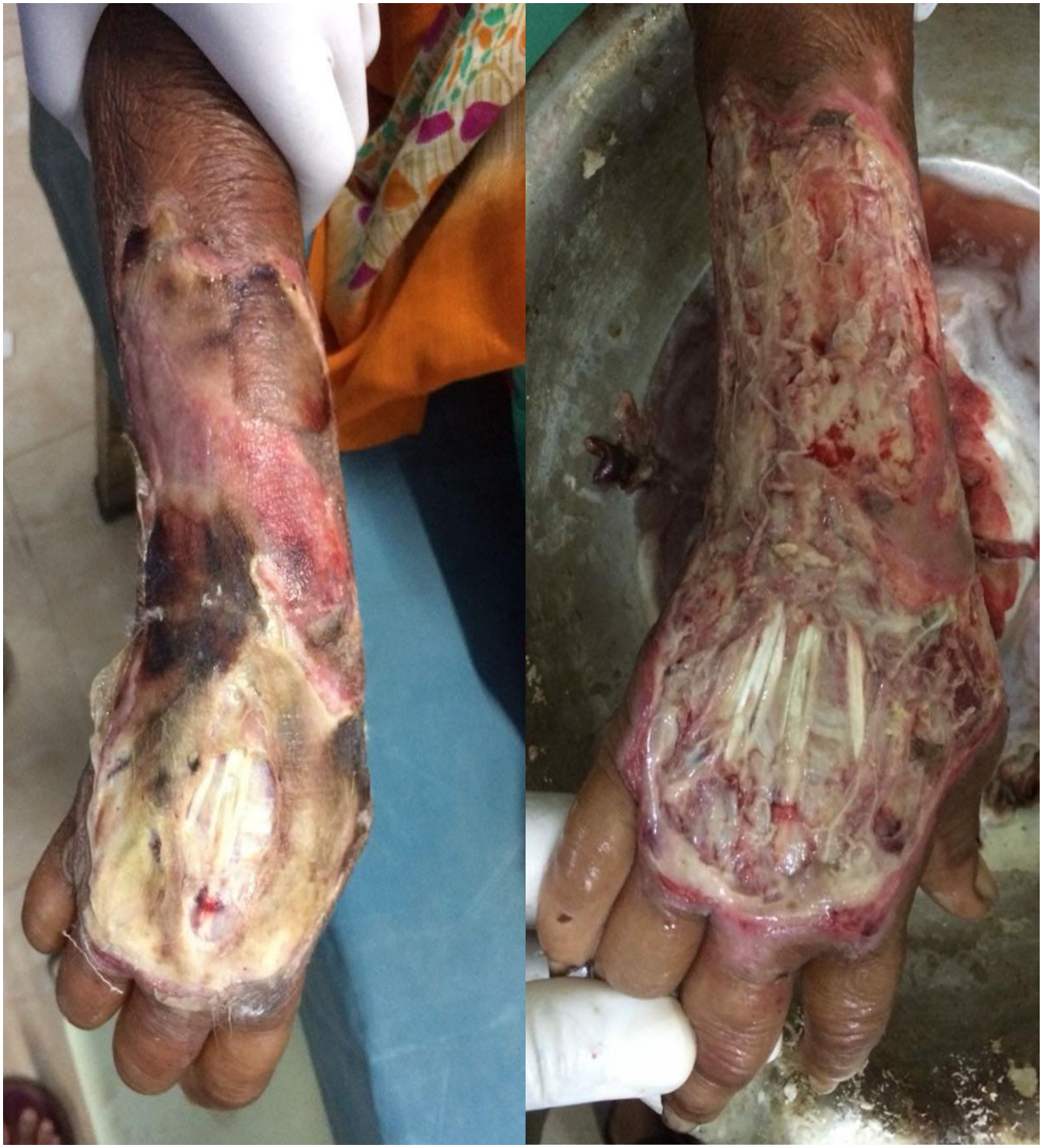
- If Vibrio infection is suspected, the early use of tetracyclines (including doxycycline and minocycline) and third-generation cephalosporins is crucial for the survival of the patient.
- Finally, type 4 NF can be treated with amphotericin B or fluoroconazoles, but the results of this treatment are generally disappointing.

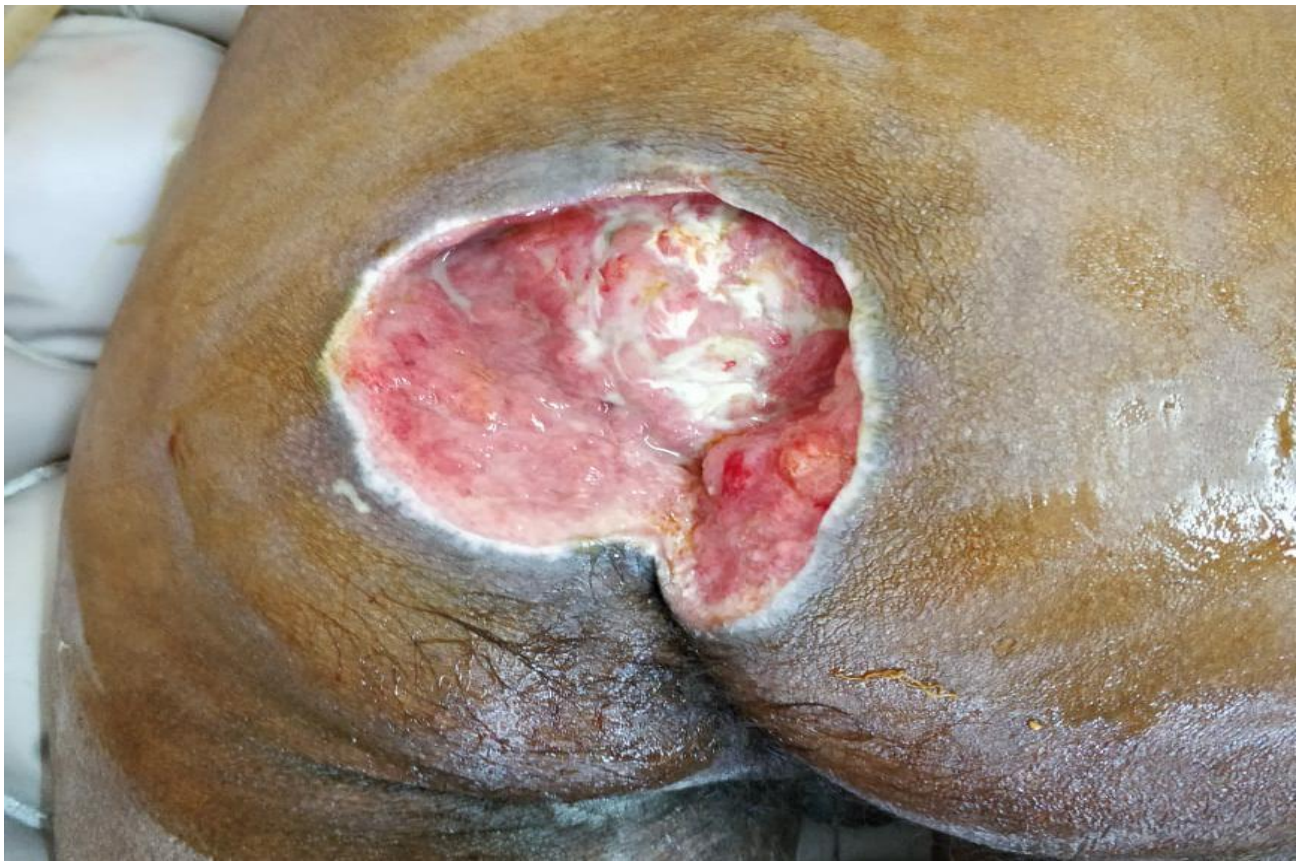
SURGICAL MANAGEMENT

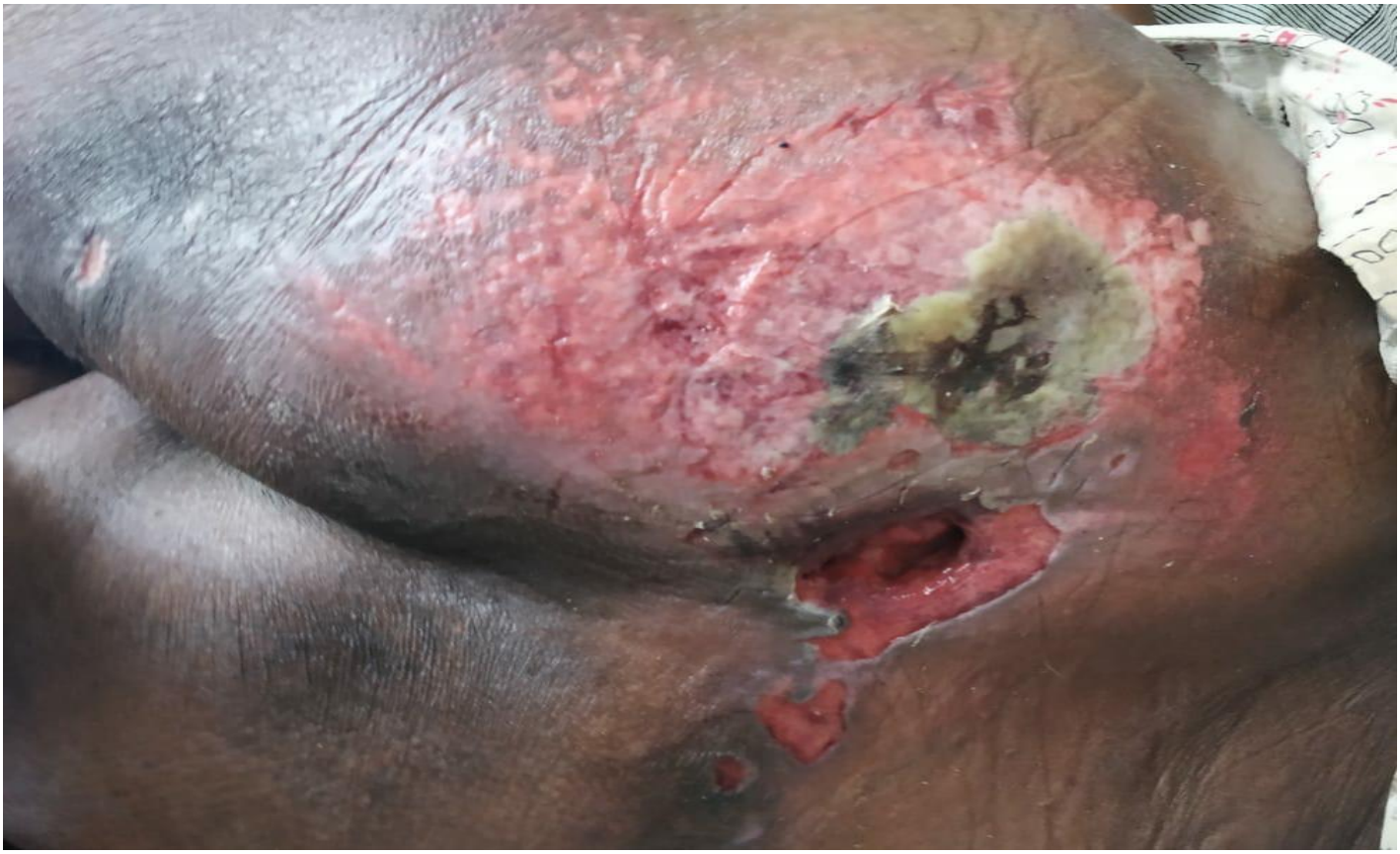
- Emergency surgical debridement of the affected tissues is the primary management modality for NF.
- Surgical debridement, necrosectomy, and fasciotomy are the main aspects of surgical treatment.
- Surgical intervention is life-saving and must be performed as early as possible, since a delay in treatment beyond 12 h in fulminant forms of NF can prove fatal.
- Surgical debridement should be repeated during the next 24 h or later, depending on the clinical course of the necrotizing infection and vital functions.
- Many studies have pointed out that timing and the extent of the first debridement are the most important risk factors in terms of increased mortality rate.
- Incisions are performed parallel to Langer's lines to achieve better surgical wound healing and less scarring.

- Surgery also minimizes the overall tissue loss as it inhibits infection spread to the fascial plane, reducing the need for amputation.
- After the release of pus and/or hemorrhagic fluid through incisions, ventricle incisions are made, keeping the wound open in order to allow drainage and to remove additional necrotic tissue.
- Patients with NF should be closely monitored during the next 24 h, surgical wounds and tissue viability should be checked.
- Uncontrolled wounds need second sitting surgery for control of infection.
- Patients with NF can require from 5 up to 40 additional operations, depending on the timing of the first surgical debridement, the adequacy of the primary debridement and necrosectomy, signs of hemodynamic instability, and concomitant illnesses, all of which are associated with a high mortality rate.
- The extent of tissue extracted depends on the body region, which is infected. As a general rule, debridement will extend until healthy tissue is found, though some authors recommend that excision should be limited to the edges of infection
- careful debridement of the potentially salvageable soft tissue is usually required)
- Nutritional support is required from the first day of the patient's admission to hospital (preferably the ICU), to replace lost proteins and fluid from large wounds and/or the resultant toxic shock.

- Metabolic demands are similar to those of other major trauma or burns, which means that the patient needs twice the basic caloric requirements.







Necrotizing fasciitis of the abdominal wall

- Requires special consideration. Skin incision must be performed in the longitudinal direction along the muscle-fascial layers of the inner abdominal wall until healthy fascia is found.
- Parallel or vertical incisions are not performed because the bridges of skin and skin islands will not usually survive.
- Postoperative management of abdominal wall wounds involves serial dressing changes over the following days, until the wound is free of recurrent or ongoing infection.
- The use of a vacuum-assisted wound closing device (VAC) can also be helpful.
- Aggressive surgical debridement should be repeated in cases of infection progression across the deep fascial planes of the abdominal wall.
- The extension of infection into the bowel, resulting in bowel ischemia, bowel obstruction, and peritonitis, is not an uncommon phenomenon.
- In such cases, an exploratory laparotomy is needed to estimate the extent of infection inside the abdominal wall.
- A radical surgical debridement at the site of infection and the retroperitoneal site is performed, followed by partial bowel excision, depending on the part of the bowel (usually right colon), which has been infected.

- A diverting colostomy is performed with multiple drainages of the infected intra-abdominal fluid collections.
- Surgical management of colonic perforation complicated with peritonitis is a topic with considerable debate in the literature.
- Hartmann's resection has been considered the procedure of choice in cases with diffuse peritonitis and remains a safe technique for colectomy in a perforated colon, especially in elderly patients with multiple co-morbidities.
- The primary defect on the abdominal wall is usually large and is repaired with advanced flaps using an abdominoplasty technique, biological mesh, or skin grafts.

Fournier's gangrene.

- A pressure sore, perineal abscess, or paraplegia frequently predispose to the spread of infection into the scrotum, inguinal region, and lower abdominal wall.
- An orchiectomy, cystostomy, or diverting colostomy is often required dependent on whether the infection has extended to the scrotum, perineal area, or lower abdominal wall, respectively.
- Surgical management includes wide tissue incision, radical debridement, and drainage of the areas involved.
- The wound is washed with hydrogen peroxide, saline, and 1% povidone iodine solution. Finally, it is covered with occlusive and adsorptive dressing with antiseptic properties.
- Again the use of VAC can accelerate the recovery period, providing clean surgical wounds.
- Once the patient is clinically and hemodynamically stable, they can be submitted to reconstructive surgery.

Necrotizing fascitis of the extremities

- The extent of debridement is very important as additional fasciotomies are needed in cases with compromised tissue viability.
- The amount of tissue that needs to be excised is a controversial issue, because the skin in the extremities usually appears normal.

- Despite a normal external appearance soft tissue in patients with NF has extensive vascular microthromboses as well as vasculitis. The risk of full-thickness necrosis high, and this can complicate a primary treated surgical wound.
- **The criteria for amputation**
    - Extensive soft tissue necrosis with involvement of the underlying muscles
    - Fastly spreading infection with sepsis and large necrosis.
- Other conditions that may justify amputation, are the presence of concurrent medical disease with high anesthetic risk and the presence of shock (toxic or cardiogenic) requiring treatment with more than one inotrope.
- Furthermore, concurrent vascular insufficiency further increases the need for amputation, especially when the patient is diabetic.
- Amputation is usually considered as a shorter procedure associated with less blood loss than a radical debridement.
- This explains why patients with hypotension and shock are best treated with amputation seen to reduce mortality, patients undergoing this procedure required fewer repeat operations.

WOUND DRESSINGS

- Wound dressing is a method taking care of minimal cuts to major wounds and helping in process of healing.
- Dressings stays in touch with the wounds and aids in healing.

SEMIPERMEABLE DRESSINGS

- These dressings are made up of polyurethane it allows water,cow to pass away from wound.
- It is impermeable to bacteria and allows self debridement of narcotic slough.
- Made up of nylon frames with polyurethane adhesive tape.
- Advantage
  - Highly elastic and flexible,
  - Can adopt to any shape of wound and easily adherent.
- Disadvantage
  - Not capable for absorbing large amount of discharge from wounds.
  - Causes damage to surrounding normal skin.

SEMIPERMEABLE FOAM DRESSINGS

- These type of dressings are made up of adhesive borders with hydrophobic and hydrophilic foam.
- The water repelling properties of outer layer protect water permeation but allow gas to exchange from wound.
- Sialistic rubber foam molds and contours to wound shape.

ADVANTAGES:

- Foam dressings are capable of absorbing large amount of wound discharge depending upon the wound thickness. Adhesive and non adhesive foam dressings are available.
- Suitable for lower limb ulcers and moderate to highly discharging wounds, also with wound granulation.
- Because of their high absorptive capacity they are used in high discharge wounds.
- Disadvantage unsuitable for low discharge wounds, dry wounds.

HYDROGEL DRESSINGS

- Made from hydrophilic materials such as poly (methacrylates) and polyvinyl pyrrolidine.
- The high water content of hydrogels makes the granulation tissue moist and helps in healing.
- Smooth and elastic nature of hydrogel dressings helps in easy application and after healing easy removal.
- They provide decreased temperature coolness for wound healing.
- This type of dressings is used for dry ulcers, necrotic scars, pressure sores.
- **ADVANTAGES:** Non irritant, inert with biological tissue and permeable to metabolic chemicals. Helpful in treating chronic wounds.
- **DISADVANTAGES:** Exudate collection, necrosis of normal tissues and microorganisms proliferation.

HYDROCOLLOID DRESSINGS

- Most commonly used dressings made up of two layers, inner colloidal layer and outer hydrophobic layer.
- They can transmit water vapour but does not allow passage of bacteria.
- Ideal for wounds with mild to moderate amount of discharge such as trophic ulcers, burn wounds and post traumatic wounds.
- **ADVANTAGES**: Used in paediatric wound care management as they cause lesser pain during removal. Hydrocolloid combines with discharge from wound produces gel which provides moist environment for healing.
- **DISADVANTAGE**: Not useful for high discharge wounds.

ALGINATE DRESSINGS

- They are sodium and calcium salts of manuronic and glucaronic acid units.
- They are derived from seaweed.
- They have high absorption capacity by forming gel with exudates in wound.
- They are capable of inhibiting keratin.
- Ions in the alginates combines with blood to form a protective system helping in healing.
- Helpful for high discharge wounds like higher degree
- They usually require secondary dressings since they absorb the discharge and make the wound dry leading to long duration for healing.

BIOACTIVE WOUND DRESSINGS

- These are usually designed from natural sources like collagen, hyaluronic acid, chondroitin.
- These biomaterials play an main role in healing process.
- These type of dressings are known for their biologically destroyable, and biologically acceptable hence used depending on type of wound.
- Sometimes these dressings are enriched with antibiotics and other materials to increase the healing process.
- Collagen induces fibroblast formation and speeds the endothelial migration when it contacts with wound tissue.
- Hyaluronic acid is a mucopolysacchride component of extra cellular matrix with wound healing properties.

TISSUE ENGINEERED SKIN SUBSTITUTES

- Human skin or dermal equivalent (HSE) has two types of tissue engineered substitutes available, one mimics the layer of skin composed of Keratinocytes and fibroblast on collagen matrix (Cell containing matrix).
- Second contains only the dermal elements with fibroblast on collagen matrix (Acellular matrix).
- HSE is to secrete and stimulate wound growth factor by which epithelialization is achieved.
- Bioengineered are capable of adapting to their environment so that they are able to release growth factors and cytokines incorporated in dressings.

- Bioengineered dressings are suitable for Diabetic foot ulcer and venous leg ulcer.
- Apligraf is a FDA approved skin equivalent substitute consists of keratinocytes and fibroblast-seeded collagen for venous ulcers.
- Some skin substitutes commercially available include, Alloderm composed of normal human fibroblasts with all cellular materials removed and Integra artificial skin consists of collagen/ chondroitin 6 sulphate matrix.

MEDICATED DRESSINGS

- Medicated dressings incorporated drugs plays an important role in the healing process directly or indirectly by removal of necrotic tissues.
- This has been achieved by cleaning or debriding agents for necrotic tissue, antimicrobials which prevents infection and promotes tissue regeneration.
- Some commonly incorporated compounds include antimicrobial agents, growth factors and enzymes. Commercially available antimicrobial dressings include Cutisorb.
- Silver impregnated dressings available are Fibrous hydrocolloid, Polyurethane foam film and silicone gels.
- Antiseptic Iodine dressing acts on bacterial cells via oxidative degradation of cell components by interrupting the function of protein, which is widely effective against pathogen.

- Prolong usage of iodine leads to skin irritation and staining. The purpose of antimicrobials is mainly to prevent or combat infections especially for diabetic foot ulcers.
- Normal tissue repair process in the body is controlled by cellular activities caused by growth factors that are naturally present in our body.
- In case of chronic wounds, growth factors and cells are arrested in the wound bed within the clots that affects the healing process.
- So exogenous application of growth factors benefits the wound healing process and this was proved by numerous studies.
- Among the different growth factors, platelet derived growth factor (PDGF) is the most commonly used growth factor which promotes chemotactic recruitment and proliferation of cells and increasing angiogenesis.
- Besides, PDGF, fibroblast growth factor (FGF), epidermal growth factor (EGF), and autologous platelet thrombin are also studied extensively for their application in healing process.
- Among which, PDGF and EGF are approved by FDA for human application Enzymatic debridement of necrotic tissues without harming healthy tissue is also a crucial part to promote normal healing process.
- Papain and collagenase based ointments are currently used to digest necrotic tissue.

- Collagenase acts on the collagen by attacking native collagen and gentle on viable collagen by gradual breakdown of tissue whereas papain attacks cystein residue and associated with inflammatory response.

COMPOSITE DRESSINGS

- Composite dressings are ideal for both minimal or deeper wounds.
- These dressings have many layers and each layer is different.
- Each composite dressings have three layers.
- They can be a primary or secondary dressing on a wide variety of wounds and may be used with topical medications.
- Outer most layer prevent the wound from infection
- middle layer usually composed of absorptive material which maintains moisture environment and assist autolytic debridement
- Bottom layer composed of non adherent material which prevents from sticking to granulating tissues.
- Composite dressings have less flexibility and they are more expensive

WOUND HEALING

Classification of surgical wounds

- Clean wound
    - Herniorrhaphy.
    - Excisions.
    - Surgeries of the brain, joints, heart transplant.
    - Infective rate is less than 2%.
- Clean contaminated wound
    - Appendicectomy. Bowel surgeries, gastrojejunostomy.
    - Gallbladder, biliary and pancreatic surgeries.
    - Infective rate is 10%.
- Contaminated wound
    - Acute abdominal conditions.
    - Open fresh accidental wounds.
    - Infective rate is 15–30%.
- Dirty infected wound
    - Abscess drainage.
    - Pyocele.
    - Empyema gallbladder.
    - Faecal peritonitis.
    - Infective rate is 40–70%.

Wound healing

- It is the biological process of achieving physiological and anatomical integrity by various components like fibroblasts, collagen,and neutrophils.
- In an organised staged pathway
- Haemostasis → inflammation → proliferation → matrix synthesis (collagen and proteoglycan ground substance) → maturation → remodelling → epithelialisation → wound contraction (by myofibroblasts).

TYPES OF WOUND HEALING

- **Primary Healing (FirstIntention**)
    - It occurs in a clean incised wound or surgical wound.
    - Wound edges are approximated with sutures.
    - There is more epithelial regeneration than fibrosis.
    - Wound heals rapidly with complete closure.
    - Scar will be linear, smooth, and supple.
- **Secondary Healing (Second Intention**)
    - occurs in a wound with extensive soft tissue loss like in major trauma, burns and wound with sepsis.
    - It heals slowly with fibrosis.
    - It leads into a larger scar due to contraction.
    - It may lead into disability.

- Re-epithelialisation occurs from remaining dermal elements or wound margins.
- Wound requires secondary suturing once it granulates well.
- Secondary suturing is done after 10–14 days, once wound granulates well with proper control of infection.
- Scar in such type is prone to form incisional hernia.

WOUND HEALING BY THIRD INTENTION

- After wound debridement and control of local infection,
- wound is closed with sutures or covered using skin graft.
- Primary contaminated or mixed tissue wounds heal by tertiary intention.

STAGES OF WOUND HEALING

- Stage of inflammation.
- Stage of granulation tissue formation and organisation.
- Stage of epithelialisation.
- Stage of scar formation and resorption.
- Stage of maturation.

INFLAMMATORY PHASE (LAG OR EXUDATIVE PHASE)

- It begins immediately after formation and lasts for 72 hours. There is initial arteriolar vasoconstriction, thrombus formation, platelet aggregation due to endothelial damage and release of adenosine diphosphate (ADP).
- Later vasodilatation and increased vascular permeability develops.
- Here haemostasis, coagulation and chemotaxis occur.

PROLIFERATIVE PHASE (COLLAGEN OR FIBROBLASTIC PHASE)

- It begins from 3rd day and lasts for 3–6 weeks.
- There will be formation of granulation tissue and repair of the wound.
- Granulation tissue contains fibroblasts, neocapillaries, collagen, fibronectin and hyaluronic acid.
- Initial angiogenesis (growth of new blood vessels) occurs by release of vascular endothelial cell growth factor (VEGF) by keratinocytes; by release of TNF-α, TGF–β, PDGF, FGF by macrophages.
- Eventual fibroplasia develops by fibroblast activity with formation of the collagen and ground substance/glycosaminoglycans. Type III collagen is deposited initially in a random fashion.
- Later re-epithelialisation of the wound surface occurs by migration of basal layer of the retained epidermis which proliferates, differentiates and stratifies to form wound closure.

REMODELLING PHASE

- It begins at 6 weeks and lasts for 6 months to 1 or 2 years.
- There is maturation of collagen by cross linking and realignment of collagen fibers along the line of tension, which is responsible for tensile strength of the scar.
- There is reduced wound vascularity. Fibroblast and myofibroblast activity causes wound contraction.

- Type III collagen is replaced by type I collagen causing maturation of the collagen. Ratio of type I collagen to type III collagen becomes 4:1.
- Early extracellular matrix contains fibronectin and collagen type III; eventually it contains glycosaminoglycans and proteoglycans; final matrix contains type I collagen.
- Scar strength is 3% in 1 week; 20% in 3 weeks; 80% in 12 weeks. Final matured scar is acellular and avascular.

Skin Grafting

- It is transfer of skin from one area (donor area) to the required defective area (recipient area).
- It is an autograft.
- Types 1 partial thickness graft (Split-thickness skin graft—SSG)
    - Also called as Thiersch graft,
    - It is the removal of full epidermis + part of the dermis from the donor area.
    - It may be Thin SSG,Intermediate SSG, all depends on the amount of thickness of dermis taken.
    - Thick SSG.

- **Indications**
    - Well-granulated ulcer
    - Clean wound or defect which can not be apposed After surgery to cover and close the defect created.
    - For example: –After wide excision in malignancy–After mastectomy–After wide excision in squamous cell carcinoma.
    - Graft can survive over periosteum or paratenon or perichondrium.
- **Prerequisite**
    - Healthy granulation area.
    - Beta haemolytic streptococci load less than 10’5 per gram of tissue.
    - Otherwise graft failure will occur
- **Contraindications**
    - SSG cannot be done over bone, tendon, cartilage, joint.
- **Technique**
    - Donor area: Commonly thigh, occasionally arm, leg, forearm.
    - Knife used is Humby’s knife.
    - Blade is Eschmann blade, Down’s blade.
    - Using Humby’s knife graft is taken, punctate bleeding is observed which says that proper graft has been obtained.
    - Different instruments used to harvest the skin graft Humby’s knife Watson modification of Humby’s knife.

- Power dermatome is also used (Brown) Sterilised razor blade can be used with a specialised device to harvest small grafts under local anaesthesia.
- Donor area is dressed and dressing is opened after 10 days, not earlier.
- Recipient area is scraped well and the graft is placed after making window cuts in the graft to prevent the development of seroma.
- Graft is fixed and tie-over dressing is placed.
- If graft is placed near the joint, then the part is immobilised to prevent friction which may separate the graft.
- On 5th day, dressing is opened and observed for graft take up.
- Mercuro chrome is applied over the recipient margin to promote epithelialisation.

➢ **Stages of Graft Intake**

- Stage of plasmatic imbibition: Thin, uniform, layer of plasma forms between recipient bed and graft.
- Stage of inosculation: Linking of host and graft which is temporary.
- Stage of neovascularisation: New capillaries proliferate into graft from the recipient bed which attains circulation later.
- Graft is stored at low temperature of 4°C for not more than 21 days.

- **Disadvantages of SSG**

Contracture of graft.

- **Primary contracture** means SSG contracts significantly once graft is taken from donor area (20–30%).
- Thicker the graft more the primary contracture.
- **B. Secondary contracture**
- Happens when graft is placed in the recipient site.
- Thinner the graft more the secondary contracture.
- Seroma and haematoma formation will prevent graft take up.
- Infection.
- Loss of hair growth,
- Blunting of sensation.
- Dry, scaling of skin due to nonfunctioning of sebaceous glands.
- So after healing, oil (coconut oil) should be applied over the area.

ADVANTAGES

- Technically easier.
- Wide area of recipient can be covered.
- To cover large area like burns wound, graft size is increased by passing the graft through a Mesher which gives multiple openings to the graft, which can be stretched on the wider area like a net. It can cause expansion up to 6 times.

- Graft take up is better.
- Donor area heals on its own.

2. FULL THICKNESS GRAFT (Wolfe Graft)

- It includes both epidermis + full dermis.
- It is used over the face, eyelid, hands, fingers and over the joints.
- It is removed using scalpel blade. Underlying fat should be cleared off properly.
- Deeper raw donor area is closed by primary suturing.
- If large area of graft is taken, then that donor area has to be covered with SSG which is a disad-vantage in full thickness graft.
- Common sites of donor area
    - Post-auricular area
    - Supraclavicular area
    - Groin crease

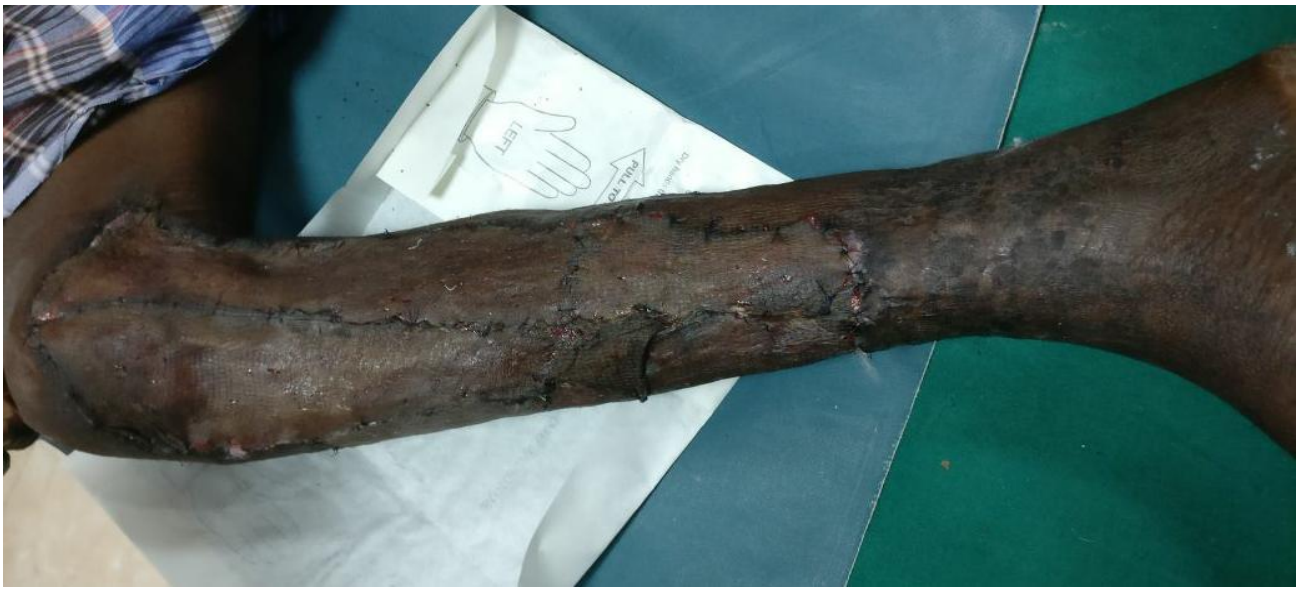
ADVANTAGES

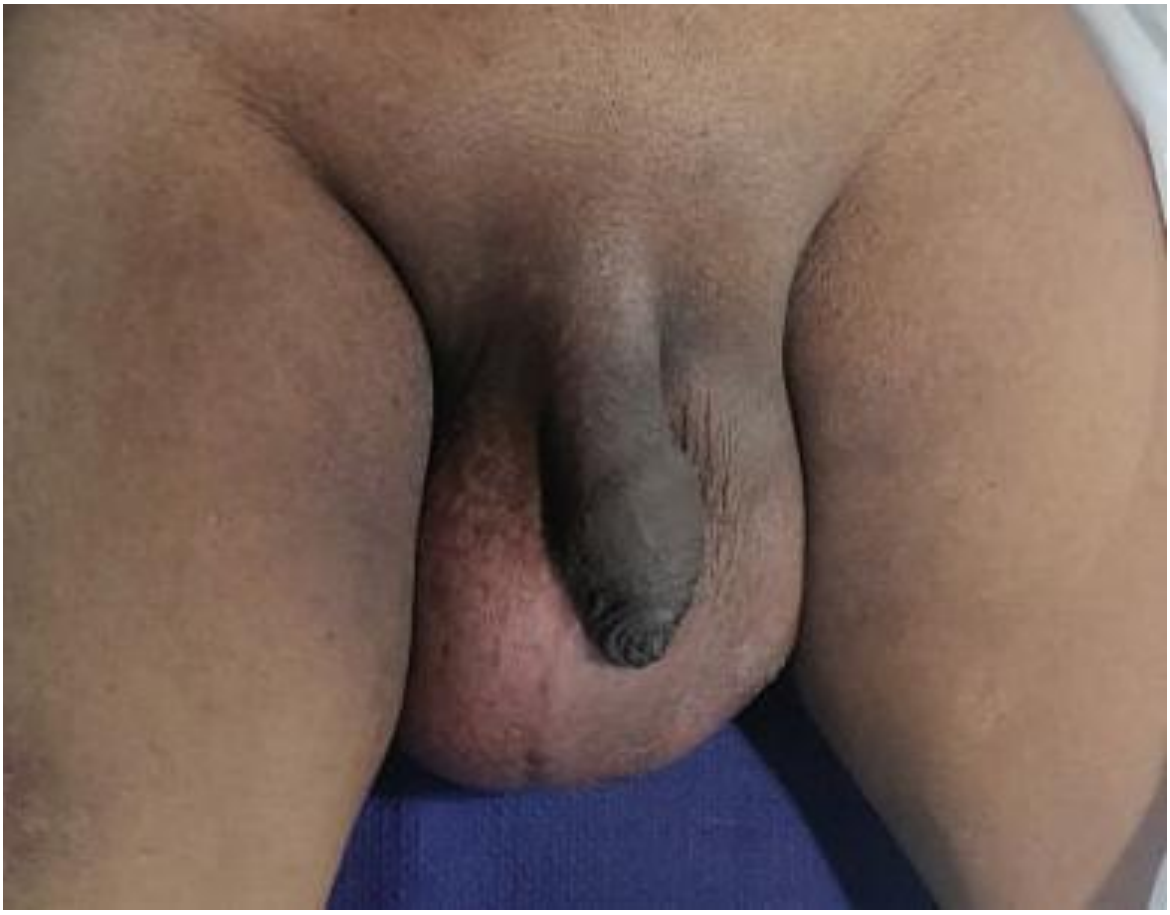
- Colour match is good. Especially for face.
- No contracture (unlike in SSG).
- Sensation, functions of sebaceous glands, hair follicles are retained better compared to SSG.
- Functional and cosmetic results are better.

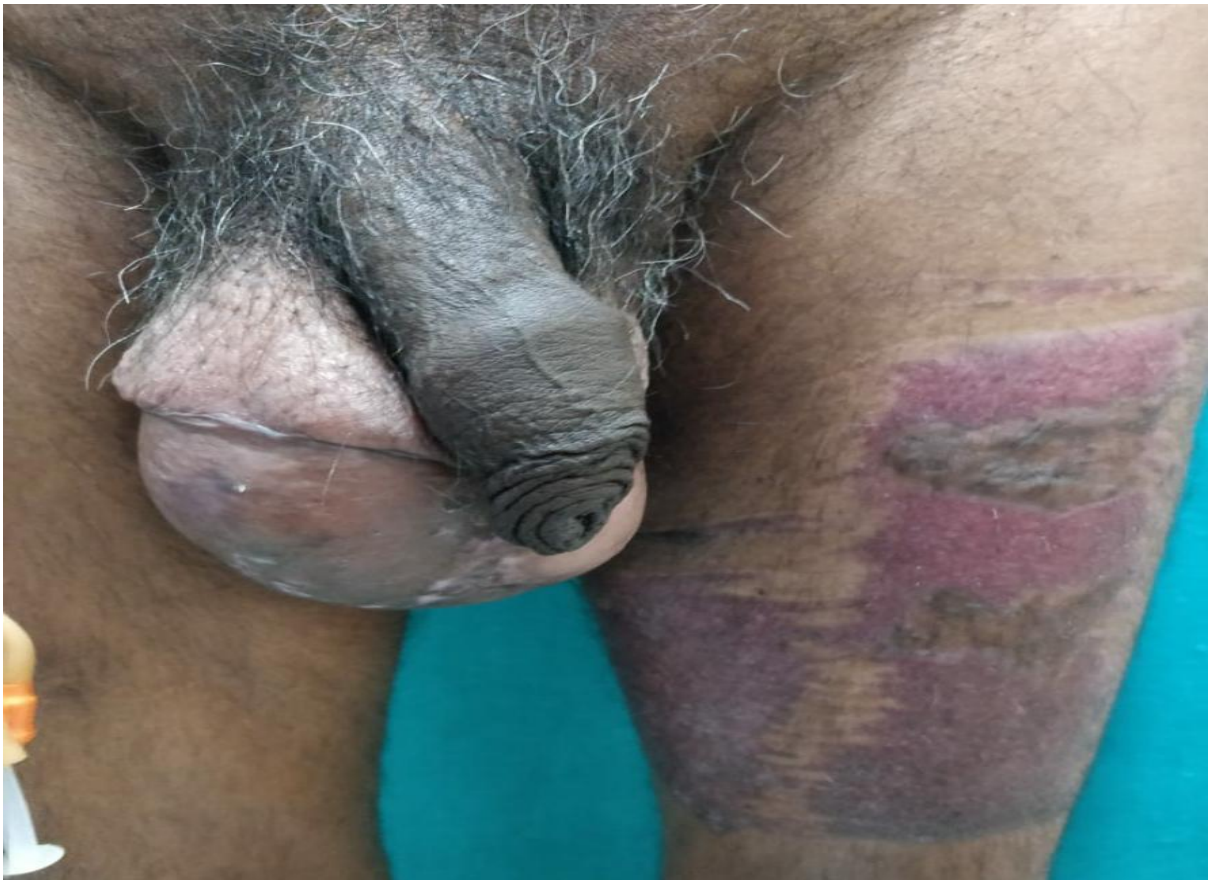
DISADVANTAGES

- It can be used only for small areas.

- Wider donor area has to be covered with SSG to close the defect.







AMPUTATIONS

INDICATIONS

- Gangrene due to atherosclerosis, embolism, TAO, diabetes, ergots.
- Trauma: To save life in crush injuries.
- Neoplasms: Osteosarcomas, Marjolin's ulcer, melanomas.
- Gas gangrene.
- Severe sepsis.
- Occasionally severe elephantiasis, madura foot, when all other methods have failed to help.
- Dead, dying, devitalised tissues.
- Severe deformity congenital or acquired

TYPES

- Non-end bearing/side bearing—Weight is taken up by the joint.
- End bearing/cone bearing—Weight is taken up by the body
- Weight bearing.
- Non-weight bearing.
- Provisional amputation with flap—later final formal amputation my be required.
- Guillotine amputation which always requires revision formal amputation.
- Formal amputation—is definitive one.
- Long posterior flap in below-knee amputation.
- Equal flaps in above-knee amputation.

IDEAL STUMP

- Should heal adequately by 1st intention.
- Should have rounded, gentle contour, with adequate muscle padding.
- Should have sufficient length to bear prosthesis.
- For B-K 7.5 (minimum) to 12.5 cm from tibial tuberosity
- For above and below elbow 20 cm stump.
- For A-K 23 cm from greater trochanter.
- Should have thin scar which does not interfere with prosthetic function.
- Should have adequate adjacent joint movement.
- Should have adequate blood supply.
- Scar should be in a place where it is not exposed to pressure.
- Scar should be freely mobile over underlying tissues.
- Skin and scar should be freely mobile over the underlying bone.
- It is achieved only if deep fascia is closed properly.
- Scar and skin should be free to achieve free movement of the prosthesis.
- Socket of prosthesis with mobile skin creates a piston to bone to move like a joint.
- Skin should not be infolded.
- Redundant soft tissue should not be there.
- Stump should be free from tenderness and conical.

VARIOUS LEVELS OF AMPUTATION

TRANS METATARSAL AMPUTATION(Gillies')

- Here amputation is done proximal to the neck of the metatarsals, distal to the base.

- **Lisfranc's amputation** (Tarsometatarsal amputation)
  - Here tarsometatarsal joint is disarticulated with a long volar flap.
  - It needs a surgical boot. But there is inevitable development of equinovarus deformity.
  - So stabilisation of midtarsal and ankle joints is needed.
  - In Hey's modification, 2nd metatarsal is cut at base instead of disarticulation.

- **Chopart's amputation** (Midtarsal amputation)
  - Here talonavicular joint and calcaneo-cuboid joints are disarticulated.
  - Tibialis anterior muscle is sutured to drilled talus bone.
  - A long volar flap is used and immobilised for 6 weeks after surgery.

- **Syme's amputation**
  - It is removal of the foot with calcaneum and cutting of tibia and fibula just above the ankle joint with retaining heel flap (dividing both malleoli).

- Heel flap is supplied by medial and lateral calcaneal vessels (branches of posterior tibial artery).
- Elephant boot is used for the limb after Syme's which is inexpensive. Many patients walk well with Syme's stump without difficulty.
- It is presently mainly used in trauma (crush injury) and malignancies in distal part of the foot.
- In vascular diseases, calcaneal vessels may not be adequate to maintain the viability of the flap. Above-knee cast is essential
- **Advantages** It is an end-bearing stump having good proprioception. Patient can walk without prosthesis. Low energy consumption ambulation is possible.
- **Disadvantages** posterior displacement of heel pad; poor cosmesis.

➢ **Boyd's amputation**

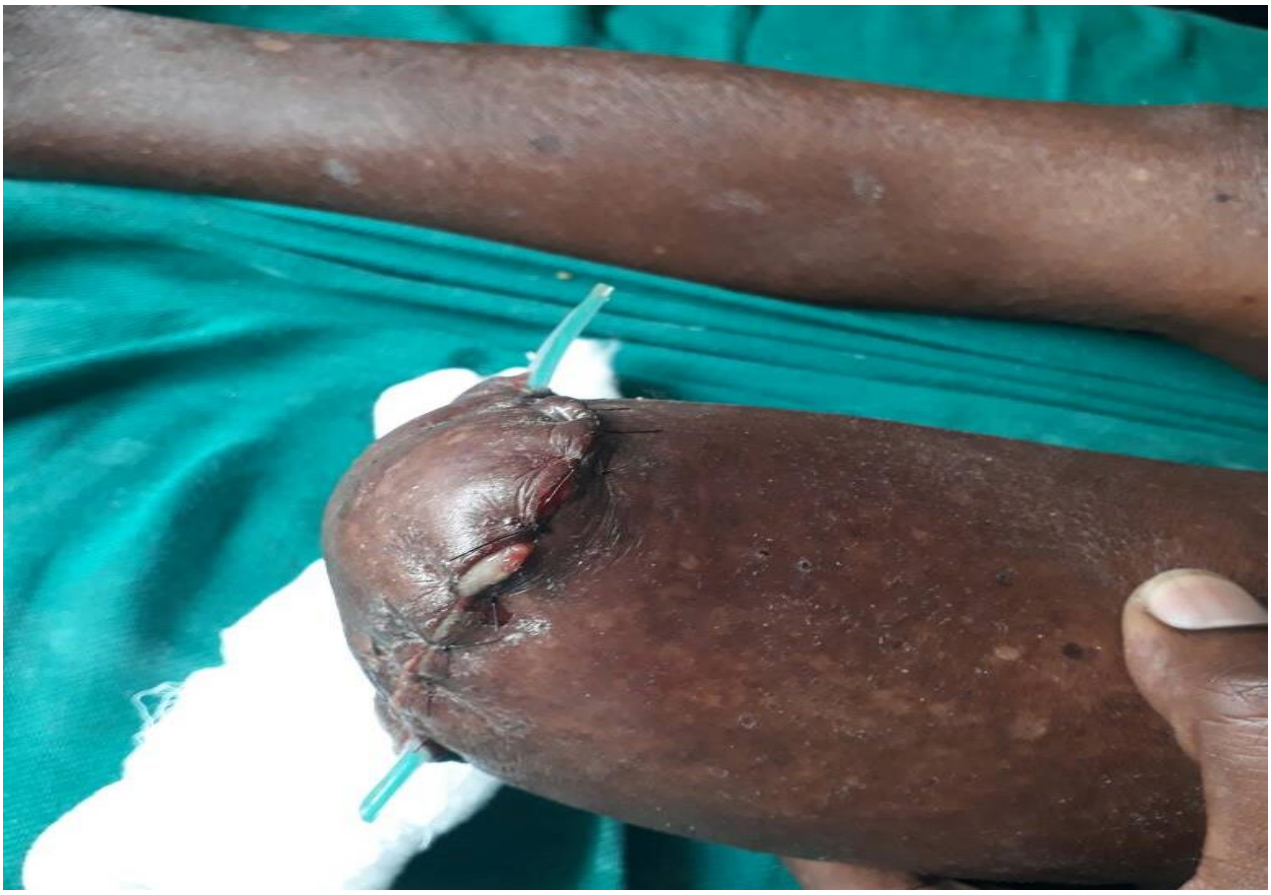
- Anterior part of the calcaneum is excised (osteotomy) just distal to the peroneal tuberosity and calcaneotibial arthrodesis is created.
- It is done to prevent posterior migration of heel pad.

BELOW KNEE AMPUTATION

➢ Here we use a long-posterior flap with scar placed over anterior aspect is used.

➢ Prosthesis placement is better here with greater range of movements without limp and without support.

- It is also called as Burgess amputation.
- Fibula should be divided first, higher than the proposed site of cut of tibia otherwise its sharp end will press on the skin flap.
- Tibial stump should be beveled anteriorly.
- Posterior muscles are sutured across the bone end, to the periosteum in front.
- In more proximal type of below-knee amputation, fibula often is removed to allow the proper use of flap.
- Length of the flap should be 11/2 times the circumference of the site (around 12 cm).
- Stump length is 14–17 cm from knee joint.
- Minimum length required for prosthesis is 8 cm.
- If need to extend more proximally, it is better to do above-knee amputation.
- Modern artificial limbs like suction socket prosthesis are used now.



PEG-LEG AMPUTATION

- It is amputation 5 cm below the knee level – proximal most below-knee amputation.
- It is not practiced nowadays.
- Here anterior flap is rotated posteriorly like a hood and patient kneels and bears weight which is well-accustomed to pressure.
- It is done whenever prosthesis cannot be used probably due to economic causes (in developing countries)

TRANS CONDYLAR (GRITTE-STOKES AMPUTATION)

- Femur is divided just above the articular
- surface and patella is anchored to the divided femur.
- There is risk of nonunion between patella and femur.
- This procedure is no longer performed.
- Above-knee amputation Usually equal anterior and posterior flaps are used.
- Lower third and middle third level amputations are done.
- Ideally the required length of the femur as stump is 25 cm from the tip of the trochanter.
- Femur length lesser than 10 cm is not possible and here one needs to do hip disarticulation.
- In children as growing epiphysis of femur is in lower end, it is essential to preserve as much length of femur as possible.
- It is done in ischaemia, trauma, sepsis, gangrene which is spreading above.

- Often patient might earlier have undergone below-knee amputation but now as indicated need above-knee amputation.
- It is usually contraindicated in children (done only in undue definitive indication) or if stump is less than 7.5 cm.
- **Advantages** are technically easy, healing chances are better and faster.
- **Disadvantages** are cosmetically poor, rehabilitation is difficult, and fitting of prosthesis is not proper, patient needs a third support for walk with often a limp.

HIP DISARTICULATION

- It is done whenever it is not possible to save the minimum 10 cm length of the femur.
- Incision used is either single posterior flap— **Solcum's approach** (better) or anterior racquet incision—**Boyd's approach.**

HIND QUATER AMPUTATION

- Inter-innominate abdominal amputation (Sir Gordon Taylor's amputation)
- Removal of one side pelvis with innominate bone, pubis, muscles and vessels.
- Original ligation of common iliac artery is modified to individual ligations of external and internal iliac vessels.
- Internal iliac artery is ligated beyond the origin of the superior gluteal artery to keep the large posterior flap viable.

- Now hind quarter name is replaced by hemipelvectomy.
- It may be standard hemipelvectomy with classic gluteal flap; extended hemipelvectomy with removal of posterior part of the scrotum.
- conservative hemipelvectomy with retaining part of the pubis, ilium bones on that side.
- Internal hemipelvectomy is new method wherein hemipelvectomy is done with preserving the limb.

KRUKENBERG'S AMPUTATION

- Done in upper limb following any trauma.
- Here forearm amputation is done in such a way that it creates a gap between radius and ulna like a claw to have a hold or grip.

INTERSCAPULO THORACIC AMPUTATION

- (Forequarter amputation) (Littlewood's posterior approach or Berger's anterior approach).
- It is removal of entire upper limb with scapula and lateral 2/3rd of the clavicle and muscles attached to it.
- It is done in malignancies involving scapula, upper part of humerus and near shoulder joint.

COMPLICATIONS OF AMPUTATION

- **EARLY COMPLICATIONS**
    - Haemorrhage
    - Hematoma
    - Infection

LATE COMPLICATIONS

- Infection of stump
- Flap necrosis
- Stump neuronal

- Stump pain
- Phantom limb
- Operation over stump
- Contracture of the joint

PROSTHESIS

- It is the substitution to a part of the body to achieve its optimum function.
- **Syme's amputation**
    - Elephant boot, Canadian Syme's prosthesis.
- **Below-knee amputation**
    - Patellar-tendon bearing prosthesis and solid ankle cushion heel.
- **Above-knee amputation**
    - Suction type prosthesis. It is placed above the stump. It is better and well-tolerated.
    - Nonsuction type prosthesis: It is placed at the ends. It requires additional support.
- **Hind-quarter amputation:**
    - Tilting table prosthesis or Canadian prosthesis is used here.
- **Below knee amputation**

- Prosthesis is leather cuff strap above femoral condyles.
- Exo- or endoskeleton is used.
- For athletics endoskeleton is preferable.

- **In Upper Limb**
  - Above-elbow prosthesis is a high technology prosthesis. It is sophisticated device with harness, socket, elbow joint unit, control cable, forearm and wrist device.
  - Below-elbow prosthesis Krukenberg's amputation does not require any prosthesis.

ADVANTAGES OF PROSTHESIS

- Cosmetic.
- Function of the part relatively can be got.
- Ambulation in lower limb prosthesis.

Disadvantages

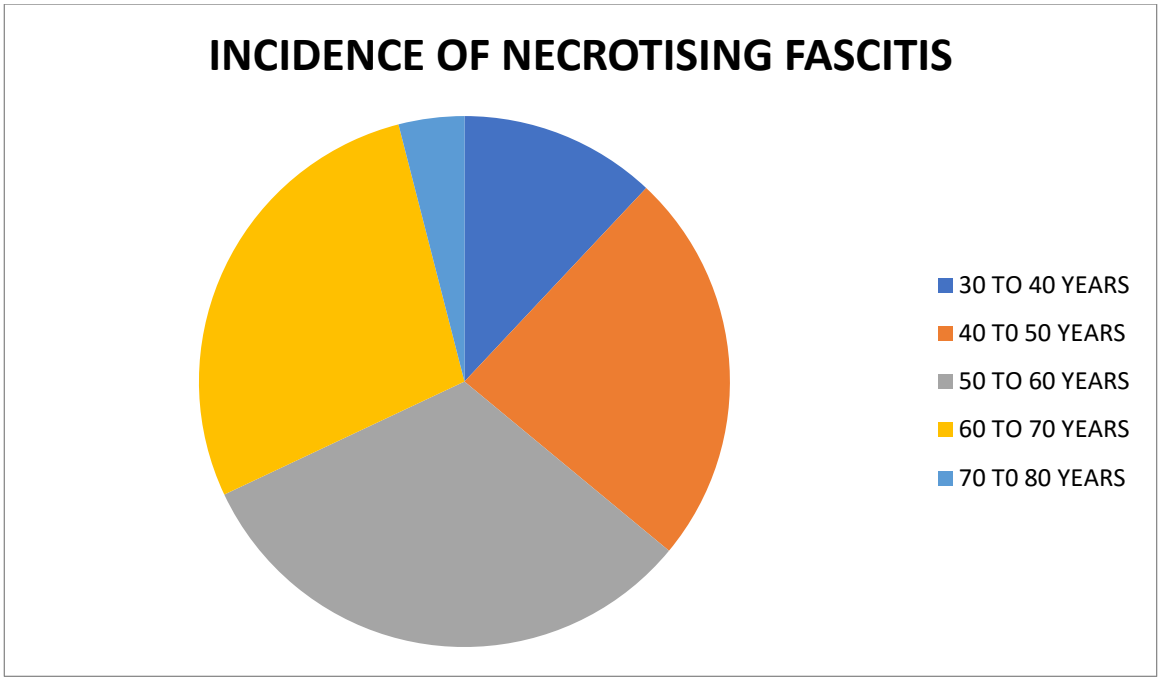
- Infection.
- Pressure ulcers.
- Joint disability.
- Prosthesis

RESULTS AND DISCUSSIONS

AGE WISE DISTRIBUTION

- Out of 100 diabetic patients with necrotizing fasciitis the maximum incidence was seen in the age group of 51-60 years (34%)
- Next is in age group of 61-70 years (27%).

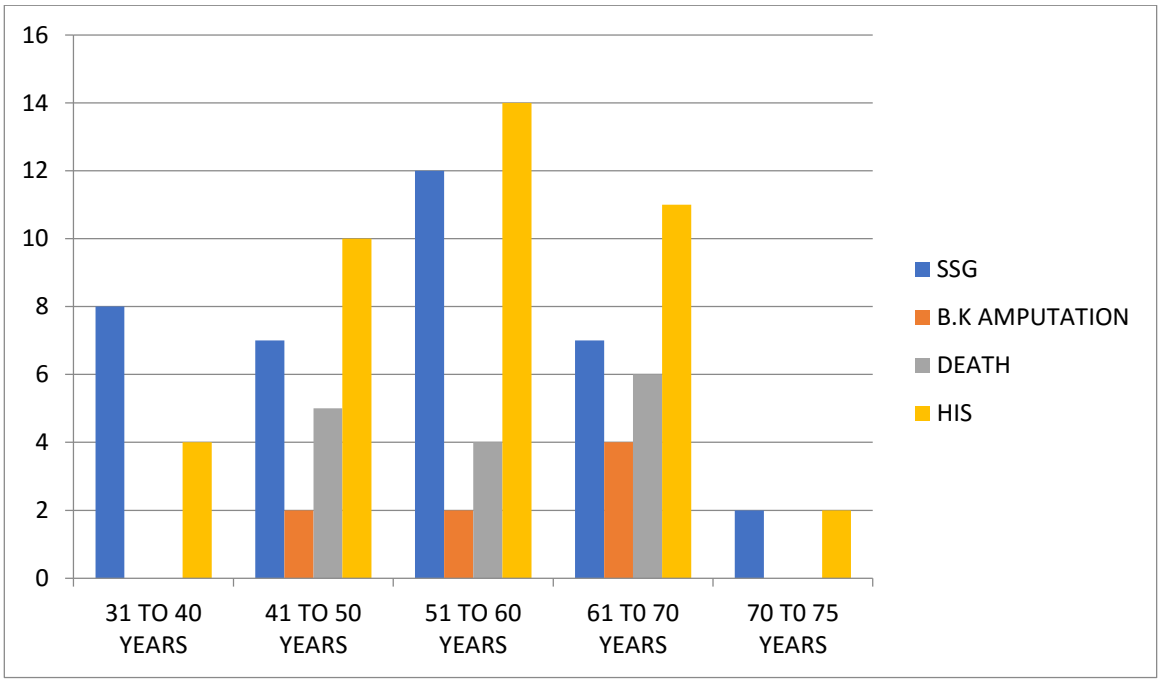
AGE	NUMBER OF PATIENTS	PERCENTAGE
31 TO 40 YEARS	11	11%
41 TO 50 YEARS	24	24%
51 TO 60 YEARS	34	34%
61 TO 70 YEARS	27	27%
71 TO 75 YEARS	4	4%



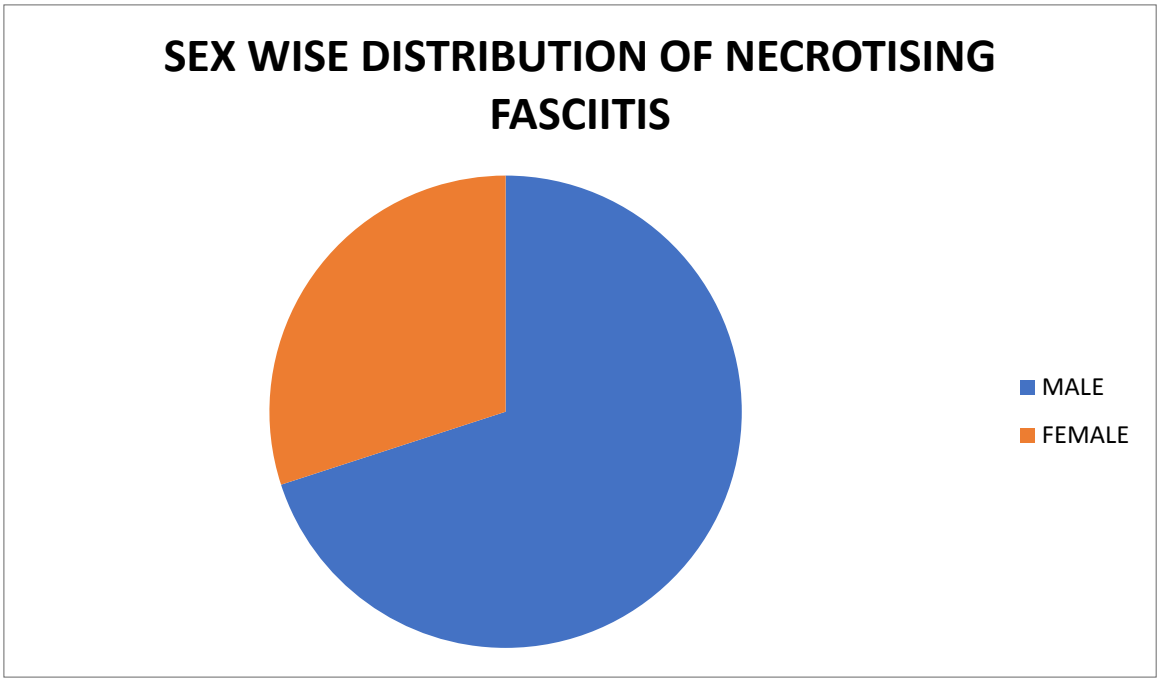
AGEWISE DISTRIBUTION OF OUTCOME OF NECROTISING FASCITIS

AGE	SSG	AMPUTATION	DEATH	HSI
31 TO 40 YEARS	8	0	0	4
41 TO 50 YEARS	7	2	5	10
51 TO 60 YEARS	12	2	4	14
61 TO 70 YEARS	7	4	6	11
71 TO 75 YEARS	2	0	0	2

- Among the age group of 31 to 40 years 8 patients had outcome SSG and 4 had outcome of HSI. No mortality and 100% healed without mortality and morbidity.
- Among the age group of 51 to 60 years mortality is more about 14 person and about 11 person in 61 to 70 years.
- Amputation is more among age group of 61 to 70 years about 4 person.



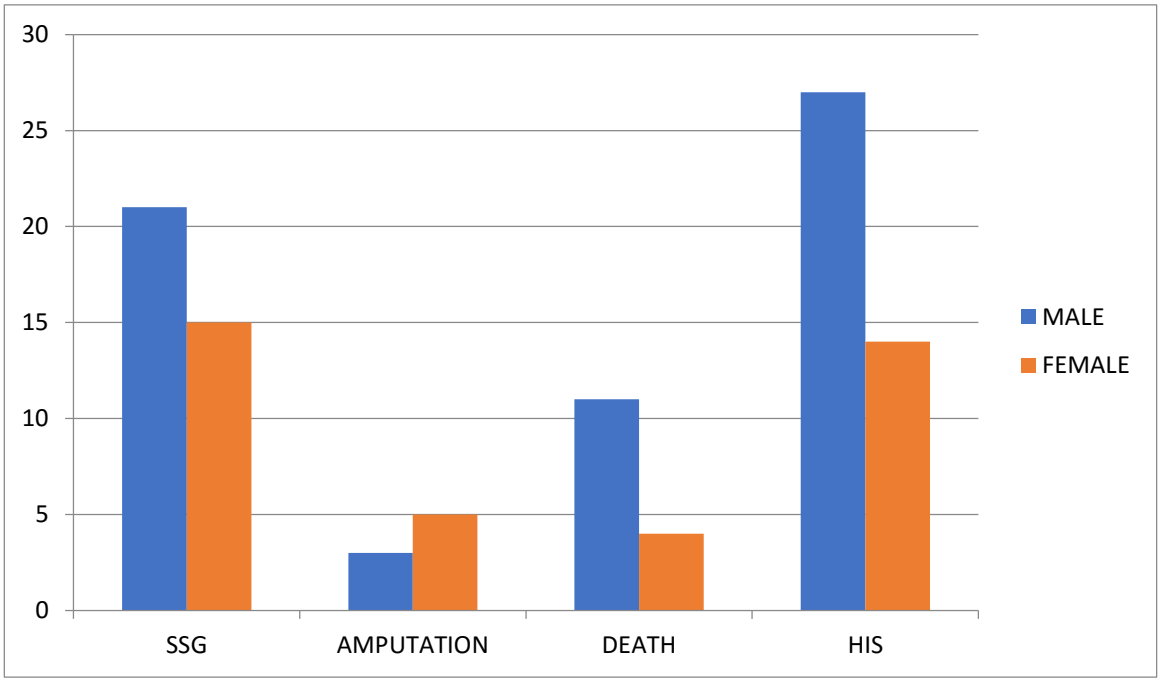
SEXWISE DISTRIBUTION OF NECROTISING FASCIITIS



- Incidence of necrotising fasciitis is more among male patients about 77% and female about 33%.

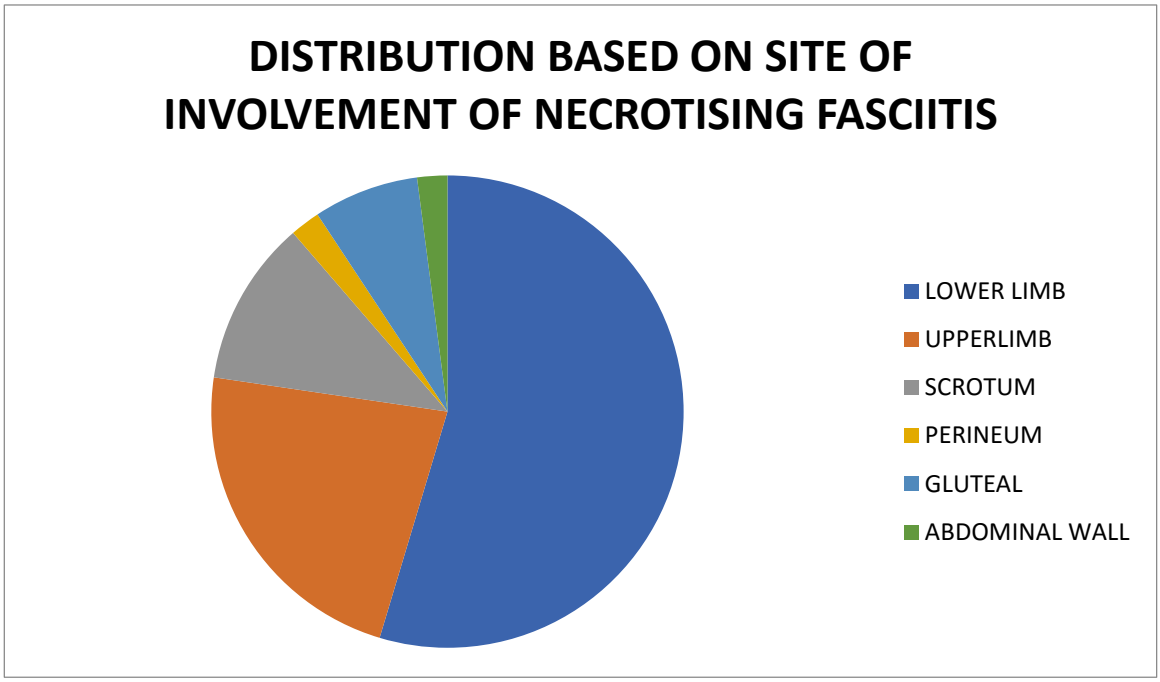
SEXWISE DISTRIBUTION OF OUTCOME OF NECROTISING FASCIITIS

OUTCOME	MALE	FEMALE
SSG	21	15
AMPUTATION	3	5
HSI	11	4
DEATH	27	14



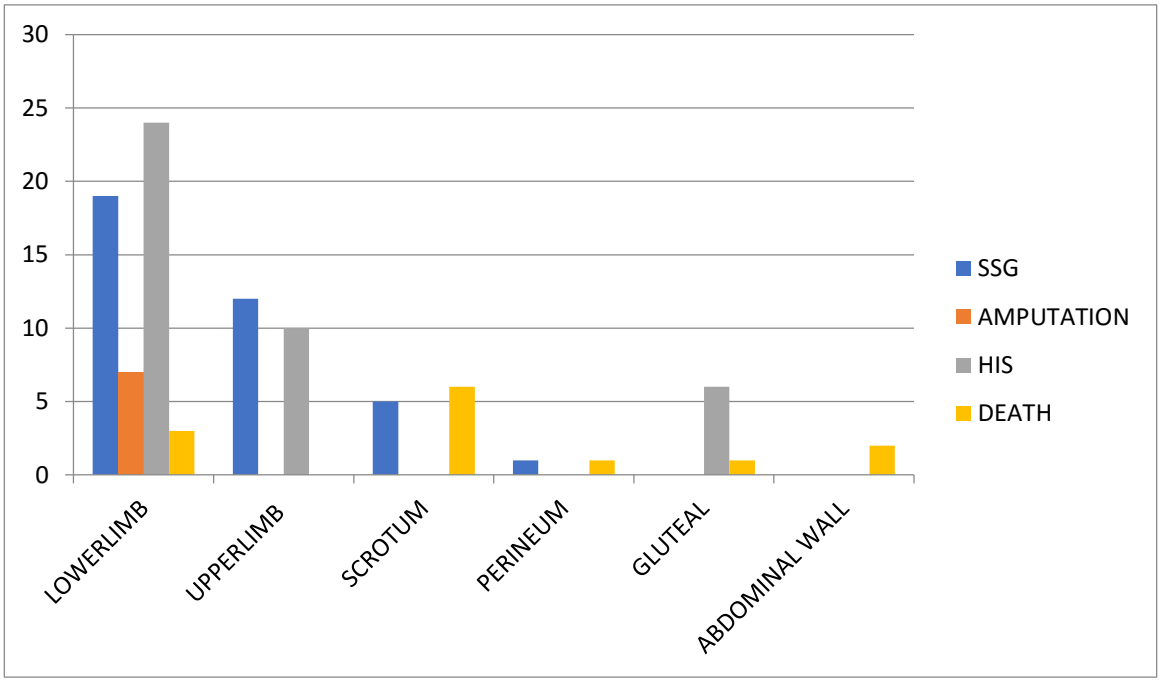
- Amputation is more among female patients about 5% and about 3% among male patients.
- Total mortality is about 15% .
- 11% among male and 4% among female patients.

DISTRIBUTION BASED ON THE SITE OF INVOLVEMENT



SITE OF INVOLVEMENT	PERCENT AFFECTED
LOWER LIMB	53%
UPPER LIMB	22%
SCROTUM	11%
PERINEUM	2%
GLUTEAL	7%
ABDOMINAL WALL	2%
PRESACRAL	3%

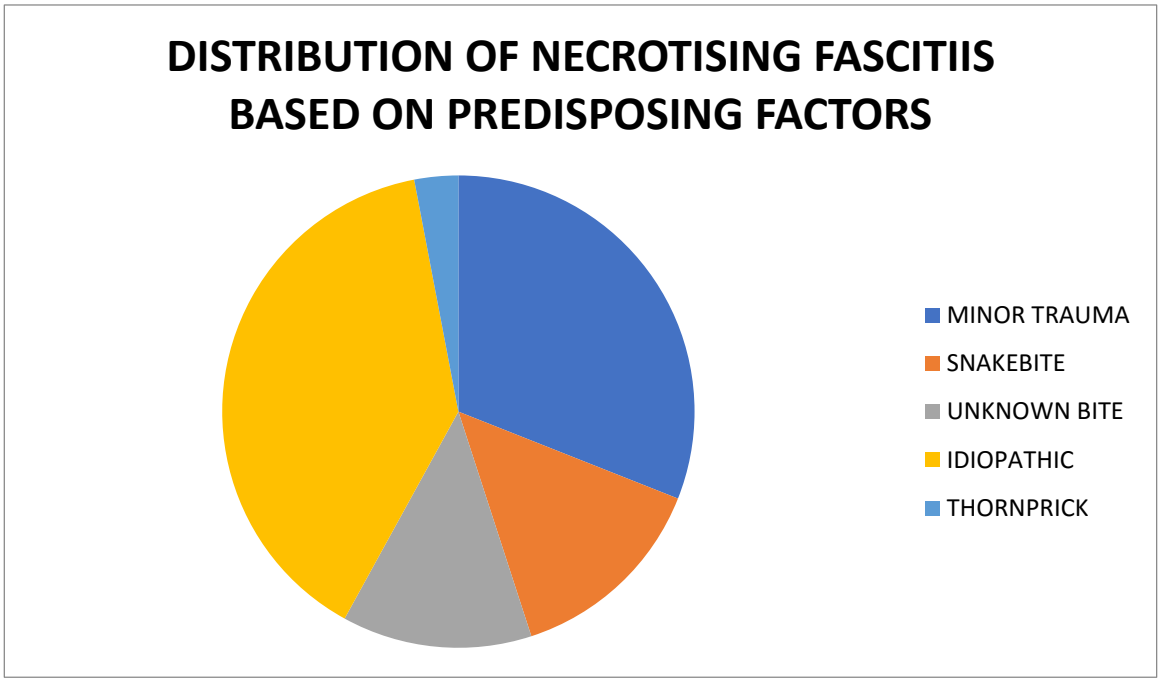
- Most common site of involvement is lower limb 53% and the least involved is abdominal wall and perineum 2%.



OUTCOME	LOWER LIMB	UPPER LIMB	SCROTUM	PERINEUM	GLUTEAL	ABDOMINAL WALL	PRESACRAL
SSG	19	12	5	1	0	0	0
AMPUTATION	7	0	0	0	0	0	0
HSI	24	10	0	0	6	0	0
DEATH	3	0	6	1	1	2	3

- Patients with involvement of lower limb has good prognosis
- Out of 53 members with lower limb involvement
  - 81% got cured with SSG
  - 13% Had amputation, only 6% died.
- Similarly, upper limb involvement had good prognosis
- Involvement of scrotum had poor prognosis with mortality of 55%
- Similarly, involvement of gluteal, perineal, presacral, abdominal wall had poor prognosis.

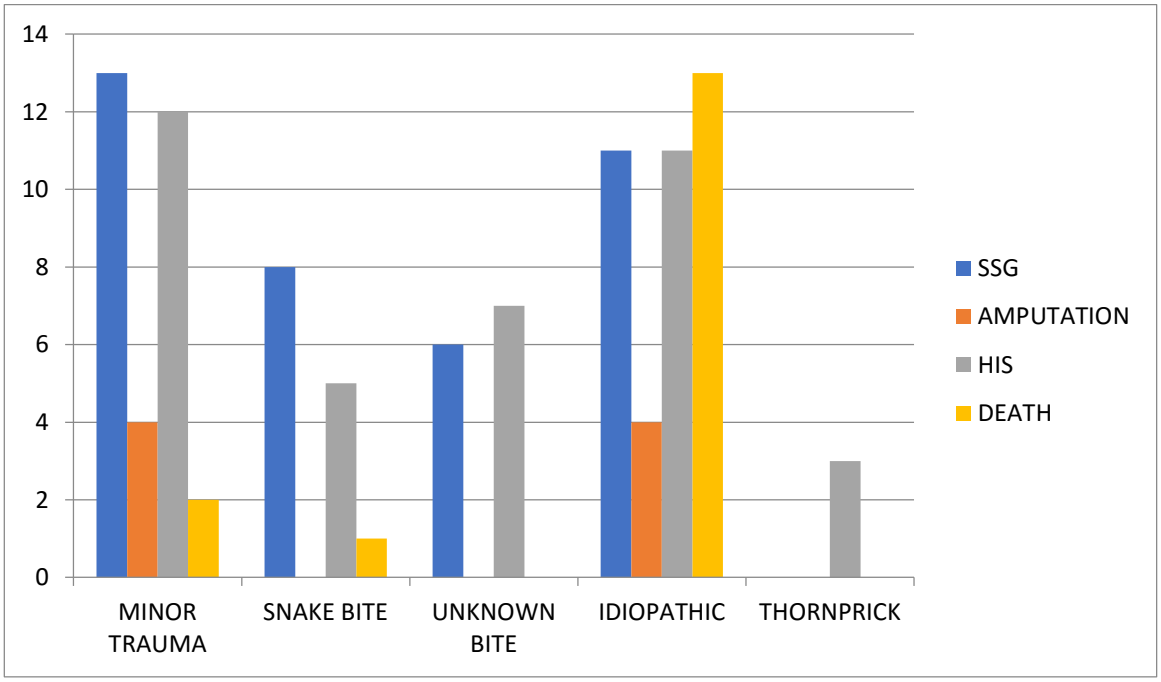
DISTRIBUTION BASED ON PREDISPOSING FACTORS



PREDISPOSING FACTOR	PERCENT AFFECTED
MINOR TRAUMA	31
SNAKE BITE	14
UNKNOWN BITE	13
IDIOPATHIC	39
THORNPRICK	3

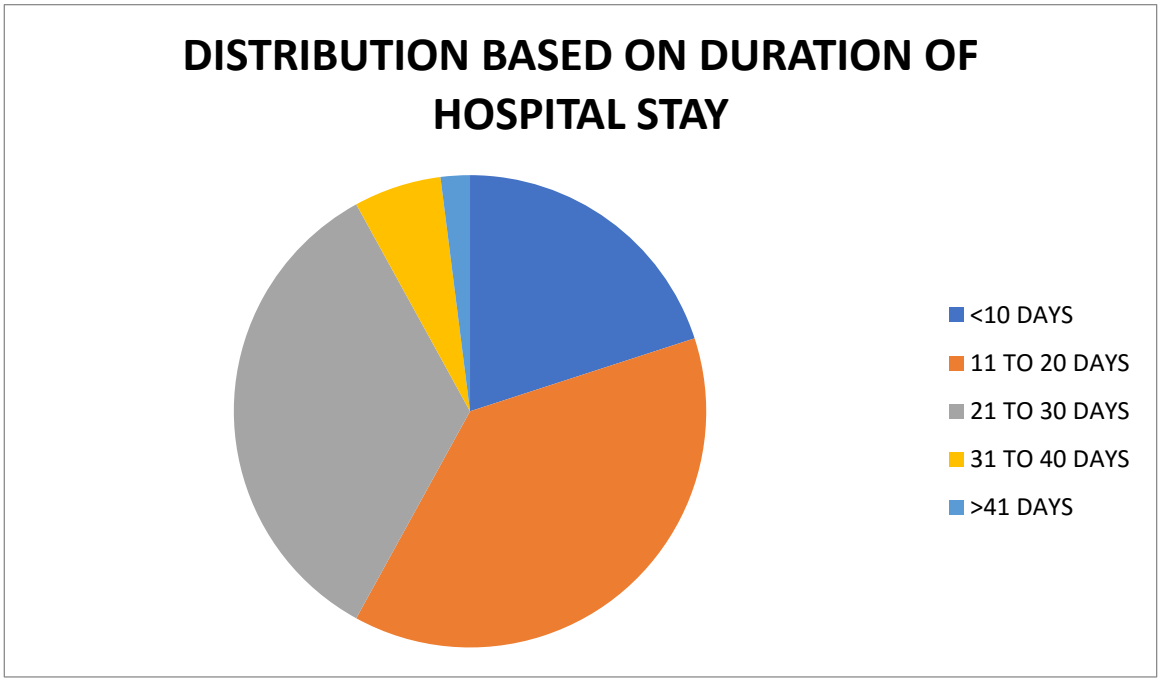
- Most common predisposing factor is UNKNOWN ETIOLOGY (IDIOPATHIC) 39%
- MINOR trauma is the next common PREDISPOSING FACTOR 31%

OUTCOME	MINOR TRAUMA	SNAKE BITE	UNKNOWN BITE	IDIOPATHIC	THORNPRICK
SSG	13	8	6	11	**0**
AMPUTATION	4	0	0	0	4
HSI	12	5	7	11	3
DEATH	2	1	0	13	0



- Mortality is Most commonly seen with idiopathic cause Out of 16 total deaths 13 had unknown etiology.
- Out of 35 patients with UNKNOWN ETIOLOGY 13 patient DIED i.e, 37% mortality

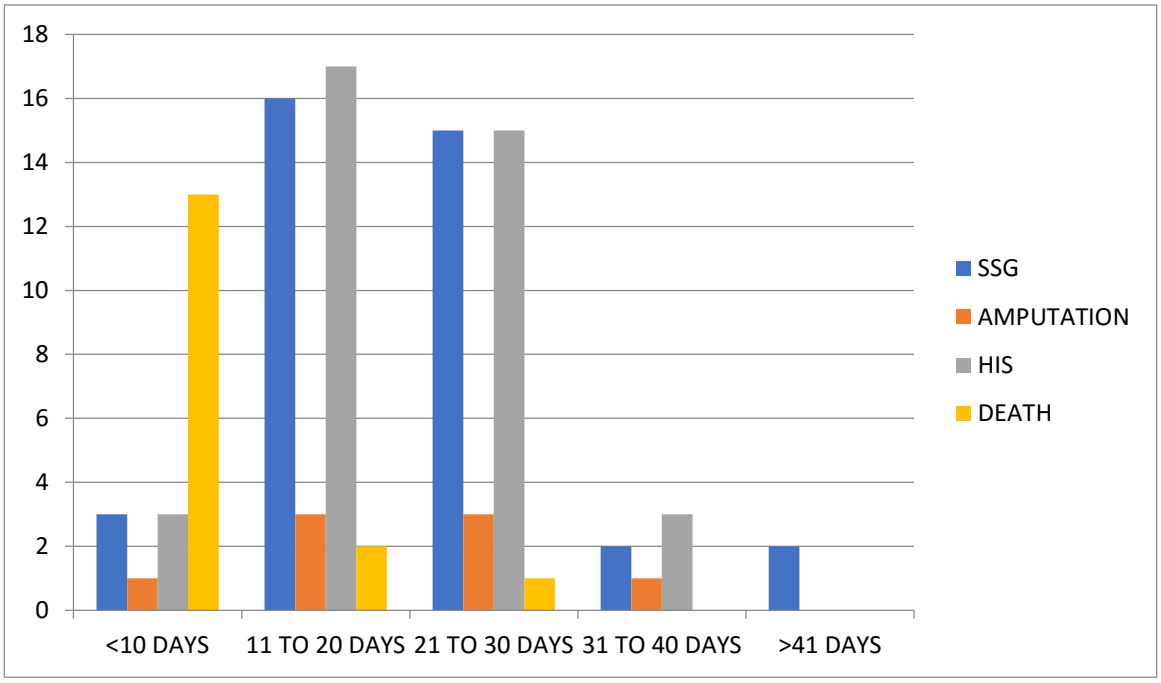
DISTRIBUTION BASED ON DURATION OF HOSPITAL STAY



- Duration of hospital stay indirectly measures the morbidity of the disease
- About 38% of patients stayed between 11 to 20 Days

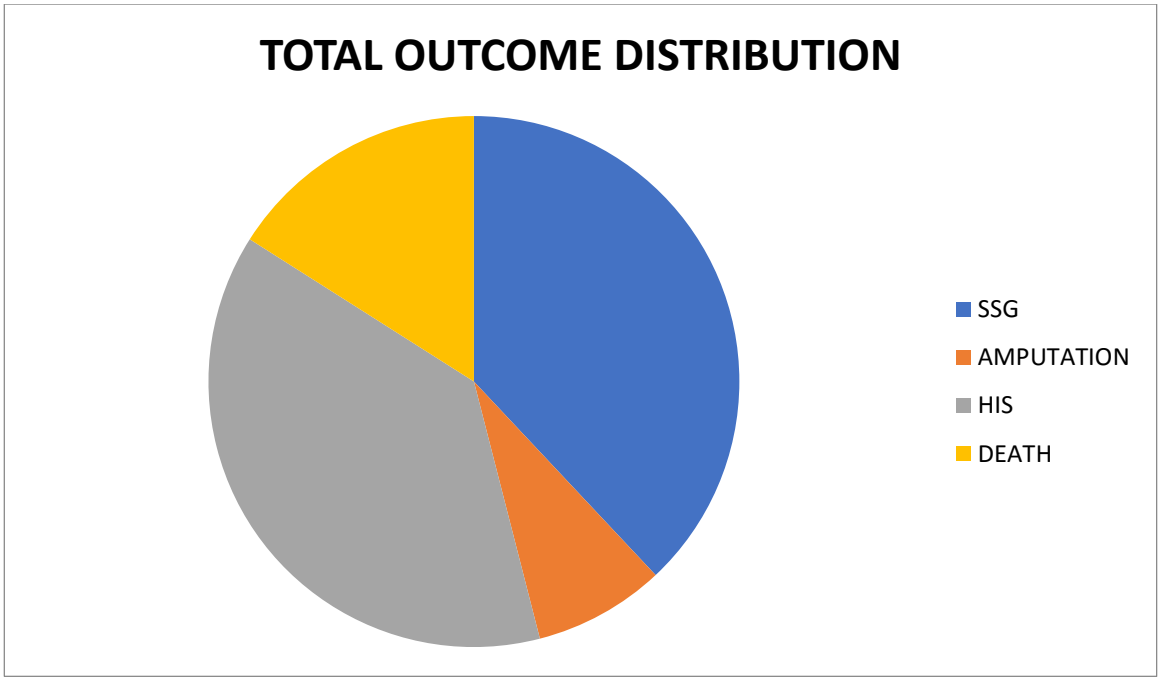
DURATION OF HOSPITAL STAY	PERCENTAGE OF PATIENTS
LESS THAN 10 DAYS	20
11 to 20 DAYS	38
21 TO 30 DAYS	34
31 TO 40 DAYS	6
41 TO 50 DAYS	2

DISTRIBUTION OF OUTCOME BASED ON THE DURATION OF HOSPITAL STAY



- Mortality is more among the patients whose duration of stay is less than 10 days.
- Out of 20 patients Death occurred for 13 patients about 65%
- Most of the patients are having duration of stay between 11 to 20 Days about 38%

DURATION OF HOSPITAL STAY	SSG	AMPUTATION	HSI	DEATH
LESS THAN 10 DAYS	3	1	3	13
11-20 DAYS	16	3	17	2
21-30 DAYS	15	3	15	1
31 TO 40 DAYS	2	1	3	0
MORE THAN 41 DAYS	2	0	0	0



OUTCOME	PERCENTAGE
SSG	38
AMPUTATION	8
HSI	38
DEATH	16

SUMMARY AND CONCLUSION

- Necrotizing fasciitis is a surgical emergency commonest risk factor is diabetes.
- This study was conducted on 100 randomly selected patient in one year period.
- Common age group affected in diabetic patient were 51 to 60yrs (34%) with a mean age of 56yrs.
- Commonly males are affected more 77 males and 33 females were affected.
- More common site of involvement is lower limb 53%.
- The commonest pre disposing factor is idiopatic (39%) followed by minor trauma (31%).
- Most common duration of hospital is between 11 to 20 Days about 38%.
- Most common outcome is split skin grafting.
- About 16 patients had death as the outcome and mortality rate is about 16%.

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PROFORMA

Name:

Age:

Sex:

Date of admission : Date of discharge:

Duration of symptoms:

History of trauma - Yes / no

Any co-morbidities:

Diabetes mellitus - yes / no

Ischemic heart disease

Personnel history:

Alcoholism - yes / no

smoking - yes / no

General examination:

Local examination:

Investigation:

Urine: sugar, Ketones

Blood sugar:

RBS FBS PPBS

Serum creatinine:

Pus culture and sensitivity:

INFORMED CONSENT FORM

Title of the Study : **A CROSS SECTIONAL STUDY TO ASSESS THE OUTCOME OF NECROTIZING FASCITIS AMONG DIABETIC PATIENTS IN CHENGALPATTU MEDICAL COLLEGE**

Name of the Participant : _____ _____.

Name of the Principal (Co-Investigator) : _____.

Name of the Institution : Government Chengalpattu Medical college and Hospital

Name and address of the sponsor / agency(ies) (If any) : _____

_____ _____

Documentation of the informed consent :

I _____have read the information in this form (or it has been read to me). I was free to ask any questions and they have been answered. I am over 18 years of age and, exercising my free power of choice, hereby give my consent to be included as a participant in **"A CROSS SECTIONAL STUDY TO ASSESS THE OUTCOME OF NECROTIZING FASCITIS AMONG DIABETIC PATIENTS IN CHENGALPATTU MEDICAL COLLEGE"**

.

1. I have read and understood this consent form and the information provided to me.

2. I have had the consent document explained to me.

3. I have been explained about the nature of the study.

4. I have been explained about my rights and responsibilities by the investigator.

5. I have been informed the investigator of all the treatments I am taking or have taken in the past _____ months including any native (alternative) treatment.

6. I have been advised about the risks associated with my participation in this study.*

7. I agree to cooperate with the investigator and I will inform him/her immediately if I suffer unusual symptoms.*

8. I have not participated in any research study within the past _____month(s).*

9. I have not donated blood within the past _____months----add if the study involves extensive blood sampling.*

10. I am aware of the fact that I can opt out of the study at any time without having to give any reason and this will not affect my future treatment in this hospital.*

11. I am also aware that the investigator may terminate my participation in the study at any time, for any reason, without my consent.

12. I hereby give permission to the investigators to release the information obtained from me as result of participation in this study to the sponsors, regulatory authorities, Govt. agencies, and IEC. I understand that they are publicly presented.

13. I have understand that my identity will be kept confidential if my data are publicly presented .

14. I have had my questions answered to my satisfaction.

15. I have decided to be in the research study.

I am aware that if I have any question during this study, I should contact the investigator. By signing this consent form I attest that the information given in this document has been clearly explained to me and understood by me, I will be given a copy of this consent document.

SIGNATURE

<u>சுயஒப்புதல்படிவம்</u>

ஆய்வுசெய்யப்படும் தலைப்பு **A CROSS SECTIONAL STUDY TO ASSESS THE OUTCOME OF NECROTIZING FASCITIS AMONG DIABETIC PATIENTS IN CHENGALPATTU MEDICAL COLLEGE**

ஆய்வுசெய்யப்படும் இடம்:

பங்குபெறுபவரின் பெயர்:

பங்குபெறுபவரின் வயது: பங்குபெறுபவரின் எண் :

மேலேகுறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கபட்டுள்ளது. நான் இவ்வாய்வில் தன்னிச்சையாக பங்கேற்கின்றேன். எந்தகாரணத்தினாலோ, எந்த சட்டசிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகிக்கொள்ளலாம் என்றும் அறிந்துகொண்டேன்.

இந்த ஆய்வுசம்பந்தமாகவோ, இதைசார்ந்து மேலும் ஆய்வுமேற்கொள்ளும்போதும் இந்த ஆய்வில்பங்கு பெறும் மருத்துவர், என்னுடைய மருத்துவஅறிக்கைகளைபார்ப்பதற்கு என் அனுமதிதேவைஇல்லை என அறிந்துகொள்கிறேன். இந்த ஆய்வின் மூலம்கிடைக்கும் தகவலையோ, முடிவையோ பயன்படுத்திக்கொள்ள மறுக்க மாட்டேன்.இந்த ஆய்வில் பங்குகொள்ள ஒப்புக்கொள்கிறேன். இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்று உறுதியளிக்கிறேன்.

பங்கேற்பவரின்கையொப்பம்: சாட்சியாளரின்கையொப்பம்:

இடம்: இடம்: தேதி:

தேதி :

பங்கேற்பவரின் பெயர் மற்றும் விலாசம்:

ஆய்வாளரின் கையொப்பம்: இடம்:

தேதி:

ABBREVIATIONS

NF – Necrotizing fasciitis

SSG – Split skin grafting

HSI – Healed by Secondary Intention

B.K – Below knee

RL – Right Leg

LL – Left Leg

LT – Left Thigh

RT – Right Thigh

RH – Right Hand

LH – Left Hand

LF – Left Foot

RF – Right Foot

RA – Right Arm

LA – Left Arm

MASTER CHART

S.NO	NAME	AGE	SEX	I.P.NO	D.O.A	D.O.D	DURATION OF STAY	PREDISPOSING FACTOR	SITE AFFECTED	OUTCOME
1	GNANASEKAR	46	M	26671	4.4.18	28.4.18	24 days	MINOR TRAUMA	SCROTUM	SSG
2	MUNUSAMY	55	M	20646	5.4.18	2.5.18	27 days	MINOR TRAUMA	RL	B.K AMPUTATION
3	KRISHNAVENI	44	F	46659	4.4.18	22.4.18	18 days	POST SNAKE BITE	RL	SSG
4	VALLIYAMMAL	50	F	46634	8.4.18	5.5.18	27 days	IDIOPATHIC	LL	SSG
5	VENUGOPAL	70	M	48409	14.4.18	10.5.18	26 days	MINOR TRAUMA	LA	HSI
6	SIVA	53	M	49300	20.4.18	4.5.18	14	IDIOPATHIC	LL	HSI
7	KASI	70	M	54235	22.4.18	23.4.18	1 day	IDIOPATHIC	SCROTUM	DEATH
8	SADASIVAM	60	M	10798	25.4.18	19.5.18	24 days	MINOR TRAUMA	RL	SSG
9	MUNIYAMMAL	72	F	36776	28.4.18	16.5.18	18 days	MINOR TRAUMA	RA	HSI
10	RAMAMOORTHY	36	M	38467	5.5.18	26.5.18	21 days	IDIOPATHIC	SCROTUM	SSG
11	MOHAN	50	M	38354	6.5.18	30.5.18	24 days	SNAKEBITE	LF	HSI
12	VEDHACHALAM	48	M	38296	8.5.18	12.6.18	35 days	THORN PRICK	LL	HSI

13	DHANABHAGYAM	75	F	46492	4.5.18	24.5.18	20 days	POST TRAUMA	LF	SSG
14	MATHIYALAGAN	37	M	66892	9.5.18	16.5.19	7 days	THORN PRICK	LU	HSI
15	VARADHARAJ	72	M	67462	13.5.18	5.6.19	23 days	IDIOPATHIC	RL	HSI
16	INDRANI	59	F	66804	16.5.18	1.6.18	17 days	POST TRAUMA	RL	SSG
17	RESHMA	45	F	668622	17.5.18	27.6.18	42 days	INSECT BITE	RL	SSG
18	SAROJA	65	F	44297	23.5.18	12.6.18	21 days	IDIOPATHIC	LU	SSG
19	SHANTHA	60	F	49828	26.5.18	9.6.18	15 days	MINOR TRAUMA	RF	HSI
20	BALARAMAN	70	M	57950	29.5.18	21.6.18	24 days	SNAKEBITE	LL	HSI
21	SAMBATH	51	M	62644	31.5.18	15.6.18	16 days	MINOR TRAUMA	RH	SSG
22	ARUMUGAM	50	M	61206	3.6.18	19.6.18	17 days	THORN PRICK	RL	HSI
23	MURUGAN	42	M	62629	6.6.18	26.6.18	21 days	UNKNOWN BITE	LL	HSI
24	AMMAVASAI	62	M	62096	8.6.18	30.6.18	23 days	IDIOPATHIC	SROTUM	SSG
25	VELLAYAMMAL	66	F	62162	13.6.18	7.7.18	25 days	MINOR TRAUMA	RL	HSI
26	CHANDRA BABU	38	M	43698	17.6.18	14.7.18	28 days	SNAKE BITE	RL	SSG

27	PAVITRA	35	M	47175	21.6.18	6.7.18	16 days	MINOR TRAUMA	RH	SSG
28	KASI	70	M	21518	25.6.18	29.6.18	5days	IDIOPATHIC	LL	DIED
29	PONNI	60	F	38623	27.6.18	18.7.18	22 days	IDIOPATHIC	RF	HSI
30	RANI	60	F	368262	30.6.18	28.7.18	29 days	MINOR TRAUMA	LL	SSG
31	KUMAR	35	M	45531	2.7.18	25.7.18	23 days	MINOR TRAUMA	LA	HSI
32	ELLAPPAN	70	M	48466	5.7.18	12.8.18	39 days	IDIOPATHIC	LH	SSG
33	VENKATESAN	45	M	76429	10.7.18	16.7.18	7 days	IDIOPATHIC	PERINIUM	DEATH
34	RAVI	52	M	49525	12.7.18	29.7.18	18 days	MINOR TRAUMA	GLUTEAL	HSI
35	RAJESHWARI	60	F	46422	18.7.18	5.8.18	19 days	UNKNOWN BITE	RL	SSG
36	KALA	56	F	39253	23.7.18	31.7.18	9 days	IDIOPATHI	LL	HSI
37	GOPIKRISHNAN	65	M	366262	29.7.18	19.8.19	22 days	IDIOPATHIC	SCROTUM	DIED
38	MARIYAMMAL	72	F	366246	31.7.18	9.8.18	10 days	SNAKEBITE	LF	SSG
39	SIVAGAMI	65	F	366282	4.8.18	15.818	12 days	MINOR TRAUMA	LL	BK AMPUTATION
40	KUPPAN	55	M	44929	9.8.19	26.8.18	28 days	IDIOPATHIC	RF	HSI

41	ARULAPPAN	38	M	42449	15.8.18	30.818	16 days	IDIOPATHIC	PERINIUM	SSG
42	MUTHUKRISHNAN	49	M	44645	16.8.18	18.8.18	3 days	IDIOPATHIC	SCROTUM	DIED
43	EZHUMALAI	56	M	34966	26.8.18	6.9.18	12 days	UNKNOWN BITE	LL	HSI
44	VEDHACHALAM	46	M	42922	31.8.18	12.9.18	13 days	MINOR TRAUMA	LA	SSG
45	KARTHIKEYAN	36	M	44328	4.9.18	19.9.18	16 days	IDIOPATHIC	RF	SSG
46	ANANDHA KRISHNAN	51	M	43299	9.9.18	25.9.18	17 days	SNAKEBITE	LL	DEATH
47	SUBHAMMA	46	F	42674	12.9.18	23.9.18	12 days	IDIOPATHIC	LL	BK AMPUTATION
48	RAVI	59	M	34292	18.9.18	4.10.18	17 days	IDIOPATHIC	SCROTUM	SSG
49	RAGUPATHY	60	M	43162	23.9.18	1.10.18	9 days	UNKNOWN BITE	LH	SSG
50	ANANDHA KRISHNAN	49	M	33465	26.9.18	12.10.18	17 days	MINOR TRAUMA	LF	HSI
51	SAVITRI	50	F	51566	29.918	5.10.18	7 days	IDIOPATHIC	RL	DIED
52	GANTHA	65	F	49002	3.10.18	13.10.18	11 days	SNAKE BITE	RH	SSG
53	PONNAYAN	42	M	43695	5.10.18	23.10.18	19 days	MINOR TRAUMA	RF	HSI

54	GUNASEKAR	50	M	53244	9.10.18	9.10.18	1 day	IDIOPATHIC	ABDOMINAL WALL	DIED
55	KALIYAMOORTHY	62	M	50390	14.10.18	28.10.18	15 days	UNKNOWN BITE	LL	HSI
56	KANAGA	45	F	51357	18.10.18	24.10.18	7 days	MINOR TRAUMA	LF	SSG
57	THANGAVEL	60	M	484119	24.10.18	16.11.18	24 days	UNKNOWN BITE	LA	SSG
58	ANTONY DASS	65	M	68827	28.10.18	4.11.18	8 days	IDIOPATHIC	PRESACRAL	DIED
59	ARUMUGAM	42	M	46479	2.11.18	18.11.18	17 days	SNAKEBITE	RH	SSG
60	POOVARASAN	61	M	50445	5.11.18	28.11.18	24 days	MINOR TRAUMA	LT	SSG
61	VELU	62	M	35542	5.11.18	9.12.18	35 days	IDIOPATHIC	LL	BK AMPUTATION
62	NAGARAJAN	45	M	46492	7.11.18	13.11.18	7 days	IDIOPATHIC	GLUTEAL	HSI
63	SAGHAYAM	52	M	35938	11.11.18	14.11.18	4 days	MINOR TRAUMA	SCROTUM	DIED
64	JOTHI	55	F	43113	18.11.18	30.12.18	43 days	SNAKEBITE	RF	SSG
65	SADAYANDI	54	M	56276	24.11.18	17.12.18	24 days	UNKNOWN BITE	LF	SSG
66	POOSANAM	70	F	56254	28.11.18	13.12.18	16 days	IDIOPATHIC	GLUTEAL	DIED

67	KAMARAJ	43	M	57164	30.11.18	15.12.18	16 days	MINOR TRAUMA	RF	HSI
68	SHANKAR	39	M	248083	5.12.18	30.12.18	26 days	UNKNOWN BITE	RH	SSG
69	SAROJA	69	F	46361	9.12.18	25.12.18	17 days	UNKNOWN BITE	LF	HSI
70	RANI	56	F	22419	13.12.18	24.12.18	12 days	IDIOPATHIC	GLUTEAL	HSI
71	DILLIGANESH	62	M	24408	17.12.18	29.12.18	13 days	IDIOPATHIC	RL	SSG
72	SUBRAMANI	34	M	24106	25.12.18	19.1.19	26 days	MINOR TRAUMA	LH	SSG
73	SHANTHI	60	F	23154	2.1.19	23.1.19	22 days	UNKNOWN BITE	LF	HSI
74	PAARI	55	M	24391	6.1.19	9.1.19	4	IDIOPATHIC	ABDOMINAL WALL	DIED
75	VENGADESAN	35	M	47401	17.1.19	31.1.19	15 days	MINOR TRAUMA	RL	SSG
76	GOVINDHAN	52	M	47524	19.1.19	2.2.19	16 days	SNAKEBITE	LF	HSI
77	VANITHA	45	F	24035	21.1.19	19.2.19	30 days	IDIOPATHIC	GLUTEAL	HSI
78	NAGENDRAN	56	M	47188	22.1.19	27.1.19	6 days	IDIOPATHIC	SCROTUM	DIED

79	CHANDRAN	62	M	96980	28.1.19	19.2.19	23 days	MINOR TRAUMA	RF	BK AMPUTATION
80	PONNIYAMMAL	60	F	38623	3.2.19	28.2.19	26 days	UNKNOWN BITE	RL	HSI
81	MANIKARAJ	63	M	46031	5.2.19	3.3.19	27 days	IDIOPATHIC	RH	SSG
82	HEMALATHA	60	F	37692	10.2.19	16.3.19	35 days	MINOR TRAUMA	RT	SSG
83	VARADHAN	62	M	39589	19.2.19	23.3.19	33 days	UNKNOWN	LL	HSI
84	ALAMELU	63	F	34320	28.2.19	17.3.19	18 days	SNAKEBITE	LF	HSI
85	REETA	46	F	39219	4.3.19	19.3.19	16 days	MINOR TRAUMA	LL	BK AMPUTATION
86	DHAYALAN	56	M	45376	8.3.19	27.3.19	30 days	IDIOPATHIC	RA	HSI
87	RAJAMANNAR	66	M	43876	14.3.19	12.4.19	30 days	IDIOPATHIC	SCROTUM	SSG
88	MARIYAPPAN	50	M	464403	16.3.19	23.3.19	8 days	TRAUMA	PRESACRAL	DIED
89	DHAYALAN	60	M	455373	19.3.19	24.4.19	37 days	SNAKEBITE	LL	HSI
90	SHANMUGAM	35	M	22146	22.3.19	11.4.19	21 days	IDIOPATHIC	LA	HSI

91	JAYARANI	70	F	642692	26.3.19	21.4.19	27 days	IDIOPATHIC	RL	BK AMPUTATION
92	PERUMAL	50	M	44927	29.3.19	15.419	18 days	SNAKEBITE	RF	SSG
93	LOGANADHAN	42	M	48326	31.3.19	29.4.19	30 days	UNKNOWN BITE	LH	HSI
94	THILAGA	45	F	21548	1.4.19	23.4.19	23 days	MINOR TRAUMA	LA	HSI
95	VARADHAN	62	M	24356	4.4.19	4.4.19	1 day	IDIOPATHIC	PRESACRAL	DIED
96	SAMUEL	53	M	25549	6.4.19	25.4.19	20 days	SNAKEBITE	RH	SSG
97	AYYAKANU	70	M	31653	8.4.19	10.4.19	3 days	IDIOPATHIC	SCROTUM	DIED
98	LAKSHMI	52	F	31563	11.4.19	26.4.19	16 days	IDIOPATHIC	LF	HSI
99	RAGAN	66	M	32355	15.4.19	29.4.19	15 days	MINOR TRAUMA	GLUTEAL	HSI
100	KANNAMAL	63	F	31534	16.4.19	23.4.19	8 days	IDIOPATHIC	LL	BK AMPUTATION