Indian J Surg (September–October 2009) 71:254–257
DOI: 10.1007/s12262-009-0076-6

ORIGINAL ARTICLE

Integrated clinical care pathway for managing necrotising soft tissue infections

Amit Kumar Shah · N. Bharath Kumar · R. P. S. Gambhir · Rajan Chaudhry

Received: 8 April 2009 / Accepted: 2 May 2009

Abstract

Background Necrotising soft tissue infections (NSTI) are relatively common infections with high morbidity and mortality rate, as they often present late in their course. Quick and aggressive surgical treatment improves survival and decreases hospital stay.

Materials and methods All patients with NSTI managed at our centre from June 2007 to January 2009 were included in this prospective study. We evaluated various parameters like age, co-morbidities, biochemical parameters, time interval between admission and first operative intervention, against duration of hospital stay and out come of the case.

Results Fifty-four patients with NSTI were admitted and treated during the study period. Male to female ratio was 6:1. Mean time interval between admission and operative intervention was 6 hours. Mean period of hospitalisation was 53 days and we had limb salvage rate of 100% and one mortality (1.85%). Diabetes mellitus was the most common co-morbid condition and *Staphylococcus aureus* the most common isolate. Presence of leucocytosis, hyponatraemia, hypoalbuminaemia, anaemia and deranged renal functions were found to be poor prognostic factors.

Conclusion Late and varied presentation is the rule rather than exception with NSTI. Early recognition of the condition, with emergency operative intervention and repeated debridement by a dedicated surgical team, is the key to patient survival and limb salvage.

Keywords Necrotising soft tissue infections · Fournier's gangrene · Meleney's ulcers · Necrotising fasciitis · Sepsis · Debridement · APACHE · LRINEC

Introduction

Necrotising soft tissue infections (NSTI) are certainly not new. They were first described by Jones in 1871 in the US Civil War as *hospital gangrene* related to group A β-haemolytic streptococci infections and *Staphylococcus aureus* [1]. Fournier's gangrene and Meleney's ulcer also are NSTIs [2, 3]. The term "necrotising fasciitis" was coined in 1952 by Wilson [4] and today it refers to "*as any infection of the soft tissue that is associated with necrosis requiring operative intervention and this usually occurs in the context of a critically ill patient".* NSTI is a progressive, rapidly spreading infection, which can involve the skin, subcutaneous tissue, fasciae and muscles. Mortality has not changed significantly for several decades, and still remains high, ranging from 24–34% [5].

Materials and methods

All consecutive patients admitted with diagnosis of NSTI were included in this prospective study. A detailed data-

A. K. Shah · N. B. Kumar · R. P. S. Gambhir · R. Chaudhry
Department of Surgery,
Armed Forces Medical College,
Pune - 411040, Maharashtra, India

A. K. Shah (✉)
E-mail: amit_akshatshah@yahoo.co.in

sheet was filled for each patient which recorded age, sex, comorbidity, duration of symptoms, body surface area (BSA) involvement, presence of any hypotension (SBP <90 mm of mercury), leucocytosis, haemoglobin, renal parameters, and serum levels of sodium, albumin and sugar at the time of admission. Patients were managed with repeated debridement; initial broad spectrum antibiotics followed by culture-based antimicrobials, daily dressings and reconstruction with split skin craft (SSG). The various recorded parameters were analysed against the final outcome and the duration of hospital stay. Statistical analysis was done using the software Epi 6 (A database and statistics software for public health professionals). A p value of <0.05 was considered significant.

Results

Fifty-four patients were admitted with diagnosis of NSTI at our centre during the study period of 18 months. They were in the age group of 17–82 years (mean – 54.96 years).

Clinical parameters at presentation and their effect on mean hospital stay is as shown in Table 1.

It is obvious that age >60 years, symptoms of >10 days duration and systolic BP of <90 mmHg at admission were associated with statistically significant longer hospital stays. Biochemical parameters at admission and their effect on mean hospital stay is shown in Table 2.

It is obvious that leucocytosis >15,400/mm^3, blood sugar >180 mg/dl, serum albumin <3 gm/dl, serum creatinine >1.5 mg%, and sodium <135 mEq/l were associated with prolonged hospital stay.

A multivariate analysis using comorbidity diabetes mellitus (n = 32), chronic renal failure (CRF) (n = 15), hypertension (n = 11) and ishaemic heart disease (IHD) (n = 6) against age <60 years or >60 years was used which showed statistical significance for DM and CRF when considered along with age >60 years (Chi-square test = 7.46 degree of freedom = 2, p value = 0.0039).

Serum creatinine was included for multivariate analysis with haemoglobin and BSA and was found to have significant correlation. The result showed an anaemic patient who has CRF is a poor prognostic factor irrespective of his BSA involvement (Chi-square test = 7.63 degree of freedom = 2, p value = 0.022084).

At the time of first debridement, tissue culture was routinely done as a part of the protocol. Thirty-seven out of 54 patients had polymicrobial infections at admission. We had no anaerobic or fungal organism grown in the cultures. The microbial distribution from the isolates is shown in Table 3.

As a rule we took up all patients for an early debridement and our mean time to debridement after admission, investigation and resuscitation was 6.2 hours (range 2.1–10.4 hours).

Discussion

NSTI are often fulminant infections caused by the synergistic presence of various aerobic or anaerobic, bacteria. NSTI may appear in any anatomical region, the abdomen, perineum and lower limbs are the most common sites of such infections. Diagnosis of NSTI is based on a constellation of symptoms, physical signs, and laboratory assessment. The main symptom described is pain out of proportion to the physical findings. Fluctuation, tenderness and exudates, not necessarily malodorous or purulent, might exist and skin is warm to palpation and is often red-bluish due to vascular thrombosis. Large haemorrhagic bullae, skin necrosis, sensory and motor deficits and crepitus on palpation (hard

Table 1 Clinical parameters and outcome

Parameters	n	Mean hospital stay	p value
Age			
<60 years	n = 38	49 days	0.00314
>60 years	n = 16	61 days	
Sex			
Male	n = 45	51.84 days	0.0521
Female	n = 9	56.11 days	
Duration of symptoms			
<10 days	n = 35	51.12 days	0.0121
>10 days	n = 19	53.47 days	
BSA			
<9%	n = 21	52.90 days	0.2104
>9%	n = 33	52.33 days	
SBP			
>90 mm of Hg	n = 38	50.47 days	0.0012
<90 mm of Hg	n = 16	57.50 days	

Table 2 Biochemical parameters and hospital stay

Lab parameters	n	Mean hospital stay	p value
Haemoglobin			
<10 gm%	n = 18	53.2 days	0.0625
>10 gm%	n = 36	52.2 days	
TLC			
<15,400/mm^3	n = 28	51.1 days	0.0432
>15,400/mm^3	n = 26	54.11 days	
Blood sugar			
<180 mg%	n = 24	48.25 days	0.0012
>180 mg%	n = 30	56 days	
Serum albumin			
<03 gm%	n = 10	69.5 days	0.0001
>03 gm%	n = 44	48.70 days	
Serum creatinine			
<1.5 mg%	n = 38	49.57 days	0.0001
>1.5 mg%	n = 16	61.5 days	
Serum sodium			
<135 mEq/L	n = 16	63 days	0.0001
>135 mEq/L	n = 38	48.15 days	

Table 3 Causative agents grown in the debrided tissue

Microbes	n
Staphylococcus aureus	34
E. coli	13
Enterococci	9
Proteus spp	9
Pseudomonas aeruginosa	7

signs) are all late features. Systematic manifestations, such as hypotension, fever (temperature >38°C), tachycardia, tachypnoea, mental disturbance, tremor, and laboratory findings of marked increase in white blood cell count and metabolic acidosis are indices of development of sepsis. Adjuncts like X-ray, MRI, incision biopsy and finger test have all been described however they have limited clinical utility [6–8].

Quick and aggressive surgical treatment improves survival compared to delayed surgical intervention. Wong et al. proved that delay in surgical debridement of more than 24 hours after hospital admission was the single independent factor that influenced mortality [9]. We also practiced an early surgical intervention in cases of NSTI and were able to achieve gratifying results with one mortality and no limb loss and attribute this to an early surgical intervention (range 2.1–10.4 hours).

Elliot et al. showed that diabetes mellitus did not influence mortality unless it was associated with age >60 years and the presence of acute renal insufficiency [10]. In our study we had only one death. Diabetes mellitus and CRF alone did not affect the hospital stay though when combined with age more than 60 years they were found to be significant predictor for a prolonged hospital stay.

The degree of body area involvement, incomplete surgical debridement, WBC >15,400/mm^3, serum sodium <135 mEq/L and increased serum lactic acid >54.1 mg/dl at hospital admission have also been shown to be associated with increased mortality [5]. In our study SBP <90 mmHg, TLC >15,400/mm^3, serum creatinine level >1.5 mg%, serum sodium levels <135 mEq/l and serum albumin levels <3 gm% were found to be independent predictors of prolonged hospital stay. Haemoglobin level <10 mg% and BSA involvement >09% however were not found to be independent predictors of prolonged hospital stay. When these three factors were combined with age more than 60 years, diabetes mellitus and/or CRF, significance was established with multiple regression analysis.

NSTI mortality rate is reported to reach 25–36%, however in our study we had only one mortality (1.8%) [5]. During the first 10 days from initial surgical debridement, death is attributed to septic shock and later is due to multiple organ insufficiency. A number of scoring systems have been developed to predict mortality rate however we found them cumbersome for practical use in most peripheral centres [11, 12].

Predisposing factors of NSTI include advanced age, diabetes mellitus, malnutrition or obesity, chronic alcoholism, drug abuse, corticosteroid use, immunosuppression, AIDS, chronic obstructive lung disease (COPD) together with the chronic use of steroids, serious trauma, and chronic venous or lymph insufficiency with tissue oedema. In our study 81% of patients had one or more predisposing factors.

Pathogenesis of severe NSTI is known to be multibacterial. Infections, especially, in the abdominal wall and perianal area are multibacterial with both aerobic and anaerobic Gram-positive and -negative organisms. However, infections in limbs are usually due to a single microorganism arising from the skin flora such as *Staphylococcus pyogenes*. Recently, Group A streptococcal (GAS) infections have received much attention recently as aetiologic agents of NSTI [13–17]. Streptococcal NSTIs can occur in otherwise healthy people at any age and may cause rapid onset of shock and multiple-organ failure [18]. In our series 37 out of 54 the infections were polymicrobial. We did not have any anaerobic and fungal growth on culture.

All patients admitted with a diagnosis of NSTI were started empirically on broad-spectrum antibiotics [19]. We used injectable ceftazidime, aminoglycoside or fluoroquinolone and clindamycin as first-line empirical antibiotics, based on our own institutional audit. On availability of culture report the antibiotic regimen was suitably modified.

Once the diagnosis of NSTI is made, resuscitation and treatment proceed simultaneously. In the operating room aggressive and wide debridement of all necrotic tissue with decompression of fascial planes is done. Repeated debridements at 24 hours interval, 1 are often required. In our study on an average each patient was debrided approximately 3 times. Regular saline dressings were done till the ulcer bed was healthy for split-thickness skin graft. In our study 53 patients (98%) were discharged to home with no functional loss, while one patient died on 4th day of admission due to overwhelming septic shock.

Amputation may be required in patients with septicaemia however we did not require resorting to amputation in any of our patients. Colostomy in patients with perineal wounds to prevent faecal contamination was also not required in any one of our patients with perineal wound (n = 7).

Other therapeutic adjuncts to surgical therapy like hyperbaric oxygen, plasmapheresis, intravenous immunoglobulin and activated protein-C, though described in literature are not available for use in most of our centres [20, 21].

Conclusion

High index of suspicion, early and aggressive surgical debridement are the mainstay of NSTI management. A dedicated multidisciplinary healthcare team, following a well-defined integrated clinical pathway of care is essential if we want to salvage limb and life of patients with NSTI.

Conflict of interest The authors do not have any disclosable interest

References

1. Sutherland ME, Meyer AA (1994) Necrotizing soft-tissue infections. Surg Clin North Am 74:591–607
2. Fournier JA (1883) Gangrene foudroyante de la verge. Semin Med 3:345–347
3. Meleney FL (1924) Hemolytic streptococcus gangrene. Arch Surg 9:317–364
4. Wilson B (1952) Necrotizing fasciitis. Am Surg 18:416–431
5. Yaghubiian A, de Virgillio C, Dauphine C, Lewis RJ, Mathew L (2007) Use of Admission Serum Lactate and Sodium Levels to Predict Mortality in Necrotizing Soft-Tissue Infections. Arch Surg 142(9):840–846
6. Arslan A, Pierre-Jerome C, Borthne A (2000) Necrotizing fasciitis: unreliable MRI for disease in the preoperative diagnosis. Eur J Radiol 36:139–143
7. Yamaoka M, Furusawa K, Uematsu T, Yasuda K (1994) Early evaluation of necrotizing fasciitis with use of CT. J Craniomaxillofac Surg 22:268–271
8. Theis JC, Rietveld J, Danesh-Clough T (2002) Severe necrotizing soft tissue infections in orthopaedic surgery. J Orthop Surg (Hong-Kong) 10:108–113
9. Wong CH, Chang HC, Pasupathy S, Khin LW, Tan JL, Low CO (2003) Necrotizing fascitis: clinical presentation, microbiology, and determinants of mortality. J Bone Joint Surg Am 85(8):1454–1460
10. Eliot DC, Kufera JA, Myers RA (1996) Necrotizing soft tissue infections: risk factors for mortality and strategies for management. Ann Surg 224(5):672–683
11. Pessa ME, Howard RJ (1985) Necrotizing fasciitis. Surg Gynecol Obstet 161:357–361
12. Wong CH, Khin LW, Heng KS, Tan KC, Low CO (2004) The LRINEC (Laboratory Risk Indicator for Necrotizing Fasciitis) score: a tool for distinguishing necrotizing fasciitis from other soft tissue infections. Crit Care Med 32(7): 1535–1541
13. Weinbren MJ, Perinpanagayam RM (1992) Streptococcal necrotizing fasciitis. J Infect 25:299–302
14. Donaldson PMW, Naylor B, Lowe JW, et al. (1993) Rapidly fatal necrotizing fasciitis caused by Streptococcus pyogenes. J Clin Pathol 46:617–620
15. Chelsom J, Halstensen A, Haga T, Hoiby EA (1994) Necrotising fasciitis due to group A streptococci in western Norway: Incidence and clinical features. Lancet 344(8930): 1111–1115
16. Marshall DH, Jordan DR, Gilberg SM, et al. (1997) Periocular necrotizing fasciitis: A review of five cases. Ophthalmology 104:1857–1862
17. Kliska DL, Thiede B, Caracciolo J, et al. (1997) Invasive Group A Streptococcal infections in North Carolina: epidemiology, clinical features, and genetic and serotype analysis of causative organisms. J Infect Dis 176:992–1000
18. Hackett SP, Stevens DL (1992) Streptococcal toxic shock syndrome: Synthesis of tumor necrosis factor and interleukin-1 by monocytes stimulated with pyrogenic exotoxin A and streptolysin O. J Infect Dis 165:879–885
19. Bisno AL, Stevens DL (1996) Streptococcal infections of skin and soft tissues. N Engl J Med 334:240–245
20. Riseman JF, Zamboni WA, Curtis A, et al. (1990) Hyperbaric oxygen therapy for necrotizing fasciitis reduces mortality and the need for debridement. Surgery 108:847–850
21. Cainzos, Miguel; Gonzalez-Rodriguez, Francisco J (2007) Necrotizing soft tissue infections. The surgical patient. Curr Opin Crit Care 13(4):433–439