NECROTISING FASCITIS PROMPT AND AGGRESSIVE SURGICAL APPROACH IS THE CORNERSTONE OF MANAGEMENT

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ABSTRACT
Objectives: *To determine the role of prompt and aggressive surgical debridement on the mortality and morbidity associated with necrotizing fascitis and to determine the role of bacteriology and antibiotic sensitivity in the outcome of this debilitating condition.*
Study Design: *This is a retrospective study.*
Setting & Duration: *Department of Surgery, Ward D, Khyber Teaching Hospital, Peshawar from June 2007 to June 2009.*
Methodology: *Forty cases were selected who were diagnosed and managed in this single surgical unit. All patients underwent immediate major surgical debridements in operation theatre with broad spectrum antibiotics coverage and hemodynamic support with regular dressings and debridements.*
Results: *The mean age of presentation was 43.5 years (range 16-78 years) Most of the patients presented late (more than 5 days) after the onset of the condition .The predisposing causes were found in most of the cases. Once necrotizing fascitis was suspected a quick and prompt surgical approach was initiated, with immediate starting of antibiotics and major debridement in operation theatre and regular minor debridements in ward and if needed in theatre. The mortality was 6 during present study.*
Conclusion: *Early diagnosis, radical surgical debridement of all involved necrotic tissue, broad spectrum antibiotics, and aggressive nutritional support are specific treatment options for the management of necrotizing fascitis.*

KEY WORDS: *Necrotizing Fascitis, Surgical Debridement, IV Antibiotics*

INTRODUCTION

Necrotizing fascitis (NF) is soft tissue infection characterized by rapidly spreading inflammation and subsequent necrosis of muscle fascia, subcutaneous fat and in some cases the epidermis and systemic signs of toxicity.[1,2] Necrosis is usually limited in depth to the plane of muscle fascia and skin may be spared in some cases. Two clinical types exist.[3,4]

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Type I necrotizing fasciitis is a polymicrobial infection caused by aerobic and anaerobic bacteria and occurs most commonly in patients with underlying medical or surgical problems.

Type II necrotizing fasciitis refers to a monomicrobial infection caused most commonly by group A streptococcus, necrotizing fascitis caused by cMRSA (community-associated methicillin-resistant Staphylococcus aureus as a monomicrobial infection has also been described).[4]

Type II can occur in any age group and among patients who do not have complicated medical illnesses.[5] Predisposing factors include a history of blunt trauma, varicella (chickenpox), injection drug use, a penetrating injury such as laceration, surgical procedures, childbirth, exposure to a "case," and burns.[6,7]

Two other types of infection commonly considered as

types of necrotizing fascitis are Fournier's Gangrene and cervical necrotizing fascitis.

Similarly there is frequent overlap between necrotizing fascitis and spontaneous gangrenous myositis. Early recognition of necrotizing fasciitis is important since there may be a remarkably rapid progression from an in apparent process to one associated with extensive destruction of tissue, systemic toxicity, loss of limb or death.[8,9] In this condition the mortality and morbidity are very high due to late presentation and some time late diagnosis of the condition, as in many cases early clinical diagnosis of an area of necrotizing fascitis is difficult and can only be confirmed after surgical intervention in theatre.

METHODOLOGY

This study was carried out in surgical "D" ward of Khyber Teaching Hospital for two years from June 2007 to June 2009. A total of 40 patients were included in this study. Most patients presented late and in most cases predisposing causes were present.

Inclusion criteria
All patients with necrotizing fascitis.

Exclusion criteria
All patients with other skin infections and Fornier's gangrene.

Unexplained pain, which increases rapidly over time, may be the first manifestation of necrotizing fasciitis. When necrotizing fascitis was suspected especially in patients with soft tissue infection with associated systemic findings, such as fever, tachycardia, hypotension, tense edema outside the involved skin, disproportionate pain, blisters/bullae, crepitus, and subcutaneous gas. The following principles were adopted. Intravenous antibiotics were started immediately which included ampicillin or ampicillin-sulbactam combined with metronidazole. Those patients who have had prior hospitalization or if they had used antibiotics recently we used ticarcillin-clavulanate, piperacillin-tazobactam, fluoroquinolone or a third generation cephalosporin. These antibiotics were used until the results of specific cultures and sensitivity of the organisms were available.

Emergency aggressive and extensive debridement of all necrotic tissues (skin, subcutaneous tissues and fascia) was carried out until healthy bleeding tissue was reached. The wound was thoroughly washed with hydrogen peroxide, pyodine and saline. Excised tissues were sent for histopathological examination and culture. After not less than 12 hours the wound was inspected and further debridement carried out in case it was needed. Apart from two main OT days in the main operation theatre during hospital stay all the wounds were regularly examined in the morning and evening rounds.

Daily twice dressings and necrosectomies were carried out at the bed side. Once healthy granulation tissue appeared at the local wound dressings were done with normal saline till culture from the wound did not isolate any organisms, definitive procedures for the closure of the wound were then undertaken. Depending upon the defect in the skin, age of the patient, and the viability of the tissues definitive procedures like secondary closure, split thickness skin grafting, and fasciocutaneous flap were carried out. Following recovery patients were discharged and were followed for 3-6 months.

RESULTS

During the two years duration of this study an overall 40 patients admitted to our unit and diagnosed with necrotizing fascitis were studied. The mean age of presentation was 43.5 years (range 16-78 years). Most of the patients presented late after 5 to 6 days of the initiation of symptoms, 29 (72.5%) patients presented with pain, fever, swelling and erythema of the affected area, in 17(42.6%) patients crepitus was noted on initial examination. 9(22.5%) patients reported flu like illness before development of symptoms of skin infection, 27(67.7%) patients reported fever associated with body aches. Regarding predisposing conditions 5 patients were post lapratomy (2 firearms), 4 patients reported

Table I. Isolated organisms and their percentage

Organisms	No.	%
Streptococcus species	8	20
Corynebacteriam	5	12.5
Staphylococcus species	7	17.5
Escherichia coli (E. coli)	9	22.5
Bacteroides species	6	15
Pseudomonas	5	12.5
Clostridium welchii	4	10
Prevotella	3	7.5
Proteus species	7	17.5
Peptostreprococcus	3	7.5
Porphyromonas species	2	5
Enterococci	7	17.5

Predisposing Factors	%	No. of Dedridements	Hospital Stay (Avg)	Outcome Died/Recovered	No. of Patients
Idiopathic	17.5	4	22	2/5	7
Trauma	10	4	9	0/4	4
I/M Injections	7.5	3	21	0/4	3
Boil on skin	5	3	26	0/2	2
Psoas Abscesses	5	4	17	0/2	2
Post Lapratomy	12.5	6	32	1/5	5
Carbuncle	10	4	21	0/4	4
Post append	5	3	39	0/2	2
Post ileostomy	5	5	26	0/2	2
Firearm	5	4	17	1/2	2
Palmer fissure	2.5	3	13	0/1	1
Snake bite	5	5	41	1/1	2
IV Drug Users	5	4	22	1/1	2
FA injuries to legs	5	3	29		2

Table II. Presentation and outcome of patients

minor trauma, 4 patients had carbuncles on their back 3 patients had intra gluteal/ intra deltoid injections, 2 patients were snake bites, 2 patients had boil on their skin, 2 patients had psoas abscesses, 2 post appendecectomy, 2 post ileostomy, 2 patients had fire arm injuries to legs and 2 were IV drug abusers and, 1 has a palmer fissure while in remaining patients no cause could be identified .

Common underlying medical conditions with these cases were diabetes mellitus in 23(57.50%) of patients, chronic renal failure in 9(22.50%), hepatic failure in 7(17.5%) and tuberculosis in 5(12.50%). Out of these 11(27.5%) patients were having more than one of the above underlying conditions. Aggressive surgical debridement was done in all cases. 17 patients were operated within 24 hours of admission and 9 patients were operated within 3 to 4 days, in 6 patients the delay was due to patients seeking late help from hospital and in the remaining patients the delay was mostly due to slower development of symptoms. Antibiotics were started immediately and pus for culture and sensitivity was taken from all cases with first debridement session and antibiotics were then changed accordingly.

DISCUSSION

In 1924 Meleney and Cullen separately described an infective process in the subcutaneous tissues, calling it skin gangrene and synergistic gangrene respectively, the term 'necrotizing fascitis, was introduced by Wilson in 1952.[1] The bacteriology of the condition is extremely variable and it has changed over the years in literature.[2,3] A variety of synergistic infections have been reported but no specific combination has been found to be particularly associated with this condition. In this study we also found multibacterial etiology in almost all cases. Bacteria can be isolated from almost all cases of type I necrotizing fasciitis. In two series from Maryland and Singapore, two-thirds had both mixed aerobic and anaerobic bacteria.[3,6]

Type II necrotizing fasciitis is caused by GAS and was previously called "streptococcal gangrene.[7] There was a dramatic increase in the number of invasive infections such as necrotizing fasciitis caused by GAS during the 1990s. There are reports suggesting an association between the use of nonsteroidal antiinflammatory drugs (NSAIDs) and development of GAS necrotizing infection[10], although a review of the literature including five prospective studies did not demonstrate a correlation.[8] In this study 12 patients had history of NSAID use during previous 3 to 4 days. However it can be said that NSAIDs may mask the usual signs of inflammation, thereby delaying the diagnosis. The laboratory findings generally are nonspecific. Blood tests typically demonstrate a leukocytosis with a marked left shift coagulopathy, and elevations in the serum lactate, creatine kinase and

creatinine concentrations.[9,10,11] There has been several studies regarding the role of Intravenous immune globulin in neutralization of circulating streptococcal when hypotension is present. There seems little question that some batches of IVIG contain neutralizing antibodies against some of the clostridial toxins and streptococcal superantigens.[12,13] We could not use IVIGs because they are not widely available in Pakistan and most patients can not afford them.

Even with optimal therapy, necrotizing fasciitis is associated with considerable mortality. The mortality rates in different studies have included 21 percent in type I necrotizing fasciitis[6], 14 to 34 percent in type II necrotizing fasciitis in which streptococcal toxic shock syndrome is commonly associated with mortality in patients with GAS infection[14,15], 22 percent in patients with cervical necrotizing fasciitis[3], and 22 to 40 percent in those with Fournier's gangrene.[4,5,16] Hyperbaric oxygen therapy may decrease mortality and limit the extent of debridement in Fournier's gangrene[17,18] and necrotizing fasciitis[19,20], but results are conflicting. One observational study of 26 patients with Fournier's gangrene found a significantly lower mortality rate among HBO-treated patients (7 versus 42 percent).[18] A second observational study of 29 patients with necrotizing fasciitis noted significantly fewer debridements and a lower mortality rate (23 versus 66 percent) when HBO was employed.[20] In contrast, a subsequent larger study of 42 patients with Fournier's gangrene suggested increased mortality, morbidity, and cost of therapy among patients treated with HBO.[21] We didnot use HBO therapy in our present study. In our series of 40 patients 6 patients died so our mortality was 15%. Many other factors contribute to mortality. Because large prospective studies have not been performed, such factors cannot be stated with certainty. The following are among the possible prognostic factors: the duration of time from onset of infection to definitive treatment; the type, extent and adequacy of surgical debridement; and infection of the head and neck, thorax, and the abdomen, which are more complex in terms of the surgical debridement.

CONCLUSION

The key to the management of patients with necrotizing fascitis is immediate extensive aggressive surgical debridement of all the necrotic tissues. Simultaneously intravenous fluids therapy and broad spectrum antibiotics therapy should be commenced and systemic ailments such as diabetes should be dealt with.

REFERENCES

1. Anaya D A, Patchen Dellinger E. Necrotizing soft-tissue infection: diagnosis and management. Clin Infect Dis 2007; 44: 705.
2. Gozal D, Ziser A, Shupak A. Necrotizing fasciitis. Arch Surg 1986; 121: 233.
3. Brook I, Frazier E H. Clinical and microbiological features of necrotizing fasciitis. Journ Clin Microbiol 1995; 33: 2382.
4. Laucks S S 2nd. Fournier's gangrene. Surg Clin North Am 1994; 74: 1339.
5. Wong C H, Chang H C, Pasupathy S. Necrotizing fasciitis: clinical presentation, microbiology, and determinants of mortality. Journ Bone Joint Surg Am 2003; 85-A: 1454.
6. Wong C H, Chang H C, Pasupathy S. Necrotizing fasciitis: clinical presentation, microbiology, and determinants of mortality. Journ Bone Joint Surg Am 2003; 85-A: 1454.
7. Eneli I, Davies HD. Epidemiology and outcome of necrotizing fasciitis in children: an active surveillance study of the Canadian Paediatric Surveillance Program. Journ Pediatr 2007; 151: 79.
7. Bisno A L, Stevens D L. Streptococcal infections of skin and soft tissues. N Engl Journ Med 1996; 334: 240.
8. Aronoff D M, Bloch K C. Assessing the relationship between the use of nonsteroidal antiinflammatory drugs and necrotizing fasciitis caused by group A streptococcus. Medicine (Baltimore) 2003; 82: 225.
9. Stevens D L, Tanner M H, Winship J. Severe group A streptococcal infections associated with a toxic shock-like syndrome and scarlet fever toxin A. N Engl Journ Med 1989; 321: 1.
10. Stevens D L. Could nonsteroidal antiinflammatory drugs (NSAIDs) enhance the progression of bacterial infections to toxic shock syndrome? Clin Infect Dis 1995; 21: 977.
11. Butterworth S A, Murphy J J. Necrotizing soft tissue infections are they different in healthy vs immuno-compromised children?. Journ Pediatr Surg 2006; 41: 935.
12. Norrby-Teglund A, Kaul R, Low D E. Plasma from patients with severe invasive group A streptococcal infections treated with normal polyspecific IgG

inhibits streptococcal superantigen-induced T cell proliferation and cytokine production. J Immunol 1996; 156: 3057.

13. Norroby-Teglund A, Basma H. Varying titers of neutralizing antibodies to streptococcal superantigens in different preprations of normal polyspecific immunoglobulin G: Implications for therapeutic efficacy. Clin Infect Dis 1998; 26: 631.

14. Kaul R, McGeer A, Low D E. Population-based surveillance for group A streptococcal necrotizing fasciitis: Clinical features, prognostic indicators, and microbiological analysis of seventy-seven cases. Ontario Group A Streptococcal Study. Am Journ Med 1997; 103: 18.

15. Darenberg J, Luca-Harari B, Jasir A. Molecular and clinical characteristics of invasive group A streptococcal infection in Sweden. Clin Infect Dis 2007; 45: 450.

16. Asfar K, Baraka A, Juma T, Ma'Rafie A, Alladeen T, Al Sayer A. Necrotizing Fascitis. BJS 1991; 78: 838-40.

17. Pizzorno R, Bonini F, Donelli A. Hyperbaric oxygen therapy in the treatment of Fournier's disease in 11 male patients. Journ Urol 1997; 158: 837.

18. Hollabaugh R S, Dmochowski R R, Hickerson W L, Cox C E. Fournier's gangrene: Therapeutic impact of hyperbaric oxygen. Plast Reconstr Surg 1998; 101: 94.

19. Green R J, Dafoe D C, Raffin T A. Necrotizing fasciitis. Chest 1996; 110: 219.

20. Riseman J A, Zamboni W A, Curtis A. Hyperbaric oxygen therapy for necrotizing fasciitis reduces mortality and the need for debridements. Surgery 1990; 108: 847.

21. Mindrup S R, Kealey G P, Fallon B. Hyperbaric oxygen for the treatment of fournier's gangrene. Journ Urol 2005; 173: 1975.