Clinical Journal of Plastic and Reconstructive Surgery
An official Journal of KGMU Plastic Surgery Education Foundation

REVIEW ARTICLE
Volume 3 | Issue 1 | January-June 2025
www.cjprs.in

Necrotizing Fasciitis: Epidemiology, Diagnosis and Management

Vineeta Mittal[1], **Vikramjeet Singh**[1], **Vidush Kumar**[2]

Keywords:
Hyperbaric oxygen therapy, LRINEC score, Necrotizing fasciitis, Polymicrobial infection.

doi: 10.61081/cjprs/3v1i105

ABSTRACT

Necrotizing fasciitis (NF) is a fulminant soft tissue infection characterized by rapid fascial and subcutaneous necrosis. Although classically associated with toxin-producing bacteria, fungal pathogens are increasingly recognized as emerging causative agents. The disease typically advances within hours of symptom onset, underscoring the importance of early recognition and intervention. Diagnostic delay is common, as superficial findings often underestimate the underlying extent of infection. Severe pain disproportionate to clinical examination, along with signs of systemic toxicity, should alert clinicians to the possibility of NF. Management requires broad-spectrum antimicrobial therapy, urgent and radical surgical debridement, and intensive supportive care. Despite timely treatment, multiple debridements and prolonged hospitalization are frequently necessary. Mortality remains high, ranging between 30% and 90%, and is strongly influenced by age, comorbidities, infecting organisms, and time to definitive therapy. This review summarizes the epidemiology, etiology, and pathogenesis of NF; highlights advance in microbiological understanding; and discusses diagnostic challenges and evolving treatment strategies. Particular emphasis is placed on phased management, the role of multidisciplinary teams, and implications for patient outcomes.

Clinical Journal of Plastic and Reconstructive Surgery, 2025;3(1).

INTRODUCTION

Necrotizing fasciitis (NF) is a rapidly progressive and invasive infection of the skin, subcutaneous tissue, and deep fascia, with relative sparing of muscle. It is a critical surgical emergency due to its aggressive spread and potential for high mortality.[1]

Descriptions of NF can be traced back to antiquity. Hippocrates, in the 5th century BC, provided one of the earliest clinical accounts. During the American Civil War, Joseph Jones described it as "hospital gangrene" in 1871. Fournier later documented necrotizing infections of the perineum in 1884, while Meleney reported an epidemic of hemolytic streptococcal NF in Beijing in 1924. The currently accepted term, "Necrotizing Fasciitis," was first introduced by Wilson in 1952.[2,3]

Epidemiology

Globally, NF is relatively rare, with 500–1000 new cases reported annually. Its prevalence is estimated at 0.40 per 100,000 population. Men are affected more often than women, with a male-to-female ratio of approximately 3:1, largely attributable to the higher incidence of Fournier's gangrene in men. Although the disease can occur at any age, middle-aged and elderly patients are most commonly affected.[4]

Microbiology

NF may present as either a polymicrobial or monomicrobial infection. Polymicrobial infections often involve anaerobes and Gram-negative bacilli,

[1]*Department of Microbiology, Dr Ram Manohar Lohia Institute of Medical Sciences, Lucknow, UP, India*
[2]*Department of Plastic Surgery, AIIMS, Bhubaneswar, Odisha, India*
***Correspondence:** *Vineeta Mittal (vineetamittal@yahoo.co.in)*
***Funding:** None declared.*
***How to cite:** Mittal V, Singh V, Kumar V. Necrotizing Fasciitis: Epidemiology, Diagnosis and Management. Clinical Journal of Plastic and Reconstructive Surgery. 2025;3(1):18–26*

particularly members of the Enterobacteriaceae family. Monomicrobial cases are most frequently caused by Group A β-hemolytic *Streptococcus pyogenes* (GAS) or community-acquired methicillin-resistant *Staphylococcus aureus* (CA-MRSA).[5] GAS is the leading pathogen, implicated in up to 60% of NF cases. M1 and M3 serotypes are particularly virulent, producing pyrogenic exotoxins that drive rapid fascial spread and systemic illness. Individuals lacking protective immunity against streptococcal M protein or exotoxins are especially vulnerable to streptococcal toxic shock syndrome.

Clostridial species such as *Clostridium perfringens, Clostridium septicum, Clostridium histolyticum, Clostridium sordellii, Clostridium novyi*, and *Clostridium fallax* are also important pathogens. These organisms secrete a wide spectrum of toxins and enzymes—including collagenases, phospholipases, and hyaluronidases—that disrupt connective tissue integrity, promote rapid tissue necrosis, and cause gas gangrene. Infections caused by anaerobes are often fulminant, with high mortality if not treated promptly, especially within the first 48 hours.[6]

CA-MRSA has emerged as a significant cause of NF in recent years, due in part to production of Panton–Valentine leukocidin (PVL). Hyperacute cases associated with *Vibrio vulnificus, Vibrio parahaemolyticus,* and *Vibrio damsela*are linked to exposure to warm coastal waters in Asia and South America, although infections have also been reported in cooler European regions such as Scandinavia, Belgium, and New England.[7] Rare but severe NF caused by *Acinetobacter baumannii* has been reported, particularly in polytrauma patients.[8] In addition, multidrug-resistant pathogens such as *Proteus mirabilis* and *Escherichia coli* have been associated with NF following elective orthopedic and general surgical procedures, leading to worse clinical outcomes.[9] Fungal NF, though uncommon, is increasingly recognized. *Apophysomyces variabilis*, a soil-dwelling fungus found in tropical and subtropical regions, has been reported as a causative agent. Rodriguez and colleagues described NF due to *A. variabilis* in an immunocompetent individual following severe trauma.[10]

Pathogenesis

A breach in the mucous membrane barrier of gastrointestinal tract and genitourinary tract leads to entrance of microorganisms which causes NF. Pyrogenic exotoxins acts as superantigens and bind to the Human leukocytic antigen complex class II portion of antigen presenting cells and Vβ segments of the T-cell receptor in a restricted pattern. This is associated with release of interleukin 1,6 and tumour necrosis factor along with lymphokines like interleukin 2 and interferon. Thrombosis of subcutaneous blood vasculature due to release of these cytokines results in gangrene development in overlying skin. In cases of delayed diagnosis and treatment a surge in mortality and morbidity is observed. Patients suffering from diabetes mellitus, peripheral vascular disease or intravenous drug abusers usually diagnosed late and cause diagnostic dilemma for clinicians. Groin, abdomen and extremities are commonest sites involved in these patients.[11]

Classification

Necrotizing fasciitis is classified on the basis of area involved and microorganisms responsible for causation of NF. Idiopathic NF is defined as NF occurring without any initiating event. According to the body area involved with the NF it is classified into: 1) Cervical necrotizing fasciitis; 2) Abdominal necrotizing fasciitis (Meleney's gangrene); 3) Fournier's gangrene.[12] On the basis of microorganism responsible for causation of NF; it is classified into four groups (Table 1).

Type I Necrotizing Fasciitis

This is polymicrobial in origin, often bowel flora derived; tissue bacterial culture will show growth of both anaerobes and aerobes. This is most common type of NF; frequently occurring in patients with diabetes mellitus. Perineum and trunk of the body are most commonly affected in NF type I. Newborns suffering from omphalitis may have Type I NF as life-threatening complication. Type I NF infection is most common type approximately 55–75% of all NF.[12]

Type II Necrotizing Fasciitis

It is a monobacterial, skin or throat derived infection, the causative organism associated with this NF will be either Group A/B hemolytic Streptococci

Table 1: Types of Necrotizing Fasciitis.[41]

Microbiological type	Pathogens involved	Site of NF infection	Co-morbidities associated	Clinical progress
Type I (polymicrobial)	Obligate and facultative anaerobes	Trunk and perineum	Diabetes mellitus	More indolent, better prognosis, easier to recognize clinically
Type II (monomicrobial)	Beta-hemolytic streptococcus A	Limbs	Trauma	Aggressive presentations, easily missed
Type III	*Clostridium* species Gram-negative bacteria *Vibrio* spp.	Limbs, trunk, and perineum	Trauma Seafood consumption (for *Aeromonas*)	Water contamination wounds
Type IV	*Candida* spp., *Aspergillus,* Zygomycetes	Limbs, trunk, perineum	Immunosuppression	Aggressive with rapid extension especially if immunocompromised

or Staphylococci. Isolated cases of Type II NF due to MRSA are increasing alarmingly in recent years.[12] Type 2 NF commonly occurs in young and immunocompetent patients. Extremities are frequently involved with this infection. Usually, these patients have toxic shock syndrome and multiple organ dysfunctions.[13,14]

Type III Necrotizing Fasciitis

Gram-negative bacteria like *Vibrio* spp and *Aeromonas* spp are responsible for this fulminant type of NF. Type III NF occurs after punctured wound from fish or cut injury exposed to the seawater. It leads to hyper acute infection, causing septic shock and Multi organ dysfunction within 12 to 24 hours of injury; a prompt diagnosis is warranted, if not detected early it will lead to mortality.[13-15]

Type IV Necrotizing Fasciitis

Immunocompromised or severe trauma patients usually presents with Type IV NF. It is caused by fungal infection. Fungal organisms responsible for this type of NF have propensity for rapid and severe spread. Fungal agents may be Candida *spp* or *Aspergillus* spp or *Mucormycosis.* [16,17]

Predisposing Factors

It includes intravenous drug use, surgery, peripheral vascular disease, diabetes mellitus, penetrating traumatic injury to the abdomen or diabetes mellitus.[11,18] It may gain entry through portals like minimal tear during trauma or operation, malignancy, diverticulum, haemorrhoidal, urethral tear leading to leakage in the perineal region causing Fournier's gangrene, this condition is characterized by enlarged swelling in scrotum and penis with extensive spread to abdominal wall, perineum or legs. It may also be associated with childbirth, varicella infection or muscle strain.[19] Wong *et al.* reported diabetes mellitus as the commonest morbidity in 71% of the studied population of Singapore.[11] In the same study, history of substance abuse in the form of alcoholism was observed in only 3% population but 23% of total patients presented with peripheral vascular disease. In contrary to the study of Wong *et al.*, Park *et al.* found that diabetes mellitus accounted for only 15.2% cases and major cause of morbidity was alcoholism in 53.5% of studied population.[20]

Clinical Features

NF most commonly involves extremities due to more exposure during trauma. Prompt diagnosis may be delayed when pain or unexplained pyrexia is the only presentation. Most common presentation of NF is severe pain with swelling and redness in 78%–100% of patients. Fever is the second most common symptom seen in 86% of NF patients while 33% of patients will have hypotension. Local crepitus and blistering are commonly seen in 40% of NF patients. At the time of presentation 24% and 23% of them had local hypoesthesia and disorientation respectively.[21] If untreated in first 36 hours, swelling then starts developing followed by brawny oedema and tenderness. As the disease progresses epidermis becomes dark red and indurated along with blue or purple fluid filled bullae in next 3 to 4 days.

Later the friable skin becomes maroon or black colored. At the same time extensive thrombosis of dermal papillae occurs.[22] NF infection rapidly progresses to deep fascia through venous channels and lymphatics causes the tissue to take on a brownish gray appearance. In later stages patient becomes toxic and suffers from shock and multiorgan failure.[23]

Periorbital NF can occur at any age after any trauma to soft tissues of orbit or face. There is absence of subcutaneous tissue in skin of eyelids. The skin and the underlying periorbita are separated by a barrier formed by blood vasculature of orbicularis, thus restricting the orbital spread of infection. In periorbital NF, infection begins as a superficial cellulitis after any trauma due to breach in the orbicularis barrier. As periorbital NF progresses with alarming rapidity to the superficial and deep fascial planes, it leads to subtle or non-specific (e.g., pain, localised tenderness, and oedema) signs and symptoms to severe features like necrosis, blistering, along with systemic toxicity. If left untreated, periorbital NF progresses to complications like blindness, meningitis, orbital lesions and other neurological disorders, and finally death. An aggressive approach warranted for periorbital NF treatment that includes antibiotics administration and surgical debridement.[24]

DIAGNOSIS

a) Haematological Findings – Though the findings are not specific, yet it includes an elevated leucocyte count, blood urea nitrogen and serum creatinine levels, abnormal coagulation profiles, and decreased platelet and fibrinogen levels. Hyperglycemia, elevated lactate levels, hypocalcemia, hypoalbuminemia, and anemia are other laboratory findings commonly observed in NF patients.

Based on laboratory tests routinely performed for the diagnostic evaluation of severe soft tissue infections, Wong et al developed scoring system for distinguishing and diagnosing NF from other soft tissue infections. The laboratory risk indicators for necrotizing fasciitis (LRINEC) score is a numerical score with a maximum total of 13. If LRINEC score is 5 or less, than it has low risk for development of NF. A score of 6 to 7 has intermediate risk with 50–75% chances of NF. A LRINEC score of 8 and above

Table 2: Variables of LRINEC score.

Value	LRINEC Score points	Value	LRINEC Score points
1. C-Reactive Protein (mg/L)		4. Sodium level (mmol/L)	
<150	0	>135	0
>150	4	<135	2
2. White Blood Corpuscles counts (cells/mm^3)		5. Creatinine level (mg/dL)	
< 15	0	<1.6	0
15–25	1	>1.6	2
➢ 25	2		
3. Hemoglobin level (g/dL)		6. Glucose level (mg/dL)	
>13.5	0	<180	0
13.5–11	1	>180	1
<11	2		

observed to have stronger positive predicative value with a specificity of 95%. (Table 2) The LRINEC score is easy to calculate and facilitate early diagnosis.[25]

CRP plays an important but non-specific diagnostic marker in early detection of NF. In a study by Thomas et al, Patients suffering from cellulitis usually shows an elevated CRP of 49 mg/dL. NF patients displayed more than 5-fold higher CRP levels with 254 mg/dL (median, range 72 to 592) in this study of Thomas *et al.,* he observed statistically significant difference in CRP levels of cellulitis and NF patients (p <0.001).[26]

Role of Procalcitonin in NF – Procalcitonin (PCT) is defined as precursor hormone of calcitonin, which is involved in the calcium homeostasis. Its normal level in the serum is as low as 0.01-µg/mL. PCT acts as proinflammatory stimulus, and their levels are raised during bacterial infections and related sepsis. Elevated PCT represents severe progressive tissue damage and thus, indicates worsening of the underlying NF. PCT evaluation on postoperative day acts as a significant indicator of successful surgical intervention. PCT is not only an important diagnostic marker but also it helps in foreseeing the prognosis of organ damage and predicts mortality.[27]

b) Microbiological diagnosis- Sample collection – Purulent wound specimens characterized by ulceration or necrosis with little moisture, can be collected with two swabs, but this is generally inferior

to aspiration or biopsy. Deep lesions that communicate with the surface are most problematic. Surgical debridement and sampling are recommended. If surgery is not performed, effort should be made to aspirate a "pocket" of infected material that is not open to the surface. As a last option, fresh specimen should be expressed from deep within the wound. A swab used to collect specimen from the surface overlying the draining wound is not acceptable. Only deep specimens collected by aspiration or during debridement offer useful culture information.[28]

Samples should be sent to Microbiology department for Gram staining, culture and antibiotic susceptibility testing or molecular testing.

c) Molecular diagnosis – Studies involving PCR technique showed that extracellular proteins responsible for "streptococcal pyrogenic toxins" plays an important role in pathogenesis and these proteins were non-uniformly distributed among bacteria responsible for Type-2 NF. Nowadays, available Multiplex PCR targets six specific genes that will differentiate the six common microorganisms responsible for NF. Primers for the rpoS gene (RPOS-F and RPOS-R), were used to target *Vibrio vulnificus*. rpoS gene is responsible for hemolytic and cytolytic activity, The toxR gene is a transcriptional activator of the cholera toxin operon and it is used to target *Vibrio cholerae* (toxR-F and toxR-R). Primers targeting specific genes have been developed for the molecular identification of organisms associated with NF. The rpoS gene (RPOS-F and RPOS-R primers), which regulates hemolytic and cytolytic functions, is used for the detection of *V vulnificus*. In *V. cholerae*, the toxR gene—a transcriptional activator of the cholera toxin operon—is targeted with toxR-F and toxR-R primers. For methicillin-resistant *S. aureus* (MRSA), diagnostic assays commonly amplify the mecA gene (mecA-F and mecA-R). Similarly, the gcat gene, encoding collagenase, is a marker for *Aeromonas hydrophila*, whereas the dnaseB gene (dnaseB-F and dnaseB-R), linked to pyrogenic activity, serves as a target for Group A *Streptococcus*.[30]

To improve diagnostic speed and cost-effectiveness compared with pulsed-field gel electrophoresis (PFGE), Obszańska and colleagues proposed two novel approaches capable of delivering results within 10 hours. The first, multiple-locus variable number tandem repeat fingerprinting (MLVF), amplifies several variable loci to generate banding patterns that are compared against reference databases. The second, multilocus variable number tandem repeat analysis (MLVA), evaluates the number of repeat sequences at each locus to produce a unique numerical profile that can be matched with existing strain databases.[31]

d) Histology - In clinically suspected cases of NF where there is no obvious diagnostic finding histopathology may be sought in order to confirm diagnosis. In comparison to Fine needle aspiration (FNA), Incisional biopsy is more sensitive technique, FNA is no longer recommended for NF diagnosis. Incisional biopsy under local anesthetic can be done near bedside and is advised in early cases. As per standard procedure, biopsy is obtained from a representative infectious area of the lesion and from the leading edge of any necrotic area.[32] Frozen-section of incisional biopsy specimen reveals vasculitic, necrosis, microorganisms, polymorphonuclear infiltration and vascular thrombosis occurring in superficial fascia, deeper layer of dermis, and adipose tissue. Areas which are spared involves superficial dermis, and underlying muscle tissue. Microscopic reports using haematoxylin eosin stain typically show necrosis of the superficial fascia with polymorphonuclear infiltration and oedema of reticular dermis, subcutaneous fat and angiothrombosis encountered at all levels, also with secondary vasculitic alterations.[33]

e) Radiodiagnosis - Imaging studies also claimed to be useful in the diagnosis of the NF, but one should be very careful not to waste the life saving time, while waiting for the imaging studies. Plain x-ray can show soft tissue swelling or subcutaneous gas and sometimes osteomyelitis of underlying bones may be seen. Computed tomography (CT) or magnetic resonance imaging (MRI) shows edematous extension in fascial plane; however, sensitivity and specificity of these diagnostic modalities are ill defined and lead to delay in definitive diagnosis and prompt treatment. In practice, clinical judgment is of paramount importance in diagnosis, as waiting for CT or MRI to make definitive diagnosis can lead to delay in treatment. Ultrasound guided aspiration of

perifascial fluid, and its culture can help in isolating the organism.[34] Computerized tomography (CT) scan is reported to have sensitivity of 50–80%; it will show fascial edema with or without gas formation. Literature suggest Magnetic resonance imaging (MRI) is highly sensitive (70–90%) in comparison to CT scan, MRI helps in determining the spread and extension of NF along the deep thick fascial planes and muscles show hyper intense T2W signal.[35]

f) Surgical Diagnosis – It is the mainstay of diagnosis of NF and comprises of two surgical modalities.

Finger test: Under local anaesthesia, 2-cm long incision is given in the affected lesion, lack of fascial resistance, active bleeding and foul-smelling fluid will constitute a positive finger test; Positive finger test is diagnostic of NF.[33]

Biopsy/Frozen section biopsy: demonstrated early diagnosis of NF and hence reduces the risk of mortality. However, a dedicated pathologist required for interpretation of NF.[34-36]

TREATMENT

NF is a fulminant condition and requires prompt and early intervention. Adequate fluid replacements along with umbrella coverage of broad-spectrum antibiotic and aggressive debridement are essential components involved in treatment of NF infection. Overall mortality rate is 15–34%, however when NF is associated with Toxic shock syndrome mortality rises to >70% and if surgical intervention is not provided promptly nearly 100% chance of mortality is present in patients suffering from NF.

a) Antibiotics: For Group A Streptococcus Clindamycin 600m IV 6 hourly along with Penicillin G 2–4 mU IV 4hourly is the primary treatment. However, in necrotising fasciitis due to Community acquired MRSA, Vancomycin 1g IV 12 hourly along with Clindamycin 600 mg IV 6 hourly is the preferred treatment of choice. NF due to polymicrobial infection (mixed aerobes/anaerobes) usually treated with a cocktail of antibiotics Ampicillin, 2 g IV 4 hourly, Clindamycin, 600–900 mg IV 6 hourly and ciprofloxacin, 400 mg IV 8 hourly. In case of clindamycin resistance, metronidazole can be used as alternative treatment for NF. [12]

b) Surgery: The NF is usually a polymicrobial, mixed rapidly spreading infection, need early and regular surgical debridement and dressings every 12 to 24 hours depending on the severity of spreading infection. Each time after debridement tissue sensitivity and culture for bacteriology and mycology along with histopathology send as it is a mixed infection. After culture report surgical debridement is supported by medical treatment.[32] For dressings of wounds normal saline, Chlorine water, diluted white vinegar is used for dressings. Role of topical antibiotic and anti-fungal ointment is controversial. After control of ongoing sepsis wound margins and bed starts granulating, supported by regular dressings with normal saline and Chlorine water. Before any procedure for cover of the wound, repeated wound margin histopathology and culture should be free from the organisms. Wound may be closed by skin grafting, flaps and sometimes tissue expanders may be needed for aesthetic purposes.[33]

c) Intravenous Immunoglobulin: Use of IVIG is reasonable in GAS infections due to presence of neutralizing antibodies that acts against antigens of GAS. There is limited evidence regarding benefit of IVIG in gram negative sepsis and as a result the IVIG can be considered as an adjuvant therapy in NF due to gram negative microorganisms. Therefore, the exact benefits of IVIG remain uncertain. Many clinicians recommend 2 g/Kg with a maintenance second dose; however, many authors still have varied opinion regarding dosage recommendation. Initially infusion is started at a rate of 20 mL/h, to a maximum of 160 mL/h. Major contraindications in which immunoglobulin infusion is not recommended are IgA deficiency or anaphylaxis history with earlier immunoglobulins infusion.[37]

d) Hyperbaric Oxygen Therapy: Hyperbaric oxygen therapy is a recognized additional treatment to surgical debridements and antibiotic therapy for NF infections. Over the years many clinical scenarios have been described, based on tissues involved, occurrence of lesion and site of infection, infective cause or particular host immunologic and vascular risk factors. In these clinical situations, there occurs to be development of hypoxia or reduced redox potential in tissues resulting in necrosis. After an infective process is initiated, metabolic products of aerobic and anaerobic metabolism tend to lower the oxidation-reduction potential, which creates a milieu

for growth of organisms whether aerobic or anaerobic microorganisms. Local hypoxia causes up-regulation of endothelial adherence molecules, leading to leukocyte adhesion and endothelial cytotoxicity. Leukocytes get sequestered in blood vessels, decreasing the local immunity and accumulation of gases in tissues due to incomplete substrate oxidation.

The significant amount of hypoxic leukocyte deregulation can be reduced by Hyperbaric oxygen therapy and provide oxygenation to other surrounding ischemic areas, thus restraining the spread and progression of infection. The dissolved oxygen in plasma which is present in large blood vessels diverts itself to areas of poorly perfused tissue, where it causes gradient exchange of oxygen from regions of very high O_2 saturation to tissues with lower oxygen gradient. Integrin inhibition decreases leukocyte adherence, reducing systemic toxicity. The increase in neutrophil's bactericidal activity is thought to be due to hyperbaric oxygen (HBO). Hyperbaric oxygen therapy is also inhibitory for growth of anaerobes, including *C. perfringens* as it decreases the production of α-toxin and for this reason HBO is believed to be beneficial in treating mixed infections.[37,38]

Complications

NF patients may have complications like septic shock or toxic shock syndrome and multi-organ dysfunction syndrome (MODS); 79% patients of complicated NF require invasive ventilation and vasopressors. In cases of rapidly progressing NF, inspite of debridement the patient may require amputation; the percentage and success rate of amputation varies depending on the comorbid factors like myonecrosis and concurrent vascular insufficiencies.[39]

Prognosis

If diagnosed early and promptly treated, most NF patients will have higher chance of survival. Whenever there is significant loss of tissue, grafting of skin or in worst circumstances amputation of the affected area is essential. Mortality is seen in 25% of patients, due to complications such as septicaemia, kidney failure and severe organ failure. Specific bacteria (aerobic or anaerobic) or fungus, patient's immune status, site of infection, and rapid initiation of treatment can influence or alter the outcome. Prognosis depends on many factors like leukocytosis, elevated blood potassium level and increased partial thromboplastin time on admission will increase the chances of mortality of NF patients. Depending on the body area involved; NF of chest, axilla, and flanks as well as gluteal region has highest mortality (25%), followed by cervical NF which has a mortality of 20% and lowest mortality (10%) is seen in NF involving perineum. Mortality of type I NF patients depends mainly on associated co-morbid conditions like diabetes mellitus and peripheral vascular diseases; type II and III NF has a mortality of 32% and 35%, respectively and type IV NF has mortality of more than 40%.[32,39]

CONCLUSION

Early and promt diagnosis of necrotizing fasciitis is difficult, even for well experienced medical professionals. However, certain clinical signs and symptoms should raise clinical suspicions. Patients suffering with endocrine disorders like diabetes mellitus, immunocompromised status and history of intravenous drug abuse are at increased risk, and in them there is an increased chance of mortality. The public should be aware of the risks and informed regarding the common clinical symptoms involved in necrotising fasciitis.

According, to the centre for disease control and prevention, there are the recommended guidelines for prevention and spread of necrotizing fascitis.[40] Good hand hygiene before and after the patient care, keeping skin intact and early medical care plays an important role in patients having infected wound with febrile episodes. Patients suffering from sore throat should visit physician.

REFERENCES

1. Sarani B, Strong M, Pascual J, Schwab CW. Necrotizing fasciitis: current concepts and review of the literature. J Am Coll Surg. 2009;208:279–288.
2. Descamps V, Aitken J, Lee M. Hippocrates on necrotizing fasciitis. Lancet.1994; 344:556.
3. Loudon I. Necrotizing fasciitis, hospital gangrene and phagedena. Lancet.1994; 344:1416 –1419.
4. Kao LS, Lew DF, Arab SN, et al. Local variations in the epidemiology, microbiology, and outcome of necrotizing

soft-tissue infections: a multicenter study. Am J Surg. 2011; 202(2):139–145.
5. Barlam TF, Kasper DL. Approach to the Acutely Ill Infected Febrile Patient. In: Fauci AS, Braunwald E, Kasper DL, Hauser SL, Longo DL, Jameson JL, Loscalzo J (eds.) Harrison's Principles of Internal Medicine vol 1.19th ed. New York: McGraw Hill Inc; 2015:780-783.
6. Pasternack MS, Swartz MN. Cellulitis, Necrotizing Fasciitis, and Subcutaneous Tissue Infections. In: Mandell GL, Bennet JE, Dolin R. (eds) Principles and practice of infectious diseases vol 2.7th ed. Philadelphia: Churchil livingstone; 2010:3191-3198.
7. Tang WM, Ho PL, Fung KK, et al. Necrotizing fasciitis of a limb. J Bone Joint Surg Br 2001; 83B:709–714.
8. Nehme A, Joukhadar NI, Saidy E, Darwiche M, Aouad D, Nour HGA. Fatal Acinetobacter baumanii Necrotizing Fasciitis following Open Reduction Internal Fixation in a Polytrauma Patient. Case Reports in Infectious Diseases, vol. 2018, Article ID 4176320. https://doi.org/10.1155/2018/4176320.
9. Yeika EV, Foryoung JB, Efie DT, Nkwetateba EA, Tolefac PN, Ngowe MN. Multidrug resistant Proteus mirabilis and Escherichia coli causing fulminant necrotising fasciitis: a case report. BMC Res Notes 2018; 11:322 https://doi.org/10.1186/s13104-018-3413-7
10. Rodríguez JY, Morales-López SE, Rodríguez GJ, Álvarez-Moreno CA, Ocampo W, Cepeda ML, et al. Necrotizing fasciitis caused by Apophysomyces variabilis in an immunocompetent patient. Med Mycol Case Rep. 2017; 20:4–6.
11. Wong CH, Chang HC, Pasupathy S, Khin LW, Tan JL, Low CO, "Necrotizing fasciitis: clinical presentation, microbiology, and determinants of mortality," Journal of Bone and Joint Surgery A, vol. 85, no. 8, pp. 1454–1460, 2003.
12. Andreasen TJ, Green SD, Childers BJ. Massive infectious soft-tissue injury: diagnosis and management of necrotizing fasciitis and purpura fulminans. Plast Reconstr Surg. 2001;107: 1025–1035.
13. Green RJ, Dafoe DC, Raffin TA. Necrotizing fasciitis. Chest. 1996;110: 219–229.
14. Miller LG, Perdreau-Remington F, Rieg G, et al. Necrotizing fasciitis caused by community-associated methicillin-resistant Staphylococcus aureus in Los Angeles. N Engl J Med. 2005; 352: 1445–1453.
15. Tsai YH, Huang TJ, Hsu RW, et al. Necrotizing soft-tissue infections and primary sepsis caused by Vibrio vulnificus and Vibrio cholerae non-O1. J Trauma. 2009; 66: 899 –905.
16. Cook DA, Heiner JP, Rao VK. Necrotizing candidal fasciitis following hip surgery. Orthopedics 1990; 13: 768-770.
17. Jain D, Kumar Y, Vasishta RK, et al. Zygomycotic necrotizing fasciitis in immunocompetent patients: a series of 18 cases. Mod Pathol 2006; 19:1221-1226.
18. Cheung JPY, Fung B, Tang WM, Ip WY, "A Review of Necrotizing Fasciitis in the Extremities," Hong Kong Medical Journal, Vol. 15, No. 1, 2009, pp. 44-52.
19. Shaikh N, "Necrotizing Fasciitis, a Decade of Surgical Intensive Care Experience," Indian Journal of Critical Care Medicine, Vol. 10, No. 4, 2006, pp. 225-229.
20. Park KH, Jung SI, Jung YS, Shin JH, Hwang JH. Marine bacteria as a leading cause of necrotizing fasciitis in coastal areas of South Korea. Am J Trop Med Hyg 2009;80(4):646–50
21. Brook I and Frazier EH, "Clinical and Microbiological Features of Necrotizing Fasciitis," Journal of Clinical Microbiology, Vol. 33, No. 9, 1995, pp. 2382-2387.
22. Tang WM, Ho P L, Fung KK, Yen KY and Leong JC, "Necrotizing Fasciitis of a Limb," Journal of Bone & Joint Surgery, British Volume, Vol. 83, No. 5, 2001, pp. 709-714. doi:10.1302/0301-620X.83B5.10987
23. Wang YS, et al., "Staging of Necrotizing Fasciitis Based on the Evolving Cutaneous Features," International Journal of Dermatology, Vol. 46, No. 10, 2007, pp. 1036-1040. doi:10.1111/j.1365-4632.2007.03201.
24. Lazzeri D, Lazzeri S, Figus M, et al. Periorbital necrotizing fasciitis. Br J Ophthalmol 2010;94:1577–1585
25. Wong CH, Khin LW, Heng KS, et al. The LRINEC (Laboratory Risk Indicatotors for Necrotizing Fasciitis) Score. A Tool for Distinguish Necrotizing Fasciitis from Cellulites. Critical Care Medicine, Vol. 32, 2004, pp. 1535-1540. doi:10.1097/01.CCM.0000129486.35458.7D
26. Borschitz T, Schlicht S, Siegel E et al. Improvement of a clinical score for necrotizing fasciitis: 'pain out of proportion' and high CRP levels aid the diagnosis. PLoS ONE 2015; 10(7): e0132775.
27. Zil-E-Ali A, Fayyaz M, Fatima A, Ahmed Z. Diagnosing Necrotizing Fasciitis using Procalcitonin and a laboratory risk indicator: brief overview. Cureus 2018; 10(6):e2754. https://doi.org/10.7759/cureus.2754
28. Thomson RB. Specimen Collection, Transport and Processing: Bacteriology. In: Murray PR, Baron EJ, Landry ML, Jorgensen JH, Pfaller MA (eds.) Manual of Clinical Microbiology. 9th ed. Washington D.C: ASM Press;2007:291-333.
29. Streptococcus, Enterococcus and similar organisms. In: Forbes BA, Sahm D, Weissfeld AS. (eds).Bailey and Scott's Diagnostic Microbiology. 13th ed. Philadelphia: Mosby Elsevier; 2014:247-264.
30. Tsai, Y.-H., Chen, P.-H., Yu, P.-A., Chen, C.-L., Kuo, L.T., Huang, K.-C. A multiplex PCR assay for detection of Vibrio vulnificus, Aeromonas hydrophila, methicillin-resistant Staphylococcus aureus, Streptococcus pyogenes, and Streptococcus agalactiae from the isolates of patients with necrotizing fasciitis. Int J Infect Dis. 2019;81:73–80
31. Obszańska K, Borek AL, Hryniewicz W, Sitkiewicz I (2012) Multiple locus VNTR fingerprinting (MLVF) of Streptococcus pyogenes. Virulence 3: 539-542.
32. Edlich RF, Cross CL, Dahlstrom JJ, Long WB III. Modern concepts of the diagnosis and treatment of necrotizing fasciitis. J Emerg Med. 2010;89:7–36.
33. Anaya D, Dellinger E. Necrotising soft tissue infection: diagnosis and management. Clin Infect Dis 2007; 4: 705
34. Arslan A, Pierre-Jerome C, Borthne A. Necrotising fasciitis: unreliabile MRI findings in the preoperative diagnosis. Eur J Radiol. 2000; 36:139–143.

35. Loyer E, DuBrow R, David C. Imaging of superficial soft-tissue infections: sonographic findings in cases of cellulitis and abscess. Am J Roentgenol. 1996; 166:149–152.
36. Majeski J and Majeski E, "Necrotizing Fasciitis: Im-proved Survival with Early Recognition by Tissue Biopsy and Aggressive Debridement," Southern Medical Journal, Vol. 90, No. 11, 1997, pp. 1065-1068.
37. Provan D, Nokes TJC, Agrawal S, Winer J, Wood P. Clinical guidelines for immunoglobulin use. 2nd ed. London: Department of Health; 2008:62.
38. Morgan MS. Diagnosis and management of necrotizing faciitis: a multiparametric approach. Journal of Hospital Infection 2010. 75; 249 – 257
39. Hsiao CT, Weng HH, Yuan YD, Chen CT and Chen IC, "Predictors of Mortality in Patients with Ne-crotizing Fasciitis," The American Journal of Emergency Medicine, 2008; 26(2): 170-175.
40. Group A Streptococcal (GAS) Disease, Center for Disease Control and Prevention. Http/www.cdc.gov/ncidod/dbmd/diseaseinf/groupastreptococal.g.html
41. Davoudian P, Flint NJ. Necrotizing fasciitis. Contin Educ Anaesth Crit Care Pain. 2012;12(5):245–50.