VALIDATION OF LRINEC SCORING SYSTEM FOR DIAGNOSIS OF NECROTIZING FASCIITIS IN PATIENTS PRESENTING WITH SOFT TISSUE INFECTIONS

DISSERTATION SUBMITTED FOR

MASTER OF SURGERY

BRANCH - I (GENERAL SURGERY)

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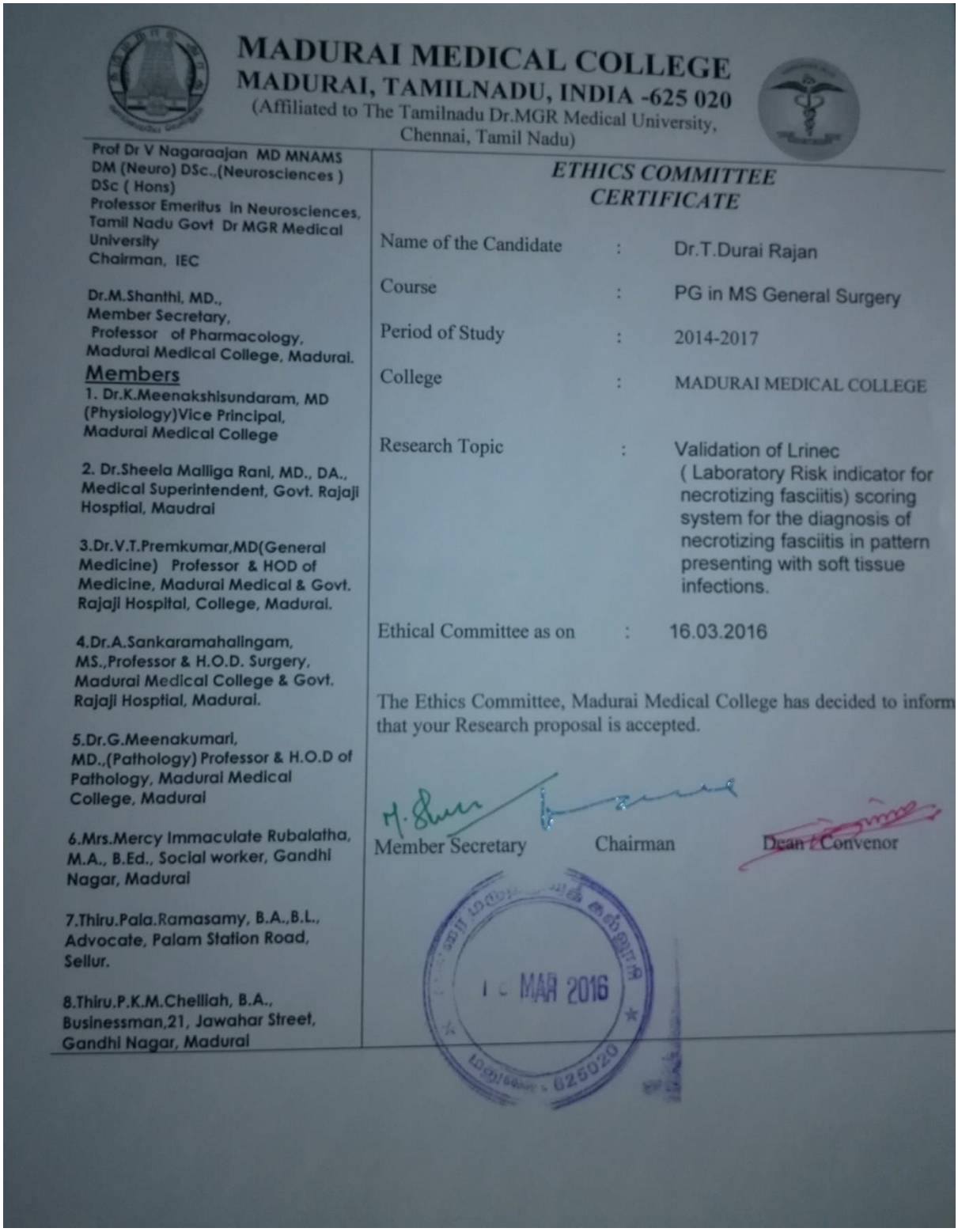
THE TAMILNADU

DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI



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College	:	MADURAI MEDICAL COLLEGE
Research Topic	:	Validation of Lrinec (Laboratory Risk indicator for necrotizing fasciitis) scoring system for the diagnosis of necrotizing fasciitis in pattern presenting with soft tissue infections.
Ethical Committee as on	:	16.03.2016

The Ethics Committee, Madurai Medical College has decided to inform that your Research proposal is accepted.

Member Secretary Chairman Dean / Convenor

16 MAR 2016

ETHICAL COMMITTEE CLEARANCE:

BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled **"VALIDATION OF LRINEC SCORING SYSTEM FOR DIAGNOSIS OF NECROTIZING FASCIITIS IN PATIENTS PRESENTING WITH SOFT TISSUE INFECTIONs "** submitted by Dr.T,DURAI RAJAN to the Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of M.S. Degree Branch I (General Surgery) is a bonafide research work was carried out by him under my direct supervision & guidance.

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CERTIFICATE BY THE DEAN

This is to certify that the dissertation entitled entitled **"VALIDATION OF LRINEC SCORING SYSTEM FOR DIAGNOSIS OF NECROTIZING FASCIITIS IN PATIENTS PRESENTING WITH SOFT TISSUE INFECTIONS "**is a bonafide research work done by **DR.T.DURAI RAJAN.,** Post Graduate Student, Department of General Surgery, MADURAI MEDICAL COLLEGE AND GOVERNMENT RAJAJI HOSPITAL, MADURAI, under the guidance and supervision of General Surgery, MADURAI MEDICAL COLLEGE AND GOVERNMENT RAJAJI HOSPITAL, MADURAI.

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DECLARATION

I, Dr.T.DURAI RAJAN declare that, I carried out this work on **"VALIDATION OF LRINEC SCORING SYSTEM FOR DIAGNOSIS OF NECROTIZING FASCIITIS IN PATIENTS PRESENTING WITH SOFT TISSUE INFECTIONS "** at the Department of General Surgery, Govt. Rajaji Hospital during the period of march 2016 to September 2016. I also declare that this bonafide work or a part of this work was not submitted by me or any others for any award, degree, diploma to any other University, Board either in India or abroad.

This is submitted to The TamilnaduDr.M.G.R.Medical University, Chennai in partial fulfillment of the rules and regulations for the M.S. degree examination in General Surgery.

Place: Madurai

Date: Dr.T.DURAI RAJAN

ACKNOWLEDGEMENT

At the outset, I wish to thank our Dean **DR.M.R.VAIRAMUTHURAJU ,M.D** .for permitting me to use the facilities of Madurai Medical College and government Rajaji Hospital to conduct this study.

I thank sincerely**, Prof.DR.MARUTHU PANDIAN M.S.,FICS.,FAIS** Professor and Head of the Department, Department of General Surgery for his valuable advice and cooperation in completing this study.

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My patients, who form the most integral part of the work, were always kind and cooperative. I pray to God give them courage and strength to endure their illness, hope all of them go into complete remission.

Place: Madurai.

Date

VALIDATION OF LRINEC(LABORATORY RISK INDICATORS OF NECROTISING FASCIITIS)SCORING SYSTEM FOR DIAGNOSIS OF NECROTIZING FASCIITIS IN PATIENTS PRESENTING WITH SOFT TISSUE INFECTIONS

- ***AIM:*** *To validate the LRINEC scoring system for the diagnosis of necrotising fasciitis among patients with soft tissue infections*

- ***SECONDARY OBJECTIVES :***

TO MEASURE

The relations of the variables used in the LRINEC scoring system with necrotizing soft tissue infections.

ELIGIBILITY CRITERIA:

Inclusion Criteria : All patients presenting to Govt.Rajaji Hospital, with symptoms suggestive of soft tissue infections during the study period.

Exclusion Criteria :

Patients below 15 yrs or above 75 yrs of age

Patient who has undergone surgical debridement for present episode of Soft tissue infection

Patient with boils or furuncles with no evidence of cellulitis

METHODOLOGY

Materials and methods:

STUDY AREA: GRH,MADURAI.

METHOD OF COLLECTION OF DATA:

- Patients presenting with symptoms suggestive of soft tissue infections will

  undergo clinical examinations and the below mentioned investigations.
- LRINEC scoring system will be applied to each of the study subjects.
- The confirmatory diagnosis for necrotising fasciitis will be done vide

  histopathology for all patients.

STUDY DESIGN : PROSPECTIVE OBSERVATIONAL STUDY

STUDY ENDPOINT:

PATIENT FOLLOW UP WAS UPTO 3 months

DATA ANALYSIS STATISTICAL ANALYSIS

LIST OF ABBREVIATIONS

1. **Crp - C reactive protein**
2. **CT - Computerisied tomography**
3. **CA-MRSA - Community acquired methicillin resistant staphylococcus aureus**
4. **Cr - Creatinine**
5. **FG - Fournier's gangrene**
6. **GAS - Group A Streptococcus**
7. **HBO - Hyperbaric oxygen**
8. **Hb - Hemoglobin**
9. **HPE - Histopathological examination**
10. **LRINEC - Laboratory risk indicator for necrotizing fasciitis**
11. **MRI - Magnetic resonance imaging**
12. **NSTI - Non soft tissue infection**
13. **NPV - Negative predictive value**
14. **PPV - Positive predictive value**
15. **PVD - Peripheral vascular disease**
16. **RBS - Random blood sugar**
17. **Sr.Na - Serum sodium**
18. **TC - Total count**

INTRODUCTION

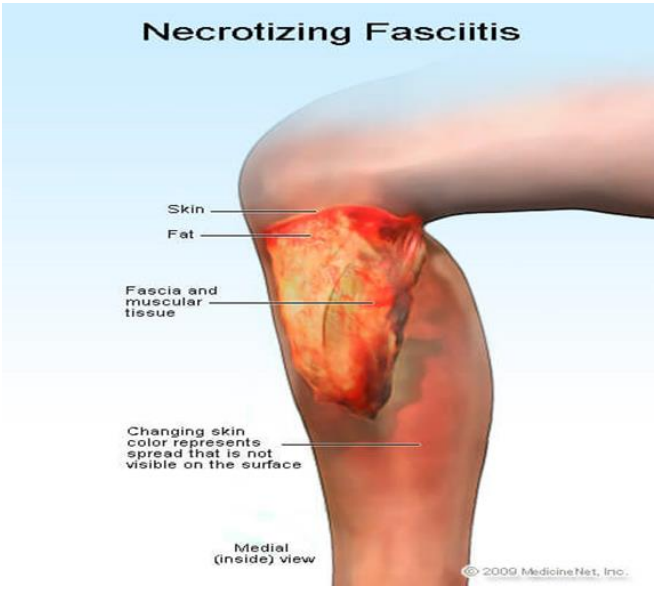
Necrotising fasciitis is one of a group of highly lethal infections that cause rapidly spreading necrosis of fascia and subcutaneous tissues, sometimes involving muscles and skin. They were previously known by such names as hospital gangrene, gas gangrene, and Fournier's gangrene and are now referred to by the generic term "necrotising soft tissue infections".

A mixed pattern of organisms is responsible: coliforms, staphylococci, Bacteroides spp., anaerobic streptococci and peptostreptococci have all been implicated, acting in synergy. Abdominal wall infections are known as Meleney's synergistic hospital gangrene and scrotal infection as Fournier's gangrene. Patients are almost always immunocompromised with conditions such as diabetes mellitus.

The wound initiating the infection may have been minor, but severely contaminated wounds are more likely to be the cause. Severe wound pain, signs of spreading inflammation with crepitus and smell are all signs of the infection spreading. Untreated, it will lead to widespread gangrene .

The sub dermal spread of gangrene is always much more extensive than appears from initial examination.

The key to successful treatment lies in early diagnosis and appropriate management. This condition is catastrophic if missed. Even with surgery, mortality is 20-40%. Delay in diagnosis increases mortality, and those who survive need more extensive surgery, reconstruction, and often amputation. With early diagnosis outcome is much improved and significant long term disability is reduced or prevented.



The differentiation of necrotizing fasciitis from other soft tissue infections is therefore critically important. Delayed recognition and diagnosis is one of the main reasons for the high mortality rate . Early operative debridement is a major

determinant of outcome in necrotizing fasciitis. However, early recognition is difficult clinically.

In this study we describe a novel, simple, and objective scoring system, the Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) score[8], based on routine laboratory investigations readily available at most centres, that can help distinguish necrotizing fasciitis from other soft tissue infections.

We have checked for the validation of LRINEC scoring system for the diagnosis of necrotizing fasciitis in patients with soft tissue infections and prospectively examined patients with soft tissue infections treated at GOVT RAJAJI HOSPITAL, MADURAI over the last 1 year . Certain conclusions have been arrived at which are presented in latter part of the dissertation.

REVIEW OF LITERATURE

HISTORIC OVERVIEW

Necrotising soft tissue infections have been recognised and reported for centuries, the earliest dating back to Hippocrates in the 5th century BC [9]. Such infections represent a large spectrum of clinical entities, ranging from mild pyodermas to life threatening necrotising fasciitis. Although these infections are most commonly caused by streptococcal and staphylococcal species, a multitude of other organisms have also been implicated.

Necrotising soft tissue infections were first described by Jones in 1871 and at the time were termed “hospital gangrene”. A variant of the disease has become known as Fournier’s Gangrene after necrotizing infection of the perineum was described by Jean Alfred Fournier in 1884 .

The term necrotising fasciitis was first used by Wilson in 1952 to describe the most consistent feature of the infection, necrosis of the fascia and subcutaneous tissue with relative sparing of the underlying muscle. It can progress rapidly to systemic toxicity and even death if not promptly diagnosed and treated. Once suspected, management should consist of immediate resuscitation, early surgical debridement, and administration of broad spectrum intravenous antibiotics.

However, still today, different terms are used to define and classify NSTIs, leading to confusion when referring to infections that have common pathophysiological and clinical characteristics and, most importantly, share a common management strategy. We encourage the use of the term “necrotizing soft-tissue infections” to encompass all of these necrotizing infections and advocate an approach to all of them that uses the same principles for diagnostic and treatment strategies.

Diagnosis is often delayed because of the paucity of symptoms and the unfamiliarity of the condition among clinicians. This will allow for earlier diagnosis and expedited treatment, which are essential for improving outcomes and decreasing mortality in patients with NSTI.

EPIDEMIOLOGY

The incidence of NSTI in the United States is estimated to be 500–1500 cases per year. A recent study that established the incidence of soft-tissue infection, using insurance databases from various states in the United States, determined the incidence of NSTI to be 0.04 cases per 1000 person-years.

Establishing the diagnosis of NSTI is probably the greatest challenge in managing these infections. Delay of diagnosis leads to delayed surgical debridement, which leads to higher mortality.

It is for this reason that familiarity with the clinical characteristics, diagnostic tools, and principles of management is important when treating patients with NSTI.

Conditions associated with necrotizing soft tissue infection include diabetes, drug use, obesity, immunosuppression, recent surgery, and traumatic wounds. The key to successful treatment lies in early diagnosis and appropriate management. This condition is catastrophic if missed. Even with surgery, mortality is 20-40%. Delay in diagnosis increases mortality, and those who survive need more extensive surgery, reconstruction, and often amputation. With early diagnosis outcome is much improved and significant long term disability is reduced or prevented.

There is no age or sex predilection for necrotizing fasciitis. The disease occurs more frequently in diabetics, alcoholics, immunosuppressed, IV drug users and patients with peripheral vascular disease. However necrotizing fasciitis occurs in young and healthy individuals.

Relevant anatomy

The integument or skin is the largest organ of the body, making up 16% of body weight, with a surface area of 1.8 m2.It has several functions the most important being to form a physical barrier to the environment, allowing and limiting the inward and outward passage of water, electrolytes and various substances while providing protection against micro-organisms, ultraviolet radiation, toxic agents and mechanical insults. There are three structural layers to the skin: the epidermis, the dermis and subcutis.

Hair, nails, sebaceous, sweat and apocrine glands are regarded as derivatives of skin . Skin is a dynamic organ in a constant state of change, as cells of the outer layers are continuously shed and replaced by inner cells moving up to the surface.

Although structurally consistent throughout the body, skin varies in thickness according to anatomical site and age of the individual.

Skin anatomy

The epidermis is the outer layer, serving as the physical and chemical barrier between the interior body and exterior environment; the dermis is the deeper

layer providing the structural support of the skin, below which is a loose connective tissue layer, the subcutis or hypodermis which is an important depot of fat (see Table 1.1).

Epidermis

The epidermis is stratiated squamous epithelium. The main cells of the epidermis are the keratinocytes, which synthesise the protein keratin. Protein bridges called desmosomes connect the keratinocytes, which are in a constant state of transition from the deeper layers to the superficial.

Skin layer Description

Epidermis The external layer mainly composed of layers of keratinocytes but also containing melanocytes, Langerhans cells and Merkel cells. Basement membrane The multilayered structure forming the dermoepidermal junction. Dermis The area of supportive connective tissue between the epidermis and the underlying subcutis: contains sweat glands, hair roots, nervous cells and fibres, blood and lymph vessels. Subcutis The layer of loose connective tissue and fat beneath the dermis.

Stratum basale The innermost layer of the epidermis which lies adjacent to the dermis comprises mainly dividing and non-dividing keratinocytes, which are attached to the basement membrane by hemidesmosomes. As keratinocytes divide and differentiate, they move from this deeper layer to the surface.

Making up a small proportion of the basal cell population is the pigment (melanin) producing melanocytes. These cells are characterised by dendritric processes, which stretch between relatively large numbers of neighbouring keratinocytes. Melanin accumulates in melanosomes that are transferred to the adjacent keratinocytes where they remain as granules. Melanin pigment provides protection against ultraviolet (UV) radiation; chronic exposure to light increases the ratio of melanocytes to keratinocytes, so more are found in facial skin compared to the lower back and a greater number on the outer arm compared to the inner arm. The number of melanocytes is the same in equivalent body sites in white and black skin but the distribution and rate of production of melanin is different. Intrinsic ageing diminishes the melanocyte population. Merkel cells are also found in the basal layer with large numbers in touch sensitive sites such as the fingertips and lips. They are closely associated with cutaneous nerves and seem to be involved in light touch sensation.

Stratum spinosum as basal cells reproduce and mature, they move towards the outer layer of skin, initially forming the stratum spinosum.

Intercellular bridges, the desmosomes, which appear as `prickles' at a microscopic level, connect the cells. Langerhans cells are dendritic, immunologically active cells derived from the bone marrow,

and are found on all epidermal surfaces but are mainly located in the middle of this layer. They play a significant role in immune reactions of the skin, acting as antigen-presenting cells.

Stratum granulosum continuing their transition to the surface the cells continue to flatten, lose their nuclei and their cytoplasm appears granular at this level.

Stratum corneum The final outcome of keratinocyte maturation is found in the stratum corneum, which is made up of layers of hexagonal-shaped, non-viable cornified cells known as corneocytes. In most areas of the skin,there are10±30 layers of stacked corneocytes with the palms and soles having the most. Each corneocyte is surrounded by a protein envelope and is filled with water-retaining keratin proteins. The cellular shape and orientation of the keratin proteins add strength to the stratum corneum. Surrounding the cells in the extracellular space are stacked layers of lipid bilayers (see Figure 1.3). The resulting structure provides the natural physical and water-retaining barrier of the skin. The corneocyte layer can absorb three times its weight in water but if its water content drops below 10% it no longer remains pliable and cracks. The movement of epidermal cells to this layer usually takes about 28 days and is known as the epidermal transit time.

Dermoepidermal junction/basement membrane

This is a complex structure composed of two layers. Abnormalities here result in the expression of rare skin diseases such as bullous pemphigoid and

epidermolysis bullosa. The structure is highly irregular, with dermal papillae from the papillary dermis projecting perpendicular to the skin surface. It is via diffusion at this junction that the epidermis obtains nutrients and disposes of waste. The dermoepidermal junction flattens during ageing which accounts in part for some of the visual signs of ageing.

Dermis

The dermis varies in thickness, ranging from 0.6 mm on the eyelids to 3 mm on the back, palms and soles. It is found below the epidermis and is composed of a tough, supportive cell matrix. Two layers comprise the dermis: a thin papillary layer a thicker reticular layer. The papillary dermis lies below and connects with the epidermis. It contains thin loosely arranged collagen fibres. Thicker bundles of collagen run parallel to the skin surface in the deeper reticular layer, which extends from the base of the papillary layer to the subcutis tissue. The dermis is made up of fibroblasts, which produce collagen, elastin and structural proteoglycans, together with immunocompetent mast cells and macrophages. Collagen fibres make up 70% of the dermis, giving it strength and toughness. Elastin maintains normal elasticity and flexibility while proteoglycans provide viscosity and hydration. Embedded within the fibrous tissue of the dermis are the dermal vasculature, lymphatics, nervous cells and fibres, sweat glands, hair roots and small quantities of striated muscle.

Subcutis

This is made up of loose connective tissue and fat, which can be up to 3 cm thick on the abdomen.

Blood and lymphatic vessels

The dermis receives a rich blood supply. A superficial artery plexus is formed at the papillary and reticular dermal boundary by branches of the subcutis artery.

Branches from this plexus form capillary loops in the papillae of the dermis, each with a single loop of capillary vessels, one arterial and one venous. The veins drain into mid-dermal and subcutaneous venous networks. Dilatation or constriction of these capillary loops plays a direct role in thermoregulation of the skin. Lymphatic drainage of the skin occurs through abundant lymphatic meshes that originate in the papillae and feed into larger lymphatic vessels that drain into regional lymph nodes.

Nerve supply

The skin has a rich innervation with the hands, face and genitalia having the highest density of nerves. All cutaneous nerves have their cell bodies in the

dorsal root ganglia and both myelinated and non-myelinated fibres are found. Free sensory nerve endings lie in the dermis where they detect pain, itch and temperature. Specialised corpuscular receptors also lie in the dermis allowing sensations of touch to be received by Meissner's corpuscles and pressure and vibration by Pacinian corpuscles. The autonomic nervous system supplies the motor innervation of the skin: adrenergic fibres innervate blood vessels, hair erector muscles and apocrine glands while cholinergic fibres innervate eccrine sweat glands. The endocrine system regulates the sebaceous glands, which are not innervated by autonomic fibres.

ETIOLOGY AND PREDISPOSING FACTORS

Necrotising fasciitis affects all age groups but is particularly rare in childhood. It is more common in patients with diabetes, chronic hepatitis, and malignancy (particularly leukaemia) and in people who inject drugs. Iatrogenic immunosuppression also increases the risk. Intra-abdominal malignancy or sepsis can lead to necrotising fasciitis of the abdominal wall.

Varicella infection is a recognised risk factor in children . Any puncture wound or surgical procedure can introduce infection, including such minor procedures

as acupuncture or intramuscular injection. However, about 25% of cases occur in patients without co morbidity or precedent trauma.

The aetiology of necrotising fasciitis is not fully understood, and in many cases no identifiable cause can be found. However, patients often have some prior history of trauma (which may even be trivial), such as an insect bite, scratch, or abrasion · The disease does not seem to have any predilection for age but occurs slightly more often in male patients .· Most patients who develop necrotising fasciitis have pre-existing conditions that render them susceptible to infection. Conditions that result in immunosuppression, such as advanced age, chronic renal failure, peripheral vascular disease, diabetes mellitus, and drug misuse seem to be risk factors .

Box 1: Predisposing factors for necrotising fasciitis

Comorbid conditions
Immunosuppression
Diabetes
Chronic disease
Drugs—for example, steroids
Malnutrition
Age > 60
Intravenous drug misuse
Peripheral vascular disease
Renal failure
Underlying malignancy
Obesity

Aetiological factors
Blunt or penetrating trauma
Soft tissue infections
Surgery
Intravenous drug use
Childbirth
Burns
Muscle injuries

Diabetes is a particularly important risk factor; several forms of necrotizing infection have been described more frequently among diabetics. These include nonclostridial anaerobic cellulitis, synergistic necrotizing cellulitis, and type I necrotizing fasciitis [1, 18]. These infections occur most frequently in the lower extremities. In addition, diabetic patients are also predisposed to developing necrotizing fasciitis in the head and neck region and the perineum.

There are two bacterial forms of necrotizing fasciitis: type I and type II.

●**Type I** necrotizing fasciitis is a mixed infection caused by aerobic and anaerobic bacteria. Risk factors include diabetes, peripheral vascular disease (PVD), immune compromised state, and recent surgery, including minor procedures such as circumcision in newborn infants. Patients with diabetes and/or PVD frequently have lower extremity involvement. Neonates usually have abdominal or perineal involvement.

●**Type II** necrotizing fasciitis due to group A *Streptococcus* (GAS) or other beta-haemolytic streptococci, either alone or in combination with other species, most commonly *S. aureus*. It can occur among healthy individuals with no past medical history, in any age group. Predisposing factors include a history of skin injury, such as laceration or burn, blunt trauma, recent surgery, childbirth, injection drug use, and Varicella infection (chickenpox) · In cases with no clear portal of entry, the pathogenesis of infection likely consists of haematogenous translocation of GAS from the throat (asymptomatic or symptomatic pharyngitis) to a site of blunt trauma or muscle strain [sa].

Idiopathic necrotizing fasciitis is likely to be due to S.pyogenes and occur in the extremities as opposed to necrotizing fasciitis from other known etiologies.

It is thought to result from either inoculation with undetected breaks in the epidermis or haematogenous spread from another source.

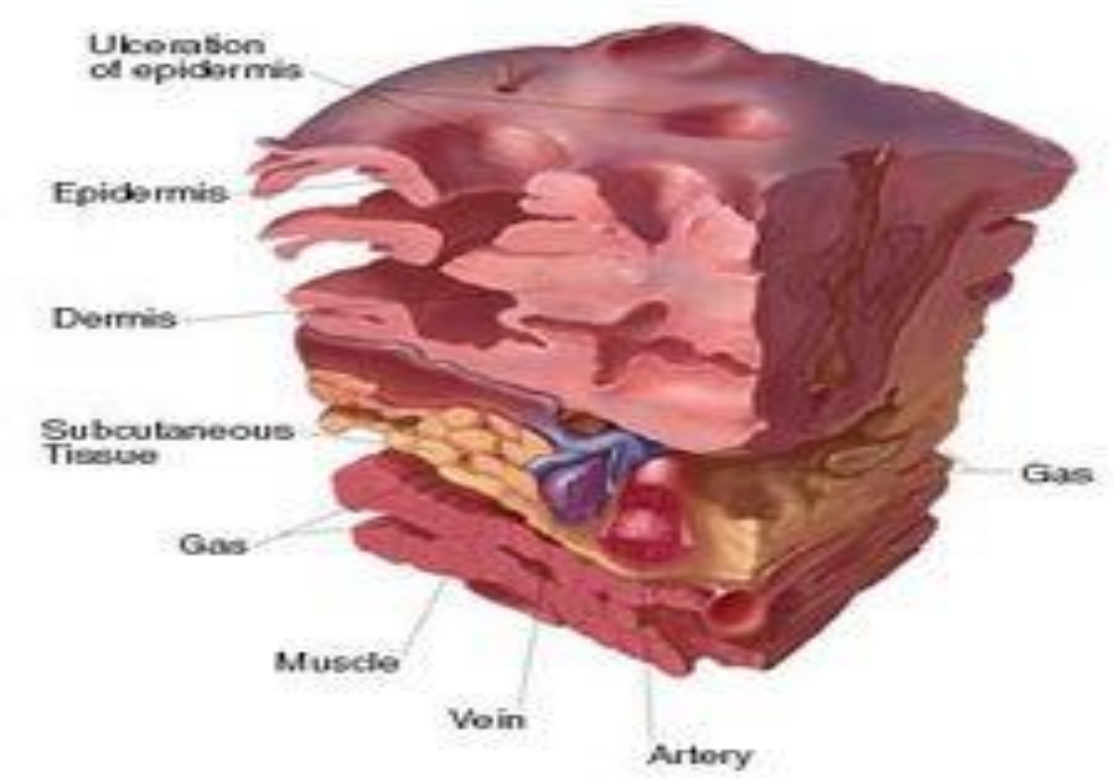


FIG- ANATOMY OF SKIN

PRESENTATION AND DIAGNOSIS

Necrotizing fasciitis is an infection of the deeper tissues that results in progressive destruction of the muscle fascia and overlying subcutaneous fat; muscle tissue is frequently spared because of its generous blood supply. Infection typically spreads along the muscle fascia due to its relatively poor blood supply; initially, the overlying tissue can appear unaffected. It is this feature that makes necrotizing fasciitis difficult to diagnose without surgical intervention.

Early recognition of necrotizing infection is critical; rapid progression to extensive destruction can occur, leading to systemic toxicity, limb loss, and/or death. Both clinical clues and diagnostic tools should be used in combination to help make an early diagnosis

Necrotising fasciitis is notoriously difficult to diagnose. The initial symptoms are non-specific up to the point when the patient rapidly deteriorates, and septicaemia develops, often accompanied by shock or confusion. However, this clinical course is often slower than might be expected. Fever or pain develops first, so the patient often presents initially to primary care or the emergency department. The pain may seem to be disproportionate to the clinical findings. Cellulitic skin changes may develop next. The presentation may mimic haematoma, bursitis, phlebitis, sciatica, cellulitis, septic arthritis, or deep venous

thrombosis. The classic textbook picture of haemorrhagic bullae, crepitus, and skin necrosis often does not occur until day 5 or late. The patient may seem systemically well until relatively late.

The patients who present the greatest diagnostic difficulty are those presenting with pain but without fever or systemic signs. Pain is caused by tissue necrosis, but the nerves can also be infarcted as perforating vessels to the tissues are occluded by thrombus during the necrotic process. This can result in exquisite pain and tenderness but also in sensory loss to the overlying skin. The area may be tender or tense. Pain is often very severe, preventing weight bearing or use of the limb but may be mild until late in the process. People who inject drugs often present without systemic signs . Even in patients with systemic signs, the severity of the skin infection is often not apparent initially. The skin may look normal, or there may be erythema suggestive of cellulitis. In true fasciitis there will be no ascending lymphangitis, but this may be present in other, more superficial necrotising soft tissue infections.

Necrotizing fasciitis is usually an acute process but rarely may follow a subacute progressive course. The affected area may be erythematous (without

sharp margins), swollen, warm, shiny, and exquisitely tender. The subcutaneous tissue may be firm and indurated such that the underlying muscle groups cannot be distinctly palpated. Lymphangitis and lymphadenitis are infrequent.

The process progresses rapidly over several days, with changes in skin colour from red-purple to patches of blue-gray. Within three to five days after onset, skin breakdown with bullae (containing thick pink or purple fluid) and frank cutaneous gangrene can be seen. By this time, the involved area is no longer tender but has become anaesthetic secondary to thrombosis of small blood vessels and destruction of superficial nerves in the subcutaneous tissue. The development of anaesthesia may precede the appearance of skin necrosis and provide a clue to the presence of necrotizing fasciitis [30].

Marked swelling and edema may produce a compartment syndrome with complicating myonecrosis requiring fasciotomy. Measurement of compartment pressure may aid the evaluation early in the course of infection when marked pain and swelling are present without concomitant skin changes that would indicate the diagnosis . Subcutaneous gas is often present in the polymicrobial form of necrotizing fasciitis, particularly in patients with diabetes.

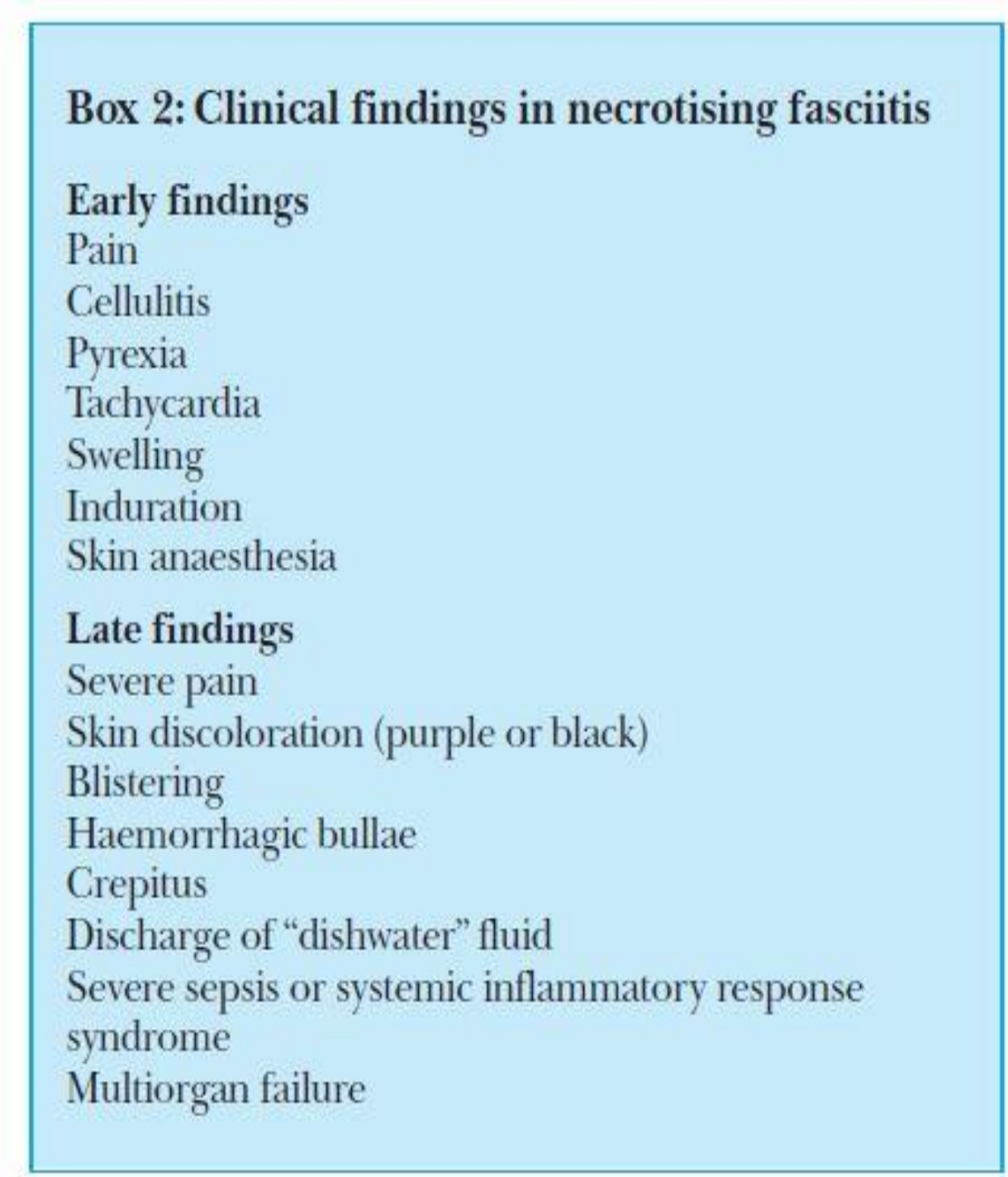
In advanced infection, fever, tachycardia, and systemic toxicity are generally observed, with temperature elevation in the range of 38.9° to 40.5°C (102° to 105°F). Other symptoms include malaise, myalgias, diarrhea, and anorexia. Hypotension may be present initially or develop with progressive infection.

In patients with fever, suspicion may be aroused by something being “not quite right” for a diagnosis of cellulitis.

The classic cyanotic and bullous skin changes may only appear late in the process;

however, the site of infection may appear unusual. The pain may seem too severe for cellulitis, despite relatively mild skin signs, or there may be overlying sensory loss.

The progression of the illness also suggests the diagnosis. The patient may seem relatively well initially, but will deteriorate despite treatment with antibiotics. Close observation is important for identifying those patients whose disease is not progressing as expected



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Certain diagnostic signs that we have come to associate with necrotising fasciitis are not always present. The findings of crepitus and soft tissue air on plain radiograph are seen in only 37% and 57% of patients, respectively.

However, great caution should be taken. Even though they are pathognomonic for the disease, their absence should not exclude the diagnosis.

Other findings that are common in necrotising fasciitis include a raised white cell count as well as raised concentrations of glucose, urea, and creatinine. Hypoalbuminaemia, acidosis, and an altered coagulation profile may also be present. No investigations are diagnostic, but blood test abnormalities such as a raised C reactive protein concentration occur relatively early, reflecting the systemic inflammatory response. The most accurate diagnostic scoring system to date is the Laboratory Risk Indicator for Necrotizing Fasciitis · A score of ≥6 was 93% sensitive and 92% specific for necrotising fasciitis in a Singaporean population but achieved only 74% sensitivity and 81% specificity in a UK validation study (H Y Sultan et al, unpublished UK data, 2011). Score demonstrates the relative importance of certain laboratory tests. Hyponatraemia in the presence of sepsis and clinical signs of soft tissue infection should be considered highly suspicious for a necrotising soft tissue infection.

Two studies have been reported to help discriminate between necrotizing and non-necrotizing infections. In the first, Wall et al performed a retrospective study and compared a set of admission variables of patients with NSTI and patients with non-necrotizing soft-tissue infection. After univariate and multivariate analysis, they found that having a WBC count 115,400 cells/ mm^3 or a serum sodium level 135 mmol/ L was associated with NSTI and that a

combination of both increased the likelihood of NSTI. Wall and colleagues' method proved to be a very sensitive tool, with a negative predictive value (NPV) of 99%, but not very specific, with a positive predictive value (PPV) of only 26%. In conclusion, Wall and colleagues' method is a good tool to rule out NSTI, but it is not as good for confirming its presence.

More recently, Wong et al. [8] created a score (laboratory risk indicator for necrotizing fasciitis score) to discriminate between NSTI and non-necrotizing soft-tissue infection. They compared a set of laboratory variables between patients with and without NSTI and identified 6 independent variables associated with NSTI. Each variable, if present, gives a specific number of points toward the final score. The total score had a range of 0–13, and patients were categorized according to the risk of NSTI among 3 groups. After internal validation, Wong and colleagues showed that, for intermediate and high-risk patients , the score had a PPV of 92% and a NPV of 96%. This constitutes a great tool for both confirming and discarding NSTI and has the advantage that it is based on laboratory variables that are widely available across different institutions. Wong and colleagues' method is useful, however, only in the context of a diagnosed or strongly suspected severe soft-tissue infection.

Table 1. Six different variables included in the laboratory risk indicator for necrotizing fasciitis (LRINEC) score to help discriminate between necrotizing and nonnecrotizing soft-tissue infections.

Value	LRINEC score, points
C-reactive protein, mg/L	
<150	0
>150	4
WBC count, cells/mm³	
<15	0
15–25	1
>25	2
Hemoglobin level, g/dL	
>13.5	0
11–13.5	1
<11	2
Sodium level, mmol/L	
≥135	0
<135	2
Creatinine level, mg/dL	
≤1.6	0
>1.6	2
Glucose level, mg/dL	
≤180	0
>180	1

Computed tomography [32] can show fascial swelling, inflammation, and sometimes soft tissue gas and is sensitive (100% in one small series) but less specific20; magnetic resonance [33] imaging is also sensitive21 but often not feasible or available. Ultrasonography [34] can be diagnostic but requires a highly skilled operator. Although plain radiography is not the imaging of choice when the diagnosis of necrotising fasciitis is suspected because sensitivity and specificity are low, it may show subcutaneous gas.

There have been studies with ultrasonography, CT, and MRI, which try to identify specific findings present in patients with NSTI [34-39]. Most of these findings have shown that increased thickness of the fascial layer with or without enhancement can be associated with NSTI. The primary limitation of these studies is that they tend to compare the involved site (usually a limb) with the contralateral or uninvolved limb, rather than comparing it with a nonnecrotizing soft-tissue infection. The studies show similar results as clinical findings: high sensitivity but low specificity. Additional studies with better methodology need to be performed; however, the use of ultrasonography, CT, and MRI can be helpful for patients with other sources of infection and for those for whom additional anatomic information may be valuable.

The mainstay for investigation and treatment remains surgical exploration. The decision to explore the soft tissues should be made early. An incision over the site of maximal skin change is needed to assess the underlying tissues. Healthy subcutaneous fat and fascia indicates that further resection is not needed, and the morbidity to the patient is limited to a short scar.

However, if exploration shows necrotic fascia, fat, or the “dishwater” appearance of liquefied necrotic tissue, resection can be done until healthy tissues are reached. This can be facilitated by the “finger sweep test” (necrotic fascia loses its adherence to surrounding tissues and the plane opens abnormally easily until the limit of the disease is reached). Where doubt over the

appearances persists, send specimens for histology to look for evidence of necrosis and microbiology for urgent Gram staining.

Differential diagnosis of necrotizing myositis and fasciitis

Clinical finding	Type I*	Type II*	Gas gangrene	Pyomyositis	Myositis viral/parasitic
Fever	++	++++	+++	++	++
Diffuse pain	+	+	+	+	++++•
Local pain	++	++++Δ	++++	++	++
Systemic toxicity	++	++++	++++	+	+
Gas in tissue	++	-	++++	-	-
Obvious portal of entry	++++	±◇	++++§	-	-
Diabetes mellitus	++++	±	-	-	-

* Type I and type II refer to the forms of necrotizing fasciitis; spontaneous gangrenous myositis is type II.
• Pain with influenza consists of diffuse myalgia; pleurodynia may be associated with severe, localized pain (eg, devil's grip); pain with trichinosis may be severe and localized.
Δ Severe pain is associated with necrotizing fasciitis due to group A streptococcal infection; the pain may not be severe in type I necrotizing fasciitis because it is commonly associated with diabetes with neuropathy.
◇ 50 percent of patients with necrotizing fasciitis due to group A streptococcal infection do not have an obvious portal of entry.
§ Gas gangrene associated with trauma may be caused by *Clostridium perfringens*, *C. septicum*, or *C. histolyticum*, which always have an obvious portal of entry; in comparison, spontaneous gas gangrene caused by *C. septicum* usually does not have an obvious portal of entry (organisms lodge in tissue as a result of bacteremia originating from a bowel portal of entry).

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MACROSCOPIC AND MICROSCOPIC TOOLS

Examination of a frozen section biopsy specimen from the compromised site that includes deep fascia and possibly muscle has been recommended as a means to achieve earlier diagnosis of NSTI in patients. Two studies evaluating this method also showed decreased mortality with historical comparisons, although this is probably related to the fact that an earlier diagnosis can be accomplished if clinicians are suspicious enough to perform the biopsy [40, 41].

In our practice, we prefer to explore the compromised area during an operation, rather than examine a frozen biopsy specimen. We have found that frozen biopsy is not very practical, because it requires availability and experience from the pathologists, and we are usually able to explore the site and identify macroscopic findings consistent with NSTI during an operation. These findings include gray necrotic tissue, lack of bleeding, thrombosed vessels, “dishwater” pus, non-contracting muscle, and a positive “finger test” result, which is characterized by lack of resistance to finger dissection in normally adherent tissues. Once NSTI is confirmed, the incision is extended, and additional debridement is performed. It is our principle and recommendation to perform exploration of the involved area through an operation whenever there is doubt and likelihood for NSTI.

MICROBIOLOGY

There are two bacterial forms of necrotizing fasciitis: type I and type II.

Type I

Type I necrotizing fasciitis is a mixed infection caused by aerobic and anaerobic bacteria. In type I infection, at least one anaerobic species (most commonly Bacteroides, Clostridium, or Peptostreptococcus) is isolated in combination with one or more facultative anaerobic streptococci (other than group A) and members of the Enterobacteriaceae (e.g., E. coli, Enterobacter, Klebsiella, Proteus) .An obligate aerobe, such as P. aeruginosa, is only rarely a component of such a mixed infection.

Type I necrotizing fasciitis can also occur in the head and neck region or in the perineum:

●Cervical necrotizing fasciitis of the neck can result from a breach in oropharynx mucous membrane integrity following surgery or instrumentation or in the setting of odontogenic infection. Fasciitis spread to the face, lower neck, and mediastinum. Factors which contributed to mediastinal involvement included prior corticosteroid use, infection by gas-producing microbes, and a pharyngeal focus of infection. Cervical (head and neck) necrotizing fasciitis is

usually a mixed (type I) infection. However, group A Streptococcus (S. pyogenes) can also cause monomicrobial necrotizing fasciitis. Necrotizing fasciitis of the head and neck is usually caused by mouth anaerobes, such as Fusobacteria, anaerobic streptococci, Bacteroides and spirochetes.

●Necrotizing infection of the male perineum, known as Fournier's gangrene, can result from a breach in the integrity of the gastrointestinal or urethral mucosa. Infection can occur in all age groups but is most common in older men. Fournier's gangrene is caused by facultative organisms (*E. coli*, *Klebsiella*, enterococci), along with anaerobes (*Bacteroides*, *Fusobacterium*, *Clostridium*, anaerobic or microaerophilic streptococci).

●In the neonate, most cases of necrotizing fasciitis are attributable to infections in association with omphalitis, balanitis, mammitis, or fetal monitoring; omphalitis is the most common predisposing condition [42, 43].

Synergistic necrotizing cellulitis is a variant of necrotizing fasciitis type I that involves the skin, muscle, fat, and fascia. It is usually occurs on the legs or perineum; diabetes is a known risk factor.

Type II

Type II necrotizing fasciitis is generally monomicrobial. It is typically caused by group A *Streptococcus (GAS)* or other beta-haemolytic streptococci either

alone or in combination with other pathogens, most commonly *S. aureus*; it has also been referred to as "streptococcal gangrene"[44].

●In type II, necrotizing fasciitis is generally mono-microbic, most commonly caused by group A *Streptococcus* (also known as haemolytic streptococcal gangrene). In communities with relatively high prevalence of community-acquired methicillin-resistant *Staphylococcus aureus* (CA-MRSA) infection, this organism is also a potential cause of monomicrobial necrotizing infection [45].

An important virulence determinant of GAS, M protein, is a filamentous protein anchored to the cell membrane. M protein has anti-phagocytic properties. Many M types of GAS have been associated with necrotizing fasciitis; types 1 and 3 are most common [46, 47]. These strains can produce one or more of the pyrogenic exotoxins A, B, or C [48, 49]. Group A streptococci may localize to the exact site of muscle injury due to increased surface expression of vimentin, which specifically binds the microbe. In an in vitro model, investigators demonstrated that injured skeletal muscle cells in tissue culture increased adherence of GAS two fold due to specific binding of GAS by vimentin on the surface of these cells [50].

Pyrogenic exotoxins lead to cytokine production, which may explain some of the clinical findings of necrotizing fasciitis. The exotoxins bind to the MHC class II portion of antigen presenting cells, such as macrophages. This complex

can then bind to a specific V beta segment of the T cell receptor in the absence of classical antigen processing by the macrophage [51]. Thus, pyrogenic exotoxins are super antigens and cause rapid proliferation of T cells bearing specific V beta repertoires. Such stimulation of the host's immune cells is associated with production of both monokines (tumour necrosis factor [TNF]-alpha, interleukin [IL]-1, and IL-6) and lymphokines (IL-2 and tumour necrosis factor-beta). Expression of these cytokines in vivo probably contributes to shock, tissue destruction, and organ failure [52, 53].

TREATMENT

Early referral to a surgeon and an early decision to explore and debride is the cornerstone of treatment. In established sepsis, debridement does not bring about a rapid change in the condition of patients. However, over the following hours they tend to stabilise but then often spend several days needing invasive support in intensive care and have an overall hospital stay averaging 33 days [54]. Subsequently, the patient will be referred to the nearest plastic surgery or burn centre for ongoing wound care and reconstruction.

Adjuvant measures include systemic support in an intensive care setting and antimicrobial treatment. Broad spectrum antibiotics such as a benzyl penicillin and flucloxacillin are used. Clindamycin has an additional role owing to its bacteriostatic mechanism. It inhibits the production of the streptococcal superantigen, which greatly contributes to septic shock [55].

Having established the diagnosis, treatment should be instituted immediately. The patient should be resuscitated according to his or her clinical state and may even require intensive care support. Intravenous antibiotics should be started at the same time and be changed according to sensitivities. Patients should be taken to theatre without delay and have aggressive surgical debridement. Further surgical exploration 24-48 hours later is mandatory to ensure that the infectious process has not extended. Repeated debridements may be necessary (as dictated by the state of the wound) until the infection has been controlled adequately.

The goal of operative management is to perform aggressive debridement of all necrotic tissue until healthy, viable (bleeding) tissue is reached. Tissue obtained in the operating room should be sent for Gram stain and culture. Subsequently, the wound is covered with a sterile dressing, re-evaluated in the operating room approximately 24 hours later, and aggressively debrided again if necrotic tissue is present[56]. The wound is closed after all necrotic tissue is completely debrided.

Antibiotic therapy

The optimal approach to empiric antibiotic therapy for necrotizing infection is uncertain; data are limited since most clinical trials exclude such patients. In general, empiric treatment of necrotizing infection should consist of broad-spectrum antimicrobial therapy, including activity against gram-positive, gram-

negative, and anaerobic organisms; special consideration for group A *Streptococcus* (GAS) and *Clostridium* species should be taken.

Acceptable regimens include administration of:

●A carbapenem or beta-lactam-beta-lactamase inhibitor, **plus**

●Clindamycin (dosed at 600 to 900 mg intravenously every eight hours in adults or 40 mg/kg per day divided every eight hours in neonates and children) for its antitoxin effects against toxin-elaborating strains of streptococci and staphylococci), **plus**

●An agent with activity against methicillin-resistant *S. aureus* (MRSA; such as vancomycin, daptomycin, or linezolid). In neonates and children, vancomycin (15 mg/kg/dose every six to eight hours) is the usual empiric antibiotic for MRSA; the six-hour dosing interval is employed for sicker children.

Options for carbapenems include imipenem, meropenem, or ertapenem. Meropenem (20 mg/kg per dose every eight hours) is the appropriate carbapenem for use in neonates with a postnatal age >7 days and children. Options for beta-lactam-beta-lactamase inhibitors include piperacillin-tazobactam, ampicillin-sulbactam, or ticarcillin-clavulanate. Patients with hypersensitivity to these agents may be treated either with an aminoglycoside or a fluoroquinolone, plus metronidazole. Antibiotic treatment should be tailored

to Gram stain, culture, and sensitivity results when available. In the setting of known group A streptococcal or other beta-haemolytic streptococcal infection, treatment may be narrowed to the combination of penicillin (4 million units intravenously every four hours in adults >60 kg in weight and with normal renal function or 300,000 units/kg per day divided every six hours in children) and clindamycin (600 to 900 mg intravenously every eight hours in adults or 40 mg/kg per day divided every eight hours in neonates and children)[21]. Therapy against MRSA may be discontinued after methicillin-resistant staphylococcal infection has been excluded.

The optimal duration of antibiotic treatment has not been defined in clinical trials. Antibiotics should be continued until no further debridements are needed and the patient's hemodynamic status has normalized; this duration must be tailored to individual patient circumstances.

History has shown that when treatment is only based on antimicrobial therapy and support, mortality approaches 100%. It is clear that early and complete debridement is essential for the treatment of NSTI. Concomitantly, appropriate broad-spectrum antibiotic coverage, combined with adequate organ support and close monitoring, helps patients during the acute phase of the disease, but it is only the complete debridement of infected tissue that controls the infection and allows for future recovery.

Intravenous immune globulin has also been used in the treatment of NSTI, particularly if the NSTI is associated with group A streptococcal infection. These studies are also controversial and difficult to compare, given the small number of patients and the different methodologies used. According to the Canadian experience, it seems reasonable to use intravenous immune globulin in patients with group A streptococcal infection who have developed streptococcal toxic shock syndrome and in those with a high mortality risk (advanced age, hypotension, and bacteraemia)[63].

Reconstructive surgery should be considered only once the patient has been stabilised and the infection fully eradicated.

Sterile dressings may be used to cover the wound in the interim.

Wound coverage can be achieved by either split thickness skin grafting or tissue transfer.

Vacuum assisted closure (VAC) therapy may be employed to promote healing and help close wounds, and this has helped in reducing the size of larger defects that would have been difficult to deal with simply on their own.

Other forms of surgery, such as amputation, may be necessary for necrotising infections of the extremities, and a defunctioning colostomy should be considered if wounds are regularly contaminated with faeces.

CONCLUSION

NSTIs are relatively infrequent but highly lethal infections.

They encompass a wide variety of soft-tissue infections associated with necrosis that shares the same diagnostic and treatment principles.

Establishing the diagnosis of NSTI is one of the biggest challenges in treating patients with NSTI.

Accuracy increases with familiarity of clinical findings and knowledge of laboratory, imaging, and macroscopic and microscopic findings, all combined with a high index of suspicion.

Surgical debridement is the primary means of treating NSTIs, and antimicrobial therapy and physiologic monitoring and support constitute adjuvant therapies.

LRINEC Score that identify high-risk patients serve to guide novel therapeutic strategies and to identify patients for future trials.

Fournier gangrene

Relevant anatomy

The corpora, urethra, testes, and cord structures are usually not involved in Fournier's gangrene, while the superficial and deep fascia and the skin are destroyed.

The complex anatomy of the male external genitalia influences the initiation and progression of Fournier's gangrene.

This infectious process involves the superficial and deep fascial planes of the genitalia.

As the microorganisms responsible for the infection multiply, infection spreads along the anatomical fascial planes, often sparing the deep muscular structures and, to variable degrees, the overlying skin.

The knowledge of the peculiar anatomy of the male lower urinary tract and external genitalia is critical for the clinician treating a man with Fournier's gangrene.

Colles' fascia completely envelops the scrotum and penis, continuiting cephalad to the level of the clavicles.

In the inguinal region, this fascial layer is known as Scarpa's fascia. Understanding the tendency of necrotizing fasciitis to spread along fascial planes and the fascial anatomy, one can see how a process that initiates in the perineum can spread to the abdominal wall, the flank, and even the chest wall.

Skin and superficial fascia

Because Fournier's gangrene is predominantly an infectious process of the superficial and deep fascial planes, appreciating the anatomic relationship of the skin and subcutaneous structures of the perineum and abdominal wall is vital.

The skin cephalad to the inguinal ligament is backed by Camper's fascia, which is a layer of fat-containing tissue of varying thickness and the superficial vessels to the skin that run through it.

Scarpa’s fascia forms another distinct layer deep to Camper fascia. In the perineum, Scarpa’s fascia blends into Colles’ fascia (superficial perineal fascia), and continuous with Dartos fascia of the penis and scrotum.

Several important anatomic relationships should be considered. A potential space between the Scarpa’s fascia and the deep fascia of the anterior wall (external abdominal oblique) allows for the extension of a perineal infection into the anterior abdominal wall.

Superiorly, Scarpa’s and Camper’s fasciae coalesce and attach to the clavicles, ultimately limiting the cephalad extension of an infection that may have originated in the perineum.

Colles’s fascia is attached to the pubic arch and the base of the perineal membrane, and it is continuous with the superficial Dartos fascia of the scrotal wall.

The perineal membrane is also known as the inferior fascia of the urogenital diaphragm and, together with Colles’ fascia, defines the superficial perineal space.

This space contains the membranous urethra, bulbar urethra, and bulbourethral glands.

In addition, this space is adjacent to the anterior anal wall and ischiorectal fossae. Infectious disease of the male urethra, bulbourethral glands, perineal

structures, or rectum can drain into the superficial perineal space and can extend into the scrotum or into the anterior abdominal wall up to the level of the clavicles.

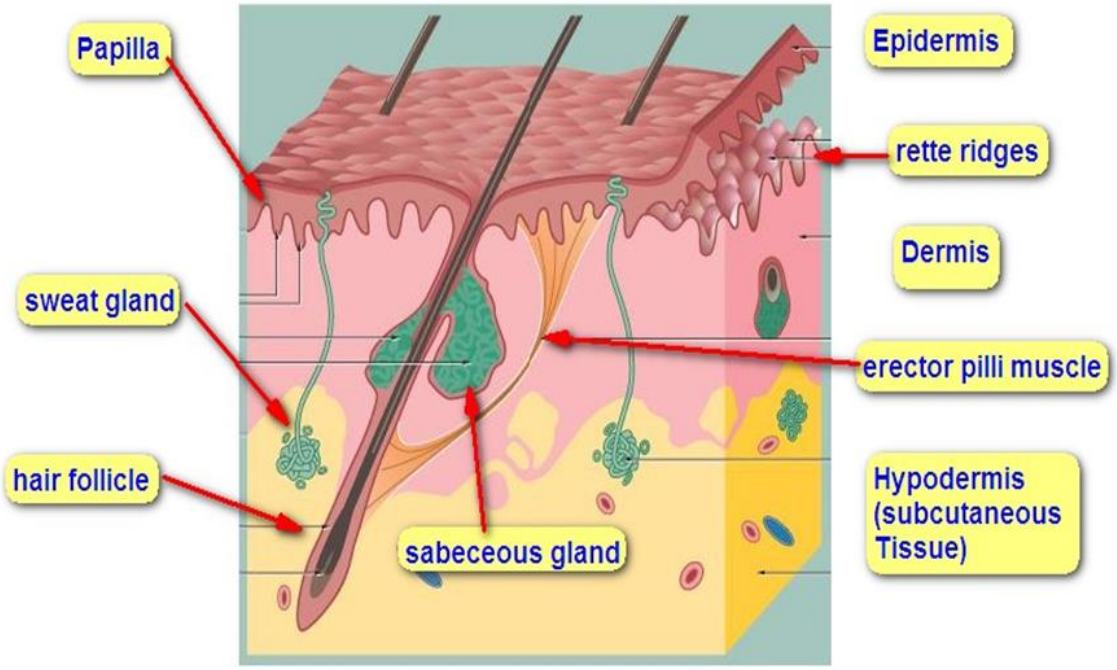


FIG- SKIN AND SUPERFICIAL FASCIA

Blood supply

Branches from the inferior epigastric and deep circumflex iliac arteries supply the lower aspect of the anterior abdominal wall.

Branches of the external and internal pudendal arteries supply the scrotal wall. With the exception of the internal pudendal artery, each of these vessels travels within

Camper’s fascia and can therefore become thrombosed in the pathogenesis of Fournier gangrene.

In the male, the testis receives its blood supply primarily from the testicular artery, a branch of the abdominal aorta.

This explains the sparing of the testis in Fournier’s gangrene.

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

Penis and scrotum

The contents of the scrotum, namely the testes, epididymides and cord structures, are invested by several fascial layers distinct from the Dartos fascia of the scrotal wall.

The most superficial layer of the testis and cord is the external spermatic fascia, which is continuous with the external oblique aponeurosis of the superficial inguinal ring .

The next deeper layer is the internal spermatic fascia, which is continuous with the transversalis fascia. The Buck fascia covers the erectile bodies of the penis, the corpora cavernosa, and the anterior urethra.

The Buck fascia fuses to the dense tunica albuginea of the corpora cavernosa, deep in the pelvis.

These fascial layers do not become involved with an infection of the superficial perineal space and can limit the depth of tissue destruction in a necrotizing infection of the genitalia.

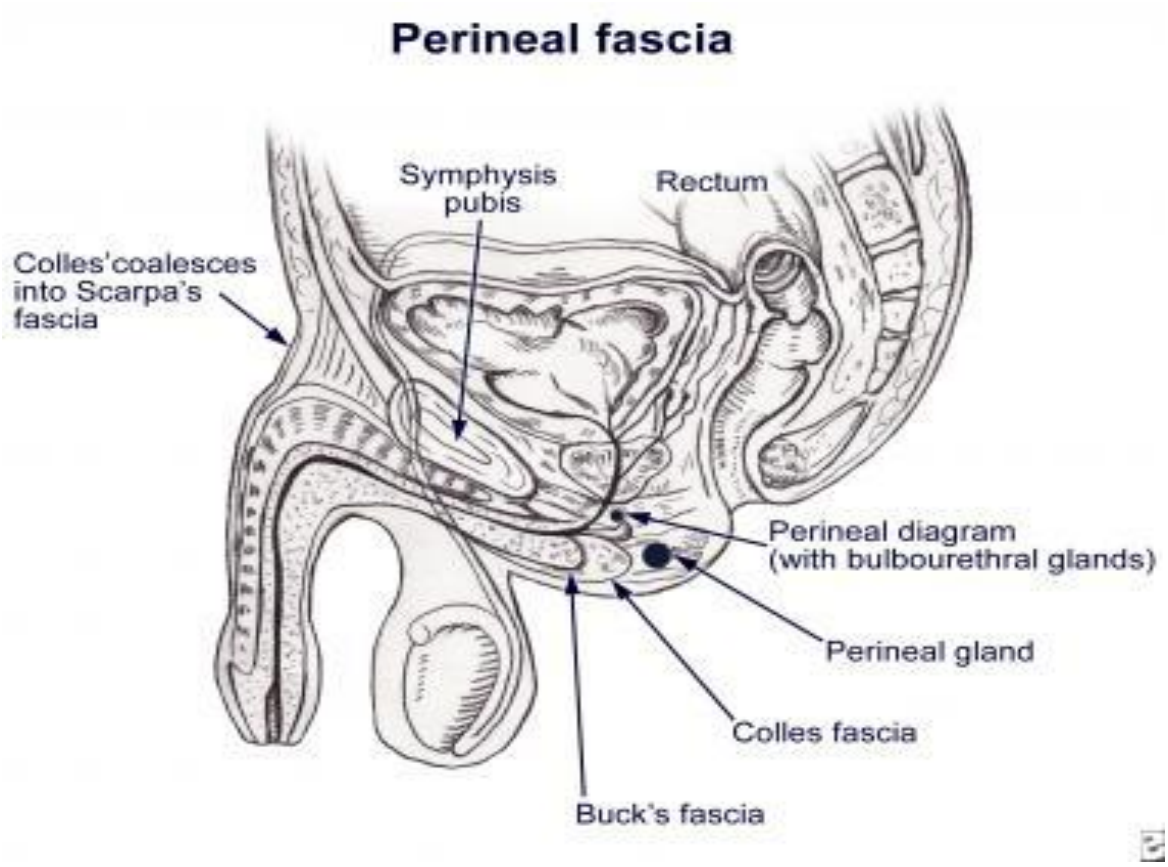


FIG – PERINEAL FASCIA

Aetiology

Although originally described as idiopathic gangrene of the genitalia, Fournier gangrene has an identifiable cause in approximately 95% of cases. The necrotizing process commonly originates from an infection in the anorectum, urogenital tract, or skin of the genitalia 10.

Trauma, recent surgery, and the presence of foreign bodies may also lead to the disease. Perianal, perirectal and ischiorectal abscesses, anal fissures; colonic perforations, urethral strictures with urinary extravasations; epididymoorchitis or hidradenitis may lead to the disease.

Urethral instrumentation, prosthetic penile implants, superficial soft-tissue injuries, intramuscular injections, genital piercings, steroid enemas (used for the treatment of radiation proctitis), blunt thoracic trauma, and penile self-injection with cocaine have been reported in the literature as causative factors.

In women, septic abortions, vulva or Bartholin gland abscesses, hysterectomy, and episiotomy are documented sources of sepsis. In men, anal intercourse may increase risk of perineal infection, either from blunt trauma to the area or by spread of rectally carried microbes.

In children, strangulated inguinal hernia, circumcision, omphalitis, insect bites, trauma, urethral instrumentation, peri-rectal abscesses, systemic infections, and burns have led to the disease.

Poor perineal hygiene or the presence of chronically indwelling catheters, such as in paraplegic patients, poses an increased risk. Wound cultures from patients with Fournier gangrene reveal that it is a polymicrobial infection with an average of four isolates per case. Escherichia coli is the predominant aerobe, and Bacteroides species the predominant anaerobe.

Other common microflora includes Proteus, Staphylococcus, Enterococcus, aerobic and anaerobic Streptococcus, Pseudomonas, Klebsiella, and Clostridium. Incidence of methicillin-resistant Staphylococcus aureus (MRSA) may be increasing .

Any condition that leads to depressed cellular immunity may predispose a patient to the development of Fournier gangrene. Examples include the following: diabetes mellitus (present in as many as 60% of cases), alcoholism, extremes of age, malignancy, chronic steroid use, cytotoxic drugs, lymphoproliferative diseases, malnutrition and HIV infection.

Pathophysiology

The following are pathognomonic findings of Fournier gangrene upon pathologic evaluation of the involved tissue: Necrosis of the superficial and deep fascial planes,

Fibrinoid coagulation of the nutrient arterioles, Polymorphonuclear cell infiltration and Microorganisms identified within the involved tissues In

necrotizing fasciitis as opposed to cellulitis, the location of the inflammation involves the subcutaneous fat, fascia, and muscle in addition to the dermis. A photomicrograph may show the presence of ulcerated epidermis and the presence of thrombosed blood vessel

Fournier's Gangrene

Infection represents an imbalance between host immunity and the virulence of the causative microorganisms. The aetiologic factors allow the portal of entry of the microorganism into the perineum. The compromised immunity provides a favourable environment to initiate the infection, and the virulence of the microorganism promotes the rapid spread of the disease.

Microorganism virulence results from the production of toxins or enzymes that create an environment conducive to rapid microbial multiplication . In a 1924 series of Chinese men with necrotizing infections, Meleney reported that streptococcal species were the predominant organisms recovered from cultures. Meleney attributed the FG to this sole genus.

Subsequent clinical series however stress the polymicrobial nature of most cases of necrotizing infection including Fournier's gangrene. Presently, recovering only streptococcal spp is unusual. Rather, streptococcal organisms are culture along with as many as five other organisms.

The commonest causative organisms are Streptococcus spp, Staphylococcus spp., Genera of Enterobacteriaceae family, anaerobic organisms and fungi. Most authorities believe the polymicrobial nature of Fournier gangrene is necessary to create the synergy of enzyme production that promotes rapid multiplication and spread of the infection.

For example, one microorganism might produce the enzymes necessary to cause coagulation of the nutrient vessels. Thrombosis of these nutrient vessels reduces local blood supply.

Thus, tissue oxygen tension falls. The resultant tissue hypoxia allows growth of facultative anaerobes and microaerophilic organisms. These latter microorganisms, in turn, may produce enzymes (e.g., lecithinase, collagenase), which lead to digestion of fascial barriers, thus fueling the rapid extension of the infection.

The fascial necrosis and digestion are hallmarks of FG. Knowledge of this provides the surgeon with a clinical marker of the extent of tissue affectation. Severe or fulminant Fournier's gangrene can spread from the fascial envelopment of the genitalia throughout the perineum, along the trunk, occasionally, into the thighs and very rarely, to the chest.

Clinical presentation

A thorough review of systems, including history of diabetes, alcohol abuse, cancer, colorectal or urogenital disease or surgery, steroid use, sexual history, and HIV status is important. Fournier's gangrene usually begins with an insidious onset of pruritus and discomfort of the external genitalia.

Early in the course of the disease, pain may be out of proportion to physical findings.

Swelling and erythema of the region follow pain, and a patient may complain of systemic symptoms such as fever or chills.

As gangrene develops, pain may subside as nerve tissue becomes necrotic. Skin overlying the affected region may be normal, erythematous, edematous, cyanotic, bronzed, indurated, blistered, and/or frankly gangrenous.

Skin appearance often underestimates the degree of underlying disease. A faeculent odor may be present secondary to infection with anaerobic bacteria. Crepitus may be present, but its absence does not exclude the presence of Clostridium species or other gas-producing organisms.

Systemic symptoms (e.g. fever, tachycardia, and hypotension) may be present. A thorough genital and perianal examination is required to detect potential portal of entry.

Investigations

The diagnosis of Fournier's gangrene is clinical and includes the history and physical examination findings, especially the anatomical involvement in the external genitalia and perineum.

Urinalysis and blood sugar measurements give evidence of metabolic derangements such as diabetes mellitus. Considering that FG is a urological emergency, treatment should not be delayed for these investigative tools.

Other investigations are essential to identify co-morbid factors as well as causative factors.

Appropriate bacteriological evaluation of pus from the gangrene, full blood count, renal and hepatic function studies are essential.

Diagnostic investigations, which may also help to determine Fournier's gangrene of scrotum following orchidectomy for prostate cancer. (Note fungal infection of perineal skin)

the extent of the disease employ radiological tools such as ultra-sound scan (USS), computerised tomographic (CT) scan and magnetic resonance imaging (MRI) 16. The hallmark in all imaging modalities is the demonstration of air in the soft tissue planes . These tools may be employed when they are available. Histopathology assessment has been reported by authors but describes

pathological consequences of the disease. Marjolin's ulcer has been reported to arise from the scar of a FG many years after successful treatment . Follow up and biopsy when indicated are necessary. Some investigations are necessary to identify the source of sepsis in FG. Thus, there may be need for cystoscopy, colonoscopy and biopsies .

Treatment

The treatment of FG depends on the status of the patient at presentation. Immediate treatment following diagnosis or suspicion includes resuscitative measures such as rehydration, blood transfusion, electrolyte replacements, multiple therapy antimicrobial agents, Oxygen and adequate analgesia.

Triple antimicrobial therapy is started empirically to cover aerobes and anaerobes as well as gram-negative and gram-positive organisms pending the result of microbial microscopy, culture and sensitivity from pus and or blood specimens.

The organisms invariably cultured from FG include staphylococci and streptococci, coliforms, pseudomonas, bacteroides and clostridia. Penicillin is used to cover streptococci while metronidazole is given against anaerobes such as bacteroides.

A broad spectrum antimicrobial agent, preferably the cephalosporins combined with gentamicin is used against gram negative organisms such as the coliforms.

Sepsis leading to multiple organ failure is thought to be the leading cause of death in Fournier's gangrene. Some authors have doubted whether antimicrobial agents are responsible for the reduction in mortality.

The use of unprocessed honey has been advocated by clinicians reporting good outcome . Honey inhibits bacterial growth due to its low pH, high viscosity, the hygroscopic effect and presence of inhibine and anti-oxidants.

Hyperbaric oxygen (HBO) was used in the erroneous belief that the crepitus in FG was of clostridial aetiology. The efficacy of hyperbaric oxygen is applicable in clostridial and nonclostridial infections .

HBO increases the oxygen tension in tissues to a level that is inhibitory and lethal to anaerobic bacteria, while limiting necrosis and enhancing demarcation of gangrene . However in a review of patients with FG and in whom half were given HBO and another half not given, there was a higher mortality among the HBO patients .

This had been observed 10 years earlier 35. Adequate nutrition is an essential part of treatment of FG. Enteral feeding is preferred to parenteral feeding. Patients with co-morbid or predisposing factors need these factors controlled. In one study, the authors concluded that diabetes control was an important prognostic measure .

Surgical treatment is the cornerstone of the treatment of FG. A mortality of 100% has been recorded in patients with necrotizing fascitis treated without surgery. Surgical treatment includes excision of all necrotic tissues. This may be repeated as necrosis is observed.

Orchidectomy may be required for testicular gangrene, a rare complication of FG. It has also been done when there was not enough scrotum to house the testis. Reconstruction may be required to restore function and cosmetic appearance.

Procedures that have been carried out vary from secondary closure of well granulated wound to flap procedures to create a neo-scrotum. The testis may be buried in inner aspect of thighs or inguinal regions temporarily, to prevent desiccation until the wound becomes clean.

In a report of FG in 10 women, the authors concluded that colostomy was an integral part of the management of FG patients requiring extensive debridement. Urinary diversion via a suprapubic cystostomy is indicated in FG of the penis. The consensus appears to be that the use of catheterization or colostomy should be pragmatic and should be decided on individual merit .

Complications

An uncomplicated Fournier's gangrene is one which is localized and resolves with the basic treatments of debridement, dressings and antimicrobial agents. Morbidity includes variable periods of hospitalization with its attendant problems such as deep vein thrombosis and pulmonary embolism.

Complicated FG is found in those patients with systemic involvement including renal, pulmonary and cardiac derangements. Complicated FG may require urgent and vigorous resuscitation in the intensive care unit and reconstructive procedures. Specific complications include auto-amputation of the penis , fatal overwhelming sepsis, tetanus and Marjolin's ulcer long after the wound has healed .

The testis may become gangrenous from FG in which sepsis has originated from the retroperitoneal space or the abdomen.

Scrotal skin loss may be severe not to accommodate the testis.

Orchidectomy has been done for this reason. Infertilty is a rare complication of Fournier's gangrene.

Methods and Materials

Study design: Hospital based - Prospective study

Study area: GOVT RAJAJI HOSPITAL, MADURAI

Study period: ONE YEAR

Sample size: 76 patients admitted to the hospital with the diagnosis of soft tissue infections were included in the study.

We obtained approval from the hospital administrators and ethical committee to conduct this study and received the participant's written consent. Patients presenting with soft tissue infections were clinically examined and subjected to a proforma. LRINEC scoring system was applied to each patient with soft tissue infections in accordance with the inclusion and exclusion criteria of the study.

INCLUSION CRITERIA

Patients presenting with soft tissue infections during the course of the study.

EXCLUSION CRITERIA

1. Patients below 15 yrs or above 75 yrs of age

2. Patient who has undergone surgical debridement for present episode Of Soft tissue infection

3. Patient with boils or furuncles with no evidence of cellulitis

Clinical Data

Patients were selected on their presentation in the Emergency triage or in the Out-Patient department. Details of cases were recorded including history, clinical examination, and investigations done after taking informed consent from the patient for the study. Score was calculated and patient was followed up during the course of stay in the hospital with respect to the management received by the patient. Final outcome was evaluated and presented below.

RESULTS :

Fig-1: Table showing age distribution of soft tissue infection among patients selected for study.

AGE	No.OF CASES	%
≤50	20	26.3
>50	56	73.7
Total	76	100.00

56 patients out of 76 selected patients where above 50 years which is the commonest age group for soft tissue infections. Below 50 years being the least common age group.

FIG.2: Bar chart showing age distribution among patients selected for study.

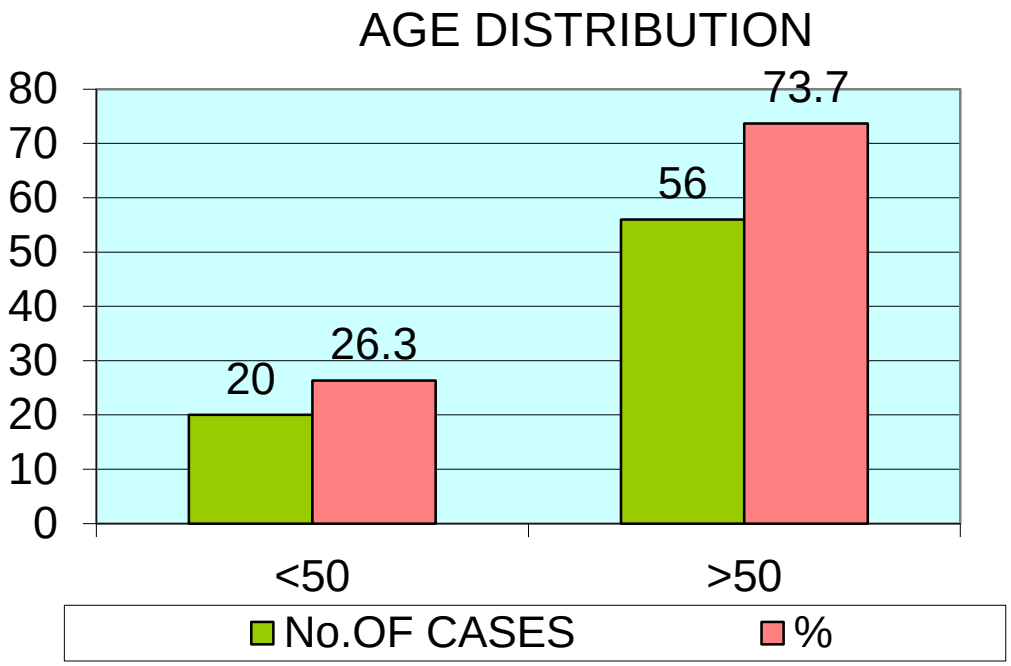


FIG.3; Table showing gender distribution among patients selected for study.

SEX	No.OF CASES	%
MALE	59	77.63
FEMALE	17	22.37
Total	76	100.00

FIG.4; Pie chart showing gender distribution among patients selected for study.78% where of male gender which is the most common group with soft tissue infections.

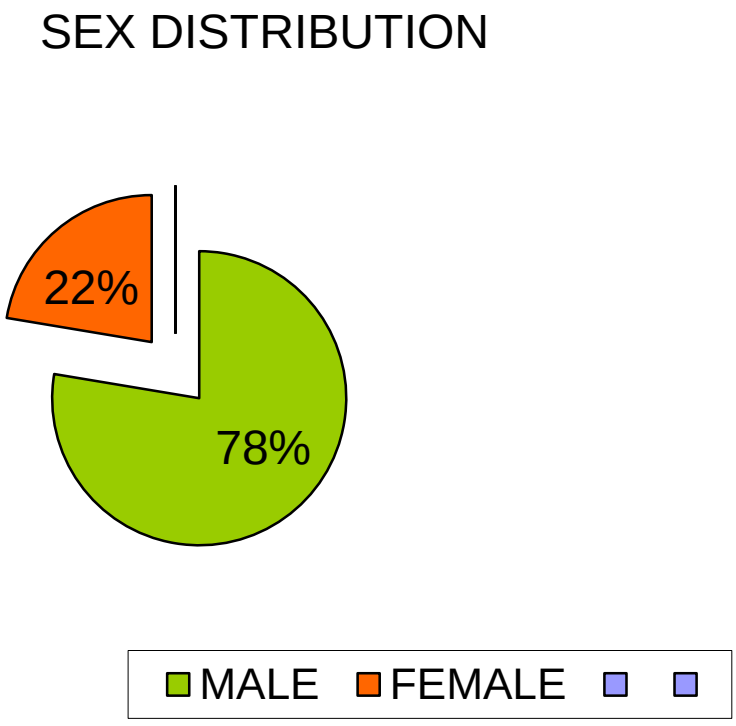


FIG.5; Table showing most common age group positive for debridement (i.e.above 50 yrs)among patients selected for study.

AGE vs DEBRIDEMENT		DEBRIDED(+)	NON DEBRIDE(-)	Total	%
≤50	20	11	9	20	26.32
>50	56	40	16	56	73.68
Total	76	51	25	76	100.00

FIG.6; Cylindrical chart showing common age group(i.e.above 50 yrs) positive for debridement among patients with soft tissue infections.

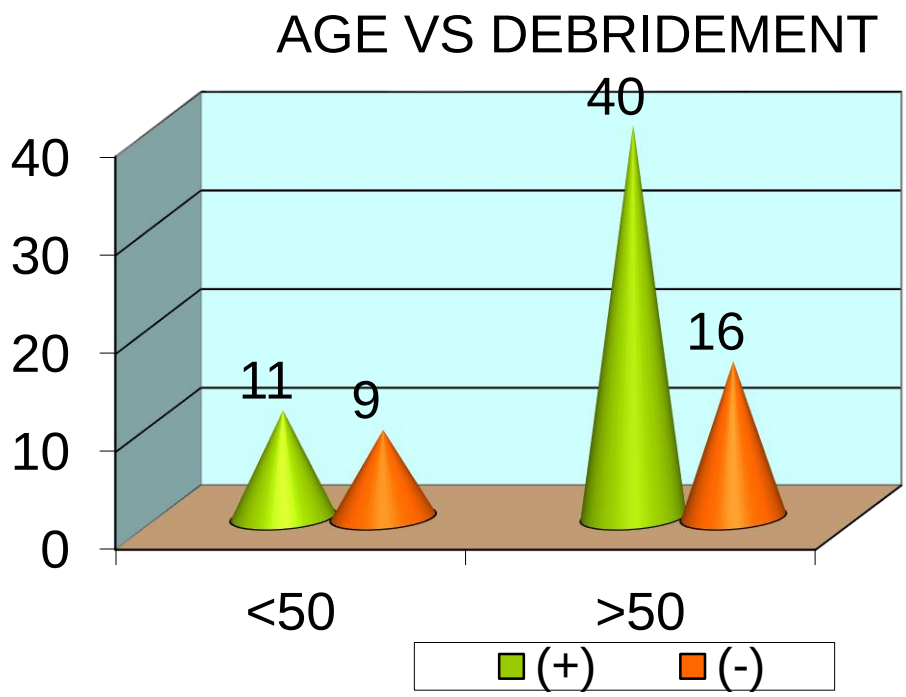


FIG.7; Table showing most common gender positive for debridement among patients selected for study.

SEX vs DEBRIDEMENT		DEBRIDED(+)	NON DEBRIDE(-)	Total	%
MALE	59	40	19	59	77.63
FEMALE	17	11	6	17	22.37
Total	76	51	25	76	100.00

FIG.8; Cylindrical chart showing male being the most common gender to be affected.

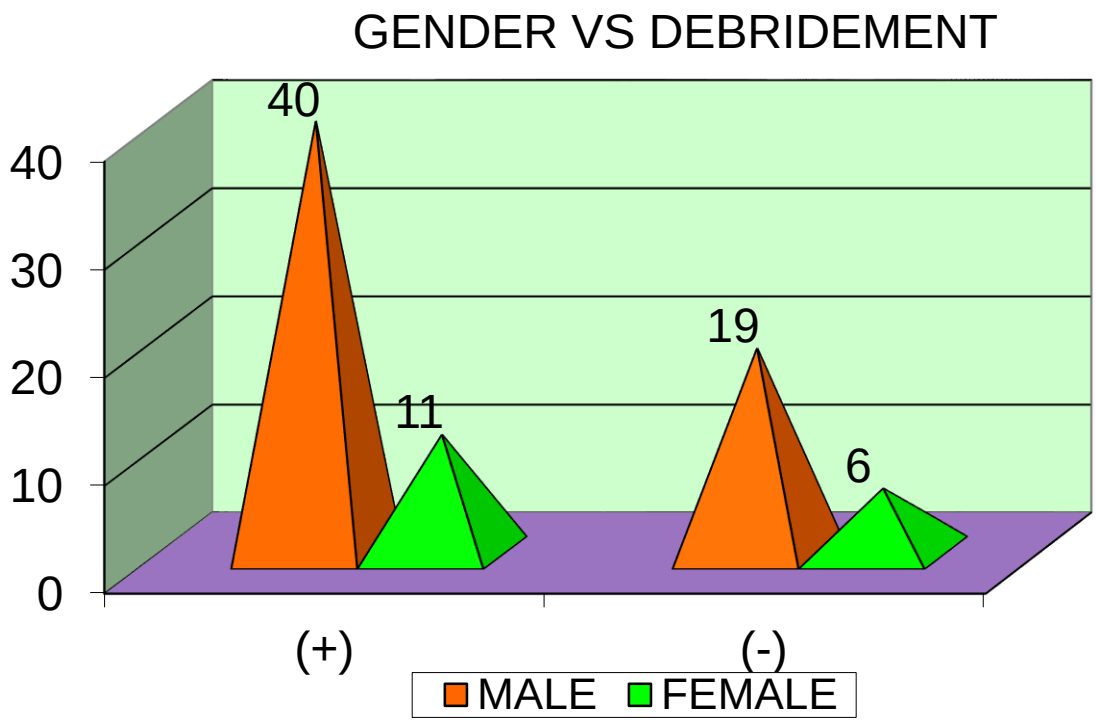


FIG.9; Table showing temperature relavence with soft tissue infections among patients selected for study.

FEVER vs DEBRIDEMENT		DEBRIDED(+)	NON DEBRIDE(-)	Total	%
FEBRILE	14	14	0	14	18.42
AFEBRILE	62	37	25	62	81.58
Total	76	51	25	76	100.00

FIG.10; Bar chart shows 18% of the operated group where febrile preoperatively.

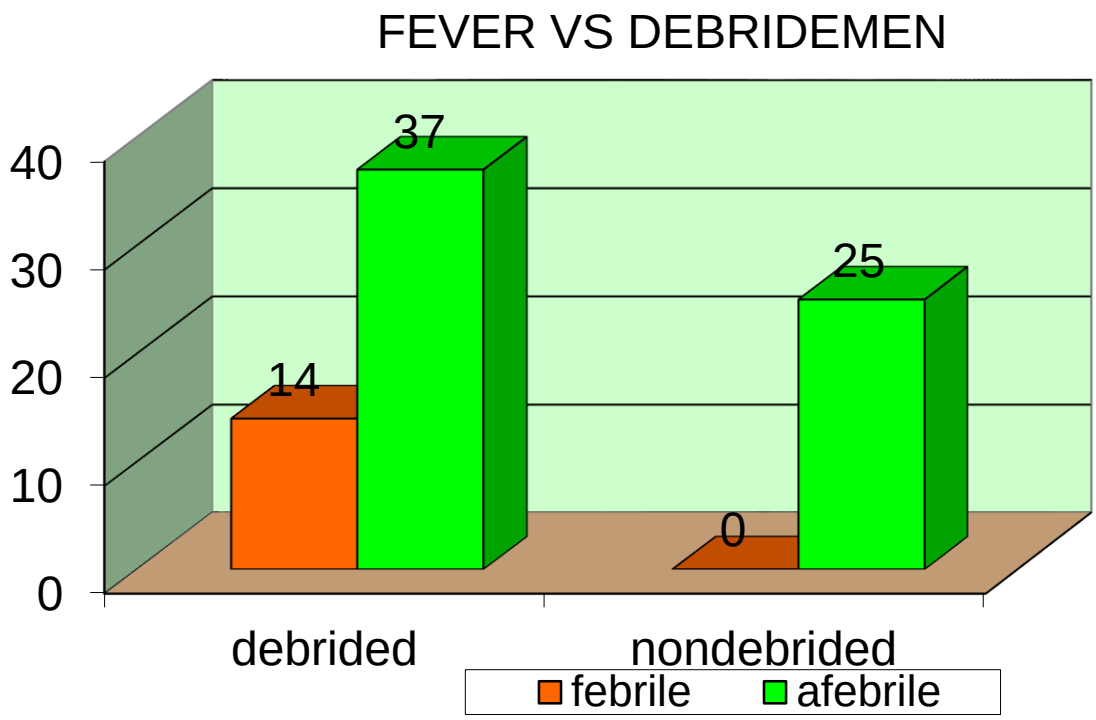


FIG.11; Table shows relevance of diabetes among patients selected for study.

DM vs DEBRIDEMENT		POSITIVE(+)	NEGATIVE(-)	Total	%
+	23	21	2	23	30.26
-	53	30	23	53	69.74
Total	76	51	25	76	100.00

FIG.12; Cylindrical chart shows 23 out of 51 operated patients where diabetic.

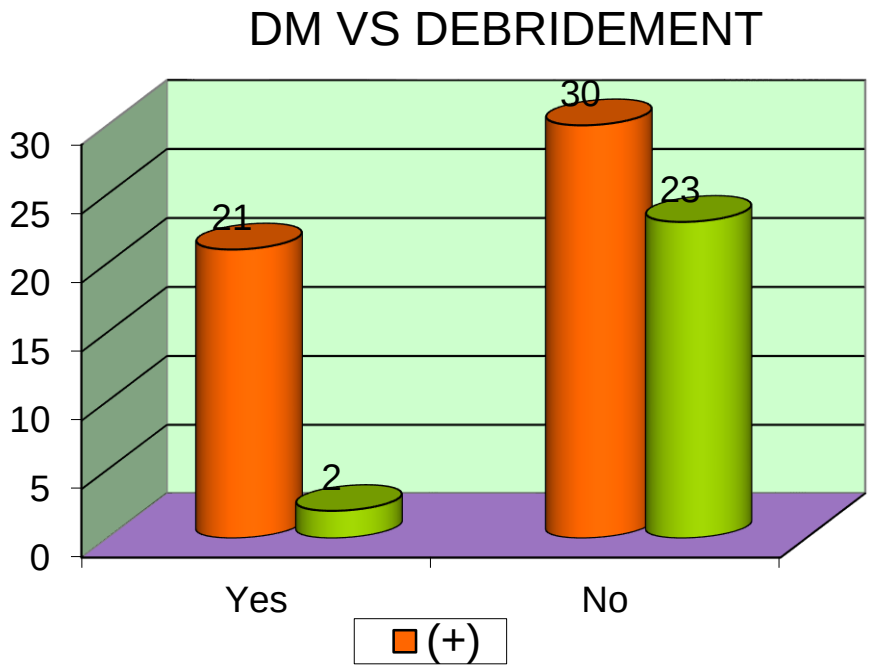


FIG.13; Table shows CRP significance in the study.

CRPvs DEBRIDEMENT		DEBRIDED(+)	NON DEBRIDE(-)	Total	%
< 150	48	26	22	48	63.16
> 150	28	25	3	28	36.84
Total	76	51	25	76	100.00

FIG.14; Bar chart shows 36% of the selected patients where with CRP>150.

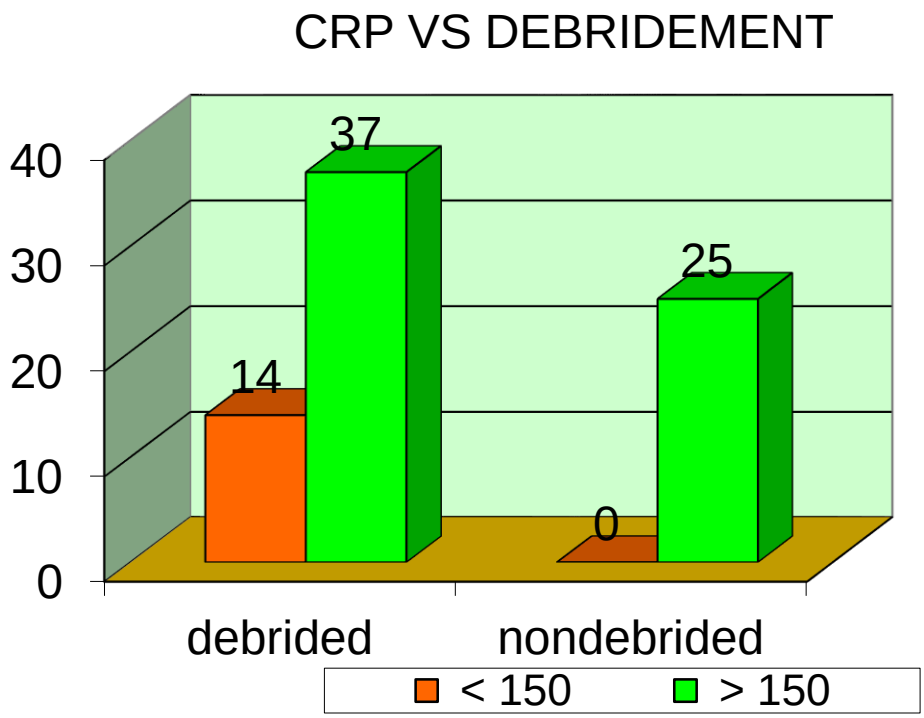


FIG.15; Table showing increased total count among patients with soft tissue infections.

TC vs DEBRIDEMENT		DEBRIDED(+)	NON DEBRIDE(-)	Total	%
< 15	18	8	10	18	23.68
15 - 25	51	38	13	51	67.11
> 25	7	5	2	7	9.21
Total	76	51	25	76	100.00

FIG.16; Bar chart shows 67% of patients among selected group where with increased total count(15-25).

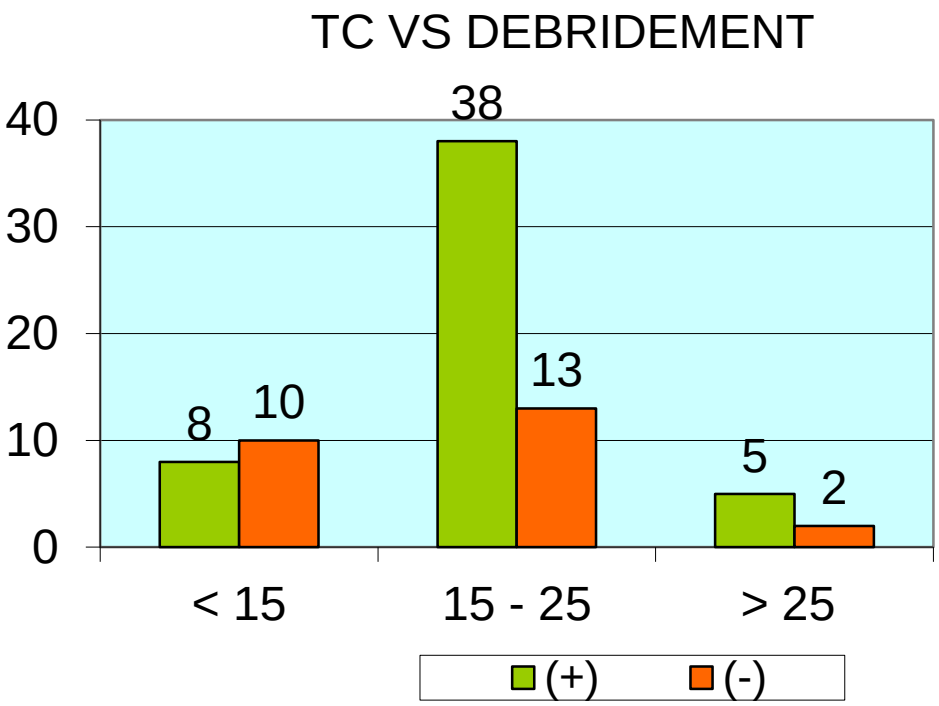


FIG.17; Table shows significance of anemia among patients with soft tissue infection.

HB vs DEBRIDEMENT		DEBRIDED(+)	NON DEBRIDE(-)	Total	%
> 13.5	12	9	3	12	15.79
11 - 13.5	28	16	12	28	36.84
< 11	36	26	10	36	47.37
Total	76	51	25	76	100.00

FIG.18; Bar chart shows 47% of patients among the selected group where with Hb<11gms.

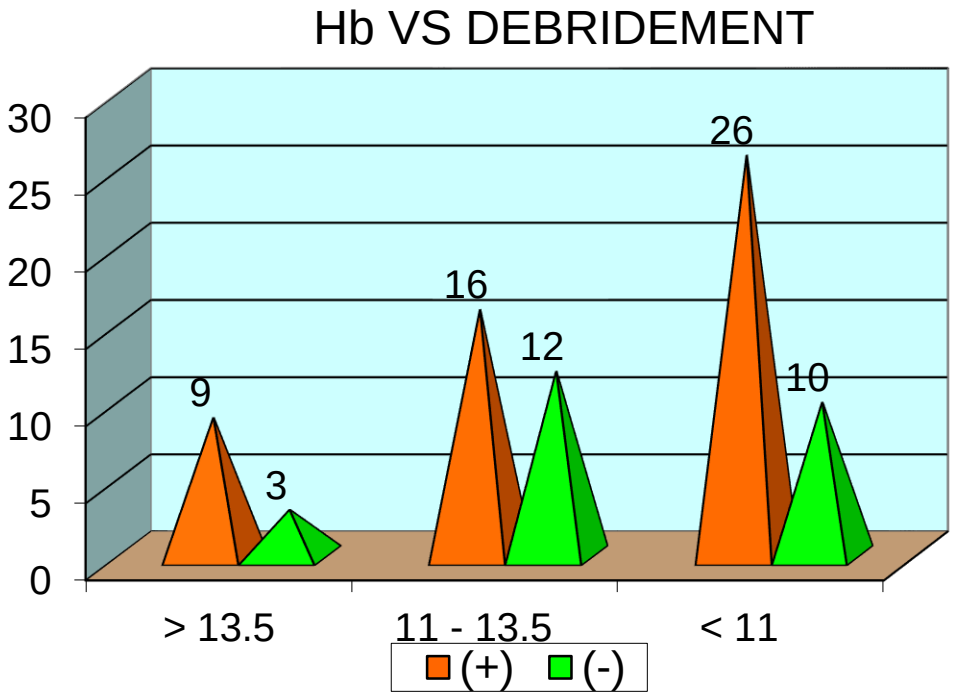


FIG.19;Table shows relevance of serum sodium among the study group.

Na vs DEBRIDEMENT		DEBRIDED(+)	NON DEBRIDE(-)	Total	%
> 135	37	21	16	37	48.68
< 135	39	30	9	39	51.32
Total	76	51	25	76	100.00

FIG.20; Cylindrical chart shows 51% of patients among the study group where with Sr.Na value <135mmol/L.

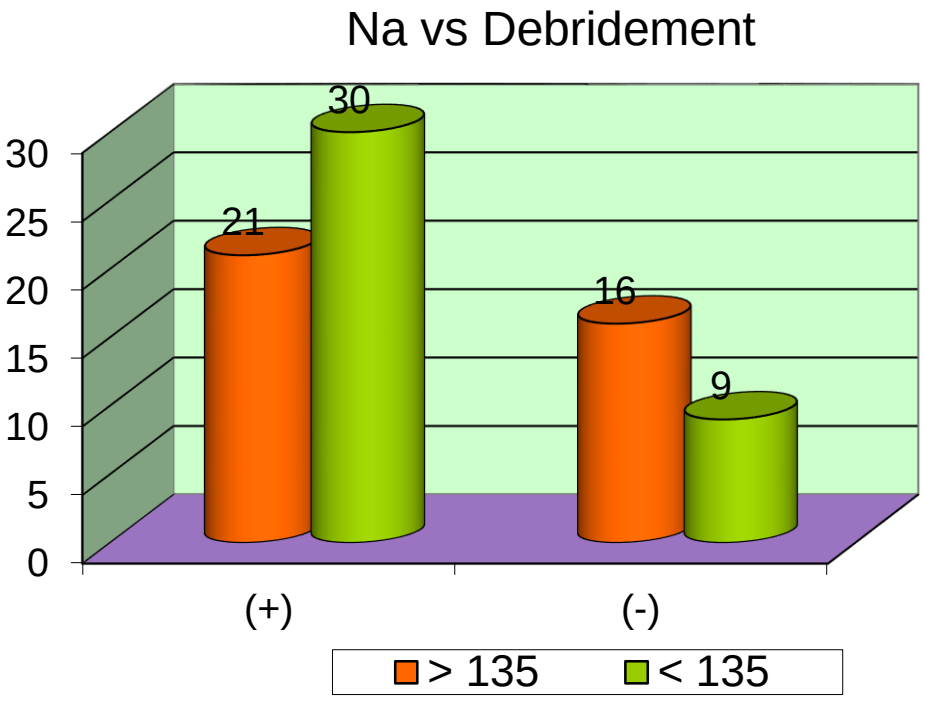


FIG.21; Table shows relevance of Sr.creatinine among the study group.

Cr vs DEBRIDEMENT		DEBRIDED(+)	NON DEBRIDE(-)	Total	%
< 1.6	39	28	11	39	51.32
> 1.6	37	23	14	37	48.68
Total	76	51	25	76	100.00

FIG.22; Bar chart shows 51% of patients among the study group where with creatinine value<1.6.

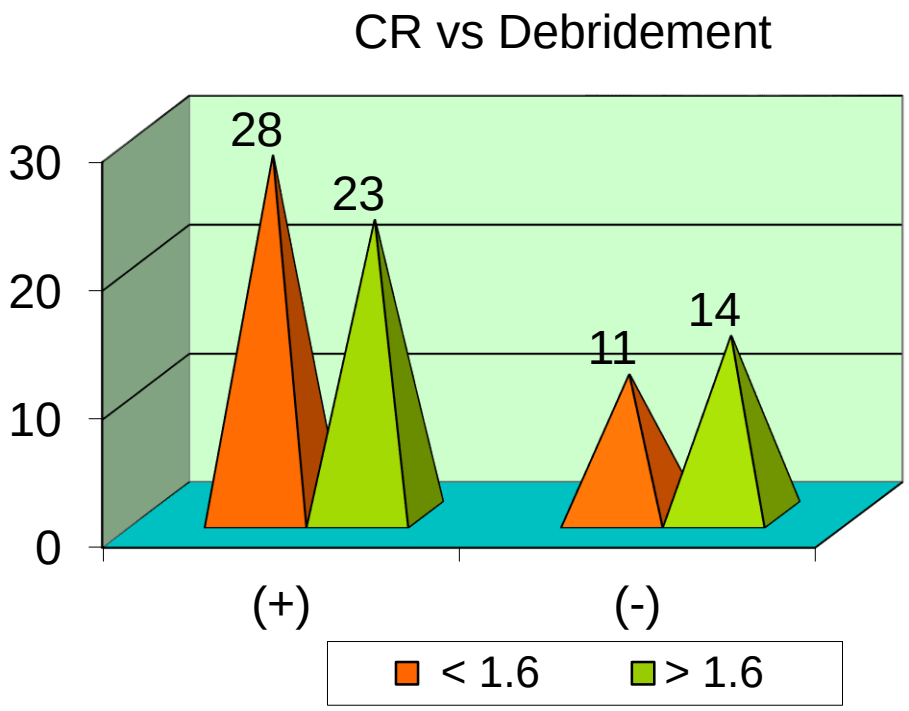


FIG.23; Table shows relevance of random blood sugar value among the study group.

RBS vs DEBRIDEMENT		DEBRIDED(+)	NON DEBRIDE(-)	Total	%
< 180	53	30	23	53	69.74
> 180	23	21	2	23	30.26
Total	76	51	25	76	100.00

FIG.24; Bar chart shows 69% of the patients among the study group where with RBS value <180mg/dl.

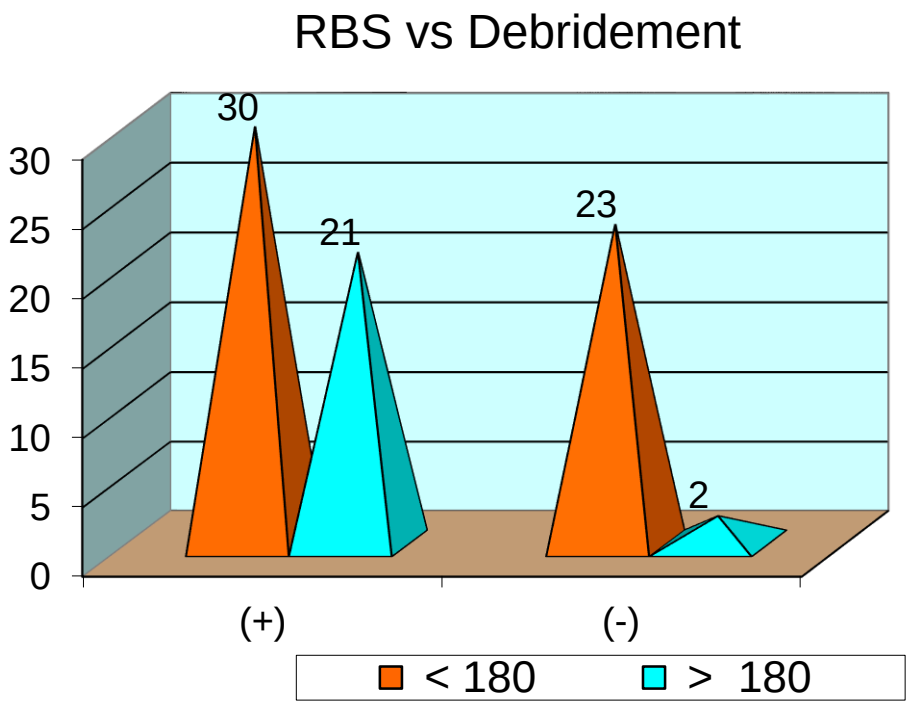


FIG.25; Table shows the LRINEC score for the study group

LRINEC vs DEBRIDEMENT		DEBRIDED(+)	NON DEBRIDE(-)	Total	%
<6		20	25	45	44.40
≥6		31	0	31	100.00
Total		51	25	76	100.00

FIG.26; Bar chart shows significance if LRINEC score among the operated group.

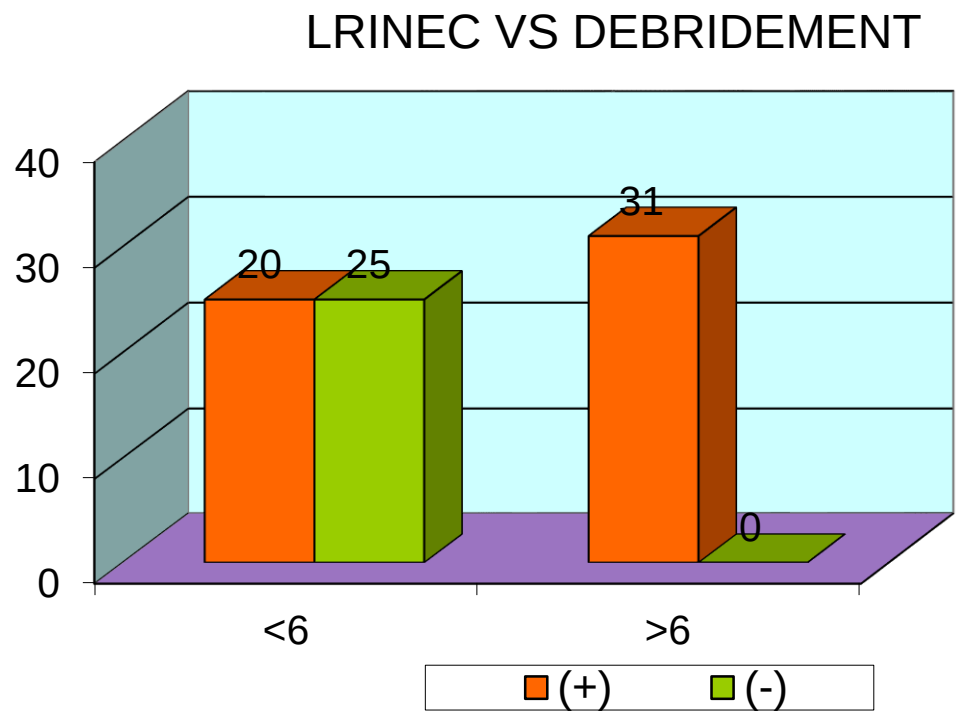


FIG.27; Table shows HPE results for the operated group.

LRINEC vs HPE		coagulous necrosis	necrosed skin,fascia	neutrophils infilration	NIL	Total	%
<6	45	4	12	4	25	45	59.21
≥6	31	6	15	10	0	31	40.79
Total	76	10	27	14	25	76	100.00

FIG.28; Bar chart shows significance of LRINEC score in confirming operated cases by histopathalogical examination.

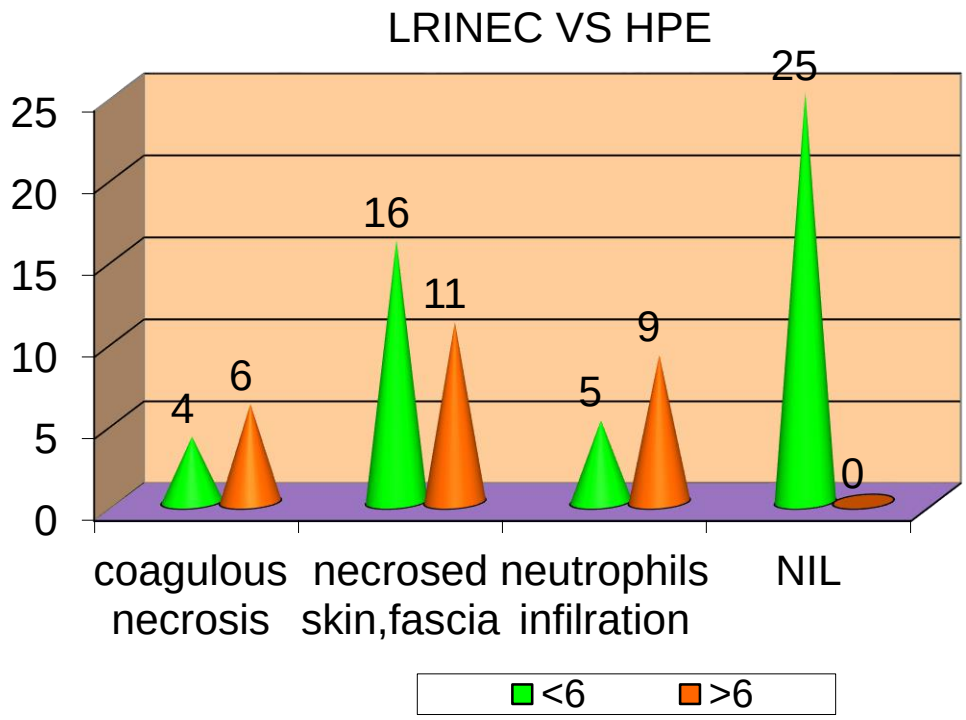


FIG.29;Table shows importance of LRINEC score in diagnosing necrotising fasciitis from other soft tissue infections.

LRINEC vs Diagnosis		cellulitis	F.G	NF	Total	%
<6	45	25	6	14	50	26.32
≥6	31	0	4	27	31	40.79
Total	76	25	10	41	76	100.00

FIG.30; Cylindrical chart shows significance of LRINEC score in diagnosing N.F

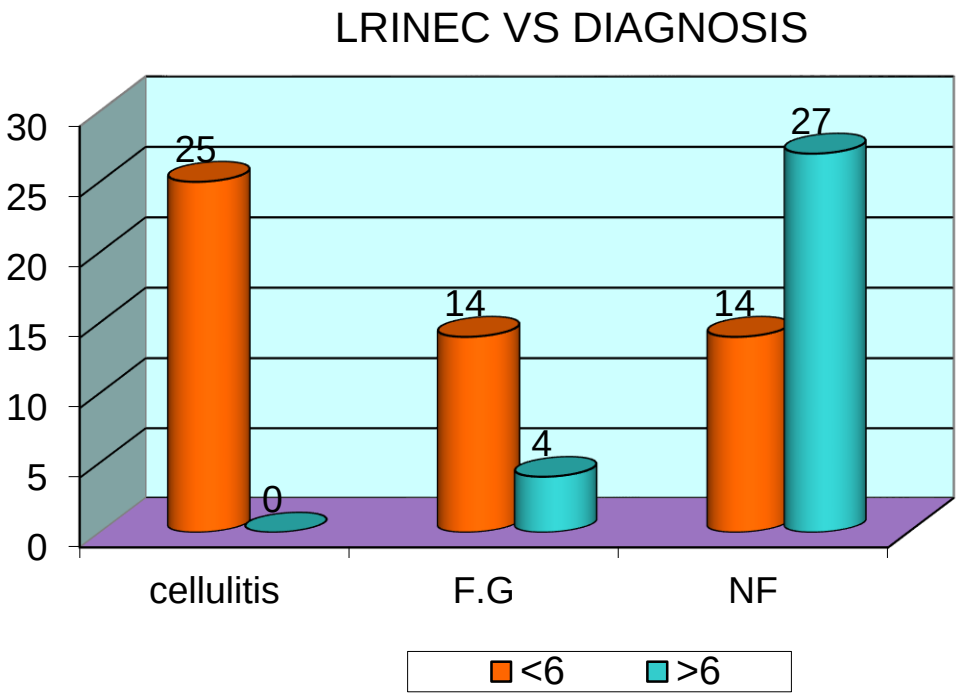
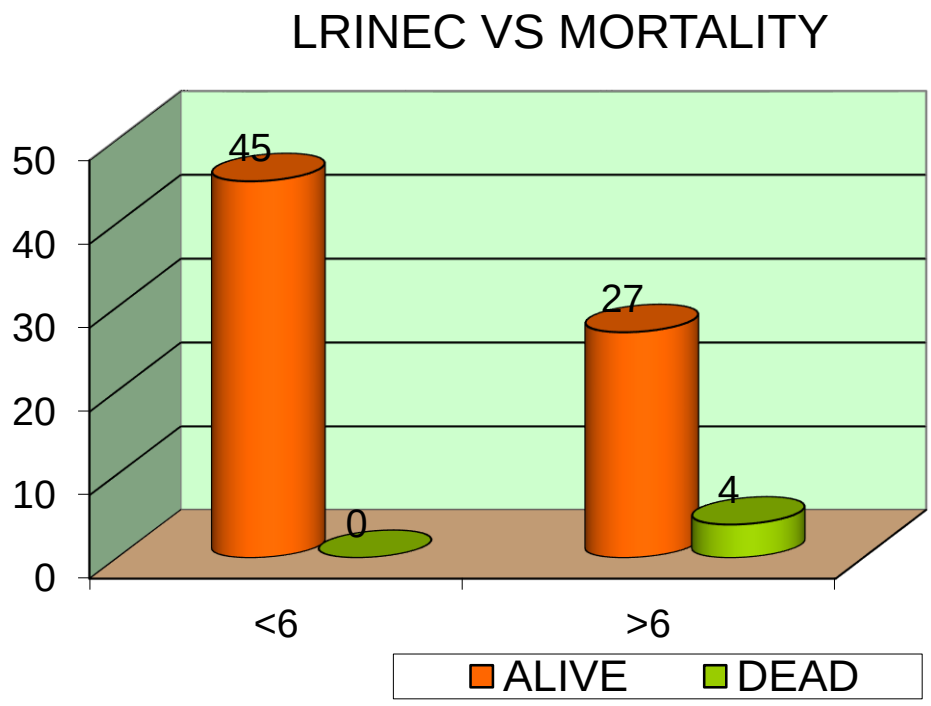


FIG.31; Table shows significance of LRINEC score in reducing mortality among study group.

LRINEC vs MORTALITY		ALIVE	DEAD	Total	%
<6	45	45	0	45	59.21
≥6	31	27	4	31	40.79
Total	76	72	4	76	100.00

FIG.32; Bar chart shows mortality rate among study group with LRINEC score above 6.



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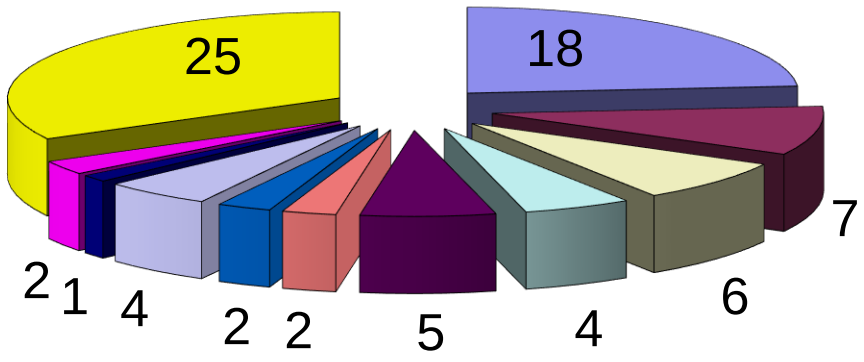
TISSUE CULTURE ORGANISM	NUMBER OF CASES
GROUPA-HEMOLYIC STREPTOCOCCI	18
POLYMICROBIAL	7
E.COLI	6
BACTEROIDS	4
PEPTOSTREPTOCOCCI	5
PROTEUS	2
CLOSTRIDIUM	2
KLEBSIELLA	4
STREPTOCOCCI	1
STREPTOCOCCI PYOGENES	2

FIG.33; Table shows tissue culture report for the study group.

TISSUE CULTURE ORGANISM



GAS
polymicrobial
E.coli
bacterioids
peptosreptococci
proteus
clostridium
Klebsiella
Sreptococci
srept.pyogenes
Non debridement

SUMMARY AND CONCLUSION

The present study comprised of 76 cases which presented with necrotizing soft tissue infections as per the inclusion and exclusion criteria.

- Present study showed that LRINEC score is capable of detecting early cases of necrotizing fasciitis among patients with severe soft tissue infections.

- Positive predictive value for the LRINEC scoring system was 94.7% with a sensitivity value of 95.6% in the present study. 51 patients with soft tissue infections were debrided based on LRINEC scoring system.

- Laboratory investigations required for the LRINEC scoring system are done on a routine basis for patients with soft tissue infections. These investigations were cheap and were readily available in the study area for the present study.

- Patients in the present study were stratified into high-, moderate-, and low-risk categories based on the LRINEC score for serious soft tissue infections warranting admission, intravenous antibiotics, and immediate further evaluation and allocation of resources.

- Common organisms like group A strpto cocci and poly microbial , pepto srepto cocci are involved in the tissue culture

- LRINEC scoring system is helpful in stratifying patients into risk categories of possibility of necrotizing fasciitis, allocating resources and ultimately aiding in the early recognition of necrotizing fasciitis.

- The LRINEC score is essentially a measure of the biochemical and hematologic disturbances associated with systemic inflammatory response syndrome.

- “Other soft tissue infections such as cellulitis and abscesses rarely cause an inflammatory state severe enough to cause such disturbances in the laboratory variables”.

- “The LRINEC score is a robust index that is capable of detecting early cases of necrotizing fasciitis and is simple enough for routine use”.

- “LRINEC scoring system is an important adjunctive tool in diagnosis of necrotizing soft tissue infections”

Drawbacks of the LRINEC scoring system

- “In patients with multiple co morbidities, the inflammatory response may be blunted and the score should be interpreted with caution”.

- “Once in the hospital, interventions to correct laboratory disturbances described (intravenous normal saline, insulin infusions, and blood transfusions). Haemoglobin, serum electrolytes, blood sugar levels normalize after initial correction and hence tends to interfere with the accuracy of the score”.

KEY TO MASTER CHART :

DIS- DISCHARGE

DM- DIABETIC MELLITUS

CRP- C REACTIVE PROTEIN

TC- TOTAL COUNT

HB- HAEMOGLOBIN

Na – SODIUM

Cr- CREATININE

RBS- RANDOM BLOOD SUGAR

LRINEC- LABORATORY RISK INDICATORS OF NECROTISING FASCIITIS

HPE- HISTOPATHOLOGICAL EXAMINATION

T, CULTURE – TISSUE CULTURE

SL.N	NAME	IP NO	AGE	SEX	swelling	ulcer	DIS	fever	DM	CRP	TC	HB	Na	Cr	RBS	LRINEC	DEB	HPE	T.CULTURE	DIAG	MOR
1	ayyanpillai	1071126	75	m	+	+	+	+	+	4	2	2	0	0	1	9	+	coagulous necrosis	polymicrobial	N.F	-
2	alagumalai	1071145	74	m	+	-	-	-	-	4	1	1	2	0	0	8	+	neutrophils infilration	polymicrobial	N.F	-
3	bommuraj	1071210	45	m	+	-	-	-	-	0	1	2	0	2	0	5	+	necrosed skin,fascia	bacterioids	N.F	-
4	parvathy	107178	65	f	+	-	-	-	-	0	1	2	2	0	0	5	-	-	-	cellulitis	-
5	malaisamy	1071210	60	m	+	-	-	-	-	4	1	2	0	0	1	8	+	coagulous necrosis	GAS	N.F	-
6	rajendiran	1071325	48	m	+	-	-	-	-	0	2	1	0	0	1	4	-	-	-	cellulitis	-
7	sundararajan	1071367	51	m	+	-	-	-	-	4	0	1	0	0	0	5	-	-	-	cellulitis	-
8	muniyamma	1071439	69	f	+	-	-	+	+	4	0	2	0	0	1	9	+	neutrophils infilration	clostridium	N.F	-
9	durairaj	1071475	68	m	+	+	+	+	-	4	1	2	2	0	1	10	+	necrosed skin,fascia	peptosreptococci	N.F	+
10	balamurugan	1071678	31	m	+	-	-	-	-	0	2	1	0	2	0	5	-	-	-	cellulitis	-
11	ponnusamy	1071789	66	m	+	-	-	-	-	0	1	1	0	2	0	4	-	-	-	celllulitis	-
12	ramayee	1080231	69	f	+	-	-	-	-	0	2	2	2	0	1	7	+	necrosed skin,fascia	GAS	N.F	-
13	venkatram	1081638	40	m	+	-	-	-	-	0	1	2	2	0	0	5	-	-	-	cellulitis	-
14	rajaram	1082568	45	m	+	-	-	-	-	0	1	1	0	2	0	4	-	-	-	cellulitis	-
15	murugan	1082429	40	m	+	+	+	+	+	4	0	1	2	2	1	10	+	coagulous necrosis	GAS	N.F	-
16	irulayee	1084871	71	f	+	-	-	-	-	4	1	2	0	0	1	8	+	necrosed skin,fascia	polymicrobial	N.F	-
17	veerappan	1083337	55	m	+	-	-	-	-	0	1	1	2	0	1	5	+	neutrophils infilration	E.coli	F.G	-
18	pandi	1084376	75	m	+	+	+	+	+	4	1	1	2	2	1	11	+	necrosed skin,fascia	bacterioids	N.F	+
19	barathiraja	1085734	47	m	+	-	-	-	-	0	1	1	0	2	0	4	-	-	-	cellulitis	-
20	ambujam	1085741	56	f	+	-	-	-	-	0	1	1	2	0	1	5	+	necrosed skin,fascia	polymicrobial	N.F	-
21	mahalingam	1086329	60	m	+	-	-	-	-	0	1	2	2	0	1	6	+	neutrophils infilration	GAS	N.F	-
22	mani	1087309	47	m	+	-	-	-	-	0	0	2	0	2	1	5	+	necrosed skin,fascia	E.coli	F.G	-
23	hussain	1087432	68	m	+	-	-	+	+	4	1	1	2	0	0	8	+	necrosed skin,fascia	peptosreptococci	N.F	-
24	begam	1089431	59	f	+	-	-	-	-	0	2	1	2	0	0	5	+	neutrophils infilration	proteus	F.G	-
25	pattappa	1094398	56	m	+	-	-	-	-	0	1	1	0	2	0	4	-	-	-	cellulitis	-
26	govindan	1095124	49	m	+	-	-	-	-	0	1	0	2	2	0	5	+	necrosed skin,fascia	clostridium	N.F	-
27	thmayan	1095289	74	m	+	-	-	-	+	0	0	2	2	2	1	7	+	coagulous necrosis	GAS	N.F	-
28	dhanam	1097467	49	f	+	-	-	-	-	0	1	2	0	2	0	5	+	necrosed skin,fascia	klebsiella	F.G	-
29	soori	1098539	71	m	+	-	-	-	-	4	1	0	0	0	0	5	+	neutrophils infilration	polymicrobial	N.F	-
30	pitchai	1092798	67	m	+	-	-	-	+	0	0	0	2	2	1	5	-	-	-	cellulitis	-
31	raman	1096087	53	m	+	-	-	-	+	4	1	0	2	0	1	8	+	necrosed skin,fascia	GAS	N.F	-
32	chitra	1097654	39	f	+	-	-	-	-	0	0	2	0	2	0	4	-	-	-	cellulitis	-
33	thilakar	1097534	69	m	+	-	-	-	-	0	2	1	2	2	0	7	+	coagulous necrosis	peptosreptococci	N.F	-
34	ulagan	1098431	58	m	+	-	-	-	-	0	1	2	2	0	0	5	+	necrosed skin,fascia	bacterioids	N.F	-
35	loganath	1098790	68	m	+	-	-	-	-	0	1	1	2	0	0	4	-	-	-	cellulitis	-
36	mallika	1098909	62	f	+	+	+	+	+	4	1	2	0	2	1	10	+	neutrophils infilration	GAS	N.F	-
37	kandha	1099075	38	m	+	-	-	+	-	4	1	1	0	2	0	8	+	necrosed skin,fascia	staph.aureus	N.F	-
38	nallayya	1100578	73	m	+	-	-	-	-	0	1	2	2	0	0	5	-	-	-	cellulitis	-
39	poovarasu	1102165	57	m	+	-	-	-	-	0	1	1	2	2	0	6	+	necrosed skin,fascia	E.coli	F.G	-

40	poongotha	1102435	61	f	+	-	-	-	-	0	0	2	0	2	0	4	-	-	-	cellulitis	-
41	ravindran	1102454	65	m	+	-	-	-	-	4	1	2	0	0	0	7	+	coagulous necrosis	GAS	N.F	-
42	eswaran	1103543	42	m	+	-	-	-	-	0	1	2	0	2	0	5	+	necrosed skin,fascia	klebsiella	F.G	-
43	sivaguru	1104564	55	m	+	-	-	-	-	0	1	1	0	2	0	4	-	-	-	cellulitis	-
44	ulagamma	1105434	66	f	+	-	-	-	-	0	1	0	2	2	0	5	+	neutrophils infilration	GAS	N.F	-
45	mannar	1105876	72	m	+	+	+	+	+	4	0	2	2	0	1	9	+	necrosed skin,fascia	GAS	N.F	-
46	needhi	1106543	47	m	+	-	-	-	-	0	1	0	2	2	0	5	+	necrosed skin,fascia	klebsiella	F.G	-
47	balan	11065321	61	m	+	-	-	-	-	4	1	1	0	0	0	6	+	necrosed skin,fascia	E.coli	F.G	-
48	gomati	1106643	52	f	+	-	-	-	-	0	0	2	0	2	0	4	-	-	-	cellulitis	-
49	mookan	1107487	53	m	+	-	-	-	-	4	1	2	0	0	0	7	+	necrosed skin,fascia	E.coli	N.F	-
50	gnanam	1108635	49	m	+	-	-	-	-	4	0	1	0	2	0	7	+	necrosed skin,fascia	GAS	N.F	-
51	gokulan	1109653	65	m	+	-	-	+	+	4	1	1	0	2	0	8	+	neutrophils infilration	polymicrobial	N.F	-
52	paamini	1109806	71	f	+	-	-	-	-	0	1	2	2	0	0	5	+	necrosed skin,fascia	GAS	N.F	-
53	oyyandi	1110212	55	m	+	-	-	-	-	4	1	0	0	0	0	5	-	-	-	cellulitis	-
54	rahman	1110432	39	m	+	-	-	-	-	0	0	2	2	0	0	4	-	-	-	cellulitis	-
55	ramasamy	1111014	67	m	+	-	-	-	-	0	1	2	2	0	0	5	+	coagulous necrosis	GAS	N.F	-
56	lakshmi	1111768	61	f	+	-	-	-	-	0	1	2	2	2	0	7	+	neutrophils infilration	peptosreptococci	N.F	-
57	duraisamy	1111780	48	m	+	-	-	-	-	0	0	1	2	2	0	5	-	-	-	cellulitis	-
58	jeyaram	1111717	75	m	+	+	+	+	+	4	1	1	2	2	1	11	+	neutrophils infilration	srept.pyogenes	N.F	+
59	soosai	1111865	53	m	+	-	-	-	-	0	1	2	0	2	0	5	+	coagulous necrosis	GAS	N.F	-
60	valli	1111982	59	f	+	-	-	+	+	4	1	0	2	0	1	8	+	neutrophils infilration	srept.pyogenes	N.F	-
61	lachmanan	1121563	45	m	+	-	-	-	-	0	0	2	0	2	0	4	-	-	-	cellulitis	-
62	kuruman	1121452	70	m	+	-	-	-	-	0	1	2	0	2	0	5	+	necrosed skin,fascia	GAS	N.F	-
63	aathimoolam	1121834	55	m	+	-	-	-	-	0	1	1	2	2	0	6	+	necrosed skin,fascia	proteus	F.G	-
64	thamarai	1124278	51	f	+	-	-	-	-	0	0	2	0	2	0	4	-	-	-	cellulitis	-
65	aravindar	1126437	44	m	+	-	-	-	-	0	1	2	2	0	0	5	+	coagulous necrosis	GAS	N.F	-
66	veeranan	1127865	72	m	+	-	-	+	+	4	1	0	2	0	1	8	+	neutrophils infilration	bacterioids	N.F	-
67	cinnaye	1130432	64	f	+	-	-	-	-	4	1	0	0	0	0	5	-	-	-	cellulitis	-
68	thiraviyam	1137641	71	m	+	-	-	-	-	0	1	1	2	0	0	4	-	-	-	cellulitis	-
69	karuppan	1138634	60	m	+	-	-	-	-	0	0	1	2	2	0	5	+	necrosed skin,fascia	polymicrobial	N.F	-
70	mookan	1140346	40	m	+	-	-	-	-	4	0	0	2	0	0	6	+	necrosed skin,fascia	E.coli	F.G	+
71	sathriyan	1140832	63	m	+	-	-	-	-	0	0	2	2	0	0	4	-	-	-	cellulitis	-
72	krishnan	1141004	54	m	+	-	-	+	+	4	1	2	0	0	1	8	+	neutrophils infilration	GAS	N.F	-
73	pandian	1141235	67	m	+	-	-	-	-	0	1	2	2	0	0	5	+	necrosed skin,fascia	peptosreptococci	N.F	-
74	thavasi	1141052	61	m	+	-	-	-	-	0	1	2	0	2	0	5	+	coagulous necrosis	klebsiella	N.F	-
75	andiappan	1142894	59	m	+	-	-	-	-	0	1	1	0	2	0	4	-	-	-	cellulitis	-
76	kulandaisamy	1142697	55	m	+	-	-	-	+	4	2	0	0	0	1	7	+	necrosed skin,fascia	GAS	N.F	-

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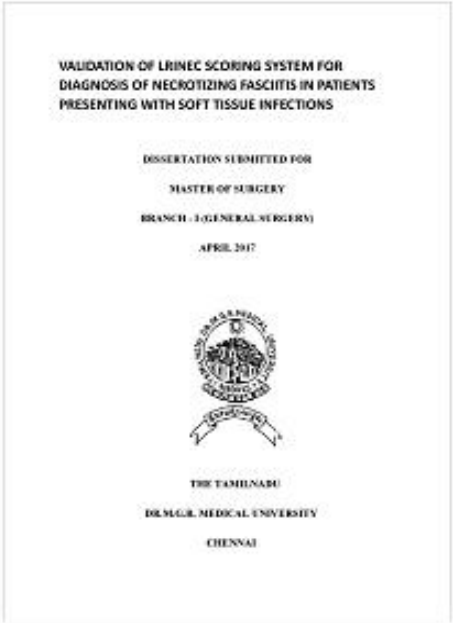

VALIDATION OF LRINEC SCORING SYSTEM FOR DIAGNOSIS OF NECROTIZING FASCIITIS IN PATIENTS PRESENTING WITH SOFT TISSUE INFECTIONS

DISSERTATION SUBMITTED FOR

MASTER OF SURGERY

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