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Original Research Article

Clinicopathological profile and management outcome of skin and soft tissue infections at tertiary care centre of sub Himalayan region

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ABSTRACT

Background: Many times, it is difficult to differentiate between Necrotizing soft tissue infections (NSTI) and Non NSTI based on symptoms, signs and investigations. Only early diagnosis and debridement can prevent high morbidity associated with it.
Methods: This prospective observational, study was conducted in the department of General Surgery, over a period of 1 year on consecutive 100 admitted patients. Clinical signs, symptoms and vital parameters of NSTI group and Non NSTI group were compared. Biochemical investigations, systemic involvement and treatment received was also evaluated. Chi-square analysis was performed to compare categorical variables.
Results: A 77.8% of the NSTI and 54.7% of the Non NSTI infections occurred in males. Significant difference was found in mean age in patients with NSTI (50.9±13.1) and Non NSTI (41.1±15). Type II DM (38.9%) and hypertension (8.3%) were more commonly associated with NSTI patients. Mean pulse rate, respiratory rate and body temperature were significantly more in NSTI group. Patients with NSTI had significantly less hemoglobin and increased creatinine as compared to Non NSTI group. In NSTI group, 83% of patients had necrotizing fasciitis along with myonecrosis followed by Fournier's gangrene (13.8%). Among Non NSTI group perianal abscess and subcutaneous abscess constituted 45.31% of patients. Methicillin sensitive staphylococcus aureus was found in 43.8% and 38.9% of Non NSTI and NSTI groups respectively.
Conclusions: Classical symptoms and signs of NSTI are not reliable and often investigations also fail to differentiate between NSTI and Non NSTI. Type 2 diabetes mellitus patients are more prone to it. High level of clinical suspicion is required for early diagnosis. Timely and adequate debridement of NSTI is necessary to decrease morbidity and mortality.

Keywords: Debridement, Multiple organ dysfunction, Non-necrotizing soft tissue infection, Necrotizing soft tissue infection, Polymicrobial

INTRODUCTION

Hippocrates first described NSTI in the 5th century BC. He described it as a complication of acute streptococcal infection, writing "many were attacked by the erysipelas all over the body when the exciting cause was a trivial accident flesh, sinews, and bones fell away in large quantities". There were many deaths.[1] But first English-language descriptions of NSTIs were by the British surgeon Leonard Gillespie and British physicians Gilbert Blaine and Thomas Trotter in the late 18th century. The disease was known as phagedaenic ulcer, gangrenous, gangrenous ulcer, malignant ulcer, putrid ulcer, or hospital gangrene.[2]

Skin and soft tissue infections (SSTI) are among the most frequent sites of human bacterial infection and represent one of the most common indications for antibiotic therapy.[3] A clinically useful distinction with important management implications, subdivides soft-tissue infections into Non NSTI i.e. sub-cutaneous abscess, cellulitis, carbuncle, acute wound infections, diabetic foot infections, surgical site infections, etc. and NSTI i.e. Fournier's gangrene, synergic necrotizing 'Cellulitis' with fasciitis and myonecrosis, gas gangrene, etc.[4]

The various methods of classification of NSTI e.g. according to site or depth of tissue involved are not clinically very useful, as the diagnostic maneuvers and treatment modalities are same. But even with optimal treatment, NSTI's portend significant morbidity and mortality rates (25%).[5] Non-necrotizing soft tissue infections usually resolve by only antibiotics or drainage of abscess/ surgical debridement plus antibiotic therapy. While NSTI is a surgical emergency as even modest delays can increase morbidity and mortality substantially when therapy is delayed longer than 24hrs.[6]

In spite of introduction of newer antibiotics, rising incidence of bacterial resistance continues to be a problem.[7] The relentless increase in methicillin resistance among *S. aureus* isolated in hospitals throughout the world has made it important to provide coverage for these organisms when treating complicated skin and soft tissue infections (cSSTIs) in hospitals.[8]

Therefore, understanding the epidemiology, etiology and timely clinical assessment of the severity of SSTI is crucial to prevent high morbidity and mortality associated with NSTI. Hence this study was conducted to look for clinico-pathological profile and management required for SSTI in a tertiary care center in sub Himalayan region.

METHODS

This prospective observational study was conducted in the Department of General Surgery over a period of 1 year on consecutive 100 admitted patients. Pregnant females and lactating mothers, patients with infections due to animal bite and patients infected with HIV, Hepatitis B and C were excluded from the study. Detailed history regarding the onset of symptoms, clinical signs, symptoms and vital parameters like pulse, blood pressure, respiratory rate, body temperature of all patients along with their co-morbid conditions was documented.

Biochemical test like hemoglobin, total leucocyte count (TLC), differential leucocyte count, serum creatinine and liver function tests [serum albumin, alanine transaminase (ALT), aspartate transaminase (AST)] were done in all patients. Detailed local examination of the infected site was recorded along with measurement of the involved area. At the time of admission, before initiation of any treatment pus/discharge or tissue sample was collected from the lesion for gram stain and culture sensitivity. Treatment end point was taken as patient becoming afebrile, normalization of TLC, wound having healthy granulation tissue, no pus discharge and resolution of erythema and peri-lesional edema.

Continuous variables with a normal distribution are presented as Mean±SD and percentages are used to describe the baseline characteristics. The t-test was used to compare continuous variables, and chi-square analysis was performed to compare categorical variables as necessary. *P* values <0.05 were considered statistically significant. All analyses were performed using SPSS software, version 13.0 (SPSS Inc, Chicago, IL).

RESULTS

In present study 77.8% of the NSTI and 54.7% of the Non NSTI infections occurred in males. (1.7:1 = M:F ratio). Authors found statistically significant difference in mean age in patients with NSTI (50.9±13.1) and Non NSTI (41.1±15) with p <0.001. The main complaints in NSTI and Non NSTI patients were pain and swelling while vesicle/ bullae, skin discoloration (75%) and erythema were significantly more common in NSTI.

Discharge from wound and fluctuation were nearly equal in both the groups and only 1 patient presented with crepitus in NSTI group. Type II DM (38.9%) and hypertension (8.3%) were found to be more commonly present in NSTI patients.

Among the vital parameters i.e. increased mean pulse rate, respiratory rate and body temperature were significantly more in NSTI group while systolic and diastolic blood pressure were in the same range in both the groups at time of admission. More than one anatomical region was involved in 8.3% of the patients in NSTI group and 4.7% of the patients in Non NSTI group.

Sixty-two percentage of the patients had lesion less than $25cm^2$. Out of which 48 patients (75%) are of Non-NSTI group and 14 patients (39%) are from NSTI group. Whereas only 20% of the patients had lesions of size more than $75cm^2$ comprising mainly 42% from the NSTI group.

Patients with NSTI had significantly less hemoglobin as compared to Non NSTI group. Mean TLC count in NSTI group ($16,015/mm^3$) was more than non NSTI group ($14,743/mm^3$). Serum creatinine was significantly more in NSTI group and 50% of the NSTI patients having raised creatinine levels. Similarly, 72% of the NSTI patients had serum albumin levels less than 3 gm%. Multiple organ dysfunction was noted in 11.1% patients. Among the NSTI group necrotizing fasciitis along with myonecrosis was found in 83.3% of patients followed by Fournier's gangrene 13.8%. Among the Non NSTI group perianal abscess along with subcutaneous abscess was found in 45.31% of patients.

Table 1: Age, clinical features and comorbidities.

Age group (yrs)	Type of infection		Total n (%)
	NSTI [N (%)]	Non-NSTI [N (%)]	
<20	0	2 (3.1)	2 (2.0)
21-30	3 (8.3)	20 (31.3)	23 (23.0)
31-40	5 (13.9)	11 (17.2)	16 (16.0)
41-50	9 (25.0)	14 (21.9)	23 (23.0)
51-60	12 (33.3)	12 (18.8)	24 (24.0)
>60	7 (19.4)	5 (7.8)	12 (12.0)
Total	36 (100)	64 (100)	100 (100)
Mean±SD	50.9±13.1	41.1±15.0	44.6±15.0
P value	0.001		
Clinical features			
Pain	25 (69.4)	44 (68.8)	69 (69.0)
Swelling	20 (55.6)	42 (65.6)	62 (62.0)
Local wound / ulcer	9 (25.0)	6 (9.4)	15 (15.0)
Discharge	7 (19.4)	19 (29.7)	26 (26.0)
Vesicles / bullae	18 (50)	4 (6.3)	22 (22.0)
Skin discoloration	27 (75.0)	2 (3.1)	30 (30.0)
Erythema	28 (77.8)	17 (26.6)	45 (45.0)
Fluctuation	19 (52.8)	38 (59.4)	57 (57.0)
Crepitus	2 (5.6)	0	2 (2)
Co-morbidities			
Para paresis	1 (2.7)	0	1 (1.0)
Coronary artery disease	0	2 (3.1)	2 (2.0)
Congestive heart failure	1 (2.7)	1 (1.6)	2 (2.0)
Chronic obstructive pulmonary disease	0	1 (1.6)	1 (1.0)
Hypertension	3 (8.3)	4 (6.3)	7 (7.0)
Hypotension	0	1 (1.6)	3 (3.0)
Multiple skin eruptions	0	1 (1.6)	1 (1.0)
Type II DM	14 (38.9)	19 (29.7)	33 (33.0)
SFJ incompetence	1 (2.7)	1 (1.6)	2 (2.0)
None	21 (58.3)	37 (57.8)	58 (58.0)

In non NSTI group 43.8%±4.7% were due to staphylococcus aureus (MSSA) and coagulase negative staphylococci followed by *E. coli* and *Enterococcus faecalis* (23.4±9.3%). No growth was found in 4 (6.3%) patients and 2 patients (3.1%) polymicrobial infection. Among the NSTI group most common organism were MSSA (38.9%) followed by *E. coli* (13.9%) and *Streptococcus pyogenes* (11.1%) while only 1 patient was due to MRSA. 30.6% (11) of patients in NSTI group had polymicrobial infection. Multiple debridement's were required more frequently in NSTI patients (average 3.5) as compared to Non NSTI (1.5). 68.8% of Non NSTI group required I and D and debridement was needed only in 23.4%. In the NSTI group 100% of the patients required debridement / fasciotomy and 16.7% of the patients had to undergo amputation of the lower limb/ toes. While there was no mortality in Non NSTI group, three patients died due to NSTI. Mean hospital stay was 19 days in NSTI as compared to 5 days in Non NSTI patients.

DISCUSSION

In India SSTI and NSTI are commonly occurring infections. SSTI are usually found in 4th decade and age above 50 is a risk factor for NSTI.[9] Mean age of NSTI patients was 50.9 years as compared to Non NSTI patients (41.1 years) in present study. Among symptoms, fever has never been found to be a constant feature of SSTI. Of the 734 patients enrolled, only 96 (13.1%) had fever in a study by Mongelluzo J et al.[10] Lipsky et al showed that fever was present at the baseline in only 8.2% of the patients, while about 30.6% of the NSTI patients and 26.6% of the Non-NSTI patients were febrile in present study.[11] So, fever cannot be a reliable sign for early diagnosis of SSTI.

Table 2: Site of lesion, area involved and total leucocyte count.

	Type of infection		Total n (%)	p
	NSTI [N (%)]	Non-NSTI [N (%)]		
Site of Lesion				
Upper limb	3 (8.3)	3 (4.7)	6 (6.0)	
Lower limb	22 (61.1)	23 (35.9)	45 (45.0)	
Chest wall	1 (2.8)	1 (1.6)	2 (2.0)	
Abdominal Wall	1 (2.8)	18 (28.1)	19 (19.0)	
Gluteal and Perineal region	6 (16.7)	15 (23.4)	21 (21.0)	
Head and neck	0	1 (1.6)	1 (1.0)	
More than one anatomical region	3 (8.3)	3 (4.7)	6 (6.0)	
Area in cm^2				
Greater than 75	15 (42)	5 (8)	20 (20)	0.459
Between 50 and 75	3 (8)	3 (5)	6 (6)	0.626
Between 25 and 50	4 (11)	8 (13)	12 (12)	0.789
Less than 25	14 (39)	48 (75)	62 (62)	0.604
Total	36	64	100	
Hematological investigation				
TLC / mm^3	16,015.5±5263.2	14,743.3±6570.1	14491.3±5196.1	P- 0.322
Neutrophils	79.5±10.6	78.8±9.0	79.1±9.6	P-0.716
Lymphocyte	15.0±9.3	15.5±6.9	15.3±7.8	P-0.759
Eosinophil	1.8±3.4	2.0±3.1	1.9±3.2	P-0.742
Monocytes	3.6±2.7	3.7±2.7	3.6±2.7	P-0.898
Basophils	0.08±0.5	0.02±0.12	0.4±0.3	P-0.305

Table 3: Biochemical investigations.

	Type of infection		Total n (%)	χ^2 value	p value
	NSTI [N (%)]	Non-NSTI [N (%)]			
Serum Creatinine (mg/dl)					
Less than 1.2	18 (50.0)	57 (89.1)	73 (73.0)		
More than 1.2	18 (50.0)	7 (10.9)	25 (25.0)	18.75	0.000
Total	36 (100)	64 (100)	100 (100)		
Hemoglobin Levels (gm/dl)					
Less than 8	1 (2.8)	2 (3.1)	3 (3.0)		
8-10.99	17 (47.2)	13 (20.3)	30 (30.0)		
11-13.5	14 (38.9)	33 (51.6)	47 (47.0)	8.580	0.035
More than 13.5	4 (11.1)	16 (25.0)	20 (20.0)		
Total	36 (100)	64 (100)	100 (100)		
Serum albumin (g/dl)					
Greater than 3.5	6 (17%)	1 (8%)	14%	0.0942	
Between 3 and 3.5	4 (11%)	6 (46%)	20%		
Less than 3	26 (72%)	6 (46%)	65%		
Total	36 (100%)	13 (100%)	100%		

Classic symptoms associated with NSTI are pain, anxiety, diaphoresis and organ dysfunction that may worsen rapidly. Pain is usually out of proportion to physical findings which is due to severe damage of the deep layers of skin produced by bacterial toxins, however in some patients there may be little or no pain.[12] Local erythema and swelling are the most common signs of NSTI (40-50% of the patients).[11] Skin discoloration with erythema is an ominous sign of NSTI, which was seen in 75% patients of NSTI as compared to only 3.1% in Non NSTI group. Singh G et al found similar results along with purulent or serous discharge in 72% of patients of NSTI.[13] Crepitus and blistering considered an important sign of NSTI also has varied presence. In a retrospective

analysis of NSTI Elliot et al found crepitus and blistering absent in 63% and 76% of the patients respectively.[14] Author found vesicle/bullae in 50% of NSTI patients while only two patients had crepitus. Unfortunately, these signs and symptoms often appear later in the course of necrotizing infections and may not be present.

Table 4: Type of infection.

Type of infection	Frequency	Percent
Non NSTI		
Subcutaneous abscess	14	13%
Cellulitis	7	6%
Peri anal abscess	15	13%
Breast abscess	11	10%
Carbuncle	3	3%
Acute wound infections	7	6%
Chronic wound infection	11	10%
Infected burns	0	0
Surgical site infections	8	7%
NSTI		
Fournier's gangrene	5	4%
Synergic necrotizing 'cellulitis' with fasciitis and myonecrosis	30	27%
Crepitant/clostridial myonecrosis/gas gangrene	1	1%
Non-crepitant /non-clostridial necrotizing infection	0	0
Total	112	100

Table 5: Causative organism and treatment.

	Type of infection		Total n (%)
	NSTI [N (%)]	Non-NSTI [N (%)]	
Pathogen			
Staphylococcus aureus (MSSA)	14 (38.9)	28 (43.80)	43 (43.0)
Staphylococcus aureus (MRSA)	1 (2.8)	0	5 (5.0)
Coagulase negative staphylococci	2 (5.6)	3 (4.7)	1 (1.0)
Staphylococcus epidermis	0	1 (1.6)	1 (1.0)
Streptococcus pyogenes	4(11.1)	2 (3.1)	6 (6.0)
E. coli	5 (13.9)	15 (23.4)	20 (20.0)
Enterococcus faecalis	3 (8.3)	6 (9.3)	9 (9.0)
Enterobacter spp.	3 (8.3)	2 (3.1)	5 (5.0)
Pseudomonas aerogenosa	2 (5.6)	2 (3.1)	4 (3.0)
Cirobacterfreundii	3 (8.3)	0	3 (3.0)
Citrobacterkoseri	0	2 (3.1)	2 (2.0)
Proteus mirabilis	2 (5.6)	0	2 (2.0)
Proteus vulgaris	1 (2.8)	0	1 (1.0)
Klebsiellaoxytoca	2 (5.6)	1 (1.6)	3 (3.0)
Acinetobacter calcocaticus baumani complex	1 (2.8)	0	1 (1.0)
Clostridium perfringens	1 (2.8)	0	1 (1.0)
No growth	2 (5.6)	4 (6.3)	6 (6.0)
Polymicrobial	11 (30.6)	2 (3.1)	13 (13.0)
Treatment			
IandD	14 (38.9)	44 (68.8)	58
Debridement /s	36 (100)	15 (23.4)	51
Amputation	6 (16.7)	2 (3.1)	8
SS grafting	12 (33.3)	0	12
Secondary suturing	3 (8.3)	5 (7.8)	8
Fasciotomy	5 (13.9)	0	5
Conservative	0	8 (12.5)	8
Number of debridement			
1-5	2.8/36	2.1/15	2.6/51
0	NA	0/49	0/49

According to separate studies by Wong CH et al and Wall DB et al the diagnosis based on clinical features is challenging, because an early NSTI can be clinically indistinguishable from non-necrotizing infection such as cellulitis or phlegmon.[15,16] Hence low threshold for suspecting NSTI should be kept while managing cases of SSTI.

In patients presenting with shock, a suspicion of NSTI should be raised and a prompt surgical intervention should be done.[17] A 8.33% of patients of NSTI presented in shock to us and required debridement under anaesthesia and ICU care. The patients had type II diabetes mellitus in 38.9% of the NSTI and 29.7% of the Non NSTI patients. Diabetes mellitus, renal insufficiency and peripheral vascular disease are more frequently found in of patients of SSTI (60-70%), especially NSTI.[11]

Lower limbs are the most common site of SSTI followed by abdomen and perineal region probably due to more chances of trivial injuries which are over looked.[9,11,18] While perineal and gluteal region is 2nd most common site for NSTI (16%) as compared to non NSTI, where abdominal region is more common site (28%) in present study (Table 2). Due to its nature of rapid spread more surface area is involved in patients of NSTI, which is associated with increased morbidity (Table 2).

Hematological investigations may differentiate between NSTI and Non NSTI as more derangement is found in patients of NSTI.[16,18] NSTI patients are more commonly associated with hemoglobin less than 10gm%.[18] In present study also significantly more NSTI patients had less than 10gm% hemoglobin. Leucocytosis of more than 15 X 10^9/L has been found to be a common feature in NSTI, Wall et al found that a white blood cell (WBC) count <15,000 cells/mm^3 and a serum sodium level greater than 135mmol/L had a negative predictive value of 99% and a 90% sensitivity for detecting NSTIs and considered it to be an useful parameter that may help to distinguish NSTI from non-NSTI infections, particularly when classic "hard" signs of NF are absent.[16]

Development of systemic organ dysfunction in patients with NSTI is associated with higher morbidity and mortality rates. Renal function derangement is often the most common.[15] The difference in serum creatinine levels was statistically significant between the NSTI and Non NSTI groups in present study (Table 3).

NSTI is associated with derangement in liver function and loss of serum albumin from the intravascular compartment. Previous studies have documented that low serum albumin levels are associated with immune-compromised status and higher incidence of surgical intervention and mortality.[19] In present study, 72% of NSTI patients had serum albumin less than 3.0gm/dl.

Various scores like LRINEC are used for diagnosing Necrotizing Fasciitis.[15] But it has never been validated and the authors themselves noted that many other conditions might cause similar laboratory derangements.[20]

SSTI often involve gram-positive and gram-negative aerobic and anaerobic bacteria as well.[4] NSTI may be monomicrobial or polymicrobial and are commonly caused by *Staphylococcus aureus, Staphylococcus epidermidis, Streptococcus, E. coli, Klebsiella, Proteus, Enterococcus, Pseudomonas* etc.[4] Polymicrobial infection up to 69% had been reported by Mc-Henry et al.[21] Both NSTI and Non NSTI are most commonly caused by Staph aureus, approximately in 50% of patients.[7] Author also found same group of organisms with nearly equal distribution in both NSI and Non NSTI patients except *E. coli* which was found to be more prevent in Non NSTI group (23.4%) and 30% patients in NSTI group had polymicrobial infection (Table 5). Author found only one patient with MRSA in NSTI group. Joshi et al in a multicentre study conducted in tertiary care centers found prevalence of MRSA ranging from 7.5% to 41% in various hospitals of Delhi.[22] Miller LG has also shown less than 10% prevalence of MRSA.[23] Hence prevalence of MRSA is varied according to different level of hospitals and regions.

Non NSTI cases are usually managed successfully by antibiotics or they may require incision and drainage or minor debridement. But adequate treatment of NSTI requires not only antimicrobial therapy, fluid resuscitation, electrolyte imbalance and acid base correction but early and complete debridement of infected tissue is key to successful treatment.[24-27] All the patients in NSTI group required debridement under general anesthesia as compared to only 15% Non NSTI patients and mean number of major debridement's required in NSTI group (3.5 vs 1.5) were more and statistically significant similar to other studies.[28,29]

Prolonged hospital stay has been associated with NSTI due to repeated major debridement's and systemic involvement compared to Non NSTI patients similar to present study, 18.6 days.[30] Therefore, early diagnosis and debridement is required for adequate management of NSTI to avoid high morbidity and prolonged hospital stay.

Limitation of the study was being a tertiary care center, before admission most of the patients had already undergone some sort of treatment so we could not assess how many cases of Non NSTI progresses to NSTI despite of proper treatment, what were the exact initial differences in their symptoms and signs at presentation.

CONCLUSION

Classical symptoms and signs of NSTI are not reliable and many times investigations also fail to differentiate between NSTI and Non NSTI. Multiple organ dysfunctions portend a severe morbidity and even mortality. NSTI are commonly polymicrobial and type 2 diabetes mellitus patients are more prone to it. High level of clinical suspicion is required for early diagnosis and debridement of NSTI to decrease morbidity.

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