

Necrotizing Fasciitis:

A Plea for Early Diagnosis and Treatment

JOHN D. CUNNINGHAM, M.D., LESTER SILVER, M.D.¹, AND DONALD RUDIKOFF, M.D.²

Abstract

Introduction: Necrotizing fasciitis is an uncommon infectious entity that poses difficult diagnostic and therapeutic management decisions.

Purpose: This paper addresses the presentation, evaluation and management of the patient with a necrotizing soft tissue infection.

Case Report: A 54-year-old man presented to his physician with pain and swelling of the left anterior chest wall following a presumed insect bite several days prior. He was treated with oral antibiotics but returned to the office three days later with increased swelling, pain, and erythema in the axilla. Necrotizing fasciitis was diagnosed. He was hospitalized and taken to the operating room for debridement of the chest wall. Extensive necrosis of the skin, subcutaneous tissue and muscle was encountered. Muscle debridement extended from the pectoralis major and both obliques anteriorly to the latissimus dorsi and para-spinalis muscles posteriorly. Multiple operative debridements were performed over several days. The patient developed septic shock requiring blood pressure support, and multiple organ system failure requiring hemodialysis, prolonged ventilatory support and eventual tracheostomy. Split-thickness skin grafts were placed during the third operative debridement and concluded on the 15th day of hospitalization. The patient eventually recovered and on the 53rd hospital day was transferred to the rehabilitation service, where he spent the next four weeks recovering movement in the left arm.

Conclusion: Necrotizing fasciitis is an infectious entity that requires rapid diagnosis, surgical debridement, and tissue coverage if the patient is to survive.

Key Words: Necrotizing fasciitis, soft tissue infection, myonecrosis, surgical debridement.

Introduction

NECROTIZING FASCIITIS IS AN UNCOMMON, critically serious infection of the subcutaneous tissue and fascia with relative sparing of the skin and muscle, both of which may be infected secondarily. Necrotizing soft tissue infections (NSTIs) are characterized by subepithelial invasion of the soft tissue by microorganisms, resulting in tissue edema, vascular thrombosis, and lymphocytic infiltration. Muscle necrosis may result from untreated infection. The diagnosis is often missed initially because of the paucity of clinical signs and symptoms or the clinician's lack of familiarity with the condition. Once the diagnosis is suspected, the patient needs to be adequately stabilized and then taken to the operating room for surgical exploration and debridement. Negative outcomes result from delay in diagnosis, inadequate surgical debridement, and complications of sepsis (1–6). The in-hospital mortality rate ranges from 6–33% (1–7); for those patients who do survive, significant disfigurement, disability and long-term rehabilitation are the rule.

Case Report

A 54-year-old, left-handed man developed pain in the left chest wall and shoulder a few days after working in the crawl space of his house. An area of redness, pain, and tenderness on the left chest and axilla was diagnosed as cellulitis due to an insect bite. The patient was started on oral azithromycin and non-steroidal, anti-inflammatory analgesics. He returned three days later and was referred for dermatological consultation where a diagnosis of necrotizing fasciitis was made. The man was agitated, demanding analgesics for severe pain in the left chest that radiated to the back. He momentarily lost consciousness in the dermatologist's office and was rushed to the emergency room.

His past medical history was significant for mild hypertension and an episode of perineal cellulitis two years previously. There was no previous history of diabetes mellitus, corticosteroid use, intravenous drug abuse, alcoholism, or peripheral vascular disease. He was allergic to penicillin.

Physical examination in the emergency room revealed a temperature of 36.5°C, pulse 90, respiratory rate 18, and blood pressure 80/55 mm Hg. The patient was awake but lethargic and there was generalized skin mottling over the entire body. An area of reddish-purple discoloration

From the Departments of ¹Surgery and ²Dermatology, Mount Sinai School of Medicine, New York, NY 10029.

Address correspondence to John D. Cunningham, M.D., 120 Summit Avenue, Summit, NJ 07060.

extended over the left axilla to the back and flank (Figs. 1, 2). This area was edematous, boggy and tender to palpation. The remainder of the examination was unremarkable.

Laboratory values on admission were significant for a white blood cell count of 7,600 with



Fig. 1. The patient is supine on the operating room table, intubated, with the left chest exposed showing the area of red-to-purple discoloration in the left axilla.

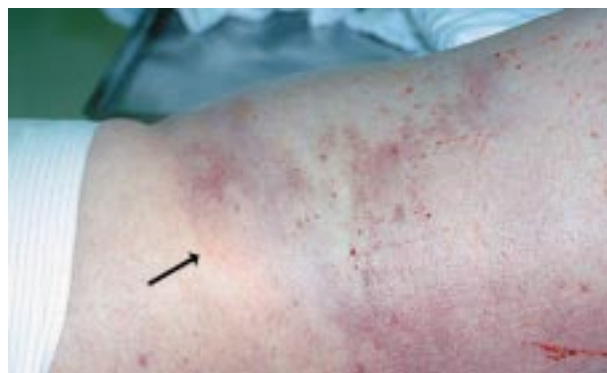


Fig. 2. The patient has been placed in the right lateral decubitus position. Extensive cyanosis is noted on the lower back with an advancing erythematous margin (arrow).

69% band cells, BUN of 32 mg/dL, creatinine of 3.0 mg/dL, and blood sugar of 240 mg/dL. An intravenous catheter was placed in the groin and treatment with vancomycin, aztreonam, and metronidazole was started. After adequate fluid resuscitation, the patient was taken to the operating room for operative debridement by a team of general and plastic surgeons.

In the operating room, he was intubated and positioned in the left lateral decubitus position. An incision was initiated in the left axilla, extended caudad to include the left chest wall down to the groin, and extended posteriorly to the midline of the back. The skin, subcutaneous tissue and muscle were all necrotic and the perforating vessels were thrombosed. The latissimus dorsi, and parts of the serratus, pectoralis major, external oblique and internal oblique were all resected (Figs. 3, 4). The wounds were packed with Betadine-soaked gauze and the patient was



Fig. 3. The patient after complete surgical debridement, with extension of the resection from the left shoulder down, including some of the pectoralis, down to the iliac crest and extending to the midline anteriorly over the abdominal cavity.



Fig. 4. The patient in the right lateral decubitus position, showing the extensive debridement from the left axilla to the iliac crest.

taken to the surgical intensive care unit. Gram stain from the first operation revealed Gram-positive cocci in clusters, and cultures grew group A beta-hemolytic streptococcus. His condition worsened that night and he was started on intravenous norepinephrine (Levophed®) to maintain a mean blood pressure of 60–70 mm Hg. He underwent further debridement on the 2nd and 3rd hospital days; no further infection was noted. On the third day, the wounds were covered in part by split-thickness skin grafts. The patient required three more operative treatments to cover all of the wounds with split-thickness skin grafts. The estimated body surface area that had been debrided and grafted was 18%. Pathological examination showed necrotic skin, adipose tissue and muscle with numerous bacteria.

The patient's overall condition during this time remained critical. Blood pressure support with intravenous norepinephrine (Levophed®) was required for the first nine days in the intensive care unit (ICU). He also received five units of packed red blood cells and six units of fresh frozen plasma. Although urine output was present, his creatinine clearance was low, thereby requiring the use of continuous veno-venous hemo-filtration (CVVH) to remove excess fluid. On the eighth hospital day, when the creatinine clearance improved, CVVH was discontinued. The patient began to show signs of improvement, with resolution of his multi-system organ failure. He remained sedated and intubated for daily bedside dressing changes. Total parenteral nutrition (TPN) was started on the fourth day since attempts at enteral nutrition failed as a result of a paralytic ileus. Enteral nutrition was successfully instituted a few days later and TPN was discontinued. The patient underwent tracheostomy on the 21st hospital day. His course was complicated by an episode of sinus arrest that required a temporary trans-venous pacemaker, and also by Gram-negative line sepsis. After four weeks in

the ICU, he was transferred to the surgical floor for an additional two weeks. He then spent another four weeks on the rehabilitation service, improving function in his left hand, arm and shoulder (Fig. 5).

Discussion

NSTIs have been described in the literature for centuries under a variety of names, including malignant ulcer, putrid ulcer, phagedena, Fournier's gangrene and hospital gangrene. A number of retrospective reviews have attempted to distinguish different necrotizing soft tissue infections based on anatomic location, bacterial flora, presence or absence of crepitance, and clinical progression (8). All of these classification schemes serve little purpose, since the initial treatment and management of all NSTIs are the same. Therefore, these sub-categories of NSTIs are important only from an academic point of view and one should maintain a broad therapeutic approach when dealing with any of these infections.

Epidemiology and Pre-existing Conditions

NSTI can occur at any age, including infancy (9). In selected larger series, the youngest person afflicted was 9 years old and the oldest 90, while the mean age varied from 32 years to 57 (1–7). The ratio of men to women is 1.4:1 but the reason for this difference is not explained (1–7). The majority of patients who develop NSTI have pre-existing conditions that render them susceptible to infection. These conditions may adversely affect their prognosis (1–7). The common denominator appears to be an immunosuppressive effect resulting from advanced age, diabetes mellitus, acute or chronic renal failure, local defects such as peripheral vascular disease or lymphedema that predispose to local cellulitis. A list of conditions which have been associated with NSTI is shown in

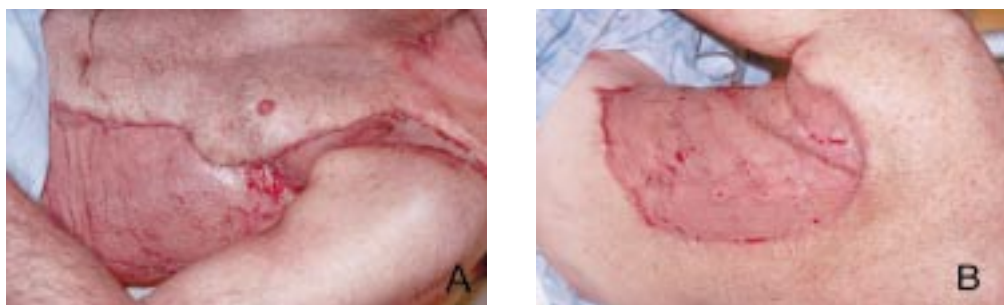


Fig. 5. Anterior (A) and posterior (B) views of the split-thickness skin graft.

Table 1 (1–7). Significant differences in the cause of NSTI exist from study to study and most likely reflect the distinct patient populations seen at each institution. Many patients have more than one pre-existing factor and the number of different factors may be related to outcome (4).

A number of other causes of NSTI have been implicated, as shown in Table 2 in some series (1–7). The etiologies most frequently implicated are intravenous drug abuse, previous trauma and soft tissue infections; however, many adult cases are idiopathic. Most NSTIs in infants are a result of secondary infection of omphalitis, balanitis, mammitis, postoperative complications, and fetal monitoring.

Presenting Symptoms and Physical Findings

Many patients with NSTI present initially to a primary care physician or dermatologist. There may be a history of soft tissue injury from an animal or insect bite, blunt or penetrating trauma, or subcutaneous injections of insulin or illicit drugs. Sometimes an inconsequential scratch or abrasion may be implicated. NSTI also occurs in the hospital setting as a postoperative complication. Distinguishing NSTI from other less severe infections is difficult but crucial since NSTI is a surgical emergency.

A majority of NSTI patients (47–94%) present with an erythematous, tender, swollen area resembling cellulitis with disproportionately

severe pain at the site of involvement (2, 4–6). Other less common presenting complaints include fever, chills, weakness, confusion, and rash. The most common physical findings are listed in Table 2. Due to the myriad presenting signs and symptoms, the diagnosis is often not apparent initially. Health-care providers must be cautious when confronted with such patients. Certain findings once believed to be classic are not as common as previously thought. The presence of crepitus on physical examination or soft tissue air on plain x-ray is seen in only 37% and 57% of patients respectively (4). The absence of such “classic” findings does not rule out NSTI, so one should not be lulled into a state of complacency if they are not present (2, 4, 6). On the other hand, the presence of these findings is pathognomonic for NSTI.

As the infectious process progresses, the skin characteristically becomes more erythematous, painful and swollen, with indistinct borders. The skin develops a violaceous hue, may become necrotic with bullae formation, and eventually appears hemorrhagic and gangrenous (10). There may be involvement of more than one area, separated by islands of normal skin. Involvement of the muscle and/or nerves may lead to weakness and loss of sensory innervation.

Symptoms of NSTI are often present for several days before the patient presents. As a result of this delay, signs of advanced infection may be present. Hypotension, tachycardia, mental

TABLE 1
Pre-existing Conditions that Have Been Associated with Necrotizing Soft Tissue Infections and Presumed to Be Causes of These Types of Infections.

Pre-existing Factor	Incidence	Reference
Diabetes mellitus	21–64%	1, 2, 4, 6, 7
Chronic renal insufficiency	10%	4
Peripheral vascular disease	15–80%	2, 4–7
Acute renal failure	15%	4
Age > 60	31%	4
Intravenous drug abuse	8–67%	1–4, 6
Obesity	18–46%	1, 2, 4, 6, 7
Chronic alcoholism	12–31%	1, 2, 4
Malnutrition	18–40%	1, 2, 4–6
Steroids	12%	4
Paraplegia	3%	6
Cancer	2–12%	1, 4
Etiology		
Infections, soft tissue	11–90%	1, 5, 6
Intravenous drug abuse	27–56%	1–4, 6
Post-operative infection	3–14%	1, 6, 7
Perirectal abscess	9–15%	1, 6
Trauma	15–55%	1, 3, 6
Idiopathic	2–38%	1, 3, 6, 7
Insect bites	6–7%	3, 6

TABLE 2

Signs and Symptoms Found at the Time of Admission for Patients Presenting with Necrotizing Soft Tissue Infections.

	Frequency	Reference
Symptom		
Pain	47–94%	2, 4–6
Mental obtundation	18%	4
Sign		
Swelling	67–75%	2–4
Erythema/cellulitis	66–100%	1–7
Crepitus	12–36%	2, 4, 6
Fever > 100.5°F	32–100%	1, 2, 4, 6, 7
Skin slough/necrosis	31–47%	1–4, 7
Blistering/bullae	10–24%	3, 4, 7
Skin discoloration	18%	4
Hypotension	11%	4
Laboratory findings		
Leukocytosis (>10,000/mm ³)	49–88%	1, 2, 4, 6
Anemia	38–64%	2, 4, 6
Acidosis (<7.36)	33–35%	1, 4, 6
X-ray findings		
Soft tissue air		
Plain x-ray	17–57%	1, 2, 4, 7
CT	12%	4

obtundation, skin mottling, acute renal failure, lactic acidosis, leukocytosis, and increased cardiac output with concomitant, reduced peripheral-vascular resistance may be encountered (Table 2). Some laboratory findings common in NSTI, but by no means diagnostic, include elevated white blood cell count, glucose, lactate, BUN and creatinine levels. Anemia, hypocalcemia, hypoalbuminemia, an altered coagulation profile, and decreased oxygenation may also be present (1, 2, 4, 6). Such findings are not present in all patients but may be helpful in suggesting the diagnosis (Table 2).

Radiographic evaluation has been used primarily for patients whose diagnosis was in doubt. Plain films of the involved area may show evidence of soft tissue air (2–4, 7). This is a significant finding in any patient and needs to be addressed immediately with surgical exploration. Computed tomography (CT) and magnetic resonance imaging (MRI) have also been employed in equivocal cases and have sometimes been helpful in defining the presence and extent of infection (4, 11, 12). It cannot be overemphasized, however, that these studies are only adjuncts in the evaluation of patients with potential NSTI and should not be relied upon to exclude that diagnosis. The diagnosis is still primarily a clinical one. Most important, the extent of debridement is determined by physical findings at the time of surgery and not by CT findings.

Some authors advocate the use of fine-needle aspiration for diagnosing NSTI but this procedure has limited usefulness and can be misleading. A positive aspiration confirms infection, but a negative examination does not exclude it. If the diagnosis is suspected, it is our opinion that a more definitive procedure such as incisional biopsy or surgical exploration is indicated (3).

A more recent approach to the diagnosis of NSTI is bedside incisional biopsy down to the fascial level (13). This biopsy is immediately sent for frozen section culture and Gram stain. This analysis is far more accurate than that performed on a fine-needle aspirate. Incisional biopsy appears to be the only reasonable approach to the diagnosis other than a trip to the operating room. Punch biopsy should not be used as an alternative to scalpel incisional biopsy, since it can easily miss the pathology.

Sites of Presentation

Although NSTIs can occur anywhere on the body, they are more common in certain locations. Perineal soft tissue infections, termed Fournier's gangrene, are seen in up to 36% of cases (1, 3, 4) and the extremities are involved in 36–53% of NSTI. The trunk accounts for 18–64% of cases (1–7). The distribution reflects the particular patient population seen at each institution.

Bugs and Drugs

All tissues obtained at the time of initial surgical debridement should be subjected to aerobic and anaerobic cultures and Gram staining. Although the Gram stain has been suggested as a guide to initial antibiotic therapy of NSTI, it is of limited value given the polymicrobial etiology of most cases. Recently, two cases of Group A streptococcal (GAS) associated NSTI were reported in which GAS was identified using the standard rapid streptococcal diagnostic kit (14).

Cultures of NSTI have yielded a wide variety of pathogenic organisms in different studies (Table 3). Factors that affect bacterial isolates identified include handling of specimens at surgery, use of aerobic and anaerobic culture media, delay in plating in the laboratory and final interpretation of cultures. Other variables that determine the bacteriology of NSTI include site of infection and clinical setting. Postoperative abdominal wound infections and perineal infections may yield gastrointestinal flora.

Infection with a single pathogen occurs in 15–29% of cases and polymicrobial infections are seen in the remainder (1, 3, 4, 6). Of the latter, roughly one-half will have mixed aerobic flora, and the other half mixed aerobic and anaerobic growth (1–7). In a small number of cases (7–15%), only anaerobic bacteria are identified, and rarely no organisms are isolated (1, 2, 4).

GAS infections have received much attention recently as etiologic agents of NSTI (15–19), referred to in the lay press as “flesh-eating bacteria.” In a recent study, GAS were isolated in only 16% of patients with necrotizing fasciitis (20). Of the 8 patients in whom GAS was isolated, one grew only streptococci, and in six it was accompanied in two patients each by *Staphylococcus aureus*, *Acinetobacter anitratus* and *Klebsiella pneumoniae*. There was mixed growth in the remaining patient. Another recent study identified GAS in 10 of 14 patients (71%) with necro-

tizing fasciitis (8). In a study of necrotizing skin infections in children, GAS was seen as a single organism in only 25% percent of cases while the remaining cases were polymicrobial (21).

An important aspect of streptococcal NSTIs is that they can occur in otherwise healthy people at any age and may cause rapid onset of shock and multiple-organ failure. They may follow minor or major trauma, injection of illicit drugs, accidental needle sticks and varicella infections in children and adults (22–25). Elderly individuals and patients with underlying medical disease are at greater risk for serious GAS infections, necrotizing fasciitis and shock (26). Necrotizing GAS infections may occur anywhere on the body, including the trunk, extremities and even the periorcular area (18).

Streptococcal toxic-shock syndrome is a complication of GAS infections. It is associated with necrotizing fasciitis in 50% of cases (27). Patients present with cutaneous pain, fever, chills, muscle ache and malaise. They may develop nausea, vomiting, diarrhea, tachycardia, tachypnea, hypotension and renal failure. Cases of so-called “toxic strep syndrome” are most commonly associated with group A streptococci M1 and M3. Streptococcal pyrogenic exotoxins which cause the rash of scarlet fever are also known to act as superantigens. It has been suggested that shock may be mediated by massive release of cytokines such as tumor necrosis factor alpha and interleukin-1 beta induced by streptococcal pyrogenic exotoxin A (SPEA) and streptolysin O (SLO) (28).

Patients suspected of having NSTI should be started empirically on broad-spectrum antibiotics covering the most commonly encountered pathogens (Table 3). The most frequently advocated antibiotic regimen includes ampicillin/penicillin, gentamicin and anaerobic coverage with either metronidazole or clindamycin (1–7). Penicillin-allergic patients may be started on aztreonam or vancomycin in place of ampicillin

TABLE 3

A List of the Most Common Bacterial Isolates for Patients Presenting with Necrotizing Soft Tissue Infections.

Gram-positive organisms*	(%)	Gram negative organisms*	(%)
Group A streptococci	18–46	<i>E. coli</i>	8–28
Enterococci	16–34	Enterobacter	2–12
Coagulase (-) Staph	15–37	Pseudomonas species	9–20
<i>Staph. aureus</i>	9–37	Proteus species	6–12
<i>Staph. epidermidis</i>	18	Serratia species	2–6
Clostridial species	5–21	Bacteroides	18–48
Mixed Gram positive	10	Mixed Gram negative	16

* References 1–7.

or penicillin. There is experimental evidence suggesting that clindamycin may be superior to penicillin in overwhelming streptococcal infections. Penicillin may be less effective due to an "inoculum effect" of large numbers of slower-growing organisms with decreased expression of certain penicillin-binding proteins (27). Clindamycin, which works by inhibiting protein synthesis, is not subject to such effects; moreover, it suppresses the production of certain bacterial toxins. It may be given in conjunction with penicillin.

Subsequent antibiotic management is guided by the sensitivities of the organisms identified from intraoperative cultures. Incorrect handling of specimens may result in falsely negative anaerobic cultures, so antibiotic coverage of anaerobic organisms is still advisable. Antibiotic coverage should be continued until the infection is under control and for at least 48 hours after the temperature and white blood cell count have returned to normal.

Treatment

Once the diagnosis of NSTI is made, treatment should be instituted promptly. Resuscitation, based on the clinical state of the patient, includes aggressive fluid replacement to manage acute renal failure from ongoing sepsis and shock. Intravenous antibiotics are given and appropriate measures are taken to maintain cardiac output and pulmonary stability. Urine output should be monitored via an indwelling urinary catheter.

The patient should be brought to the operating room without unnecessary delay and undergo aggressive and extensive operative debridement by a team of general and plastic surgeons (1–7). The skin, soft tissue and muscle should be debrided until there is no further evidence of infected tissue, based solely on the findings at surgery (Figs. 3, 4). The first operative debridement is the most important one for the survival of the patient. It is preferable to remove more tissue than necessary than to leave any actively infected or necrotic tissue. The patient should then be returned to the operating room daily to confirm that there has been no extension of the infectious process and to debride any skin and soft tissue edges that have become desiccated. The total number of trips to the operating room is based on the condition of the wound and whether the infection has been adequately controlled. Once the infection is controlled, daily dressings can be done at the bedside, with sedation. Our approach is to cover soft tissues with split-thickness skin

grafts once the infection has been eradicated. Only rarely are more extensive procedures needed in the acute setting. For our patient, this was begun on the third visit to the operating room and was completed by the 15th day of hospitalization. Prompt closure of wounds is important to eliminate a portal of entry for continued bacterial infection, to prevent ongoing serum loss, and to allow for early rehabilitation. After the patient has recovered from the acute process, the plastic surgeon may perform tissue transfer to close persistent defects or repair areas that need more than skin coverage.

The role of amputation in controlling NSTI is controversial. If infection can only be eradicated by amputation, it should be done promptly and without hesitation. Controversy also exists concerning the role of colostomy in patients with perineal wounds. If there is regular fecal contamination of the wounds, colostomy should be performed. Because NSTI can involve the abdominal wall in the usual sites of colostomy placement, this procedure should be performed only after control of the infection and in an uninvolved area.

While some authors have advocated the use of hyperbaric oxygen in the treatment of NSTI, in addition to operative debridement, its usefulness is controversial (4, 29, 30). It has never been shown to improve survival rates when compared to standard operative and supportive therapy (4, 29, 30). Moreover, few institutions have facilities for hyperbaric treatment and patients are often too ill to be transported there for treatments.

Postoperative Course and Complications

Patients with NSTI can develop multiple-organ-system failure and should be managed in an ICU. Respiratory insufficiency is common and prolonged mechanical ventilation and tracheostomy are often required. Renal failure is usually transient and can be managed medically. Either intermittent hemodialysis or continuous arterio-venous or veno-venous hemofiltration may be used, as was the case in our patient, to ameliorate the problems associated with acute renal failure. Renal function usually improves once infection has been controlled. Blood pressure support is often required in the immediate postoperative setting due to septic shock. Central venous and cardiac pressures are routinely monitored with a Swan-Ganz catheter and adrenergic agents are used to maintain blood pressure and cardiac output. Hemodynamic parameters usually improve with control of infection.

When the patient has completely recovered from the acute effects of NSTI, rehabilitation should be instituted as soon as possible, to ensure complete functional recovery. The particular therapy will depend on the area affected, the amount of tissue removed, the muscles involved and the duration of the hospital stay. Physical therapy can often be started in the ICU and then be continued when the patient is transferred to the surgical floor. If long-term reconditioning is necessary, the patient should be transferred to a skilled rehabilitation facility prior to returning home.

Prognostic Factors for Mortality

The reported mortality rate for NSTI varies from 6% to 33% (1–7). The relative importance of some prognostic factors has been investigated (Table 4), but they are only of academic interest since the physician has little or no control over most of them.

Elliott et al. demonstrated that a combination of factors is more predictive of outcome than any individual factor (4). In their study, diabetes mellitus did not predict outcome, but when combined with peripheral vascular disease, renal failure, and age greater than 60, it was associated with a significant increase in mortality (4). Elliott et al. also showed that elevated serum creatinine, blood lactate, and organ failure at the time of admission predicted a poor outcome (4). The white blood cell count, serum glucose level, and platelet counts were not associated with an unfavorable prognosis (4). While the site of infection has been suggested to impact on survival, it is difficult to ascertain because studies have had different distributions of involvement. Despite this, truncal and perineal

infections are thought to have higher mortalities (4). Delay in diagnosis and surgical management are frequently implicated as important predictors of increased mortality (1–6). Inasmuch as patients often delay in seeking medical attention, the physician has limited control over mortality rates. When the patient does finally present, the diagnosis is often missed. Sometimes, the diagnosis of NSTI is not entertained until the situation worsens. Delay in diagnosis allows more time for the infection to advance and this adversely affects outcome (1–6). An increased index of suspicion for NSTI may hasten the diagnosis and treatment, thus improving the prognosis.

The major factor that physicians can control is the extent of debridement, and this appears to be the most important determinant of survival (1–7). Inadequate surgical debridement at the initial operative procedure results in adverse outcomes, with disease progression, multi-system failure and death.

Conclusions

NSTIs are relatively uncommon infections that often present for medical attention late in their course. The diagnosis is often missed at initial presentation, allowing further progression of the infectious process. Patients most commonly present with pain at a soft tissue site, with erythema and tenderness. The diagnosis is made clinically based on the visual findings in the infected area and by a high index of suspicion on the part of the clinician. Laboratory tests and plain x-rays may support the diagnosis but are frequently normal despite ongoing infection. CT and MRI are sometimes useful, but the critical condition of the patient often precludes their use. They should not be relied upon to exclude the diagnosis of NSTI if the diagnosis is suggested. Once the diagnosis has been made, the patient is stabilized and taken to the operating room for debridement. Surgical debridement should be performed daily until the acute infection has been controlled. Outcomes are based on the promptness of diagnosis, surgical treatment, and the management of postoperative complications. A multi-disciplinary team of health-care personnel, including general surgeons, plastic surgeons, infectious disease specialists, intensivists, rehabilitation staff and nursing staff, are needed to provide the extensive resources and time it takes for patient recovery.

References

1. Bosshardt TI, Henderson VJ, Organ CH. Necrotizing soft-tissue infections. *Arch Surg* 1996; 131:846–854.

TABLE 4

Factors that Have Been Evaluated to Determine the Mortality Rate in Patients with Necrotizing Soft Tissue Infections.

No effect on mortality	Increases mortality
Race	Cardiac disease
Blood glucose	Presence of malignancy
Platelet count	Malnutrition
Hepatic disease	Intravenous drug abuse
HIV	Renal failure
Albumin level	Peripheral vascular disease
Myonecrosis	Elevated serum lactate
Abnormal vital signs	Obesity
Pulmonary disease	Body surface involved
	Diabetes mellitus
	Age > 60
	Delay in operative debridement
	Limited incision and drainage

2. McHenry CR, Piotrowski JJ, Teprinic D, Malangoni MA. Determinants of mortality for necrotizing soft-tissue infections. *Ann Surg* 1995; 221:558–565.
3. Lille St, Sato TT, Engrav LH, et al. Necrotizing soft tissue infections: Obstacles in diagnosis. *J Am Coll Surg* 1996; 182:7–11.
4. Elliott DC, Kufera JA, Myers RAM. Necrotizing soft tissue infections. Risk factors for mortality and strategies for management. *Ann Surg* 1996; 224:672–683.
5. Majeski JA, Alexander JW. Early diagnosis, nutritional support, and immediate extensive debridement improve survival in necrotizing fasciitis. *Am J Surg* 1983; 145:784–787.
6. Sudarsky LA, Laschinger JC, Coppa GF, Spencer FC. Improved results from a standardized approach in treating patients with necrotizing fasciitis. *Ann Surg* 1987; 206:661–665.
7. Rubinstien E, Dehertogh D, Brettman L. Severe necrotizing soft tissue infections: Report of 22 cases. *Conn Med* 1995; 59:67–72.
8. Jarrett P, Rademaker M, Duffill M. The clinical spectrum of necrotizing fasciitis. A review of 15 cases. *Aust N Z J Med* 1997; 27:29–34.
9. Hsieh WS, Yang PH, Chao HC, Lai JY. Neonatal necrotizing fasciitis: A report of three cases and review of the literature. *Pediatrics* 1999; 103(4):e53.
10. Buchanan Necrotizing fasciitis due to group A beta-hemolytic streptococci. *Arch Dermatol* 1970; 101(6):664–668.
11. Beltran J. MR Imaging of soft tissue infection. *Magn Reson Imaging Clin N Am* 1995; 3:743–751.
12. Rahmouni A, Chosidow O, Mathieu D, et al. MR imaging in acute infectious cellulitis. *Radiology* 1994; 192:493–496.
13. Majeski J, Majeski E. Necrotizing fasciitis: Improved survival with early recognition by tissue biopsy and aggressive surgical treatment. *South Med J* 1997; 90:1065–1068.
14. Ault MJ, Geiderman J, Sokolov R. Rapid identification of Group A streptococcus as the cause of necrotizing fasciitis. *Ann Emerg Med* 1996; 28:227–230.
15. Weinbren MJ, Perinpanagayam RM. Streptococcal necrotizing fasciitis. *J Infect* 1992; 25:299–302.
16. Donaldson PMW, Naylor B, Lowe JW, et al. Rapidly fatal necrotizing fasciitis caused by *Streptococcus pyogenes*. *J Clin Pathol* 1993; 46:617–620.
17. Chelsom J, Halstensen A, Haga T, Hoiby EA. Necrotising fasciitis due to group A streptococci in western Norway: Incidence and clinical features. *Lancet* 1994; 344(8930):1111–1115.
18. Marshall DH, Jordan DR, Gilberg SM, et al. Periocular necrotizing fasciitis: A review of five cases. *Ophthalmology* 1997; 104:1857–1862.
19. Kliska DL, Thiede B, Caracciolo J, et al. Invasive Group A Streptococcal infections in North Carolina: epidemiology, clinical features, and genetic and serotype analysis of causative organisms. *J Infect Dis* 1997; 176:992–1000.
20. Singh G, Ray P, Sinha SK, et al. Bacteriology of necrotizing infections of soft tissues. *Aust N Z J Surg* 1996; 66:747–750.
21. Brook I. Aerobic and anaerobic microbiology of necrotizing fasciitis in children. *Pediatr Dermatol* 1996; 13:281–284.
22. Sutherland ME, Meyer AA. Necrotizing soft-tissue infections. *Surg Clin North Am* 1994; 74:591–607.
23. Hagberg C, Radulescu A, Rex JH. Necrotizing fasciitis due to group A streptococcus after an accidental needle-stick injury [letter; comment]. *N Engl J Med* 1997; 337:1699.
24. Aebi C, Ahmed A, Ramilo O. Bacterial complications of primary varicella in children. *Clin Infect Dis* 1996; 23:698–705.
25. Waldhausen JH, Holterman MJ, Sawin RS. Surgical implications of necrotizing fasciitis in children with chickenpox. *J Pediatr Surg* 1996; 31:1138–1141.
26. Davies HD, McGeer A, Schwartz B, et al. Invasive group A streptococcal infections in Ontario, Canada. Ontario Group A Streptococcal Study Group. *N Engl J Med* 1996; 335:547–554.
27. Bisno AL, Stevens DL. Streptococcal infections of skin and soft tissues. *N Engl J Med* 1996; 334:240–245.
28. Hackett SP, Stevens DL. 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* 1992; 165:879–885.
29. Riseman JF, Zamboni WA, Curtis A, et al. Hyperbaric oxygen therapy for necrotizing fasciitis reduces mortality and the need for debridements. *Surgery* 1990; 108:847–850.
30. Pessa ME, Howard RJ. Necrotizing fasciitis. *Surg Gynecol Obstet* 1985; 161:357–361.