

TO STUDY THE CLINICAL CHARACTERISTICS, MANAGEMENT AND OUTCOMES IN PATIENTS OF NECROTISING FASCIITIS WITH AND WITHOUT DIABETES MELLITUS

General Surgery

Dr. Nishant	PGJR3, Department Of General Surgery, Hind Institute Of Medical Sciences, Barabanki, Uttar Pradesh, India.
Dr. Vijay Kumar Goel*	Professor And Head Of Department Of General Surgery, Hind Institute Of Medical Sciences, Barabanki, Uttar Pradesh, India. *Corresponding Author
Dr. Nikhil Tiwari	Assistant Professor, Department Of General Surgery, Hind Institute Of Medical Sciences, Barabanki, Uttar Pradesh, India
Dr. Dharmendra Uraiya	Professor, Department Of General Medicine, Hind Institute Of Medical Sciences, Barabanki, Uttar Pradesh, India.
Dr. Suprabha Chaudhary	Assistant Professor, Department Of General Surgery, Hind Institute Of Medical Sciences, Barabanki, Uttar Pradesh, India.

ABSTRACT

This prospective cohort study was conducted with the objective of studying the clinical characteristics, management and outcomes of necrotizing fasciitis (NF) in patients with and without diabetes mellitus (DM). The research aimed to evaluate management strategies, morbidity, and mortality rates, focusing on the distinct bacteriological profiles of both cohorts. This study was conducted over 12 months period at the Hind Institute of Medical Sciences, involving 84 patients, categorized into two groups: those with DM and those without. NF was more prevalent in males, and in both groups, the lower extremities were predominantly affected. Notably, diabetic patients required more frequent surgical debridements and experienced longer hospital stays (14.52 days compared to 7.57 days for non-diabetics). The study concludes that DM significantly impacts NF severity and treatment, necessitating more intensive management and a multidisciplinary approach, emphasizing early diagnosis and strict glycemic control to improve patient outcomes. **Summary:** This study explores the differences in outcomes of Necrotizing Fasciitis (NF) between diabetic and non-diabetic patients. NF, a critical soft tissue infection, exhibits a higher severity and poorer prognosis in individuals with Diabetes Mellitus (DM). The research involved 84 patients, equally divided into two groups, assessing demographics, clinical features, microbiological profiles, management approaches, and treatment outcomes. Results indicated that diabetic patients had a higher mean age, longer hospital stays, and a greater incidence of severe infections, along with increased rates of polymicrobial infections. Additionally, diabetic individuals exhibited a higher prevalence of complications and a lower rate of successful healing compared to their non-diabetic counterparts. The findings underscore the importance of early diagnosis, tailored treatment, and strict glycemic control in improving NF outcomes among diabetic patients. Future research should focus on adjunctive therapies and developing specific risk assessment models for this population.

KEYWORDS

Necrotizing Fasciitis (NF), Diabetes Mellitus (DM), Glycemic Control, Polymicrobial Infections

INTRODUCTION

Necrotising fasciitis (NF) is a serious soft tissue infection that spreads quickly and is typified by widespread fascial and surrounding tissue necrosis. With high rates of morbidity and mortality, this aggressive illness poses a serious risk to patient health and requires immediate medical attention. Although NF can happen to people who do not already have any health issues, its effects are more noticeable in people who have diabetes mellitus (DM). Diabetes weakens the immune system, which makes the environment conducive to infection and increases the severity of clinical symptoms. [1]

Necrotizing Fasciitis Can Be Classified Into Two Main Types

- Type 1 NF
- Type 2 NF

Usually polymicrobial, type 1 NF often involves a combination of aerobic and anaerobic bacteria, including Streptococcus, Staphylococcus, Klebsiella, Escherichia coli, and different strains of Bacteroides. Patients with underlying diseases like diabetes and immunosuppression frequently have this kind.

Conversely, **Type 2 NF** is primarily monomicrobial, most commonly attributed to Group A Streptococcus (GAS), and can also involve Methicillin-Resistant Staphylococcus Aureus (MRSA). Type 2 NF typically presents more acutely, often associated with a history of trauma or surgical procedures.

The overall incidence of NF is estimated at approximately 0.4 to 1.0 cases per 100,000 individuals annually in the United States^[2]; however, this condition is becoming increasingly prevalent, particularly with pre-existing health conditions.

In India, incidence rates are significantly higher, ranging from 1.3 to 3.2 cases per 100,000 populations particularly in southern regions of the country.^[3]

This rise is often linked to the increasing burden of diabetes and other comorbidities, which play a critical role in the pathogenesis of NF.

Recent advancements in our understanding of necrotizing fasciitis incorporate improved diagnostic techniques, such as imaging methods and molecular microbiology for more accurate pathogen identification.

The evolution of management strategies emphasizes the importance of timely surgical debridement and comprehensive antibiotic therapy tailored to the causative organisms.^[4]

Pathophysiology Of Necrotizing Fasciitis

Microbial virulence factors, host immune responses, and underlying comorbidities interact intricately in the pathophysiology of NF, which leads to the condition's severe consequences and quick progression.

Usually, germs are introduced through skin breaks like trauma, surgical wounds, or even small skin diseases, which is how the infection starts. These pathogens' virulence factors, which include toxins, enzymes, and the capacity to elude the host's immune system, are essential for promoting the quick spread of infection^[1]

On a cellular level, NF is characterized by extensive tissue necrosis resulting from the release of pro-inflammatory cytokines and enzymes by bacteria. This inflammatory response leads to increased vascular permeability resulting in edema, ischemia and tissue damage.

The rapidity of tissue destruction often exceeds the body's capacity to mount an effective immune response culminating in systemic involvement, sepsis and multi-organ failure.^[2]

Diabetes can impair various components of the immune system including neutrophil function which is essential for combating infections. Studies have shown that hyperglycemia adversely affects

the migration and phagocytic ability of neutrophils leading to a diminished inflammatory response to infections.^[5] Hyperglycemia promotes the production of Advanced Glycation End Products (AGEs), which can enhance inflammation and impair wound healing.^[6] Diabetic neuropathy can mask early signs of infection as patients may not perceive pain or discomfort in affected areas.^[7] Patients with diabetes often have additional comorbid conditions such as obesity and renal impairment, which can further complicate the clinical course of NF.^[8]

Clinical Features

Diabetes patients frequently have more subtle beginning symptoms, which might cause a delay in diagnosis. Compared to those without diabetes, the initial symptoms, such as localized pain and edema, may be less severe.^[9] Localized pain, edema, erythema, and a localized increase in body temperature over the affected area are early signs of presentation.

Symptoms of the system include chills and fever.^[10] The development of blisters or bullae, skin discoloration that progresses to necrosis, and crepitus, which indicates gas production within tissues, are examples of late signs.^[10]

Necrotizing fasciitis is scored using the Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) score.^[11]

Laboratory Risk Indicator For Necrotizing Fasciitis (LRINEC) Scoring^[11]

Variable	Value	Score
C-Reactive protein (mg/L)	≤150	0
	>150	4
Total white blood cell count (1000 cells/μL)	<15	0
	15-25	1
	>25	2
Hemoglobin (g/dL)	>13.5	0
	11-13.5	1
	<11	2
Sodium (mmol/L)	≥135	0
	<135	2
Creatinine (mg/dL)	≤1.6	0
	>1.6	2
Glucose (mg/dL)	≤180	0
	>180	1

Lrinec Risk Assessment

Risk Category	LRINEC Points	Probability for Presence of NF
Low	≤5	<50%
Medium	6-7	50-75%
High	≥8	>75%

Diagnostic modalities ⁽¹²⁾			
Imaging		Laboratory investigations	
X - ray	Helps to rule out fractures or bone involvement	HbA1C	Glycaemic index
Ultrasono graphy	Identifies fluid collections, abscesses & gas within soft tissues, increased echogenicity in the fascia	Culture & sensitivity	Pus/ Discharge/ Blood
Computed tomograp hy	Gives detailed images that highlight the extent of NF	C – reactive proteins & Procalcitonin	Inflammatory markers
Magnetic resonance imaging	Shows extensive oedema in the fascial planes, muscle involvement and the presence of abscesses or necrotic tissue	Other (s)	CBC/ LFT/ RFT/ Coagulation profile

MATERIALANDMETHOD

This prospective cohort study was conducted at the Department of General Surgery, Hind Institute of Medical Sciences, Barabanki, from January 2024 to December 2024, encompassing 84 patients aged 18 to 60 years to evaluate the clinical characteristics, management and outcomes in patients diagnosed with necrotizing fasciitis (NF) with and without diabetes mellitus (DM). Inclusion criteria consisted of patients with clinically confirmed cases of NF and immunocompromised patients (excluding DM), patients with malignancy, end-stage renal disease, congestive heart failure and chronic liver disease were excluded.

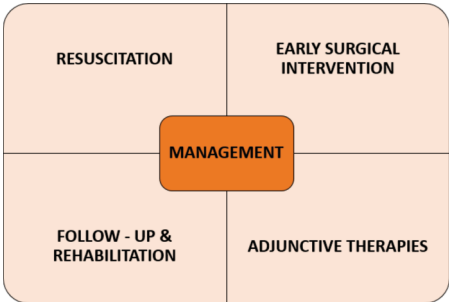
Patients Diagnosed With Necrotizing Fasciitis Was Categorized Into Two Groups:

Group A: Patients diagnosed with necrotizing fasciitis with diabetes mellitus.

Group B: Patients diagnosed with necrotizing fasciitis without

diabetes mellitus.

Management Protocol



RESULT

Table 1: Age Distribution

Age Statistics	Group A (Diabetic)	Group B (Non-Diabetic)
Total Patients	42 years	42 years
Mean Age	46.2 years	43.2 years
Median Age	46 years	42.5 years
Minimum Age	29 years	26 years
Maximum Age	59 years	58 years

Table 2: Gender Distribution

Gender	Group A (Diabetic)	Group B (Non-Diabetic)
Male	33 (78.6%)	35 (83.3%)
Female	9 (21.4%)	7 (16.7%)

Table 3: Involved Site Distribution

Involved Site	Group A (Diabetic)	Group B (Non-Diabetic)	Observation
Upper Extremity	9 (21.4%)	15(35.7%)	More common in non-diabetics
Lower Extremity	33 (78.5%)	26(61.9%)	More common in diabetics
Perianal Region	3 (7%)	2 (5%)	Slightly higher in diabetics
Trunk	1 (2%)	1 (2%)	Equal distribution
Head and Neck	0 (0%)	0 (0%)	No cases in either group

Table 4: Distribution Of Etiological Factor

Etiological Factor	Group A (Diabetic)	Group B (Non-Diabetic)	Observations
Insect Bite	2 (4.8%)	1 (2.4%)	Rare in both groups
Sharp Object Injury	3 (7.1%)	1 (2.4%)	Slightly more common in diabetics
Intramuscular Injection	1 (2.4%)	0 (0%)	Noted only in diabetics
Trivial Trauma	21 (50%)	31 (73.8%)	More common in non-diabetics
No Identifiable Cause	17 (40.5%)	6 (14.3%)	More frequent in diabetics

Table 5: Distribution Of Presenting Symptoms

Presenting Symptom	Group A (Diabetic)	Group B (Non-Diabetic)	Observations
Erythema	36 (85.7%)	31 (73.8%)	Common in diabetics
Abscess	20 (47.6%)	14 (33.3%)	Slightly higher in diabetics
Bullae	26 (61.9%)	11 (26.2%)	More frequent in diabetics
Edema	(88.1%)	37 (88.1%)	Equal prevalence
Pain	21 (50%)	40 (95.2%)	More common in non-diabetics
Skin necrosis	24(57.1%)	8(19%)	More common in diabetics (p value 0.0008 significant)
Induration	30(71.4%)	15(35.7%)	More common in diabetics (p value 0.00025 significant)

Table 6: Comparison Of General Examination Findings

General Examination	Group A (Diabetic)	Group B (Non-Diabetic)	Observations
Pallor	14 (33.3%)	4 (9.5%)	More common in diabetics
Icterus	1 (2.4%)	3 (7.1%)	common in non-diabetics
Cyanosis	7 (16.7%)	3 (7.1%)	More common in diabetics
Clubbing	1 (2.4%)	4 (9.5%)	More frequent in non-diabetics
Edema	37 (88.1%)	37(88.1%)	Equal prevalence

Table 7: Comparison Of Vital Signs

Vital Sign	Group A (Diabetic)	Group B (Non-Diabetic)	Observations
Systolic BP (mmHg)	107.9 ± 9.77	120.8 ± 11.85	Higher in non-diabetics
Diastolic BP (mmHg)	72.6 ± 5.29	68.8 ± 7.05	Lower in non-diabetics
Pulse Rate (bpm)	89.9 ± 15.6	89.3 ± 12.5	Similar
Respiratory Rate (bpm)	18.6 ± 2.36	17.1 ± 0.76	Slightly higher in diabetics
Temperature (°C)	36.9 ± 3.69	37.2 ± 0.80	Higher in non-diabetics

Table 8: Comparison Of Laboratory Parameters

Laboratory Test	Group A (Diabetic)	Group B (Non-Diabetic)	Observations
Hemoglobin (g/dL)	11.40 ± 2.29	12.40 ± 2.00	Lower in diabetics
Total Leukocyte Count (x1000)	16.77 ± 4.29	14.98 ± 1.58	Higher in diabetics
Platelet Count (lakh)	3.14 ± 1.41	2.38 ± 0.79	Higher in diabetics
Random Blood Sugar (mg/dL)	185.24 ± 34.86	106.47 ± 15.41	Markedly higher in diabetics
HbA1c (%)	8.73 ± 1.11	4.92 ± 0.57	Significantly higher in diabetics

Table 9: Distribution Monomicrobial Vs. Polymicrobial Microorganism

	Group A (Diabetic)	Group B (Non-Diabetic)	Observations
Monomicrobial	18 (42.8%)	27 (64.3%)	More common in non-diabetics
Polymicrobial	24 (57.2%)	15 (35.7%)	More common in diabetics

Table 10: Specific Microorganisms Identified

Microorganism	Group A (Diabetic)	Group B (Non-Diabetic)	Observations
Pseudomonas aeruginosa	20(47.6%)	11 (26.1%)	Common in both groups
Staphylococcus aureus	11 (26.1%)	6 (14.2%)	Higher in diabetics
MRSA	11(26.1%)	5 (11.9%)	More frequent in diabetics
E. coli	10(23.8%)	8 (19.0%)	Not significant (p-value >0.05)
Acinetobacter	8(19%)	7 (16.6%)	Not significant (p value - 1.0)
Klebsiella pneumoniae	5(11.9%)	4 (9.5%)	Not significant (p value- 1.0)
No Growth in Culture	4(9.5%)	12 (28.6%)	More common in non-diabetics

Table 10: Frequency Of Debridement Procedures

Number of Debridement	Group A (Diabetic)	Group-B (Non-diabetic)
One	27	35
Two	12	10
Three	3	0

Table 11: Comparison Of Amputation

Amputation	Group A (Diabetic)	Group B (Non-Diabetic)
Done	6	3

Not done	36	39
----------	----	----

Table 12: Comparison Of Split Skin Grafting

Split Skin Graft	Group A (Diabetic)	Group B (Non-Diabetic)
Done	12	20
Not Done	30	22

Table 13: Comparison Of Primary Closure

Primary closure	Group A (Diabetic)	Group B (Non-Diabetic)
Done	3	9
Not Done	39	33

Table 14: Comparison Of Management Strategies

Management	Group A (Diabetic)	Group B (Non-Diabetic)	Observations
Number of Debridements	1.45 ± 0.63	1.31 ± 0.52	Slightly higher in diabetics
Amputation Rate	0.14 ± 0.35	0.10 ± 0.30	More common in diabetics
Skin Graft (SSG)	0.36 ± 0.48	0.33 ± 0.48	Similar in both groups
Flap Cover	0.00 ± 0.00	0.00 ± 0.00	Nil in both groups
Primary Closure	0.02 ± 0.15	0.21 ± 0.42	More common in non-diabetics
Healing by Secondary intention	Predominant	Predominant	Most used approach

DISCUSSION

This study investigates the clinical characteristics, treatment strategies, and outcomes of Necrotizing Fasciitis (NF) among diabetic and non-diabetic patients. We analysed demographic factors, clinical presentations, laboratory markers, microbiological profiles, and prognosis for both groups. Our findings revealed that NF predominantly affects adults across various age groups, with diabetic patients being older (mean age of 46.2 years) than their non-diabetic counterparts (43.2 years, $p = 0.045$). A strong male predominance was observed in both groups, with diabetic males at 78.6% and non-diabetic at 83.3%.

Lower extremity cases were significantly more prevalent among diabetics (78.5%) compared to non-diabetics (61.9%). Trivial trauma was the most frequent predisposing factor, particularly among non-diabetics (73.8% vs. 50%, $p = 0.004$), while 40.5% of diabetic patients had no identifiable cause ($p < 0.001$). Clinical symptoms varied, with erythema (85.7% vs. 73.8%) and bullae (61.9% vs. 26.2%) more common in diabetics, whereas pain was reported more frequently by non-diabetics (95.2%).

Diabetic patients exhibited significantly lower systolic blood pressure (107.9 mmHg) and elevated random blood sugar levels (185.24 mg/dL) compared to non-diabetics (SBP: 120.8 mmHg; RBS: 106.47 mg/dL).

HbA1c levels were also higher in diabetics (8.73% vs. 4.92%). Notably, diabetics experienced a higher frequency of microbial infections, including Methicillin-resistant Staphylococcus aureus (MRSA).

Diabetes was associated with more frequent surgical interventions, with diabetics needing an average of 1.45 debridements compared to 1.31 for non-diabetics. Amputation rates were higher in diabetics (14%) than in non-diabetics (10%). Healing outcomes showed no significant differences in primary closure or healing by secondary intention between the groups, with p -values exceeding 0.05.

Finally, diabetic NF patients had a substantially longer hospital stay (14.52 days) versus 7.57 days for non-diabetics, attributed to poor glycemic control and delayed wound healing.

These findings underscore the complexity of managing NF in diabetic patients and advocate for tailored therapeutic approaches and timely intervention.

CONCLUSION

This study highlights significant differences in the clinical

presentation, microbial etiology, and treatment outcomes of necrotizing fasciitis (NF) between diabetic and non-diabetic patients. Diabetic individuals exhibited more severe infections, with increased skin necrosis, induration, polymicrobial growth, and a higher frequency of surgical interventions including multiple debridements and amputations. They also had longer hospital stays and a higher requirement for split-thickness skin grafting, while non-diabetics more commonly underwent primary closure. The predominance of polymicrobial infections, especially involving *Pseudomonas aeruginosa*, *Staphylococcus aureus*, and MRSA, in diabetic patients underscores the need for broad-spectrum empiric antibiotics and stringent glycemic control. Early diagnosis, aggressive surgical management, and a multidisciplinary approach are critical to reducing morbidity and mortality. The study emphasizes the importance of identifying diabetes as a prognostic factor in NF, warranting heightened vigilance and customized treatment plans. Further research is necessary to develop predictive models and evaluate adjunctive therapies to optimize outcomes in this high-risk population.

REFERENCES

1. Cheng NC, Tai HC, Chang SC, Chang CH, Lai HS. Necrotizing fasciitis in patients with diabetes mellitus: clinical characteristics and prognostic factors. *Br J Surg*. 2015 Sep;102(10):1194-201. doi: 10.1002/bjs.9862. PMID:26174070.
2. Wallace HA, Perera TB. Necrotizing Fasciitis. [Updated 2023 Feb 21]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK430756/>
3. Ajitha MB, Archana CS, Avinash K. A study on necrotizing fasciitis with thetiological factors and microbiological aspects with its prevalence. *Inte J Surgery Science*. 2020;4(2):167-71.
4. DeGenova DT, Hyland SS, Peabody T, Schmitz NP, Myers D, Patterson R, Patel J. Differences Between Patients With Diabetes Mellitus and Those Without in Cases of Necrotizing Fasciitis. *Cureus*. 2023 Mar 23;15(3):e36576. doi: 10.7759/cureus.36576. PMID: 37101987; PMCID: PMC10123239.
5. Berbudi A, Rahmadika N, Tjahjadi AI, Ruslami R. Type 2 Diabetes and its Impact on the Immune System. *Curr Diabetes Rev*. 2020;16(5):442-449. doi:10.2174/1573399815666191024085838. PMID: 31657690; PMCID: PMC7475801.
6. Khalid M, Petroianu G, Adem A. Advanced Glycation End Products and Diabetes Mellitus: Mechanisms and Perspectives. *Biomolecules*. 2022 Apr 4;12(4):542. doi: 10.3390/biom12040542. PMID: 35454131; PMCID: PMC9030615.
7. Anandhanarayanan A, Teh K, Goonoo M, et al. Diabetic Neuropathies.[Updated 2022 Mar 15]. In: Feingold KR, Ahmed SF, Anawalt B, et al., editors. *Endotext* [Internet]. South Dartmouth (MA): MDText.com, Inc.; 2000-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK279175/>
8. Bodke H, Wagh V, Kakar G. Diabetes Mellitus and Prevalence of Other Comorbid Conditions: A Systematic Review. *Cureus*. 2023 Nov 24;15(11):e49374. doi: 10.7759/cureus.49374. PMID: 38146555; PMCID: PMC10749406.
9. Hakkarainen TW, Kopari NM, Pham TN, Evans HL. Necrotizing soft tissue infections: review and current concepts in treatment, systems of care, and outcomes. *Curr P r o b l Surg*. 2014 Aug;51(8):344-62. doi: 10.1067/j.cpsurg.2014.06.001. Epub 2014 Jun 12. PMID: 25069713; PMCID: PMC4199388.
10. Misiakos EP, Bagias G, Patapis P, Sotiropoulos D, Kanavidis P, Machairas A. Current concepts in the management of necrotizing fasciitis. *Frontiers in surgery*. 2014 Sep 29;1:36.
11. Hoels V, Kempa S, Prantl L, Ochsenbauer K, Hoels J, Kehrer A, Bosselmann T. The LRINEC Score-An Indicator for the Course and Prognosis of Necrotizing Fasciitis? *J Clin Med*. 2022 Jun 22;11(13):3583. doi: 10.3390/jcm11133583. PMID: 35806870; PMCID: PMC9267597.
12. Wei XK, Huo JY, Yang Q, Li J. Early diagnosis of necrotizing fasciitis: Imaging techniques and their combined application. *Int Wound J*. 2024 Jan;21(1):e14379. doi: 10.1111/iwj.14379. Epub 2023 Sep 7. PMID: 37679292; PMCID: PMC10784425.