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A REVIEW ON NECROTISING FASCIITIS

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ABSTRACT

Necrotizing fasciitis is a rare soft tissue infection that is usually caused by virulent toxin-producing bacteria and is characterized by extensive fascial necrosis with relative sparing of the underlying skin and muscles.The disease progresses quickly, and septic shock may occur; as a result, the mortality rate is high (middle mortality 32.2 percent). The nearness of co-morbidities, for example, diabetes mellitus, immunosuppression, unremitting liquor illness, persistent renal disappointment, and liver cirrhosis, makes guessing worse. Necrotizing fasciitis (NF) is one of the most difficult surgical infections a surgeon will face. The difficulty in treating this entity is due to a combination of difficulties in diagnosis and also in early and late treatment. For the patient, such a diagnosis means prolonged hospitalization, painful bandages, a longer recovery and, in some unfortunate cases, even loss of limbs or life. Necrotizing fasciitis is a fairly common disease in surgical practice in the Indian context, resulting in a great deal of clinical experience. This article provides an overview of the MEDLINE literature with the keywords "necrotizing," "fasciitis," and "necrotizing infections" from the 1970s and cross-referenced articles.

Keywords : Necrotizing fasciitis, Diabetes mellitus, Liver cirrhosis, Necrotizing infections

INTRODUCTION

Necrotizing fasciitis (NF), also known as flesh-eating disease, is a bacterial infection that results in the death of parts of the body's soft tissue.It is a rare but potentially fatal infection involving the subcutaneous tissue and fascia. It is commonly known as *flesh-eating disease.*[1]**.** It is a excessive disorder of unexpected onset that spreads rapidly.Symptoms typically consist of pink or crimson pores and skin withinside the affected area, excessive pain, fever, and vomiting.The maximum generally affected regions are the limbs and perineum. Typically, the contamination enters the frame via a wreck withinside the pores and skin which includes a reduce or burn.Risk elements consist of bad immune feature which includes from diabetes or cancer, obesity, alcoholism, intravenous drug use, and peripheral artery disorder.It isn't always commonly unfold among humans.The disorder is classed into 4 types, relying at the infecting organism. Between fifty five and 80% of instances contain multiple sort of bacteria.Methicillin-resistant Staphylococcus aureus (MRSA) is worried in as much as a 3rd of instances. Medical imaging is frequently beneficial to affirm the diagnosis. Necrotizing fasciitis can be avoided with right wound care and handwashing.It is typically dealt with with surgical procedure to take away the inflamed tissue, and intravenous antibiotics. Often, a mixture of antibiotics is used, which includes penicillin G, clindamycin, vancomycin, and gentamicin.Delays in surgical procedure are related to a far better chance of dying.Despite exquisite treatment, the chance of dying is among 25 and 35%. Necrotizing fasciitis takes place in approximately 0.four humans in keeping with 100,000 in keeping with yr withinside the US, and approximately 1 in keeping with 100,000 in Western Europe.Both sexes are affected equally.It turns into greater not unusualplace amongst older humans and is uncommon in children.It has been defined as a minimum because the time of Hippocrates.The term "necrotizing fasciitis" first got here into use in 1952.[2]

Synonyms :

- Flesh-eating bacteria.
- Flesh-eating disease.
- Streptococcal gangrene.
- Suppurative fasciitis.

HISTORY

Hippocrates described the fall of the body and there were many deaths in the 5th century BC."The first description in English of a necrotizing soft tissue infection was made in the 18th century by the British surgeon Leonard Gillespie and the British physicians Gilbert Blaine and Thomas Trotter. At that time soft tissue infections became necrotizing as" phagenetic ulcer "(ulceration that spreads and destroys surrounding tissue)," gangrenous phagenesis "," gangrenous ulcer "," malignant ulcer "," putrid ulcer "," fulminant gangrene "," necrotizing erysipelas "," gangrenous erysipelas ", "crackling cellulitis", "gangrenous cellulitis", "Meleney cellulitis", "necrotizing synergistic cellulitis", "hemolytic streptococcal gangrene", "progressive bacterial synergistic gangrene", "necrotizing abscess" Galloping "Hospital gangrene"[3]Later , "hospital gangrene" became more common.In 1871, Confederate States surgeon Joseph Jones

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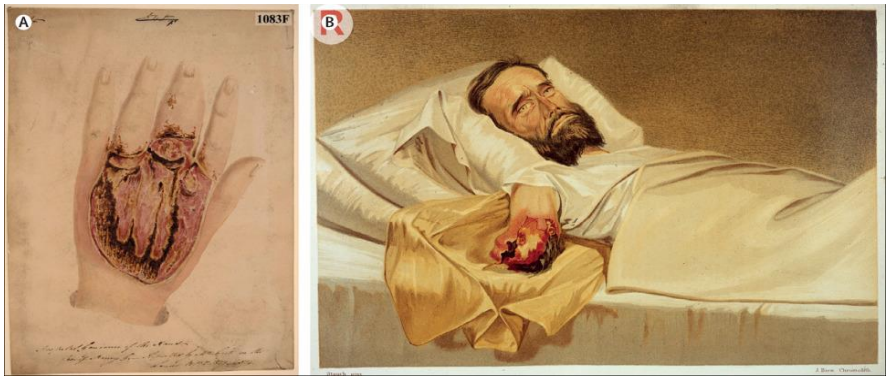
reported 2,642 cases of hospital gangrene with a mortality rate of 46%. In 1883, Dr. Jean-Alfred Fournier described the necrotizing infection of the perineum and scrotum, which is now known as Fournier's gangrene. The term "necrotizing fasciitis" was first coined in 1952 by Wilson. The definition of it has been expanded to include not only infection of the fascia, but also other soft tissue infections[4]. Although unpopular with the medical community, the term "rampant gangrene" is often used in the tabloid media to refer to outbreaks of necrotizing fasciitis.

The disease was first discovered in France in 1783 and appeared from time to time throughout the 19th and 20th centuries.The first clear description of necrotizing fasciitis was given in 1871 by Joseph Jones, a surgeon in the United States Confederate Army.The disease was generally found in military hospitals during wartime. There have been some outbreaks in public.He described it as a "hospital fire" that affected 2,642 soldiers with a death rate of 46% during the Civil War. Necrotizing fasciitis is oftencaused by group A streptococcal bacteria (GAS). This is the same type of bacteria that causes a sore throat. But different types of bacteria like staph and others have also been linked to the disease. Necrotizing fasciitis itself was described by Wilson in 1952 when he observed edema and necrosis of the subcutaneous fat and fascia with sparing of the underlying muscle in a series of 22 patients



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HISTOPATHOLOGY

Tissue removed from the operating room after debridement usually exhibits extensive superficial fascial necrosis.Most small and medium-sized blood vessels are thrombosed. Neutrophil aggregates are seen in the fascia and subcutaneous tissue.Small vessel vasculitis and extensive fat necrosis are also visible.All glands in the dermis and subcutaneous tissue are also necrotic.The Gram stain shows accumulations of different types of microorganisms

Bacteria Involved:

Types of soft tissue necrotizing infection can be divided into four classes according to the types of bacteria infecting the soft tissue. This classification system was first described by Giuliano and his colleagues in 1977.[2]

Type I infection: This is the most common type of infection, and accounts for 70 to 80% of cases. It is caused by a mixture of bacterial types, usually in abdominal or groin areas.This type of infection is usually caused by various species of Grampositive cocci, (Staphylococcus aureus, Streptococcus pyogenes, and enterococci), Gramnegative rods, (Escherichia coli, Pseudomonas aeruginosa), and anaerobes, (Bacteroides and Clostridium species). Populations of those affected are typically older with medical comorbidities such as diabetes mellitus, obesity, and immunodeficiency. Usually, trauma is not the cause of such infections[2].Previous history of abscess infection or gut perforation with bacterial translocation may be elicited.Clostridial infection accounts for 10% of type I infection. The Clostridium species involved are Clostridium perfringens, Clostridium septicum, and Clostridium sordellii, which typically cause gas burns (also known as myonecrosis).Clostridium perfringens produces two deadly toxins: alpha toxin and tetatoxin.Alphatoxin causes excessive platelet aggregation, which blocks blood vessels and deprives vital organs of oxygen supply.This creates an acidic, low-oxygen environment for bacteria to multiply. When alpha toxin is absorbed by soft tissues, it can inhibit the migration of white blood cells from blood vessels to soft tissue, impairing the function of phagocytes.The two toxins together can cause destruction of red blood cells in blood vessels, damage to the integrity of blood vessels, and suppression of heart function.Clostridiumsordellii can also produce two main toxins: all known virulent strains produce those of the lethal essential virulence factor (TcsL) toxin, and some also produce hemorrhagic

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toxin (TcsH).TcsL and TcsH are members of the large family of clostridial cytotoxins (LCC). The key virulence factor of Clostridium septicum is a pore-forming toxin called alpha-toxin, although it is not related to Clostridium perfringens alpha toxin. Myonecrotic infections caused by these types of clostridia are common in heroin users who inject themselves.People with clostridial infections often experience severe pain at the wound site, where the wound normally drains foul-smelling blood mixed with serum (serosanguineous discharge). Shock can progress rapidly after the initial injury or infection, and once shock is established, the probability of death is greater than 50%. Another bacterium that has been linked to an equally rapid disease course is a group A streptococcal infection (primarily Streptococcus pyogenes). Meanwhile, other bacterial infections can take two or more days to become symptomatic.

Type II infection: This infection represents 20 to 30% of cases and mainly affects the extremities[5].It is mainly Streptococcus pyogenes bacteria, alone or in combination with staphylococcal infections.Both types of bacteria can progress rapidly and manifest as toxic shock syndrome.Streptococcus species produce M protein, which acts as a superantigen and stimulates a massive systemic immune response that is ineffective against bacterial antigen and causesshock.A contamination by Streptococcus A (Streptococcus pyogenes) has been described in detail, either alone or in association with Staphylococcus aureus, which is classically localized on the edges of the body, but is also involved in the trunk.Gather A streptococci can survive and mimic macrophages, thus avoiding antimicrobial treatment even in those tissues that remain well supplied with blood and are considered tolerable for antimicrobial penetration.Type II is, so to speak, NSTI, which is related to toxic anesthesiadisorder.Grade II is much less common than grade I;

However, this incidence is increasing, reflecting the increasing incidence of methicillin-resistant S. aureus (MRSA) acquired in the community in some parts of the world.MRSAdiseases of sensitive tissue have been reported particularly in i.v.Drug users,competitors and institutional groups.Type II NSTIs occur regularly in healthy, adolescent, and immunocompetent patients, although there is a regular history of subsequent injury or operation to the tissue involved.Type II infection is more likely to affect young, healthy adults with a history of injury.

Type III infection:Vibrio vulnificus, a bacteria found in salt water, is a rare cause of this infection, which occurs through a tear in the skin.Type III may be gram-negative monomicrobial NF.Themost common gram-negative mindfuls are Vibrio spp., Such as V. damselae and V. vulnificus.Variety III is exceptional, but has a really high mortality rate of 30-40%, despite provocative closure and vigorous handling.The course of the disease is similar to type II, but sometimes with minor visible skin changes.[4]

Type IV infection: Some authors have described type IV infection as fungal in nature[2].Type IV Type IV shows contagious cases of Candida NF.These are exceptionally rare.Parasiticinvasion is more common in patients with traumatic wounds and burns and in patients with severe immunodeficiency.

EPIDEMIOLOGY:

In India approximately 48 cases were observed in necrotizing fasciitis.In the United States, necrotizing fasciitis affects approximately 0.4 out of 100,000 people each year[2].There are about 1,000 cases of necrotizing fasciitis per year in the United States, but the rates have increased.This could be due to increased awareness of the disorder leading to increased reporting, or due to bacterial virulence or increased bacterial resistance to antibiotics.In some parts of the world it is as common as one in 100,000 people.A higher rate of necrotizing fasciitis is seen in people with obesity or diabetes, in people with compromised immune systems or with alcohol consumption, or in people with peripheral arterialdisease.However, the disease can also occur in young, healthy adults without underlying medical conditions.NSAIDscan increase the rate of necrotizing infections by changing the immune response in the body because NSAIDs inhibit the enzymes cyclooxygenase1 and cyclooxygenase2, which are important for the production of thromboxane and prostaglandin E2.Prostaglandin is responsible for fever, inflammation, and pain. The inhibition of prostaglandin E2 production reduces inflammatory response and leukocyte adhesion, and thus reduces immune response against bacterial invasion, giving rise to softtissueinfection[4

ETIOLOGY:

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Diagnosis	Etiology	Clinical Setting
Gangrenous cellulitis +/- necrotizing fasciitis (type I necrotizing fasciitis)	Polymicrobial, including: Enterobacteriaceae facultative streptococci staphylococci anerobes (Bacteriodes species, anerobic peptococci, peptostreptococci)	
Streptococcal gangrene +/- necrotizing fasciitis (type II necrotizing fasciitis)	Group A, C, or G streptococci	Occurs on extremities at sites of trauma
Anerobic cellulitis (may complicate clostridial myonecrosis)	Clostridia species (mostly C. perfringens)	
Progressive bacterial synergistic gangrene	Microaerophilic streptococci + Gram negative bacilli and/or S. aureus	Postoperative wound complications
Synergistic necrotizing cellulitis (including Fournier's gangrene)	Polymicrobial: Bacteriodes species + peptostreptococci, Gram negative bacilli	Diabetes mellitus, obesity
Gangrenous cellulitis in immunocompromised hosts	Broad range of causative pathogens: Bacteria: Gram negative bacilli Fungi: Aspergillus, Mucor species, Rhizopus, Apophysomyces elegans	Granulocytopenia, immunosuppression, burns, diabetes mellitus
Gangrene (skin necrosis) complicating conventional cellulitis	Group A, C, or G streptococci	Severe conventional cellulitis

Necrotizing fasciitis is typically an acute process that occurs rapidly over several days.It is the direct result of a bacterial infection, which is caused by a breakdown in the integrity of the skin in approximately 80% of all cases.Gram-positive cocci, particularly Staphylococcus aureus and streptococci, are responsible for most of these single-sourceinfections.Polymicrobial infections also occur due to a combination of gram-negative and anaerobic disease. Most patients have diabetes and a history of alcoholism.Cirrhotic patients are also prone to necrotizing fasciitis.[6][7]

CAUSES

Necrotizing fasciitis is commonly caused by group A streptococcus (GAS) bacteria.Necrotizing fasciitis happens when these type of bacteria contaminate the shallow belt, a layer of connective tissue underneath the skin.Spreading of contamination through blood has been proposed for those with streptococcal pharyngitis. This isn't the as it were sort of microbes that can cause this disease. Other microscopic organisms that can cause necrotizing fasciitis include

- Aeromonas hydrophila
- Clostridium
- E.coli
- Klebsiella
- male genitalia are genitourinary
- infections and trauma. A urinary tract infection[8]
- Staphylococcus aureus
- The disease general causes
- Trauma causes the disease, from intravenous medicate infusion, affront infusion, creature and creepy crawly nibbles,
- Catheter inclusion over the skin .
- Cancer and ulcers
- spreading inflammation and necrosis of the skin[9]
- Urinary tract contamination,
- Stones,
- Bartholin organ sore are the normal cause
- side effects in the head and neck locale fair earlier to onset of necrotizing fasciitis[10]

SYMPTOMS

Symptoms can include fever, swelling, and complaints of excessive pain.Initial skin changes resemble cellulitis or an abscess, making it difficult to diagnose in the early stages.Hardening of the skin and soft tissues and swelling beyond

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the area of lesions are common in patients with early necrotizing changes.The redness and swelling usually pass into the surrounding normal tissue.The overlying skin can appear shiny and tight. The skin has redness, swelling and feels hot to touch. Patient has fever with chills. Patient moreover encounters queasiness, heaving and loose bowels.[11]
Rapid progression to shock despite antibiotic therapy is another indication of necrotizing fasciitis. Necrotizing changes in the groin are known as Fournier's gangrene. bacterial toxicity and extracellular fluid[12] .[However, 4,444 people with a weakened immune system (cancer, corticosteroid use, radiation therapy, chemotherapy, HIV / AIDS, or previous organ or bone marrow transplantation) may not have typical symptoms. Immunosuppressed people are also twice as likely to die from necrotizing infections, so greater suspicion should be maintained in this group

MODE OF TRANSMISSION:

It is mostly caused by gram negative microbios mainly e. coil[13]which causes necrotizing fasciitis, most often enters the body through a wound in the skin, such as a skin wound.Aside from necrotizing fasciitis caused by Vibrio vulnificus, you can also eat raw or undercooked fish, such as shellfish, in warm waters.Necrotizing fasciitis is passed from one individual to another from time to timeThe bacteria that cause necrotizing fasciitis can be transmitted from one individual to another through close contact, such as touching the wound of the infected individual.Although anyone can develop necrotizing fasciitis, it is rare. Most people with this disease have other health problems that can affect their body's ability to fight infections. Some diseases that weaken the body's immune system are:Direct **mode** of bacterial **transmission** is more common[14]

PATHOPHYSIOLOGY

Necrotizing fasciitis is a threatenina bacterial infection causing necrosis of the fascia , underlying skin and vasculature
↓
Necrotizing fasciitis (NF) could be a dynamic and fulminant bacterial disease of the subcutaneous tissue that spreadsrapidly through the fascia and causes extensive tissue destruction.
↓
NF can affect any part of the body and is the main serious induction of Necrotizing Sensitive Tissue Disease (NSTI); it could be an unusual but potentially fatal condition.
↓
Promoting recognition and mediation are essential since mortality depends specifically on the time of intervention.[15]
↓
The effect of bacteria Microbial attack of the subcutaneous tissue occurs through an external injury or the coordinated spread of a perforated viscus (especially large intestine, rectum) or a urogenital organ.
↓
Bacterial growth within the flat belt releases a mixture of proteins and endo and exotoxins that cause the spread of contamination through this fascia.
↓
This grip is caused by poor microcirculation, ischemia in affected tissues, and ultimately, the passage of cells and putrefaction.
↓
Thromboses of small veins and ducts that cross the wing cause significant skin ischemia.
↓
This cutaneous ischemia is the basic treatment for the introduction of NF into the soft tissues as it progresses.
↓
It is essential that clearly normal-looking skin can be seen in the early neurotic stages despite widespread contamination of the base fundamental sash.
↓
Haemorrhagic bullae, ulceration, and changes in the skin appear when the deeper structures are prematurely involved.
↓
The incipient cutaneous clinical discoveries diminish the clinical picture of the tissue disease, although the thrombosis of the vessels that penetrate the skin is the key to the pathology of the ITSN by putrefaction that takes place [16].

EVALUATION

Any rapidly progressing skin or soft tissue infection should be treated aggressively, as it is difficult to distinguish betweennonnecrotizingand necrotizing skin and soft tissue infections.[17][18].The Laboratory Risk Indicator for Necrotizing Infections (LRINEC) score was developed in a 2004 report to distinguish NSTIs from other serious soft tissue infections. The rating system depends on anomalies in six independent variables:

C- protein, mg / L: Less than 150More than 150

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Total white blood cells (WBC), cells / mm : Less than 15More than 25
Hemoglobin, g / Dl: More than 13.5Less than 11
Sodium, mmol / L :135 or more Less than 135
Creatinine, mg / dL :1.6 or less (0)more than 1.6 (2)
Glucose, mg / dL:180 or less (0)More than 180 (1)

A value of six has a positive predictive value of 92% and a negative predictive value of 96%.A score of eight or more means a 75 percent risk of necrotizing infection. The diagnosis of NSTIs remains primarily clinical.Images can be helpful in providing data when the diagnosis is uncertain. The most common simple radiographic finding is similar to cellulitis with increased thickness and opacity of the soft tissues.Computed tomography (CT) has a higher sensitivity for identifying necrotizing soft tissue infections than normal film.Plain radiographs are of no diagnostic value.Sometimes, under local anesthesia, the area can be examined with a finger for signs of necrotizing tissue.In most cases, necrotic tissue can be penetrated with little resistance. Aspiration and Gram stain can also be performed.The use of B-mode color Doppler ultrasound can aid in the early detection of bedside necrotizing fasciitis. It should be understood that no laboratory or imaging test should delay surgical interventions.

RISK FACTORS:

Risk factors for necrotizing fasciitis includes

- Diabetes.
- Immunosuppressive drugs (eg, prednisolone)
- Malnutrition.
- Age > 60 years.
- Intravenous drug misuse.
- Peripheral vascular disease.
- Renal failure.
- Hypertension.[29]
- Chronic disease.
- Immunosuppressive drugs (eg, prednisolone)
- Malnutrition.
- Age > 60 years.
- Intravenous drug misuse.
- Peripheral vascular disease.
- Renal failure.
- Hypertension.[19]

In some studies it was found that other underlying diseases such as chronic heart disease or cirrhosis of the liver were associated with higher death rates.4,444 patients with liver cirrhosis have a higher susceptibility to infection than patients without liver cirrhosis.The mechanism of increased mortality in cirrhotic patients with infection can be immunological and mechanical.Abnormalities affect immunity in cirrhotic patients with cellular and humoral immunity, dysfunction of T lymphocytes and B lymphocytes due tomalnutrition. The abnormal defense mechanism could be caused by a decrease in phagocytic activity of the reticuloendothelial system, alteration of monocyte function and incomplete chemotaxis. And also found that skin necrosis was associated with higher mortality.Infections and toxin-producing bacteria can cause skin necrosis and multiple organ failure .All necrotic tissue, including fascia, should be removed by surgical debridement to reduce bacteria, and broad-spectrum antibiotics should be administered.

DIAGNOSIS

Early diagnosis is difficult, as the disease often looks early on like a simple superficial skin infection.While a number of laboratory and imaging modalities can raise the suspicion for necrotizing fasciitis, none can rule it out.[20]The gold standard for diagnosis is a surgical exploration in a setting of high suspicion. When in doubt, a small incision can be made into the affected tissue, and if a finger easily separates the tissue along the fascial plane, the diagnosis is confirmed and an extensive debridement should be performed.

Doctors can also diagnose necrotizing fasciitis by taking a tissue sample, in addition to looking at the injury or illness (biopsy) Bloodwork is being examined for symptoms of infection and muscle injury. Imaging of the injured area (CT scan, MRI, ultrasound).

Medical Imaging:Imaging has a constrainedpositionwithinsidetheanalysis of necrotizing fasciitis.The time postpone in appearing imaging is a chiefconcern.Plain radiography may alsodisplay subcutaneous emphysema (fuelolinewithinside the subcutaneous tissue), that is strongly suggestive of necrotizing adjustments, however it isn'ttouchysufficient to discoverall of the cases, due to the fact necrotizing pores and skin infections resulting frommicroorganismapart from

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clostridial infections generally do now no longerdisplay subcutaneous emphysema.If the analysisremains in doubt, computed tomography (CT) scans and magnetic resonance imaging (MRI) are greatertouchy modalities than undeniableradiography.However, each the CT experiment and MRI aren'ttouchysufficient to rule out necrotizing adjustmentscompletely.CTexperimentmayalsodisplay fascial thickening, edema, subcutaneous fueloline, and abscess formation.In MRI, while fluid series with deep fascia involvement occurs, thickening or enhancement with assessment injection, necrotizing fasciitis have to be strongly suspected.Meanwhile, ultrasonography can display superficial abscess formation, howeverisn'ttouchysufficient to diagnose necrotizing fasciitis.CTexperiment is capable ofdiscoverapproximately 80% of cases, even as MRI may alsoselect slightly more..[21]

Scoring System :A white blood cell count of more than 15,000 cells / mm3 and a serum sodium level of less than 135 mmol / L have a 90% sensitivity in detecting changes from necrotizing soft tissue infection if the values showotherwise.Variousclassification systems are being developed to determine the likelihood of necrotizing fasciitis, but a classification system developed by Wong and his colleagues in 2004 is the most widelyused.It is the Laboratory Necrotizing Fasciitis Risk Indicator (LRINEC) that can be used to stratify people with signs of severe cellulitis or an abscess by risk to determine the likelihood of necrotizing fasciitis.It uses six laboratory values: reactive protein, total white blood cells, hemoglobin, sodium, creatinine, and blood sugar.A score of 6 or higher indicates that necrotizing fasciitis should be seriously considered.Theevaluation criteria are:

CRP (mg/L) ≥150: 4 points
WBC count (×103/mm3)
<15: 0 points
15–25: 1 point
>25: 2 points
Hemoglobin (g/dl)
>13.5: 0 points
11–13.5: 1 point
<11: 2 points
Sodium (mmol/l) <135: 2 points
Creatinine (umol/l) >141: 2 points
Glucose (mmol/l) >10: 1 point

However, the rating system has not been validated.Values would be false positives if other inflammatory conditions werepresent.Therefore, the values derived from this rating system should be interpreted with caution.Approximately 10% of the patients with necrotizing fasciitis in the original study still had a LRINEC score of <6. A validation study showed that patients with a LRINEC score ≥6a had higher rates of death and amputation.[22]

TREATMENT

These patients are extremely ill and should be transferred immediately to the intensive care unit.The sepsis causes refractory hypotension and diffuse capillary leak. Thus the patient will need aggressive resuscitation with fluids and use of inotropes to maintain blood pressure.The patient must be kept NPO (nothing by mouth) until seen by the surgeon. Nutrition is vital but only after surgery has been completed.Enteral feedings should be started as soon as the patient is hemodynamically stable.The enteral feedings may help offset the massive negative protein balance that occurs as a result of catabolism..[23]

The key concepts for the treatment / management of skin and soft tissue infections are: Early diagnosis and differentiation between necrotizing and non-necrotizingSSTIs.The early introduction of adequate empirical antibacterial coverage (broad spectrum)Appropriate source control of infection such as aggressive surgical procedures for drainage and debridement of abscesses from necrotizing soft tissue infections (NSTI).Identify the causative agent and adjust antimicrobial coverage accordingly. Antimicrobial therapy for necrotizing fasciitis is as follows: Linezolid 600 mg twice daily AND Piperacillin / Tazobactam 4/0 to 5 g LD infused over 30 min, then 16/2 g once daily via CI OR Daptomycin 6 mg / kg once daily, AND piperacillin / tazobactam 4/0 to 5 g LD infused over 30 min, then 16/2 g once daily with CI AND clindamycin 600 mg to 900mg four times a day.

SURGERY

Aggressive wound debridement should be done early, usually as soon as a diagnosis of necrotizing soft tissue infection (NSTI) is made.Surgicalcuts often go beyond the area of hardening (the hardened tissue) to remove the damaged blood vessels that are responsible for the hardening. The soft tissues of cellulite, however, are sometimes avoided from debridement to later cover the wound skin.Morethan one surgery can be used to remove additional necrotic tissue. In some cases, when a limb is affected by NSTI, amputation may be the surgical treatment of choice.Afterdebridement of the wound, appropriate dressings should be applied to avoid exposure of bones, tendons and cartilage so that these structures do not dry out and wound healing is promoted.In necrotizing infections of the perineal area (Fournier's

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gangrene), debridement and wound care in this area can be difficult due to excretions that often contaminate the area and interfere with the wound healingprocess.Therefore, regular dressing changes with a fecal management system can help keep the wound in the perineal area clean. Sometimes a colostomy may be required to drain waste to keep the wound at the perineal area clean.[34]

SOFT TISSUE RECONSTRUCTION

Once all the necrotic tissue is removed and there are signs of granulation tissue, the plastic surgeon should be consulted.In most cases, primary closure is not possible and therefore the plastic surgeon may need to reconstruct the soft tissue and close the wound with a muscle flap.Ifnot enough natural skin is available for a skin graft, it may be necessary to use artificial skin.Another method of treatment is hyperbaric oxygenation.Although the literature suggests that this modality can be used, the majority of these patients are in intensive care units connected to a variety of medical devices, making travel to the hyperbaric oxygen therapy facility difficult.Hyperbaric oxygen therapy can be effective for small wounds, but for large wounds there is no evidence that this therapy improves healing or prolongs life.Finally, it should be noted that hyperbaric oxygen therapy is a complementary treatment and does not replace surgical debridement.Treatment with HBO may be helpful when the patient is stable.Some data show that this treatment can help reduce mortality.HBO is not a substitute for surgery but a complementary treatment.

Antibiotics

Empiric antibiotics are usually initiated as soon as the diagnosis of NSTI has been made, and then later changed to culture-guided antibiotic therapy. In the case of NSTIs, empiric antibiotics are broad-spectrum, covering gram-positive (including MRSA), gram-negative, and anaerobic bacteria.While the studies compared moxifloxacin (a fluoroquinolone) and amoxicillin clavulanate (a penicillin) and assessed the appropriate duration of treatment (ranged from 7 to 21 days), no definitive conclusions could be drawn about the effectiveness of treatment, duration ideal treatment or side effects due to worse Evidence made.[35]

ADD ON THERAPY:

Hyperbaric oxygen: Although studies in humans and animals have shown that high oxygen tension in the tissues reduces edema, stimulates fibroblast growth, increases the destruction capacity of white blood cells, inhibits the release of bacterial toxins and increases the efficacy of antibiotics, not Quality studies have shown to support or refute the use of hyperbaric oxygen therapy in patients with SNIs.

Intravenous immunoglobulin (IVIG):In the treatment of NSTIs, no clear difference has been shown between the use of IVIG and placebo, and one study showed serious side effects with the use of IVIG, including acute kidney damage, allergic reactions , aseptic meningitis. syndrome, hemolytic anemia, thrombi and transmissible pathogens.

AB103:A study looked at the effectiveness of a new type of treatment that affects the immune response called AB103. The study showed no difference in mortality with this therapy, but definitive conclusions are difficult due to poor-quality evidence.

Supportive therapy: Supportive therapy, often including intravenous hydration, wound care, anticoagulants to prevent thromboembolic events, pain control, etc. it should always be provided to patients when appropriate.

Since ischemia and hypoxia interfere with adequate administration of antibiotics to the site of infection, conservative treatment with antibiotics alone is of little value in the treatment of NF .However, they play an important role in the surgical treatment of the infection. Patients should be treated immediately with broad-spectrum antibiotics if NF is suspected.The empirical use of antibiotics is based on the NF microbiological classification.Antibiotic treatment for polymicrobial infection should be based on medical history, Gram stain, and culture. Initial treatment includes ampicillin or ampicillin sulbactam in combination with metronidazole or clindamycin .Anaerobic coverage is very important in type 1 infections; Metronidazole, clindamycin, or carbapenems (imipenem) are effective antimicrobial agents.Extensive gram-negative coverage is required as first empirical therapy for recently treated with antibiotics or hospitalized patients. In these cases, antibiotics such as ampicillin-sulbactam, piperacillin-tazobactam, ticarcillin-clavulanic acid, third or fourth generation cephalosporins, or carbapenems in higher doses are used.

■Type 2 disease is treated with antibiotics against S. pyogenes and S. aureus, which normally coexist with the former.

Therefore, first or second generation cephalosporins are used to coat methicillin-sensitive Staphylococcus aureus (MSSA).

MRSA is usually covered by vancomycin or daptomycin and linezolid when S. aureus is resistant to vancomycin.

Some studies suggest that clindamycin is superior to penicillin in treating streptococcal infections , but this has not yet been satisfactorily demonstrated.

Another study has suggested that clinicians should consider adding clindamycin to beta-lactam antibiotic if NF or myositis is present .

■ Type 3 NF should be treated with clindamycin and penicillin, which cover Clostridium species.

If Vibrio is suspected, early use of tetracyclines (including doxycycline and minocycline) and cephalosporins is 3.

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■ Finally, NF type 4 can be treated with amphotericin B or fluoroconazoles, but the results of this treatment are generally disappointing.

Differential Diagnosis :

- ✓ Cellulitis.
- ✓ Epididymitis.
- ✓ Gas gangrene.
- ✓ Orchitis.
- ✓ Testicular torsion.
- ✓ Toxic shock syndrome.[24]

Complications:

- ✓ Multiorgan failure.
- ✓ Septic shock.
- ✓ Loss of extremity.
- ✓ Severe scarring.
- ✓ Toxic shock.
- ✓ Death..[24]

Preventive Measures:

- ➢ There is no sure way to prevent necrotizing fasciitis infection.
- ➢ However, you can reduce your risk by taking basic hygiene measures.
- ➢ Wash your hands frequently with soap and treat all wounds right away, including minor ones.
- ➢ If you already have a wound, take good care of it.
- ➢ Perform foot and skin checks.
- ➢ Carefully care for wounds and surgical sites.
- ➢ Wash and cover small cuts and scratches.
- ➢ Avoid sharing personal effects.
- ➢ Know your risk factors.

ENHANCING HEALTH CARE TEAM AND OUTCOMES:

Necrotization is a life-threatening condition with a very high mortality rate. Any delay in diagnosis or treatment generally leads to a poor prognosis.The disorder is best treated with a team of healthcare professionals that includes a urologist, general surgeon, infectious disease expert, intensive care physician, nephrologist, intensive care nurses, and radiologist.Necrotization is a life-threatening condition with a very high mortality rate. Any delay in diagnosis or treatment generally leads to a poor prognosis.The disorder is best treated with a team of healthcare professionals that includes a urologist, general surgeon, infectious disease expert, intensive care physician, nephrologist, intensive care nurses, and radiologist.Theroles of the nurse and the pharmacist are also crucial. The nurse is often the first to recognize that the patient is seriously ill or in pain.The nursing staff should be aware of necrotizing fasciitis and consult the surgeon as soon as possible.The pharmacist should review the culture results and make sure the patient is receiving the properantibiotics.The patient should be kept in NPO, hydrated and immediately covered with broad-spectrumantibiotics.The pharmacist must check the cultures and make sure the correct antibiotics are being used to cover the pathogen.Since many of these patients also require a stool drainage to avoid contamination of the perineum, the ostomy nurse should be consulted. These patients are best treated in the intensive care unit until signs of toxicity disappear.A wound care nurse is mandatory because most patients have large open wounds that must be bandaged daily for weeks or months.Wounds often require reconstructive surgery. Only a closely cooperative systemic approach can reduce mortality from this disease..[25][26]Necrotizing fasciitis is a serious disease with a mortality rate of 30% to 90%. Ultimately, mortality depends on the age of the patient, the type of organism, the speed of diagnosis and treatment, and the patient'scomorbidity.The worst prognosis is in patients with specific strains of streptococci.Other factors that negatively affect prognosis include loss of consciousness, shortness of breath, kidney failure, and ARD.Survival is better for patients receiving immediate radical debridement, hydration, and broad-spectrumantibiotics.Even after treatment, survivors of the disease tend to have a shorter life expectancy than their age-matched controls..[27][28]

CONCLUSION:

Necrotizing fasciitis is an uncommon disease that results in gross morbidity and mortality if not treated in its early stages.At onset, however, it is difficult to differentiate from other superficial skin conditions such as cellulitis.Necrotizing fasciitis (NECK-re-tie-zing FASH-e-i-tis) is a rare bacterial infection that spreads quickly in the body.These bacteria are found on the skin or in the nose and throat of healthy people.Many people carry these bacteria

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but don't get sick.These bacteria can also cause strep throat, scarlet fever, skin infections and rheumatic fever.Accurate diagnosis, rapid antibiotic treatment, and prompt surgery are important to stopping this infection.Accurate and prompt diagnosis, treatment with intravenous (IV) antibiotics , and surgery to remove dead tissue are vital in treating necrotizing fasciitis.As the blood supply to the infected tissue becomes impaired,antibiotics often cannot penetrate the infected tissue.

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