A Dissertation on

"COMPARATIVE STUDY ON THE INCIDENCE OF NECROTISING FASCIITISIN DIABETIC AND NON-DIABETIC AND ITS OUTCOME"

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BONAFIDE CERTIFICATE

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INTRODUCTION

Necrotizing fasciitis is rapidly progressive and destructive soft tissue infections involving the skin, subcutaneous tissue and superficial fascia, with high mortality and long term morbidity. Patient survival is inversely related to the time interval between onset of disease and initiation of appropriate treatement.

Necrotizing soft tissue infections commonly occur in immuno compromised individuals and those with diabetes mellitus or peripheral vascular disease. Mortality from necrotizing fasciitis can be reduced by proper diagnosis, adequate debridement and appropriate antibiotics.

Formation of Blister and bullae, standing out as the clinching diagnostic clues , should raise the suspicion of necrotizing fasciitis which are rarely seen in erysipelas or cellulitis.. Cellulitis accompanied by ecchymoses, bullae, dermal gangrene, extensive edema or crepitus suggests an underlying necrotizing infection and mandates operative

intervention to confirm the diagnosis and definitively treat the infection.

Broad spectrum antibiotics, aggressive surgical debridement and intensive care unit support are essential.

Here we intend to compare the incidence of necrotizing fascitis in diabetic and non diabetic patients with respect to age, sex, duration, symptoms, hospital stay and outcome of necrotizing fasciitis in our hospital. Also to study the available treatment and surgical options in our set up for the management of necrotizing fascitis.To study the residual morbidity and mortality after effective management.

AIM AND OBJECTIVE

To compare the incidence of necrotizing fascities in diabetic and non diabetic patients with respect to

-age,

-sex,

-duration of symptoms,

-common site of involvement,

-precipitating factor,

-causative organism,

-duration of hospital stay and

-outcome.

Also to study the available treatment and surgical options in our set up for the management of necrotizing fasciitis.To study the residual morbidity and mortality after effective management.

REVIEW OF LITERATURE

Wilson first introduced the term Necrotizing Fasciitis in 1952 and the key feature of this disease is fascial necrosis.

Necrotizing fasciltis is an acute, life threatening infection of the superficial fascia and subcutaneous tissue caused by a variety of aerobic and anaerobic bacteria. The clinical process at first appears to be a low grade cellulitis, but fulminant infection develops rapidly in subcutaneous fascia, which may become liquefied with accompanying fat necrosis, thrombosis of subcutaneous vessels and occasional myositis and myonecrosis. As the blood supply to the skin is compromised, cutaneous erythema and oedema progress to cyanosis, bullae and gangrene. Cutaneous gangrene is associated with fever, shock and higher mortality rate.

Necrotizing fasciitis tends to occur in diabetics, alcoholics, intravenous drug abusers, immunocompromised patients, and as a post operative complication.

A patient with necrotizing fasciitis usually presents with the clinical features of cellulitis, including erythema, swelling and local heat in the affected area. There is pain in the area concerned that is out of proportion to the severity of cellulitis.

Systemic features of toxicity, including fever, tachycardia and leucocytosis, are also out of proportion to the apparent severity. If left untreated the skin becomes shiny, hot and exquisitely tender but discrete margins do not develop.

Soft tissue gas is an uncommon feature of necrotizing fasciitis but is seen when there is anaerobic infection. The diagnosis is difficult and rests on high index of suspicion in the clinical settings outlined above.

Radiological examination in the form of plain radiograph or computed tomographic scan may show gas in the tissues. The diagnostic test is a full thickness biopsy of the affected area or surgical exploration.

Although initiation of antimicrobial therapy is essential, these medications cannot stop the progress of necrosis initiated by the toxins released by the organism .Surgery is mandatory.

Aggressive surgical wound debridement is the only treatment option, which leaves the patient with an wide spread post operative raw area.

In the post operative care patient need appropriate intravenous antibiotics, an effective wound management with adequate nutritional support.

A functional extremity can usually be salvaged in fasciitis, if not, amputation can be safely performed later. Immediate amputation is necessary when there is diffuse myositis with complete loss of blood supply or when

adequate debridement will leave a useless limb. When the viability of the remaining tissue is assured and the infection has been controlled, soft tissue deficits can be covered with skin grafts.”

The skin is connected to the underlying bone , muscles or deep fascia by a loose areolar connective tissue , this layer is referred to as superficial fascia , it is of variable thickness and fat content. The neurovascular structures course in this layer , distributing only their terminal branches to the skin.

The limbs and body are wrapped in a membrane of fibrous tissue - the deep fascia.

The Deep fascia is a dense , organized connective tissue layer , devoid of fat, that covers most of the body parallel to the skin and subcutaneous tissue (superficial fascia) .

In the face and ischioanal fossa distinct layers of deep fascia are absent.

Limb is divided into various compartments based on the location (.i.e anterior ,lateral .etc.) with each compartment containing groups of muscles with similar functions and sharing the similar vascular and nerve supply and each separated from one another by the fascia in the form of intermuscular septae that extend centrally from surrounding fascial sleeve to attach to the bones.

The deep fascia itself never passes freely over bone, where deep fascia contacts bone, it blends firmly with the periosteum.

Beneath deep fascia are the muscles, the bones, the joints with synovial sheaths and the cavities (eg:peritoneal)

WOUND HEALING

A clear understanding of healing is vital to a rational approach to the practice of surgery. The major biological processes of tissue repair include collagen metabolism, wound contraction, epithelization, and inflammation.

Wound healing has three phases

Inflammatory phase,

Proliferative phase

Remodeling phase.

Inflammatory phase:

Inflammatory phase starts immediately after wounding and lasts 2 to 3 days.

Wounding is immediately followed by coagulation, altered vascularity, and inflammation, all of which modulate wound healing.

Coagulation is mediated by platelets, and during thrombus formation, platelet factors that enhance fibroblast migration and proliferation are released.

The normal inflammatory response soon follows as small blood vessels dilate, capillary permeability increases, and peripheral neutrophils and then monocytes migrate into

the wound. As monocytes ingest material, they are transformed into macrophages that phagocytize debris as well as enzymatically destroy bacteria. Macrophages also play a role in the induction of collagen synthesis.

Prostaglandins also play a significant role in this process.

Proliferative phase

Proliferative phase lasts from third day to third week consisting mainly of fibroblast activity with production of collagen and ground substance, growth of new blood vessels as capillary loops, and re-epithelization of wound surface.

Collagen provides strength and stability for all tissues of the body. The strength and integrity of all tissue repairs relies on the cross linking and deposition of collagen.

It is not the collagen synthesis but the collagen cross linking that is the bottom line for the surgeon because it is

cross linking that provides strength and integrity to any repair.

Collagen degradation , mediated by enzyme collagenase, is equally important as collagen synthesis in wound repair. In normal unwounded dermis collagen synthesis and degradation occur in equilibrium. After wounding, however , the rates of collagen synthesis and degradation rise and fall in an ordered, sequential fashion , so that enough collagen is synthesized, cross linked, deposited, and removed to provide wound strength and integrity without excessive scarring.

There are seven genetically distinct types of human collagen. Types 1 and 3 are the major components of the skin.

Epithelization is the major healing phenomenon in the partial thickness wound. Epithelial mitosis and migration occur to restore integrity to the partial loss of skin.

Various dressings and pharmacological agents have been used to speed up the process of epithelization. Epithelisation is more rapid with a hydrophobic dressing rather than a hydrophilic dressing.

REMODELING PHASE

It is characterized by maturation of collagen (type I replacing type 3 in the ratio of 4:1).

There is realignment of collagen fibres along the lines of tension, decreased wound vascularity and wound contraction due to fibroblast and myofibroblast activity.

The myofibroblasts (a fibroblast-like cell with smooth muscle components) are the cells responsible for wound contraction.

It is commonly hypothesized that myofibroblasts are the responsible contractile cells and that it is the collagen that holds the newly contracted tissues in position.

TYPES OF WOUND HEALING

Healing by Primary intention

Healing by primary intention occurs when wound edges are approximated shortly after the primary wound has been occured. Epithelization and contraction have little to do with the healing by primary closed wounds, even though minimal epithelization occurs within 24 hours and seals the wound from bacterial contamination.

Healing by secondary intention

This is healing by natural biological processes without surgical intervention, which usually occurs in large wounds associated with skin and soft tissue loss. Although epithelization and collagen deposition are involved, contraction is the most important phenomenon in the spontaneous closure of large open wounds, Unless contraction occurs and brings dermal structures together, the granulating surface is covered only by a layer of

epithelial cells that are useless in providing any coverage with strength and integrity.

Delayed primary closure

Closure of grossly contaminated incisions/wounds should be delayed, allowing time for host inflammatory and immune responses to control contamination. Most significant is that delayed primary closure does not delay the development of wound strength.

AETIOLOGY

Necrotizing infections of soft tissues are infections by virulent bacteria that have the ability, usually by the production of toxins , to cause widespread necrosis.

The soft tissues can be subcutaneous (eg : necrotizing fasciitis) , muscle (eg : gas gangrene) and less frequently skin.

Classification of necrotizing infections by defining the layer of primary importance , the clinical syndrome, and the

dominant organisms thought responsible is depicted in the table.

Necrotizing soft tissue infections are fatal infection, producing progressive tissue destruction with signit'icant potential for soft tissue and limb loss and mortality.

Necrotising soft tissue infections may involve dermis, subcutaneous tissue , fascia, or muscle proportionately. Blood supply to the fascia is usually less than that of muscle or healthy skin making fascia more vulnerable to infectious processes.

Additionally the propensity for fluid to collect between the involved fascia and nearby tissues further weakens fascial immune function by altering host clearance of the organisms by inhibiting phagocytic function. Necrotizing fasciitis is more common because infection may spread widely across the fascial planes with minimal involvement of surrounding skin or mucosa.

Necrotizing fasciitis is defined by Bisno as : ‘Necrotizing fasciitis is a deep seated infections of the subcutaneous tissue that results in the progressive destruction of fascia with fat.

The incidence of Necrotising fascitis has been reported to be 0.40 cases per 100,000 population. Although Necrotising fascitis is rare, certain conditions can predispose patients for developing the disease, including immunocompromised states such as diabetes mellitus, HIV, and malignancy and those with intravenous drug abusers or alcohol.

Necrotizing fasciitis can also occur as a result of trauma, such as burns, abrasions and lacerations. Even minor trauma, such as insect bites thorn prick, needle sticks, can lead to Necrotising fascitis . Patients with peripheral vascular disease and atherosclerosis also have an increased risk.

However, previously healthy persons can also develop the disease. The use of nonsteroidal anti-inflammatory drugs (NSAIDs) like aspirin , with varicella infection in particular, has been implicated as a risk factor for developing Necrotising fascitis, although the association is still being debated.

The entry of bacteria can occur from any break in the skin and can occur in patients with preexisting skin conditions like psoriasis, pressure ulcers, boils, or perirectal abscesses.

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CLASSIFICATION

Necrotizing fasciitis is classified as Type I or Type 2 depending on which organisms are cultured (Table 1).

- Type I Necrotising fascitis is a polymicrobial infection from aerobic and anaerobic bacteria such as Clostridium and Bacteroides species.
- Type 2 Necrotising fascitis consists of group A Streptococcus (S. pyogenes) with or without a coexisting Staphylococcal infection.
- Type 3 Necrotising fascitis is associated with Vibrio vulnificus, Necrotizing fasciitis classification

Type 1	***Type 2***
Bacteroides Peptococcus, Pseudomonas Fusobacterium , Candida Clostridium, Cryptococcus	Group A Streptococcus +1-Staphylococcus

Coryne bacterium, Histoplasma Streptococcus (not group A), Vibrio Escherichia, Staphylococcus Enterobacter, Shigella Proteus , Neisseria Klebsiella, Pasture/la Serratia, Salmonella	

Clinically, Necrotising fascitis can be divided into three groups based on the length and extent of the disease,

- Fulminant Necrotising fascitis - Patients with fulminant disease were presented with rapid disease progression and are often in a state of shock.They have typically have symptoms for only several hours.
- Acute Necrotising fasciitis -Patients with acute disease have symptoms for several days. In most cases, large areas of their skin are involved.

- Sub-acute Necrotising fasciitis - Patients with subacute disease may have symptoms for several days to weeks. Only a localized area of skin is generally involved.

Necrotizing fasciitis can affect any area of the body, but it commonly affects the extremities.

Involvement of the external genitalia is referred to as Fournier's gangrene and usually results from infection by.wide variety of micro organisms.

There is a higher mortality rate when it affects the head, neck, chest, and abdomen because those areas tend to be refractory and are more difficult to treat.

MYONECROSIS

Bacterial myonecrosis is the preferred term when muscle invasion occurs.

It is an uncommon disease with a grave prognosis even with aggressive therapy.

Non closfridial m onecrosis is caused by the same organisms as is necrotizing fasciitis and the initial symptoms may be identical.

Chronic skin ulcers and perineal infections were the most frequent primary sites. Soft tissue gas was usually present.

Non clostridial myonecrosis is caused by a group of bacteria, the most predominant of which are the anaerobic streptococci.

If not treated it progresses to gangrene, toxaemia and shock.

Clostridial myonecrosis is caused by clostridium perfringens, clostridium novyi, and clostridium septicum.

Clostridia are obligate anaerobes which survive in the environment as spores that are not readily destroyed by disinfectants. When inoculated into normal muscle tissue they do not germinate because of high oxygen tension.

However in a wound with devitalized or ischemic tissues, the spores germinate , and the bacteria produce exotoxins that create a high mortality rate associated with clostridial myonecrosis.

PATHOPHYSIOLOGY

The bacteria enter the subcutaneous layer either through a breaks in the skin or from remote sites of infections, for example, renal calculi, intestinal fistulae, and infective endocarditis.

Host factors which weaken the resistance to the micro organism, such as a compromise in skin or mucosal integrity, immuno deficiency, and diabetes, contribute to bacterial growth.

Bacterial endo and exo toxins and enzymes, like hyaluronidase, collagenase, streptokinase, and lipase, aggravates the spread of the pathogen and tissue necrosis. Necrosis of soft tissues progresses rapidly, which may not be easily noticiable because the necrosis of subcutaneous tissue and fascia is more than that of the skin.

The micro organisms rapidly proliferate in the subcutaneous tissue, which then invade and block the blood

vessels and lymphatic channels, producing vasoconstriction and thrombosis. Because of a poor blood supply the fascial layers underneath the skin are more sensitive to the events that cause hypoxemia which lead to inhibition of the immune system to fight against the infection; moreover, hypoxic conditions in the subcutaneous tissues decreases the functions of polymorphonuclear neutrophils.

Reduced blood flow impairs the availability of antibiotics to the affected area, which result in necrosis of the skin, fascia, and muscles.

In the initial stages, superficial epidermal necrosis with pannicular edema and hemorrhage, possibly without obvious pathogen or inflammatory cells, may be found.

As the lesion progresses, more numbers of polymorphonuclear cells migrate into the dermis, subcutaneous fatty layer. Destructive inflammation and coagulation necrosis takes place in the subcutaneous tissue and fascia which may spread to the muscles, followed by

angiitis and necrosis of skin appendages in the affected area. Necrosis of the cutaneous nerves causes pain.

Blister or bulla formation is important finding of Necrotising Fascitis because they are rare in cellulitis and erysipelas. They are due to ischemia in the vessels supplying the skin with resultant vessel necrolysis and thrombosis. Rarely, associated lymphangitis, and venous thrombosis are seen.

Patients may go for overwhelming sepsis and multiple organ failure followed by death.

CLINICAL FEATURES

Early diagnosis of a necrotizing fasciitis is important if optimal outcomes have to be achieved. Distinguishing necrotizing fasciitis from cellulitis can be difficult.

Unfortunately, any delay in diagnosis is highly dangerous because the concomitant delay in appropriate wound debridement may lead to increased mortality.

Pain warmth and swelling are present in most cases but are not specific to necrotizing infections and may not be universally present.

Several clinical signs are highly specific to necrotizing fasciitis but occur late in its course. These include

- Bullae formation
- Skin ecchymosis
- Presence of gas
- Cutaneous anaesthesia

Other less specific clinical signs are-

1) Severe Pain

2) Extensive Oederna

3) Systemic toxicity

4) Progression of disease inspite of antibiotic therapy

Based on the clinical course, Necrotising Fascitis is classified into 3 groups : acute, fulminant, and subacute.

ln Acute Necrotising Fascitis, the skin becomes purple-black or gray-blue in three to four days, as tissue necrosis occurs which lead to sloughing of skin. Destruction of the affected cutaneus nerve lead to numbness. As necrosis progresses, subcutaneous tissues become a hard, wooden feel without pain, and ulcers may occur. Crepitation is commonly found in polymicrobial infections including Clostridium.

Patients had severe pain which cannot be completely relieved by anti inflammatory drugs.

Involvement of muscles lead to necrotizing myositis.

Systemic findings, such as high temperature,associated with chills, tachycardia dehydration, hypotension, and multiple organ may appear due to the release of toxins into the blood.

In immune compromised patients the classical signs of necrotizing fasciitis are absent. Due to the reduced blood supply the action of antibiotic was reduced.

Septic emboli can get lodged in distant sites leading to metastatic abscesses.

The Fulminant Necrotizing Fascitis progresses very rapidly leading to septicemia, MODS and death within 24 hours.

Patients with Sub-Acute Necrotizing Fasciitis may have symptoms persisted for few weeks to months.

Systemic manifestations of necrotizing fasciitis

Toxic appearance Fever Chills Constitutional symptoms Shock Multiorgan system failure Mental status changes	Neuralgia Weakness/fatigue Tachycardia Tachypnea Decreased urinary output Death

INVESTIGATIONS

LABORATORY RESULTS

Haemoglobin - it is a useful investigation to know the general status of the patient and to know about the fitness of the patient for distinctive operative procedures. Reduced haemoglobin may add in addition to the other causes of non healing of wound.

Total WBC count - this includes the count of polymorphs, lymphocytes and eosinophils.

Differential WBC count - A raise in polymorphs count will give a clue to the underlying infection and severity of infection.

Its increased in long standing diseases like tuberculosis etc.

Bleeding time and clotting time - altered levels may require correction when contemplating any surgery for the patient.

Fasting blood sugar - to know the presence or absence of diabetes and to assess the degree of control of diabetes.

Serum creatinine - it s the more sensitive indicator of renal function, which may be hampered in renal failure as a result of long standing diabetes mellitus or as a result of acute necrotizing fasciitis going for renal failure.

Blood urea - also indicates renal function, but may vary with hydration of the patient.

HIV 1 & 2 - for diagnosis of underlying immunodeficiency syndrome. HBsAg - for diagnosis of underlying hepatitis B infection.

Examination of the urine

For sugar - detection of glucose levels in urine to rule out diabetes mellitus For ketone bodies - to rule out complication of diabetes - diabetic ketoac idosis.

Bacteriological examination - Examination of the discharge for culture and sensitivity is important. A

baseline bacterial culture with sensitivity result is useful. It provides a guideline for starting chemotherapy.

Biopsy with frozen section

The diagnostic test is a full thickness biopsy of the affected area or surgical exploration. If necrotizing fasciitis is present , one would see watery dish pus coming out of the tissues and there would be easy separation of the skin from the fascia due to extensive necrosis of subcutaneous tissues.

Radiographic studies

Soft tissue gas is a uncommon feature of necrotizing fasciitis but is seen when there is anaerobic infection. This can be visualized on plain x rays? the involved area.

One bedside maneuver, called the >FINGER TEST" was described by Childers. After administration of local anaesthetic drug , a 2 cm incision is made into the deep fascia , with the surgeon taking note of any drainage . If

no bleeding occurs or if a musky dishwater discharge is observed, necrosis should be suspected . If minimal resistance is felt when a finger is inserted and pressure is applied to the subcutaneous tissue, the test is positive and Necrotising fascitis is indicated.

MANAGEMENT

Successful management of necrotizing fasciitis includes

- Early diagnosis
- Surgical debridement
- Amputation of extremity
- Wound care
- Antimicrobial therapy
- Intensive supportive care
- Hyperbaric oxygen

Early diagnosis with frozen section biopsy at bedside and immediate surgical debridement is critical.

Surgical debridement

Adequate surgical debridement is essential to the successful management of necrotizing fasciitis. This will

require radical excision of all necrotic tissue, drainage of involved fascial planes, and extensive fasciotomy.

Careful reevaluation of the wound and formal re-exploration in the operating theatre under general anaesthesia is also required, often on two or three further occasions.

Surgical debridement is a form of mechanical debridement which includes sharp debridement, wet to dry dressings and high pressure irrigation or intermittent lavage are well accepted treatment measures.

Thorough debridement of all non viable soft tissue and bone from the infected wound is accomplished mainly with a scalpel, tissue nippers, and or curettes.

Autolytic debridement occurs naturally in a healthy moist wound with well maintained arterial perfusion and venous drainage.

Enzymatic debridement (using topical, proteolytic enzymes such as collagenase) however, is used as an adjunctive treatment in the management of wounds.

Wound Management

Usually, a wound with a moist environment bandaged to protect it from trauma and local contamination has been shown to improve the healing process. The type of dressing depends upon several factors such as size, depth, location, and the wound surface.

DRESSINGS

The types of dressings can be broadly divided into films, composites, hydrogels, hydrocolloids, alginates, foam and other absorbtive dressings including N PWT-Negative Pressure Wound Therapy.

The choice of one over the other is made by considering the wound characteristics and treatment goals.

The amount and type of exudates that is present in the wound will direct the dressing used in wounds that have some degree of bacterial colonization.

In general hydrogels, films, and composite dressings are best for wounds with light amounts of exudate; hydrocolloids are used in wounds with moderate amounts of exudate; and alginates, foams, and NPWT are best used for wounds with heavy volumes of exudate. Wounds with large volumes of necrotic material should not be treated with dressing until a surgical debridement has been performed.

TOPICAL TREATMENTS:

Saline / amorphous hydrogels: skin cleansers	Clean / infected wounds	Undefined
Detergents / antiseptics - povidone iodine etc.	Contaminated or infected wounds	Healthy granulating wounds
Topical antibiotics	Contaminated or	Healthy

silver sulfadiazine, bacitracin, mupirocin etc	infected wounds	granulating wounds
Enzymes :collagenase, papain, urease etc	Necrotic/escharotic wounds	Healthy or infected wounds
Growth factors - becaplermin gel, platelet derived growth factors	Neuropathic diabetic foot ulcers	Infected / necrotic wounds
Dermal skin substitute- Apligraf, Dennagraft	Diabetic ulcers, Venous stasis ulcers	Lnfected / necrotic wounds

VARIOUS TYPES OF DRESSINGS

CATEGORY	INDICATIONS	CONTRA INDICATIONS
Transparent films-	Dry to minimally draining wounds	Infection: Significant Drainage: Over prominence or friction
Hydrogels - Gel sheet guaze 95% water / glycerine	Dry to minimally draining wounds	Moderate to heavy draining wounds
Foam - Polyurethane foam; Open cell absorbent	Moderate to large exudates, clean wound surface	Dry wounds
Hydrocolloids: Water with adhesion carboxymethylcellulose Pectin gelatin impermeable to oxygen	Low to moderate drainage	Heavy drainage, Sinus tracts or deep wounds

CATEGORY	INDICATIONS	CONTRA INDICATIONS
Calcium alginates- pad made of Fibre from sea wood	Heavy draining wounds	Minimal drainage or dry wounds
Collagen dressings	Low to heavily draining wounds	Dry wounds
Antimicrobial dressing - Contains silver / iodine in various preparation	Infected / clean wounds to prevent infection	Allergies to components

NEGATIVE PRESSURE WOUND THERAPY

Negative pressure wound therapy or vacuum assisted wound closure has been a tremendous advance for the wound care practitioner.

It consists of the use of a porous sponge within the wound, covered by a airtight occlusive dressing, to which a vacuum is applied.

NPWT works through a combination of mechanisms

- One important action is relief of oedema. NPWT removes the pericellular exudates and wound exudates, thereby improving interstitial diffusion of oxygen to cells.
- NPWT also removes deleterious enzymes from the wound. By removing the wound fluid and bacteria that inhibit wound healing, NPWT modifies the wound microenvironment toward one more conductive to healing.

The cyclic compression and relaxation of the wound tissue likely stimulates mechanotransductive pathways that result in increased growth factor release, matrix production, and cellular proliferation.

NPWT can also be used to assist the neovascularisation of skin grafts and tissue engineered skin substitutes.

HYPERBARIC OXYGEN

The use of hyerbaric oxygen (HBO) raises the dissolved oxygen saturation in plasma from 0.3% to nearly 7%. This rise in oxygen saturation increases the interstitial diffusion distance of oxygen four to fivefold.

The broadening use of transcutaneous oximetry has permitted evaluation of patients that will likely benefit from HBO.

HBO can be inspired by a special pressure vessel known as recompression chamber. It can be used to reduce the number of amputations in necrotizing faseitis.

ANTIBIOTICS

Before the availability of report of pus culture and sensitivity broad spectrum antibiotic must be started in all cases of necrotizing fasciitis especially in type1.A common combination of antibiotic are penicillin an aminoglycoside and a third generation cephalosporin.

Imipenem-cilastatin which acts against anerobes and Pseudomonas may also be used.

Clindarnycin is also a effective drug which can be used against streptococcal.

Vancomycin can be used against MRSA organism or to patients allergic to penicillin.

Dermagraft

This is a product of wound healing which consisted of neonatal dermal fibroblasts cultured in vitro on a biologically absorbable polyglactin mesh by tissue engineering technique. This graftskin or dermagraft

(available as Apligraft) is an allogenic bilayered, metabolically active cultured skin equivalent , which has an upper epidermal layer and a lower dermal layer and contains human skin cells.

The dermal layer is produced by human fibroblasts which organize the provided structural protein which in turn produce additional matrix proteins. The epidermal layer is produced by prompting human keratinocytes first to multiply, which then to differentiate to replicate the architecture of human epidermis. Unlike normal human skin, graft skin does not contain structures like blood vessels, hair follicles, or sweat glands or other cells like langerhan's cells, melanocytes, macrophages, or lymphocytes. Graftskin has shown to produce all cytokines and growth factors that are formed by normal skin during the process of healing.

This graft skin does not elicit any immunological response from host Graftskin is not associated with any other side effects such as wound infection and cellulitis.

PLATELET DERIVED GROWTH FACTOR (BECAPLERMIN)

Advancement in molecular biology have made possible the development of highly purified recombinant human proteins and recombinant human growth factors have evolved as effective therapeutic wound healing agents. Becaplerin — BR (rh PDGF-BBI) is quickly emerged as one of the major candidates for clinical studies. In animal models, rh PDGF-BB demonstrated wound healing activity, mainly by increasing the formation of granulation tissue. It is available in USA as Regranex gel for treatment of wounds with chronicty which helps in complete healing.

GRANULOCYTE COLONY STIMULATING FACTOR (GCSF)

GCSF is an endogenous haemopoietic growth factor which induces terminal differentiation with release of neutrophils from bone marrow. The recombinant form is used commonly to treat chemotherapy induced neutropenia.

Endogenous (GCSF) concentrations increases during bacterial infections in both neutropenic and non neutropenic states, GCSF has key role in neutrophil response to infection. GCSF improves the function of both normal and dysfunctional neutrophilic cells.

GSCF therapy was associated with earlier removal of pathogens from infected wounds, faster resolution of cellulitis, and shorter duration of intravenous antibiotic treatment.

GLYCAEMIC CONTROL

The control of glucose levels should be as strict as possible, and blood glucose levels above lOmmol/L must be avoided, as they are associated with impaired function of the leucocytes, both polymorphonuclear and mononuclear cells. This degree of glucose control should not be achieved by excessive restriction of food intake in someone with tissues te heal and an infection to combat. Insulin will often be required in those not previously receiving it, even if only temporary.

SMOKING

The patient should not smoke because of its ill effects on the peripheral circulation during wound healing process

Skin grafting

Skin grafts are a standard procedure for closing defects which cannot be primarily closed. Skin grafts consist of epidermis with some or all of the dermis.

There are two types of skin grafting -

- Split thickness graft (consists of varying quantity of dermis)
- Full thickness skin graft (consists of whole dermis)

All grafts contract immediately following removal from the donor site and then after re-vascularisation in their final location.

Primary contraction is due to the immediate recoil of freshly harvested grafts due to the presence of elastin in the

dermis. More dermis is associated with increased primary contraction.

Contraction of a healed graft lead to secondary contracture and is probably due to myofibroblast activity.

A full thickness skin graft contracts more on initial harvest but less on healing (secondary contracture) than split thickness graft. The thinner the split thickness graft, greater the contracture (secondary).

Granulating wounds healed secondarily, without any skin grafting, shows the greatest amount of contracture and are more prone for hypertrophic scarring.

Sensory return is high in full thickness skin graft due to a greater availability of neurilemmal sheaths. Hair follicles are also transferred with a full thickness skin graft.

Success of skin grafting / take depends on-

The available nutrients to the graft

Subsequent blood vessels in-growth from recipient bed

Revascularization in Skin graft occurs in 3 phases:

I phase - it involves the process of serum imbibition & lasts for 1-2 days. Here a fibrin layer is formed which bind graft to bed.

II phase - it is a phase of inosculatory in which the recipient & donor site capillaries are re-aligned, It lasts for 72 hrs.

In III phase - the graft is revascularised by these kissing capillaries.

Skin grafts needs a vascular bed and will rarely take up in bones exposed, cartilage, or over tendon which are devoid of their periosteum, perichondrium, or paratenon, respectively.

Post skin grafting immobilization is required for its take up.

Hematomas and seromas should be avoided underneath the graft.

Meshed skin grafting versus sheet skin grafting

- More area can be covered with Meshed skin graft and it allows proper drainage through the holes.
- The result is pebbled appearance, is aesthetically unacceptable.
- Sheet skin graft have a continuous, uninterrupted surface, with superior aesthetic results but it does not allow serum and blood to drain through it and it need a larger graft.

DONOR SITE FOR SKIN GRAFT

- For face defects - Grafts which are taken above the clavicle provide best color match. Also upper eyelid skin can be taken for small defects.

Common donor sites

- Scalp
- Abdominal wall
- Buttocks
- Thigh

Post operation care

- Proper immobilization
- Antibiotics for prevention of infection

SKIN FLAPS:

- It has its own blood supply.
- A skin flap has skin associated with subcutaneous tissue which are transferred from one part to another part of the body through a vascular pedicle or attachment.
- Local skin flaps has two types:
  - Flaps which rotate about pivot point.
  - Advancement flaps .

Amputation

- Amputation may be necessary for acute or chronic infections which is not responding to antibiotics and surgical debridement.
- Open amputation is indicated in this setting and may be performed using one of two methods.
- A guillotine amputation may be performed with later revision to a more proximal level after the infection is under control.
- Alternatively an open amputation may be performed at the definitive level by initially inverting the flaps and packing the wound open with secondary closure 10 to 14 days later.

In the acute setting, the most worrisome infections are those produced by gas forming organisms.

Three distinct gas forming infections must be differentiated:

- Anaerobic cellulitis/NF
- Clostridial myoneorosis
- Streptococcal myoneorosis

The systematic effects of a refractory infection may justify amputation.

OPEN AMPUTATIONS

An open amputation is one in which the skin is not closed over the end of the stamp.The operation is first of at least two operations required to construct a satisfactory stump.

It must always be followed by secondary closure, reamputation, revision or plastic repair.

The purpose of this type of amputation is to prevent or eliminate infection so that final closure of the stump may be done without breakdown of the wound.

It is indicated in infections and in severe traumatic wounds.

A wound vaccum assisted closure is applied to the open stump immediately after initial debridement. Subsequent debridements are scheduled at 48 hr intervals. Reapplied after each debridement until wound is ready for closure.

COMPLICATIONS OF AMPUTATION

- Hematoma
- Infection
- Wound necrosis
- Contractures
- Pain

AMPUTATIONS OF LOWER EXTREMITY

I. Transtibial amputations

The importance of preserving the patients own knee joint in the successful rehasilitation of a patient with a lower extremity amputation cannot be over emphasized.

Transtibial amputations can be divided into 3 levels. The appropriate level must be determined for each individual patient.

In adults, ideal bone length for a below knee amputation stump is 12.5 to 17.5 cms, depending on body height.(rule of thumbs 2.5 for each 30 cm height)

Ideal/most satisfactory level is about I 5cnis distal to medial tibial articular surface. A stump less than 12.5 cm is less sufficient.

2. Disarticulation of knee

- ❖ It results in an excellent end bearing stump.
- ❖ It use in elderly is limited.

AMPUTATIONS OF UPPER EXTREMITY

1) Forearm amputations: Trans-radial:

Either distal or proximal transradial amputation can be performed depending on extent of disease.

2) Elbow disarticulation:

Elbow joint is an excellent level for amputation because the broad flare of humeral condyles can be grasped firmly by the prosthetic socket and humeral rotation can be transmitted the prosthesis.

3) Arm / transhumeral amputation:

It is defined as an amputation at any level from the supracondylar region of the humeral distally to the level of axillary fold proximally. The level of bone section should be atleast 3.8 cm proximal to the elbow joint to allow for elbow lock mechanism.

4) Shoulder amputation:

Most amputations in the shoulder area are performed for the treatment of malignant bone or soft tissue tumors that cannot be treated by limb sparing methods and rarely for infection / trauma.

MATERIALS AND METHODS

3.1 Type of study : Prospective study.

3.2 Study approval : Prior to commencement of this study , Ethical Committee of Rajiv Gandhi Govt.Gen.Hospital and Madras Medical College had approved the thesis protocol.

3.3 Place of study : , Rajiv Gandhi Govt.Gen.Hospital and Madras Medical College,Chennai.

3.4 Period of study : One year duration starting from April 2011 to March 2012.

3.5 Sample size : 50 cases

3.6 Selection of patients:

a) Sampling method- Random.

b) Inclusion criteria-

(1) The patients clinically diagnosed as necrotising fasciitis

Exclusion criteria:

Patients

1. Who were not willing to participate in the study
2. Those with Peripheral vascular disease, IHD and CVA
3. Whose age >75yrs
4. Those underwent wound debridement outside our hospital

were excluded from the study.

3.7 Ethical consideration

All the patients/ legal guardians were given an explanation of the study and about the investigative and operative procedures with their merits and demerits, expected results, and possible complications. If he/she agreed then the case had been selected for this study. The study did not involve any additional investigation or any

significant risk. It did not cause economic burden 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.

3.8 Data collection

Data were collected by pre-tested structured questionnaire. Data were collected from all the respondents by direct interview after getting informed written consent from them or from their legal guardian.

3.10 Study procedure

The data for the study was obtained from patients (hospitalized patients) with a provisional diagnosis of necrotizing fasciitis on clinical evaluation and who are admitted at Rajiv Gandhi Government General Hospital. Patients presenting with signs and symptoms of Necrotizing Fasciitis admitted during April 2011 to March 2012 at Rajiv Gandhi Government General Hospital, were counselled for

investigation and treatment of Necrotizing Fasciitis and its complication. Of those patients admitted with necrotizing fasciitis, 50 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

1) Routine blood investigations: Hemoglobin, total leucocyte count, differential count, ESR

2) FBS, PPBS, and corresponding urine sugar on regular basis

3) Routine urinalysis: Albumin, sugars, ketones and microscopy

4) Blood urea and serum creatinine

5) Lipid profile

6) Radiograph of affected part (lower limbs)

7) Wound discharge for culture and sensitivity

8) Biopsy of the affected part

9) 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 hypoglycaernic 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.

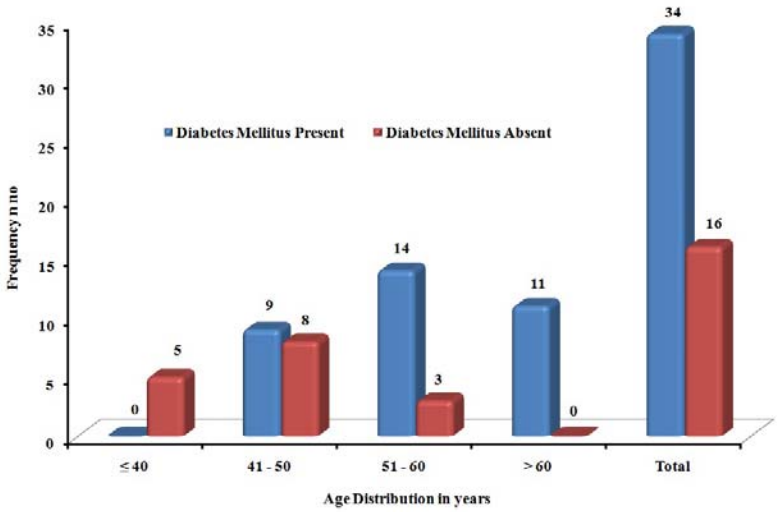
Post discharge patients were followed up to one month regularly on out patient basis for dressings, further management of diabetes and hypertension and also to review liver and renal parameters. Major amputation patients were advised for clutches and artificial prosthesis 4 weeks after surgery. In this study, we compare the incidence of necrotizing fasciitis in Diabetic and non diabetics with respect to age, sex, duration of symptoms parts commonly involved, causative organism and the outcome were analysed and discussed.

RESULTS AND DISCUSSION

AGE WISE DISTRIBUTION

Out of 34 diabetic patients with necrotizing fasciitis the maximum incidence was seen in the age group of 51-60 years (41.2%), whereas out of 16 non diabetic patient the maximum incidence was in the age group of 41-50years (50%).The mean age in diabetic is 56 and in non diabetic is 45.

Age	Diabetic		Non Diabetic		Total
	No	%	No	%	
≤ 40	0	0.0%	5	31.3%	5
41 – 50	9	26.5%	8	50.0%	17
51 – 60	14	41.2%	3	18.8%	17
> 60	11	32.4%	0	0.0%	11
Total	34		16		50
Mean ± SD	56.79±7.41		45±6.58		53.02±9.01

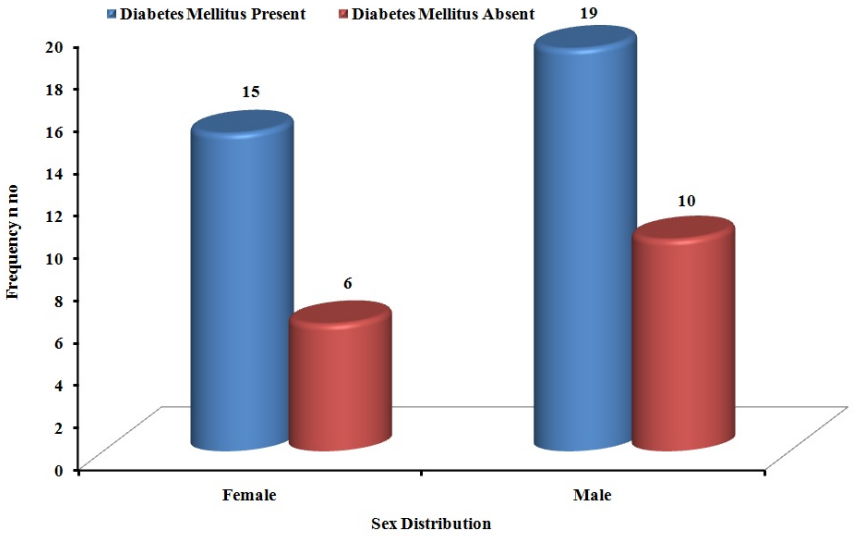


P–Value–0.000< 0.05 is statistically significant, there is difference in age group between Diabetes Present and absent - Student ‘t’ test

SEX WISE DISTRIBUTION

Out of 34 diabetic patient with necrotizing fascitis, 19 patients were male(55.9%) and 15 patients were female(44.1), whereas out of 16 non diabetic patients with necrotizing fascitis 10 patient were male(62.5%) and 6 patients were female(37.5%).From the study the incidence of necrotizing fasciitis is most common in male in both diabetic and non diabetic patients

Sex	Diabetic		Non-Diabetic		Total
	No	%	No	%	
Female	15	44.1%	6	37.5%	21
Male	19	55.9%	10	62.5%	29
Total	34		16		50



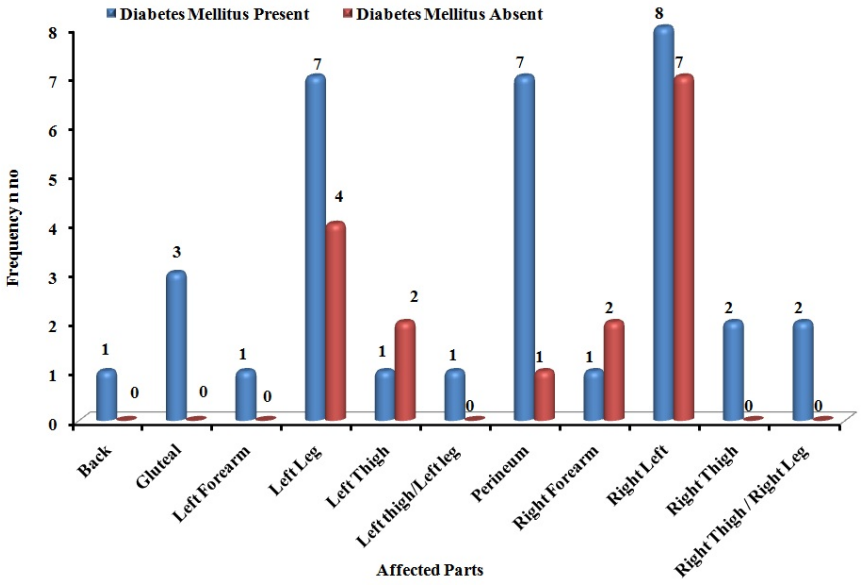
P – Value – 0.658 > 0.05 their is no statistically significant, there is no differencein sex group between Diabetes Present and absent – Chi square test – 0.196

SITE OF LESION

In this series of 34 diabetic patient with necrotizing fascitis, the lower limbs were the commonest site of involvement (41.1%), right leg (20.6%) being affected more commonly. The next most common site of involvement is the perineum (20.6%) .Where as in 16 non diadetic patient the commonest site of involvement is lower limbs (68.8%), right leg (43.8%) being affected more commonly. Perineum is involved in 6.3% only.

Affected part	Diabetic		Non-Diabetic		Total
	No	%	No	%	
Back	1	2.9%	0	0.0%	1
Gluteal	3	8.8%	0	0.0%	3
Left Forearm	1	2.9%	0	0.0%	1
Left Leg	7	20.6%	4	25.0%	11
Left Thigh	1	2.9%	2	12.5%	3
Left thigh/Left leg	1	2.9%	0	0.0%	1
Perineum	7	20.6%	1	6.3%	8

Affected part	Diabetic		Non-Diabetic		Total
	No	%	No	%	
Right Forearm	1	2.9%	2	12.5%	3
Right Leg	8	23.5%	7	43.8%	15
Right Thigh	2	5.9%	0	0.0%	2
Right Thigh / Right Leg	2	5.9%	0	0.0%	2
Total	34		16		50

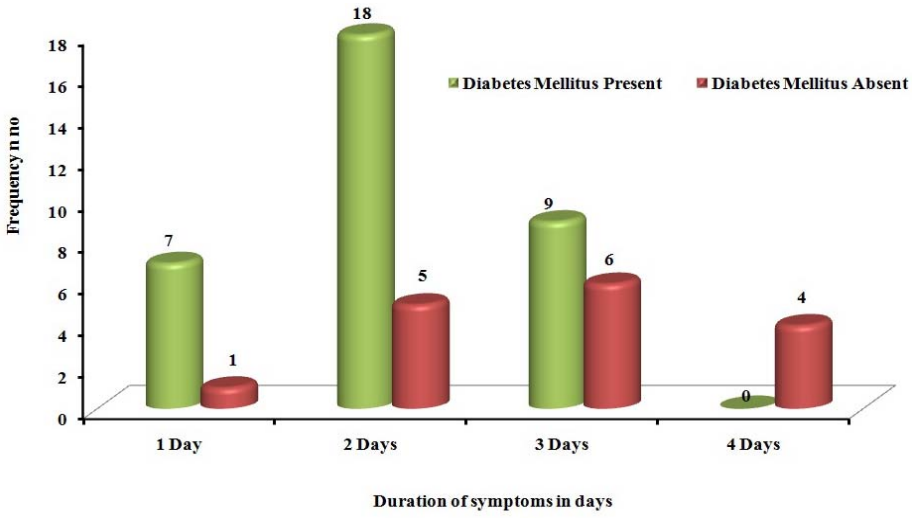


TIME BETWEEN ONSET AND HOSPITAL PRESENTATION

In this study of 34 diabetic patient with necrotizing fasciitis, 18 patients (52.9%) were presented to the hospital after a duration of 2 days from the time of onset of symptoms where as in 16 non diabetic patients with necrotizing fascitis, 9 patients (37.5%) were presented to the hospitals on third day. The average duration from the time of onset of symptoms to hospital presentation was 2.06 days in diabetic and 2.81 days in non diabetic patient.

Duration of symptoms	Diabetic		Non-Diabetic		Total
	No	%	No	%	
1 Day	7	20.6%	1	6.3%	8
2 Days	18	52.9%	5	31.3%	23
3 Days	9	26.5%	6	37.5%	15
4 Days	0	0.0%	4	25.0%	4

Duration of symptoms	Diabetic		Non-Diabetic		Total
	No	%	No	%	
Total	34		16		50
Mean ± SD	2.06 ± 0.69		2.81 ± 0.91		2.3 ± 0.84

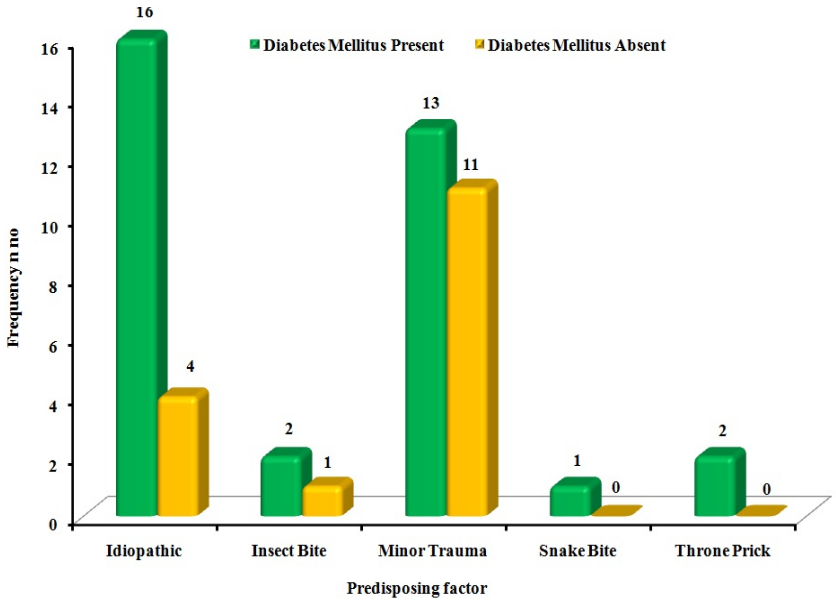


P – Value – 0.007< 0.05 is statistically significant, there is difference in duration of symptoms between Diabetes Present and absent – Student t test

PREDISPOSING FACTORS

In this study of 34 diabetic patient with necrotizing fasciitis predominant predisposing factor for developing necrotizing fasciitis was idiopathic(47.1%)followed by minor trauma(38.2%) were as in non diabetic patient minor trauma was the predominant predisposing factor(68.8%) followed by idiopathic (25%)

Predisposing factor	Diabetic		Non-Diabetic		Total
	No	%	No	%	
Idiopathic	16	47.1%	4	25.0%	20
Insect Bite	2	5.9%	1	6.3%	3
Minor Trauma	13	38.2%	11	68.8%	24
Snake Bite	1	2.9%	0	0.0%	1
Throne Prick	2	5.9%	0	0.0%	2
Total	34		16		50



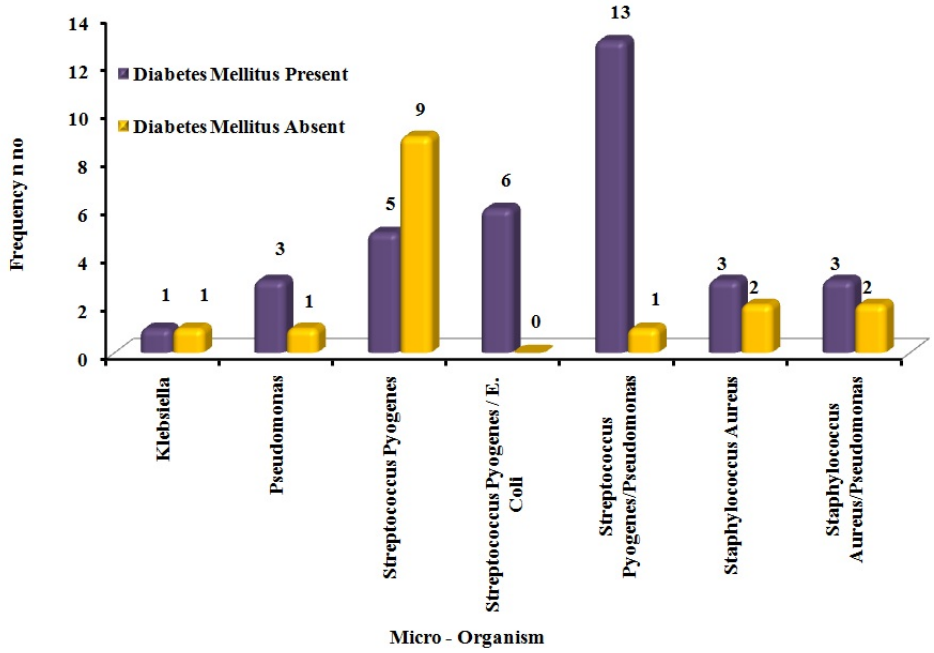
P – Value – 0.303> 0.05 their is no statistically significant, there is no difference in Predisposing Factor between Diabetes Present and absent – Chi square test – 4

ORGANISMS GROWN ON CULTURE

Most common organism found in diabetic patient with necrotizing fasciitis was poly microbial (64.6%) the commonest being streptococcal and pseudomonas (38.2%) followed by streptococcal pyogenes and Ecoli (17.6%),were as in non diabetic patient the commonest organisam is streptococcus pyogenes (56.3%).

Micro-Organism	Diabetes Mellitus		Non Diabetes Mellitus		Total
	Present		Absent		
	No	%	No	%	
Klebsiella	1	2.9%	1	6.3%	2
Pseudomonas	3	8.8%	1	6.3%	4
Streptococcus Pyogenes	5	14.7%	9	56.3%	14
Streptococcus Pyogenes / E. Coli	6	17.6%	0	0.0%	6
Streptococcus Pyogenes/ Pseudomonas	13	38.2%	1	6.3%	14

Micro-Organism	Diabetes Mellitus		Non Diabetes Mellitus		Total
	Present		Absent		
	No	%	No	%	
Staphylococcus Aureus	3	8.8%	2	12.5%	5
Staphylococcus Aureus/Pseudomonas	3	8.8%	2	12.5%	5
Total	34		16		50

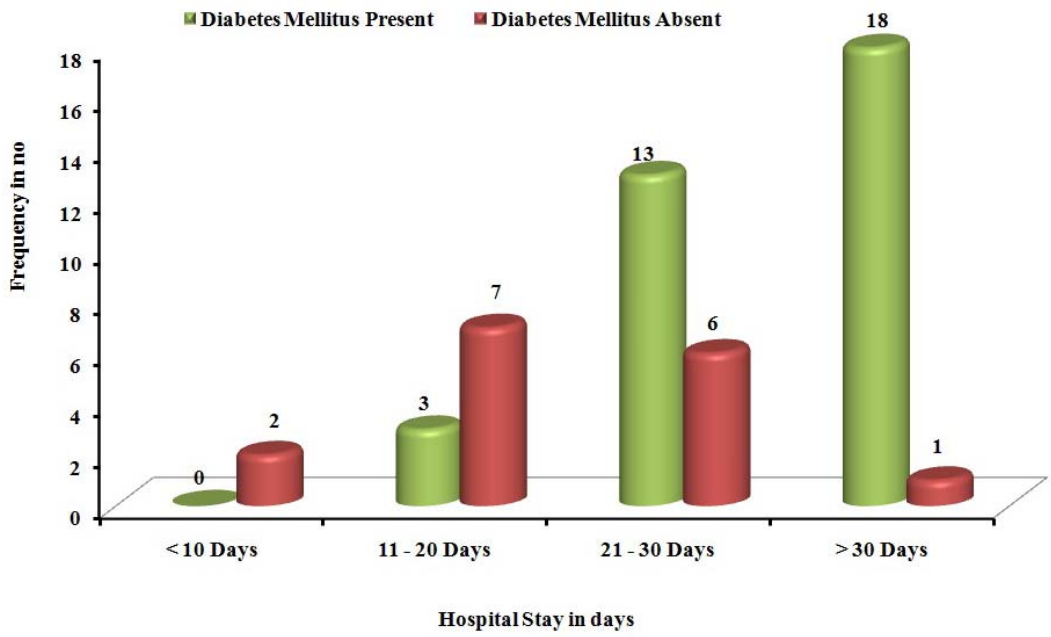


P – Value – 0.028 < 0.05 their is statistically significant, there is difference in Micro organism between Diabetes Present and absent – Chi square test – 14.2

DURATION OF HOSPITAL STAY

Average duration of hospital stay in diabetic patient is 30.9 days were as in non diabetic patient is 20.25 days

Hospital stay	Diabetic		Non-Diabetic		Total
	No	%	No	%	
< 10 Days	0	0.0%	2	12.5%	2
11 - 20 Days	3	8.8%	7	43.8%	10
21 - 30 Days	13	38.2%	6	37.5%	19
> 30 Days	18	52.9%	1	6.3%	19
Total	34		16		50
Mean±SD	30.9 ± 6.7		20.25 ± 6.61		27.52 ± 8.30

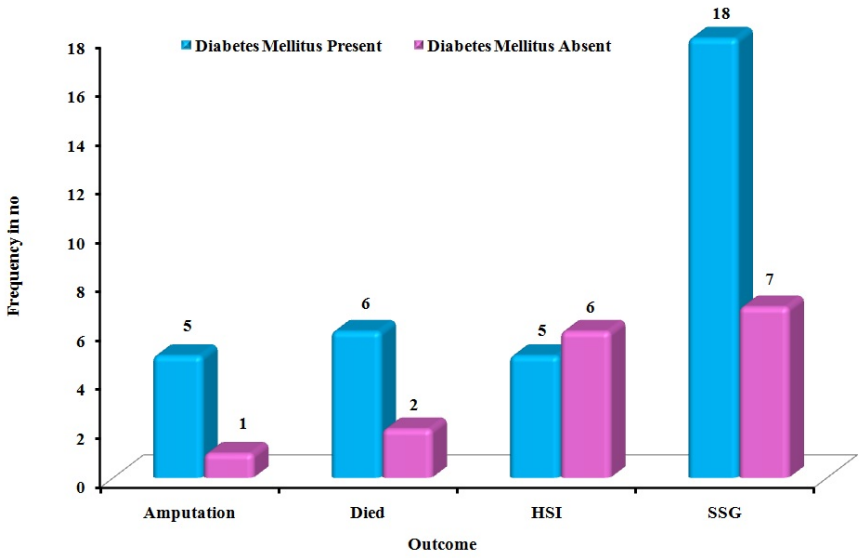


P – Value – 0.001 < 0.05 their is statistically significant, there is difference in Hospital stay between Diabetes Present and absent – Student t test

OUTCOME

Out of 34 diabetic patent with necrotizing fascitis amputation was done in 5 patient (14.7%) wound healed by secondary intention in 5 patients (14.7%), split skin graft was done in 18 patient (52.9%) and 6 patient died (17.6%). Out of 16 non diabetic patent with necrotizing fascitis amputation was done in 1 patient (6.3%) wound healed by secondary intention in 6 patients (37.5%) ,split skin graft was done in 7 patient (43.8%) and 2 patient died (12.5%)

Outcome	Diabetic		Non-Diabetic		Total
	No	%	No	%	
Amputation	5	14.7%	1	6.3%	6
Died	6	17.6%	2	12.5%	8
HIS	5	14.7%	6	37.5%	11
SSG	18	52.9%	7	43.8%	25
Total	34		16		50



P – Value – 0.310 > 0.05 their is no statistically significant, there is no difference in outcome between Diabetes Present and absent – Chi square test –3.58

SUMMARY

- Necrotizing fasciitis is a surgical emergency commonest risk factor is diabetes.
- This study was conducted on 50 randomly selected patient in one year period and the incidence among diabetes was 68% and among non-diabetic was 32%.
- Common age group affected in diabetic patient were 51-60yrs (41.2%) with a mean age of 56yrs whereas in non-diabetic patient it was 41-50yrs (50%) with a mean age of 45yrs.
- In diabetic patient 19 males (55.9%) and 15 females (44.1%) were affected. whereas in non diabetic patient 10 males (62.5%) and 6 females (37.5%) were affected.
- The average duration from the time of onset of symptoms to hospital presentation was 2.06 days in diabetic and 2.81 days in non diabetic patient.
- The commonest pre disposing factor in diabetic patient is idiopatic (47.1%) followed by minor trauma(38.2%)

whereas in non diabetic minor trauma(68.8%) is most common predisposing factor.

- Commonest site of involvement in diabetic is lower limb
- (41.1%) followed by perineum (20.6%) whereas in non-Diabetic lower limb is commonly involved(68.8%).
- In diabetic patient polymicrobial organism is common (64.6%) {(streptococcus pyogens and pseudomonas -(38.2%) followed by (streptococcal and E.Coli-17.6%)} whereas in non diabetic patient streptococcus pyogens (56.3%) is most common.
- Mean duration of hospital stay in diabetic patient is 30.9 days and in non diabetic patient is 20.5 days.
- In diabetic(52.9%) and non diabetic(43.85%) commonest mode of wound healing was by split skin graft
- Mortality rate in diabetic is (17.6%) and non diabetic is (12.5%).

CONCLUSION

- Incidence of necrotising fasciitis was more common in diabetic
- Necrotizing fasciitis in diabetic is common in the age groups of 51-60 years whereas in non diabetic patient it is 41-50 years.
- Males were affected more than female in both diabetic and non diabetic patient.
- In diabetic common precipitating factors is idiopathic whereas in non diabetic minor trauma is the common precipitating factor
- Lower extremity is the most common site of involvement in both diabetic and non diabetic patient.
- Poly microbial organism was the most common organism in diabetic whereas mono microbial was most common in non diabetic.

- Average hospital stay was more in diabetic patient.
- Mortality was more in diabetic

PROFORMA

Name:

Age: sex:

Duration of symptoms:

Any co-morbidities:

Diabetes mellitus - yes / no

Ischemic heart disease - yes / no

Personnel history:

Alcoholism - yes / no smoking - yes / no

General examination:

Local examination:

Investigation:

Urine sugar- urine ketones-

Blood sugar blood urea-

Serum creatinine:

Pus culture and sensitivity:

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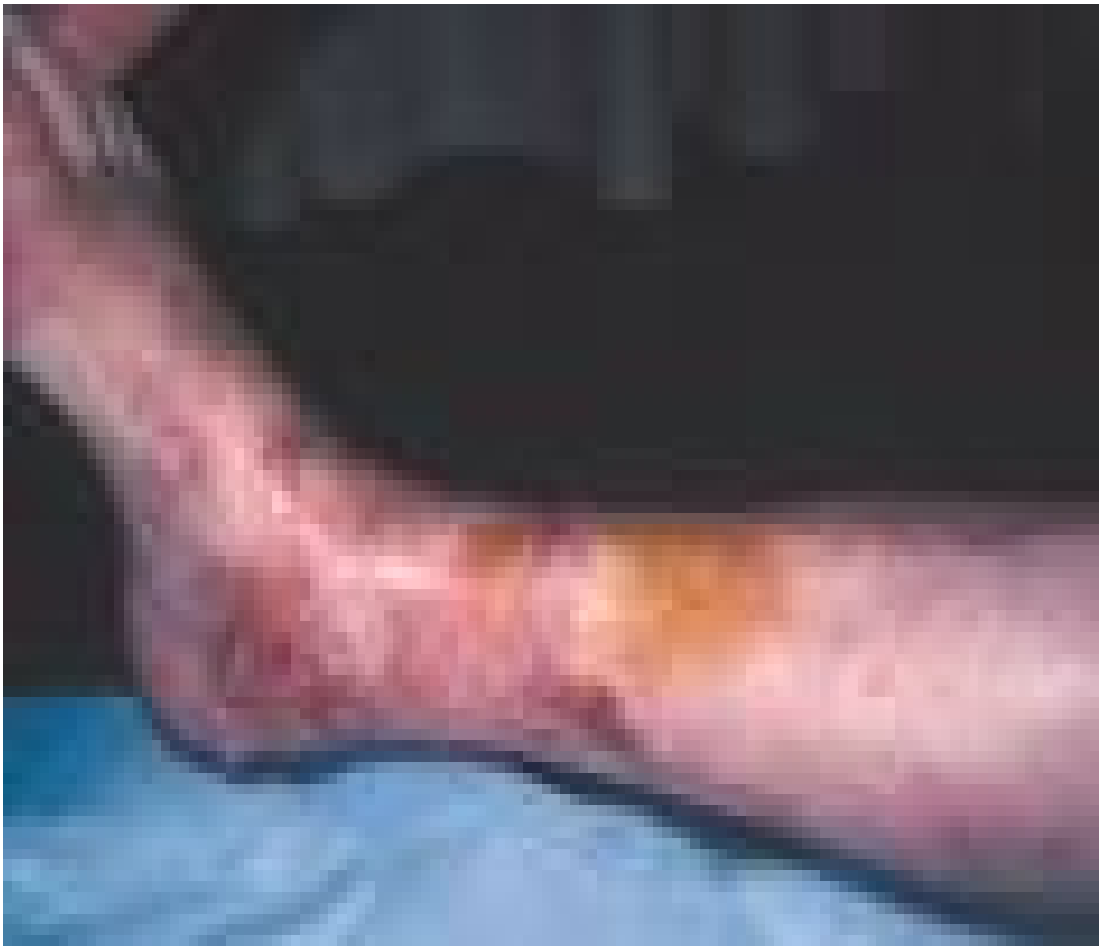
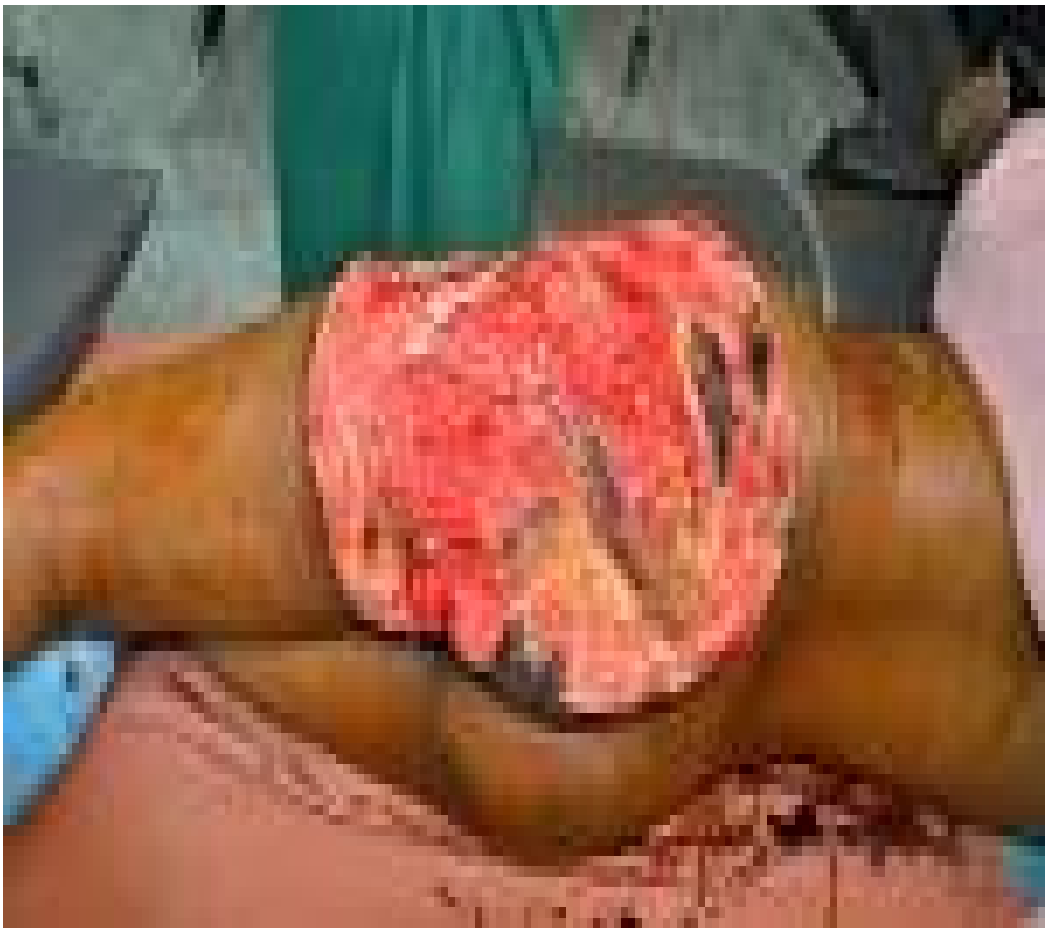
NECROTISING FASCIITIS IN LEFT LEG

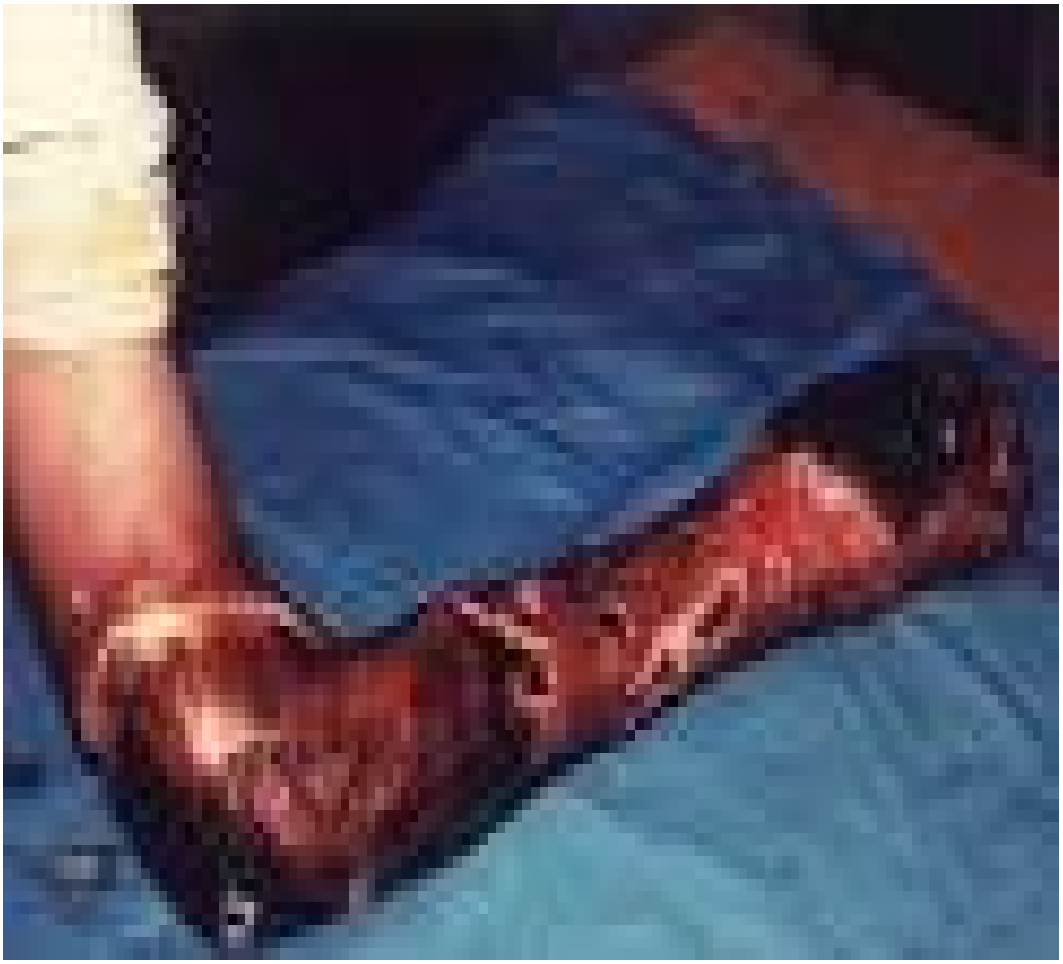
NECROTISING FASCIITIS IN RIGHT LEG



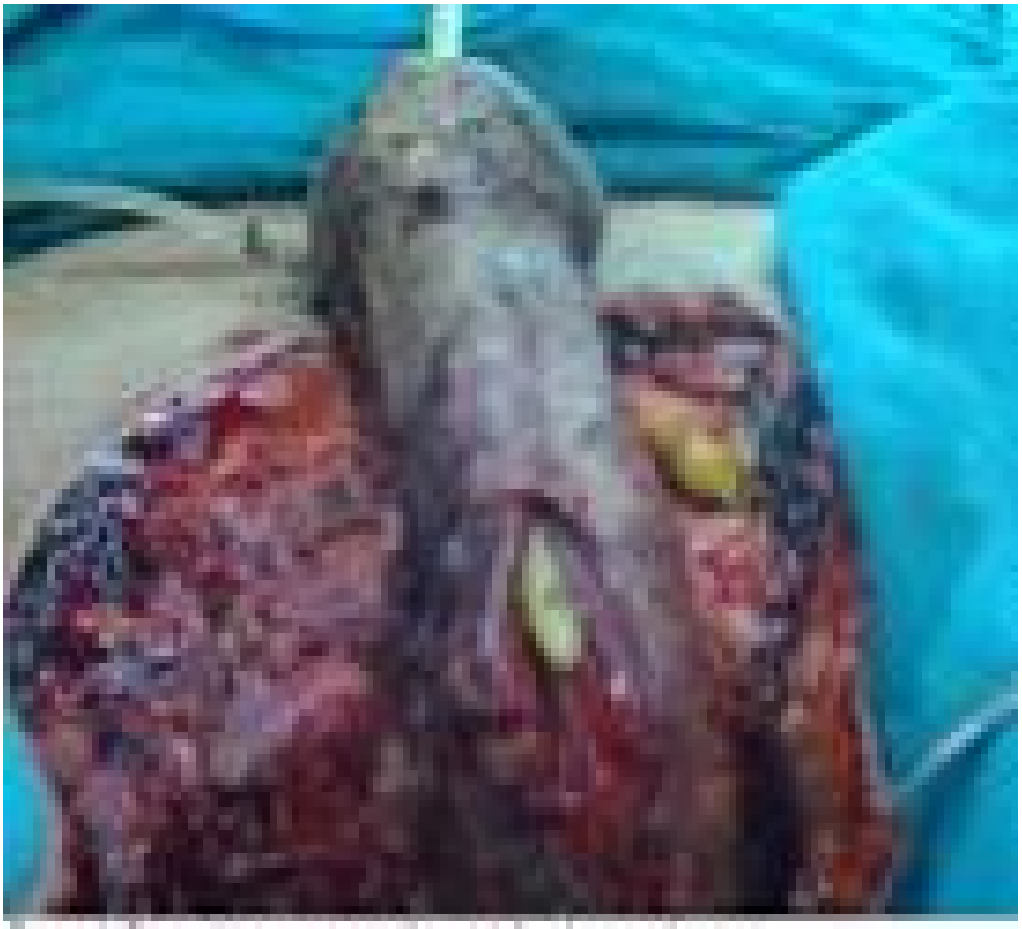
NECROTISING FASCIITIS IN GLUTEAL REGION AFTER DEBRIDEMENT



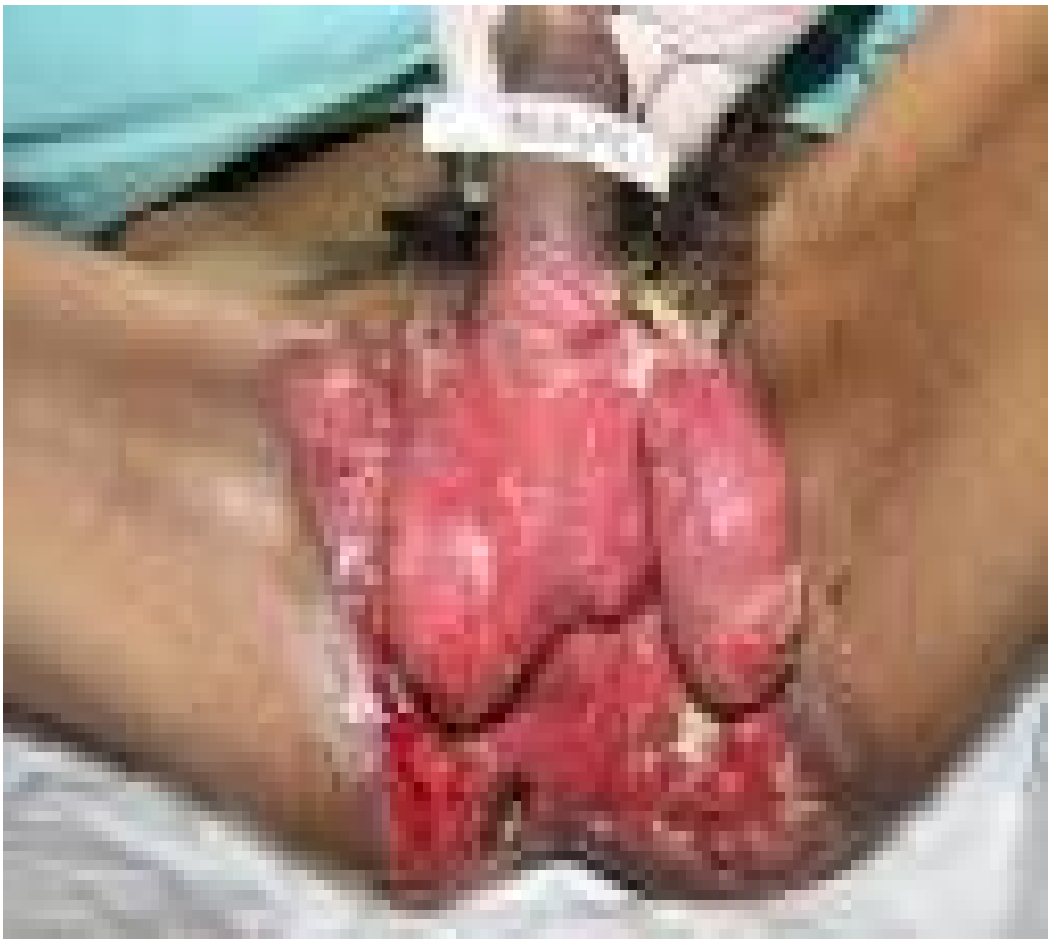
NECROTISING FASCIITIS IN FOREARM



NECROTISING FASCIITIS IN PERINIUM



FOURNIER'S GANGRENE AFTER DEBRIDEMENT



B/w With Page No: 1-64,66,68,70,72,74,76,78,80-83

B/w Without Page No: 84-90

Clr Without Page No: 91-96

Clr With Page No : 65,67,69,71,73,75,77,79

S.No	Name	Age	Sex	Affected Part	Duration of Symptoms	Diabetes Mellitus	Predisposing Factor	Micro-Organism	Hospital Stay	Outcome
1	Arumugam	50	M	RL	2	P	Idiopathic	SP/PS	34	HSI
2	Elumalai	54	M	RL	2	P	Idiopathic	SP/PS	32	HSI
3	Tamilmani	45	F	LL	4	A	Minor Trauma	SP	18	HSI
4	Kannagi	60	F	RL	2	P	Throne Prick	Klebsiella	22	SSG
5	Thangaraj	51	M	RL	3	A	Minor Trauma	SP	28	SSG
6	Sundaram	52	M	Perineum	3	P	Idiopathic	SP/PS	38	HSI
7	Manimegalai	57	F	LL	2	P	Idiopathic	SP/PS	21	HSI
8	Sundaresan	46	M	RT	2	P	Idiopathic	SP/EC	29	SSG
9	Manimaran	36	M	RF	4	A	Minor Trauma	Stap	31	SSG
10	Savithri	63	F	RL	2	P	Minor Trauma	Stap	28	SSG
11	Tajunisha	38	F	LT	2	A	Minor Trauma	Stap	27	SSG
12	Chidambaram	48	M	Gluteal	2	f	Idiopathic	PS	19	HSI
13	Marudavalli	55	F	Perineum	3	P	Minor Trauma	SP	30	SSG
14	Srinivasan	43	M	RL	1	P	Throne Prick	SP/PS	29	HSI
15	Selvam	41	M	RL	2	A	Insect Bite	Stap/PS	20	Amputation
16	Manickam	66	M	Perineum	2	P	Idiopathic	SP/EC	28	SSG
17	Ambika	61	F	RF	1	P	Idiopathic	SP/PS	31	SSG
18	Pakkiriammal	48	F	LL	3	A	Idiopathic	SP	18	SSG
19	Iqbal	38	M	LL	4	A	Idiopathic	SP	20	HSI

S.No	Name	Age	Sex	Affected Part	Duration of Symptoms	Diabetes Mellitus	Predisposing Factor	Micro-Organism	Hospital Stay	Outcome
20	Subramani	71	M	LF	1	P	Minor Trauma	SP/EC	23	SSG
21	Lakshmi	65	F	Gluteal	2	P	Idiopathic	SP/PS	36	SSG
22	Vasantha	60	F	RL	4	A	Minor Trauma	SP	20	SSG
23	Velusamy	53	M	RT	3	P	Minor Trauma	Stap/PS	24	SSG
24	Chelian	59	M	RL	1	P	Minor Trauma	SP	20	Amputation
25	Leelavathi	66	F	LL	3	P	Idiopathic	SP/PS	27	SSG
26	Venkatesan	55	M	RL	1	A	Minor Trauma	Klebsiella	26	SSG
27	Govidnan	68	M	LL	2	P	Minor Trauma	SP	18	Amputation
28	Selvi	45	F	LT	3	P	Idiopathic	SP/PS	30	SSG
29	Mayilsamy	47	M	Perineum	2	P	Minor Trauma	Stap/PS	4	Died
30	Muthuraman	42	M	LL	3	A	Idiopathic	SP	20	HSI
31	Karuppaiah	66	M	Perineum	2	P	Idiopathic	SP/PS	38	SSG
32	Tamilarasi	59	F	Back	2	P	Idiopathic	SP/EC	25	SSG
33	Munivel	46	M	RL	3	A	Minor Trauma	SP	22	HSI
34	Ganesan	60	M	LL	2	P	Snake Bite	SP	27	Amputation
35	Venugopal	62	M	Perineum	3	P	Idiopathic	SP/PS	28	SSG
36	Vembu	57	F	Gluteal	1	P	Minor Trauma	Sta	36	SSG
37	Vedavalli	62	F	RT/RL	1	P	Idiopathic	Sta/PS	6	Died
38	Joseph	46	M	RL	3	A	Minor Trauma	SP	18	HSI

S.No	Name	Age	Sex	Affected Part	Duration of Symptoms	Diabetes Mellitus	Predisposing Factor	Micro-Organism	Hospital Stay	Outcome
39	Neelakaudan	58	M	LT/LL	3	P	Idiopathic	SP/PS	27	Died
40	Balu	49	M	RT/RL	2	P	Minor Trauma	SP/EC	30	Amputation
41	Marimuthu	47	M	LT	2	A	Minor Trauma	Sta/PS	6	Died
42	Padmini	64	F	RL	2	P	Minor Trauma	Sta	35	SSG
43	Sundari	39	F	RF	3	A	Idiopathic	SP	21	HSI
44	Baskaran	57	M	Perineum	3	P	Minor Trauma	SP/PS	6	Died
45	Mallika	46	F	LL	3	P	Minor Trauma	SP	37	Amputation
46	Sivaraman	48	M	RL	1	P	Insect Bite	Pseu	4	Died
47	Manjula	57	F	LL	2	P	Minor Trauma	SP/EC	34	SGG
48	Rajeswari	57	F	LL	2	P	Insect Bite	PS	13	Died
49	Annamalai	40	M	PL	2	A	Minor Trauma	PS	22	SSG
50	Mangalam	48	F	RL	2	A	Minor Trauma	SP/PS	7	Died

P-Present, A-Absent, SP-Streptococcus Pyogenes, PS-Pseudomonas, EC-E.Coli, Stap- Staphylococcus Aureus, HIS- Healed by secondary intention, SSG- Spling Skin Graft, RL-Right Leg, LL-Left Leg, RF- Right Forearm, RT-Right Thigh, LT-Left Thigh