Aggressiveness — The key to a Successful Outcome in Necrotizing Soft Tissue Infection

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Abstract

16 patients with necrotizing soft tissue infections were managed during last three years in various service hospitals. The experience indicates that there is considerable overlap in clinical findings and bacteriology. The infections seem to be variations of the same disease process, a spreading necrotizing infection. The number of these patients suggests that there is an increasing incidence of this entity. *Staphylococcus* and coliforms were the commonest organisms cultured in most of these patients. Because of the high mortality rate upto 50% as reported, we advocate aggressive and early treatment of this condition. Urgent radical exploration, excision of all necrotic tissue and adequate drainage of the deep fascial planes was done in all patients until healthy tissue planes were reached. A strong index of suspicion aids early diagnosis which ensures a favourable outcome. Our study indicates that the lower gastrointestinal tract should be considered as a possible cause of infection in all patients with synergistic gangrene. The involvement of the perineum and scrotum was most common. All these patients were treated with a common approach of resuscitation, broad spectrum antibiotics, immediate surgical excision of all necrotic tissue, aggressive nutritional therapy and early skin coverage with 20% mortality. The infection was primary in 8, postsurgical in 4 and following trauma in 4 cases. In majority of patients, *Staphylococcus* with beta haemolytic streptococci and *E coli* were the organisms isolated initially. Mortality was highest in intensive infections extending the abdomen and chest. Aggressive, effective and early treatment of necrotizing soft tissue infections is imperative to prevent a fatal outcome. Urgent radical exploration by the most experienced surgeon available is essential and includes wide excision of all necrotic tissue and adequate drainage of the deep fascial planes until indubitably healthy tissue is experienced. The surgeon must be prepared to proceed to a laparotomy, diverting colostomy or a suprapubic cystostomy where there exists any element of doubt. Aggression is also of significance in resuscitation, early institution of empirical broad spectrum antibiotic therapy, elaborate repeated daily dressings with hydrogen peroxide and to allow further debridement till the process is controlled.

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Introduction

Necrotizing soft tissue infections include conditions of complex etiology and pathology that share a clinical picture characterized by rapidly progressive inflammation and necrosis of skin, subcutaneous fat and fascia and sometimes muscle. It results in high mortality as was seen during the World War II. The condition is alarming because it occurs not infrequently in a hospital setting and may be rapid and devastating in its progression. Risk factors include diabetes mellitus, intravenous drug abuse, peripheral vascular disease, obesity, malnutrition and immunosuppression. A number of authors have described various forms of progressive necrotizing soft tissue infections into different groups e.g. necrotizing fascitis, clostridial cellulitis, myonecrosis and synergistic necrotizing cellulitis. These entities however, all seem to be variations of same disease process. This pseudo classification has resulted in delay in institution of proper treatment and management with a subsequent increase in mortality and morbidity. 16 patients of necrotising soft tissue infections were managed in various service hospitals during last three years. A detailed study of patients together with a literature review has led to the conclusion that all these

described entities are to be managed with a unified approach - an aggressive management protocol, which has resulted in a marked reduction of mortality.

The earliest description dates back to 1871, when hospital gangrene was described by Joseph Jones, an Army surgeon during the American civil war. In 1883, Fournier described idiopathic scrotal gangrene. In 1926, progressive bacterial synergistic gangrene was described. Gas gangrene was described in 1745 by Quesnay though it was recognised since the time of Hippocrates and Celsus. Hippocrates made the following observations in 5th century BC "Many were attacked by the erysipelas all over the body when the exciting cause was a trivial accident. There were many deaths. The course of the disease was the same to whatever part of the body it spread. But the most dangerous cases....were when the pubes and the genital organs were attacked" [1]. Necrotising fascitis itself was described in 1952 by Wilson when he observed edema and necrosis of subcutaneous fat and fascia with sparing of the underlying muscle in a series of 22 patients. The classic modern description of streptococcal gangrene was reported by Meleny [2] and necrotizing fascitis due to mixed bacteria was reported by Brewer and Meleny [3].

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Material and Methods

16 patients of progressive necrotizing soft tissue infections managed at various service hospitals are presented. 3 patients were females and rest males. Age group varied from 42 years to 65 years, mean age being 53 years. Diabetes was present in 25% of the patients and 50% of the patients were obese. 5 patients had involvement of the perineum and scrotum out of which 2 patients had synergistic gangrene of the genitals secondary to colorectal disease, 5 affected the abdomen, 4 involved extremities and one each had involvement of face, breast including chest wall. The presenting signs are shown in Table 1.

Table 1

Presenting signs of necrotising soft tissue infections

Signs	%
Cellulitis	90
Oedema	85
Fever	70
Cutaneous gangrene	90
Altered sensorium	30
Skin discolouration	30
Crepitation	23
Shock	22
Jaundice	-
Vesicles or bullae	14

The most common signs were cellulitis, oedema and fever. Patients were treated by prompt surgical debridement and broad spectrum antibiotics consisting of cephalosporins or penicillins, gentamycin and metronidazole. When Gram stains and culture reports were obtained, the antibiotics were modified. Wound fluid and necrotic tissues were evaluated by Gram stain, aerobic and anaerobic cultures.

Surgical treatment included debridement of all necrotic fascia, subcutaneous tissue and muscle if involved. In one female patient with fulminant chest wall infection involving right breast, simple mastectomy was done. The open dressing method was applied to this patient. She however died of septicaemia and uncontrolled diabetes.

Results

Diabetes mellitus was a predisposing factor in 25% of patients. All patients had multiple organisms on culture. 9 patients had two organisms, 4 patients had three organisms and 3 patients had four or more organisms in their culture with an overall average of 2.6 organisms cultured. *staphylococcus* coagulase +ve and β haemolytic *streptococcus* were isolated in 75% of the patients, while 50% of the patients also had infection due to Gram-ve enteric bacteria. Hyperbaric oxygen therapy (HBOT) was given to 3 patients. The response to HBOT was favourable to the local condition of the wound. The patients usually responded to a combination of ampicillin, gentamicin and metronidazole. However, higher antibiotics like amikacine, tobramycin, cefotaxim and carbenicillin were used successfully in patients with Gram-ve septicaemia. Majority

of the patients who died had extensive involvement of abdomen, chest wall and breast. All patients had uncontrolled diabetes mellitus and septicaemia.

Discussion

Progressive necrotizing soft tissue infection is an infection of the soft tissues that spreads along the fascial planes. The sine qua non of these infections is the presence of fascial necrosis with widespread undermining of the skin. Initially the skin is not involved, but as the deeper tissues get necrosed and vessels become thrombosed, necrosis occurs. Infection can also start from the skin and then involve the deeper layers later. The affected area is red, hot, shiny and swollen without sharp margins and exquisitely tender. The condition progresses over a few hours or days with skin colour changing to blue grey in ill defined patches, followed by appearances of bullae and necrosis within 3-5 days. By this time, the involved areas are no longer tender, becoming anaesthetic secondary to thrombosis of small blood vessels and destruction of superficial nerves [4]. Underlying muscle is usually not involved. The involved fascia and subcutaneous fat are dull grey in colour and a serosanguinous exudate is present.

Systemically the patient has pain and high fever associated with toxic symptoms. Gas may be present in the tissues and crepitus may be palpable. Tachycardia, tachypnea and hypotension may be present. The commonest sites involved are the extremities, followed by the abdominal wall (Fig 1) and the perineum (Fig 2) [5]. Often the inciting event is not known. The infection may occur postoperatively or after minor trauma. The infection occurs in intravenous drug abusers, after urinary, perineal or intra-abdominal infection, vaginal delivery with episiotomy and LSCS. Cases have been reported after appendicectomy [6] and haemorrhoidectomy [7]. Necrotizing fascial plane infections of the head and neck can occur after dental or pharyngeal infections. Rarely, the condition may be idiopathic without any predisposing factors in 25% of patients inspite of newer diagnostic techniques.

Lab tests are not specific. Leucocytosis is present with massive polymorphonuclear infiltrate [8]. Anaemia is common due to haemolytic action of the bacteria on the red blood cells and bone marrow depression may occur due to toxemia. Hypocalcemia may be present due to binding of calcium to fatty acids in the necrotic tissues. Hyponatremia and hypoproteinemia are common and are caused by oedema and plasma losses into infected areas. Plain radiographs may reveal gas in the soft tissues in late stages [9]. Ultrasound is useful when there is collection of fluid and pus or involvement of superficial structures [10]. Fine needle aspiration

Fig. 1 : Necrotizing soft tissue infection involving abdominal wall (post debridement)

Fig. 2 : Extensive excision of necrosis tissue in a patient with necrotising soft tissue infection of genitals and perineum

Fig. 3 : Multiple incisions made parallel to cutaneous nerves and vessels in a patient with necrotizing soft tissue infections of chest and abdominal wall

cytology may detect organisms without indicating the depth and extent of disease [11]. Frozen section biopsy of the tissues may help in the diagnosis. Computerised tomographic (CT) scan is diagnostic, as it reveals the extent of disease which is important in initial surgical

debridement and helps in the decision for repeat debridement [12]. CT findings include asymmetric fascial thickening associated with fat stranding, focal fluid collections and gas tracking in a continuous manner along fascial planes without involving the muscles. Magnetic resonance imaging detects soft tissue fluids, visualizes pathological process but is not cost effective [13].

Once a necrotising soft tissue is recognised, prompt and aggressive treatment is essential. The four essential elements are resuscitation, broad spectrum IV antibiotic therapy, aggressive debridement and supportive treatment. Resuscitation includes fluid and electrolytic therapy and haemodynamic stabilization. Large volumes of fluid are sequestered in tissues and haemolysis occurs. Isotonic IV salt solutions are used for replacement and blood transfusion may be necessary. Calcium replacement may be required to correct hypocalcemia. Delayed treatment will increase the fluid requirement. Antibiotics are usually started empirically with penicillin, an aminoglycoside and clindamycin. Later culture results will dictate the antibiotics required. IV metronidazole or amphotericin may be required for fungal organisms. In patients with Gram-negative and Gram positive infection, imipenem/cilastin, ticarcillin/clavulanate, or ampicillin/sublactam are recommended. Vancomycin is used for its anticlostridial activity.

Once the diagnosis of necrotizing fascitis is suspected, immediate, aggressive and thorough surgical debridement should be done. The goal of surgical intervention is to remove all necrotic tissue and achieve haemostasis. The wounds are incised and drained. The incisions are made through the discoloured skin down to the fascia parallel to the cutaneous nerves and blood vessels (Fig 3). In necrotizing fascitis the grey necrotic fascia is identified and confirmed by the ease of blunt dissection. The tissue is excised for frozen sections or definitive histology and specimens are sent for Gram stain and culture. All necrotic tissue is excised, taking care to preserve the viable skin wherever possible. The deep fascia is opened to inspect the muscle and the wound is packed open for subsequent delayed closure or skin grafting. Counterincisions are made in areas of distant crepitus or oedema to assess the fascia in those locations. Gas may be present though not necessarily clostridial. Further stepwise debridement is usually required every 24-48 hours. In monomicrobial cellulitis only patchy necrotic areas will require excision. In clostridial myonecrosis, amputation of part or all of the limb may be required.

Supportive treatment includes use of respiratory support, central cardiovascular monitoring, haemodialysis, heparin and nutritional therapy in the form of oral or intravenous hyperalimentation [14]. Low

dose heparin is used to prevent deep vein thrombosis and also hastens recovery. Hyperbaric oxygen at 2-3 atmospheric pressure has been recommended for clostridial myonecrosis and for other necrotizing infections also. It inhibits clostridial growth and arrests alpha-toxin production. Hyperbaric oxygen can be used prior to definitive surgery, as it helps in defining the necrotic area. It should be administered three times a day for 48 hours, then twice a day for several days until the infection is controlled.

Necrotizing soft tissue infections still carry a substantial mortality. The most common causes of death associated with necrotizing soft tissue infections include sepsis, diffuse disseminated intravascular coagulation, respiratory failure, kidney failure and multisystem failure. A delay in operative treatment of greater than 24 hours and / or inadequate removal of infected tissue increases the morbidity and mortality rate significantly. Patients who underwent debridement within 24 hours had a mortality rate of 12.5% versus 72.7% in those where treatment was delayed by 4 days. Patients with severe underlying disease and those with chest, perineal and abdominal involvement have increased mortality as seen in this study. To summarise, it is a polymicrobial synergistic infection and a multipronged aggressive approach with intensive monitoring, broad spectrum antibiotics coverage guided by intraoperative cultures and plastic reconstructive techniques will bring down the mortality to an acceptable rate in these patients [15].

References

1. Chapnic EK, Abter EI. Necrotizing soft tissue infections. Infectious Disease Clinics of North America 1996;10:835-56.

2. Meleny FL. Haemolytic streptococcal gangrene. Arch Surg. 1924;9:317-64.
3. Brewer G, Meleny F. Progressive gangrenous infection of the skin and subcutaneous tissues, following operation for acute perforative appendicitis. Ann Surg 1926;84:438-50.
4. Janevicius R, Hann S, Batt M. Necrotizing fascitis. Surgery, Gynaecology, Obstetrics 1982;142:377-82.
5. Nehra D, Patel R, Ananthram P, Karmarkar SR. Necrotizing fascitis. Indian J Surg 1990;52:323-7.
6. Groth D, Henderson SO. Necrotizing Fascitis due to appendicitis. American J Emergency Medicine 1999;17(6):594-600.
7. Cihan A, Montes BB, Sudak G, Karamercan A., Naznedar R, Ferahkose Z. Fournier Gangrene after haemorrhoidectomy. Dis Colon Rectum 1999;42:1644-52.
8. Miller J. The importance of early diagnosis and surgical treatment of necrotizing fascitis. Surgery, Gynaecology and Obstetrics 1983;157:197-200.
9. Lille S, Sato T, Engrave L. Necrotizing soft tissue infections : obstacles in diagnosis. J Am Coll Surg 1996;182:7-11.
10. Walsha CF, Deans H. CT findings in necrotizing fascitis - a report of four cases. Clin Radiol 1996;51:429-32.
11. Stone D, Gorbach S. Necrotizing fascitis : the changing spectrum. Dermatol Clin 1997;213-20.
12. Wyoki W, Santora T, Shah R. Necrotizing fascitis : CT characteristics. Radiology 1997;203:859-63.
13. Freischlag J, Ajalat G, Busuttill R. Treatment of necrotizing soft tissue infections. Am J Surg 1985;149:751-5.
14. Majeski J, Alexander J. Early diagnosis, nutritional support, and immediate extensive debridement improve survival in necrotizing fascitis. Am J Surg 1983;145:784-7.
15. Harjai MM. Necrotizing fascitis - letter to editor. MJAFI 2000;56:273-4.

MEDICAL PHILATELY

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