Necrotizing Fasciitis

Abstract

Necrotizing fasciitis is a rare but life-threatening soft-tissue infection characterized by rapidly spreading inflammation and subsequent necrosis of the fascial planes and surrounding tissue. Infection typically follows trauma, although the inciting insult may be as minor as a scrape or an insect bite. Often caused by toxin-producing, virulent bacteria such as group A streptococcus and associated with severe systemic toxicity, necrotizing fasciitis is rapidly fatal unless diagnosed promptly and treated aggressively. Necrotizing fasciitis is often initially misdiagnosed as a more benign soft-tissue infection. The single most important variable influencing mortality is time to surgical débridement. Thus, a high degree of clinical suspicion is necessary to avert potentially disastrous consequences. Orthopaedic surgeons are often the first to evaluate patients with necrotizing fasciitis and as such must be aware of the presentation and management of this disease. Timely diagnosis, broad-spectrum antibiotic therapy, and aggressive surgical débridement of affected tissue are keys to the treatment of this serious, often life-threatening infection.

Although necrotizing fasciitis garnered tabloid fame as a sensationalistic “new” and dangerous “flesh-eating bacterium” in the 1990s, it was described by Hippocrates in the 5th century BCE. Necrotizing fasciitis has since had many names, including phagedena, phagedena gangrenosum, progressive bacterial synergistic gangrene, and nonclostridial gas gangrene. The first report in the United States was made in 1871 by Confederate Army surgeon Joseph Jones, MD, who described “hospital gangrene” with a mortality rate of almost 50%. In 1883, Fournier identified his eponymous form of gangrene and so described the pathology of necrotizing fasciitis affecting the perineum and external genitalia. Melene identified hemolytic streptococcus as the etiologic agent of the disease in 1924. Wilson coined the term “necrotizing fasciitis” in 1952 and described the inflammation and necrosis of the subcutaneous fat and deep fascia, with sparing of the muscle, that are the cardinal features of this devastating disease. This characterization agrees with the current belief that a disease process, rather than a particular organism, is implicated in this condition.

Etiology

Necrotizing fasciitis typically follows an injury to the involved site. Even a minor lesion may be sufficient to allow bacteria to breach the skin barrier. Necrotizing fasciitis has been associated with minor or blunt trauma, insect bites, surgical incisions, cuts, abrasions, contusions, injection sites, cutaneous ulcers, perirectal abscesses, incarcerated hernia, burns,
splinters, childbirth, chicken pox, penetrating injury, and muscle strains. Although the skin is the most common portal of entry, in 45% of cases, no definitive access point can be found, possibly because the inciting insult was so minor as to be forgotten. The extremities are most commonly involved, but necrotizing fasciitis can affect any body part. Infections of the trunk and perineal regions have a higher mortality rate than those of the extremities, presumably because amputation is not feasible as a life-saving option.

**Risk Factors**

Any host condition that results in an immunocompromised state is a risk factor for necrotizing fasciitis. Diabetes mellitus, the most common comorbidity, is present in 18% to 60% of cases. Other risk factors include obesity, peripheral vascular disease, intravenous drug use, alcohol abuse, malnutrition, smoking, chronic cardiac disease, chronic corticosteroid therapy, chronic immune suppression, cancer, and age. The continued or chronic use of nonsteroidal anti-inflammatory drugs has been suggested as a possible risk factor because it may mask initial symptoms and delay diagnosis. Although numerous risk factors have been identified, half of all cases of necrotizing fasciitis occur in previously healthy individuals.

**Clinical Presentation**

Necrotizing fasciitis typically begins as a slightly inflamed area of soft tissue that suddenly and dramatically progresses to overt fasciitis with systemic toxicity. Early diagnosis is made difficult by a paucity of skin findings in the first stages of disease. Consequently, the infection is often incorrectly identified as cellulitis. A high index of suspicion is critical to avoid morbidity and mortality because, although cellulitis without deeper involvement is usually treatable with antibiotics alone, necrotizing fasciitis requires surgical débridement. The triad of swelling, erythema, and inordinate pain—often disproportionate to the observed lesion—in a patient who is thought to have cellulitis should raise suspicion of necrotizing fasciitis. Pain may precede warming and induration of skin (ie, wooden skin) by several hours. Rapid migration of the margins of erythema and skin induration (>1 cm/hr) despite the use of intravenous antibiotics is another important clue in the early stages of the disease.

Classic signs of necrotizing fasciitis develop as the disease progresses. Blisters and bullae form and drain first serosanguineous and then hemorrhagic fluid (Figure 1). The skin may also show violaceous discoloration before turning frankly necrotic and sloughing (Figure 2). Crepitus may be present if there is soft-tissue gas. Edema develops rapidly. Pain disproportionate to injury seen earlier in the process gives way to analgesia as cutaneous nerves are destroyed. Superficial fat and fascia necrose, producing the watery, grayish, often foul-smelling “dishwater pus” characteristic of the disease. By this stage, the patient shows constitutional symptoms of fever, chills, hypotension, tachycardia, and possibly an altered level of consciousness, and is critically ill (Table 1).

Acute renal failure is present in 35% of patients, coagulopathy in 29%, liver function tests are abnormal in 28%, acute respiratory distress syndrome is seen in 14%, and bacteremia is seen in 46%. The clinician must not be inappropriately reassured by the absence of these findings because many patients may lack these signs on presentation. In one series, only 47% of patients presented with classic skin changes of bullae, vesicles, and necrosis, and only 51% were febrile. Disproportionately severe pain, the most sensitive symptom, is noted in nearly 100% of patients with necrotizing fasciitis.
Diagnosis

Diagnosis is primarily clinical, and no test is as valuable as a high degree of suspicion. Laboratory and radiologic evaluations can be helpful but are best used for confirmation of the diagnosis. Pursuing such tests should never delay surgical intervention. Average time from onset of initial symptoms to diagnosis is 2 to 4 days, although some cases may take weeks to be correctly identified.25

Laboratory Evaluation

Initial laboratory evaluation should include a complete blood count, comprehensive metabolic panel, and coagulation studies. Blood cultures should be taken to obtain pathologic diagnosis of involved microorganisms and antibiotic sensitivities to guide future targeted antibiotic therapy. Arterial blood gases should be assessed, especially when signs of sepsis are present.7 Azotemia, hypernatremia, hypoproteinemia, thrombocytopenia, hematuria, elevated creatine kinase and erythrocyte sedimentation rates, metabolic acidosis, hypoalbuminemia, anemia, and hyperbilirubinemia are commonly noted. Hypocalcemia secondary to extensive fat necrosis may be seen. Few metabolic abnormalities may be noted early on, but as the disease proceeds toward sepsis and organ failure, laboratory findings may become extremely deranged.

In addition to clinical monitoring, routine laboratory evaluations can be used to distinguish necrotizing fasciitis from other soft-tissue infections. Wall et al26 described a model based on the white blood cell (WBC) count and serum sodium levels. They reported that concurrent findings of a WBC count >15,400 cells/µL and a serum sodium level <135 mmol/L are 90% sensitive for necrotizing fasciitis. However, this model lacks specificity (76%) and has a poor positive predictive value (26%) for necrotizing fasciitis, suggesting that it is useful only for ruling out the disease. Another model for evaluating group A streptococcus soft-tissue infections found that C-reactive protein levels >16 mg/dL are 89% sensitive and 90% specific for necrotizing fasciitis, and that creatine kinase levels >600 U/L are 58% sensitive and 95% specific for necrotizing fasciitis as opposed to cellulitis.18 Wong et al17 developed the Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC). This method differentiates necrotizing fasciitis from other soft-tissue infections using the parameters of C-reactive protein level, total WBC count, and hemoglobin, serum sodium, creatinine, and glucose levels (Table 2). The probability of necrotizing fasciitis is <50% with a score ≤5, 50% to 75% with a score of 6 or 7, and >75% with a score ≥8 (minimum score, 0; maximum, 13). The model had a 92% positive predictive value and a 96% negative predictive value for detecting early cases of necrotizing fasciitis in patients with severe soft-tissue infections when 6 was used as the cut-off score.27

Radiologic Evaluation

Plain radiographs are most useful for detecting gas in soft tissues. However, their utility is limited, as studies are often normal until infection and necrosis are advanced, and in many cases, soft-tissue gas never presents.

Computed tomography (CT) scans are more beneficial than plain radiographs. Increased attenuation of the subcutaneous fat with stranding is seen in 80% of cases, and fascial thickening may be present.25 Images may show gas in soft tissues or tracking along fascial planes.25 CT scans are especially sensitive in iden-
tifying soft-tissue edema and often can be more useful than physical examination in defining the margins of infection. However, abnormal CT findings are not universal, and cases have been reported of necrotizing fasciitis with negative CT findings.24

Magnetic resonance imagining studies have demonstrated a high sensitivity (93% to 100%) for diagnosing necrotizing fasciitis.24 Liquefactive tissue necrosis and inflammatory edema create fascial fluid that is detected as abnormally increased signal intensity on T2-weighted images. On T1-weighted images, necrosis and edema present as variably increased signal intensity along thickened deep fascial planes.25 Although magnetic resonance imaging is very useful in delineating the extent of infection, it is of lesser priority than surgical débridement of necrotic tissue when a patient is unstable with worsening signs of sepsis.

**Biopsy**

The diagnostic benchmark for necrotizing fasciitis is the finding of fascial necrosis during surgery. Outside the operating room, tissue-based diagnostic procedures may be of use. The “finger test” is a bedside procedure that can confirm necrotizing fasciitis. Under local anesthesia, a 2-cm incision is made down to the deep fascia, and a gloved finger is inserted to its base. The presence of dishwater pus, lack of bleeding on incision, and lack of tissue resistance to blunt finger dissection define a positive test. Frozen-section biopsy can also be done at bedside. A small elliptical section of skin, deep tissue, and fascia is taken from the suspected area, along with another from the leading edge of any erythema, induration, or necrosis. The specimens are then submitted for Gram stain, frozen section, and culture. This method allows an accurate diagnosis of necrotizing fasciitis to be made within 15 minutes of biopsy and reduces the time between onset of symptoms and diagnosis. Although mortality rates are lower with this technique, it is somewhat cumbersome and requires the immediate availability of an experienced pathologist.24 Frozen-section biopsy is best used in cases in which the diagnosis is unclear and the patient is stable. Surgical intervention should never be postponed to perform such a biopsy in cases in which necrotizing fasciitis is strongly suspected.

**Mortality**

There are an estimated 500 to 1,000 cases of necrotizing fasciitis in the United States annually,4 with mortality ranging from 6% to 76%.27 A recent review of 147 patients revealed a mortality rate of 9.3%, suggesting that outcomes are improving, presumably because of more effective patient management.30 Patients aged <1 year or >60 years have the highest mortality rates.11 Thrombocytopenia, abnormal liver function tests, low serum albumin level,31 acute renal failure, and elevated blood lactate level are significantly associated with mortality,7 while advanced age, streptococcal toxic shock syndrome, and immunocompromised status are independent predictors of death.12 A study of 99 patients with necrotizing fasciitis found that the risk of death increased by 4% for every year of life.6 However, the most important clinical variable affecting mortality is the time from admission to initial débridement, making rapid diagnosis of the utmost importance.7

**Histopathology**

Necrotizing fasciitis is characterized by necrosis of the superficial fascia, fat, and nerves, with thrombosis and suppuration of the arteries and veins (Figure 3). Obliterative vasculitis of the subcutaneous vessels is seen early in the disease.13 There is expansion of fibrous septa by edema, mixed inflammatory cell infiltrate, and early fibroblastic proliferation.29 As the disease progresses, lesions develop liquefactive necrosis at all involved tissue levels. A dense neutrophil–predominant inflammatory infiltrate is present, and Gram stain of the affected tissues is usually positive.24

**Microbiology**

Aerobic, anaerobic, gram-positive and -negative organisms, and even fungi have been implicated in necrotizing fasciitis (Table 3). The infection can be classified into three groups based on Gram stain and bacterial culture results. Type 1 constitutes 80% to 90% of all cases and is a polymicrobial infection involving non–group A *Streptococcus* along with anaerobes and/or facultative anaerobes. It often involves enterobacteriaceae as well. Typically, four to five species will be cultured from the wound. This type is associated with postoperative abdominal and perineal infections; it is most common in immunosuppressed patients. Virulence is not well understood but is speculated to be the result of synergy between bacterial species.

Type 2 necrotizing fasciitis is defined by the presence of group A β-hemolytic streptococci, typically as a single agent,20 although *Staphylococcus* or other organisms may be present. This is the “flesh-eating” presentation of necrotizing fasciitis.13 Infection usually occurs in an extremity and can develop in healthy individuals.

Type 3 necrotizing fasciitis is caused by marine vibrios (gram-negative rods). *Vibrio vulnificus* is thought to be the most virulent of these agents. Infection
usually begins with a puncture wound caused by a fish, or a cut or insect bite that is then exposed to seawater or marine animals. Soft-tissue damage is thought to be mediated by an extracellular toxin synthesized by the vibrio organism.

*Klebsiella*, *Escherichia coli*, and other uncommon organisms have also been reported as causative agents of necrotizing fasciitis. Recently, cases of necrotizing fasciitis resulting from community-acquired methicillin-resistant *Staphylococcus aureus* (MRSA) have been reported.

The microbiology of type 2 necrotizing fasciitis is the best characterized of the three groups. The natural habitat of group A *Streptococcus* is human skin and mucosal surfaces. It has coevolved with humans and does not survive outside a human host. The bacterium boasts an enormous and evolving molecular diversity, driven by horizontal transmission among group A streptococci strains and also between group A and other streptococci. Acquisition of prophages confers virulence through phage-associated factors and increases bacterial survival against host defenses. This diversity renders group A *Streptococcus* one of the most common human pathogens, and it is capable of producing a variety of clinical disorders, including pharyngitis, cellulitis, impetigo, and scarlet fever. It is among the few bacteria that can cause a wound infection, cellulitis, or necrotizing fasciitis within 24 hours following surgery.

### Treatment

There are five parameters of therapy for necrotizing fasciitis: early diagnosis and débridement, broad-spectrum antibiotics, aggressive resuscitation, frequent reevaluation, and comprehensive nutritional support (Figure 4).

### Surgical Débridement

Surgical débridement is the foundation of management because it is the fastest and most effective way to reduce the bacterial load and halt the necrotic process. This is the only intervention proven to increase the rate of survival. The goal of surgery is to remove at the first débridement all necrotic tissue, including muscle and skin if necessary, in addition to the involved fascia.

The initial incision should be made directly over the involved skin, parallel to the neurovascular bundles and down to the deep fascia. The surgeon may encounter dull, gray, avascular necrotic tissue demonstrating a lack of resistance to blunt dissection, lack of bleeding of the fascia, and the presence of foul-smelling dishwater pus. The margins of débridement should be advanced until skin and fascia normally adherent to the deep fascia are encountered and must terminate in

### Table 3

<table>
<thead>
<tr>
<th>Organisms Identified in Necrotizing Fasciitis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Gram-positive aerobic bacteria</strong></td>
</tr>
<tr>
<td><em>Group A</em> β-hemolytic streptococci*</td>
</tr>
<tr>
<td><em>Group B</em> streptococci</td>
</tr>
<tr>
<td><em>Enterococci</em></td>
</tr>
<tr>
<td><em>Coagulase-negative staphylococci</em></td>
</tr>
<tr>
<td><em>Staphylococcus aureus</em></td>
</tr>
<tr>
<td><em>Bacillus</em> spp</td>
</tr>
<tr>
<td><strong>Gram-negative aerobic bacteria</strong></td>
</tr>
<tr>
<td><em>Escherichia coli</em></td>
</tr>
<tr>
<td><em>Pseudomonas aeruginosa</em></td>
</tr>
<tr>
<td><em>Enterobacter cloacae</em></td>
</tr>
<tr>
<td><em>Klebsiella</em> spp</td>
</tr>
<tr>
<td><em>Proteus</em> spp</td>
</tr>
<tr>
<td><em>Serratia</em> spp</td>
</tr>
<tr>
<td><em>Acinetobacter calcoaceticus</em></td>
</tr>
<tr>
<td><em>Citrobacter freundii</em></td>
</tr>
<tr>
<td><em>Pasteurella multocida</em></td>
</tr>
<tr>
<td><em>Anaerobic bacteria</em></td>
</tr>
<tr>
<td><em>Bacteroides</em> spp</td>
</tr>
<tr>
<td><em>Clostridium</em> spp</td>
</tr>
<tr>
<td><em>Peptostreptococcus</em> spp</td>
</tr>
<tr>
<td><em>Marine Vibrio</em> spp</td>
</tr>
<tr>
<td><em>Vibrio vulnificus</em></td>
</tr>
<tr>
<td><em>Vibrio parahaemolyticus</em></td>
</tr>
<tr>
<td><em>Vibrio damsel</em></td>
</tr>
<tr>
<td><em>Vibrio alginolyticus</em></td>
</tr>
<tr>
<td><em>Fungi</em></td>
</tr>
<tr>
<td><em>Candida</em> spp</td>
</tr>
<tr>
<td><em>Aspergillus</em> spp</td>
</tr>
<tr>
<td><em>Rhizopus</em></td>
</tr>
</tbody>
</table>

viable, vascularized tissue. Poorly perfused tissue will act as a nidus for continued bacterial proliferation.\textsuperscript{13}

Although amputation does not improve mortality in certain studies,\textsuperscript{38} it may be necessary with some very aggressive infections as a first-line surgery to avoid death. Some authors have described conservative management in pediatric patients, allowing tissue demarcation to occur over a period of several days before surgical intervention, but this approach has not been adopted at our institution for either adults or children.\textsuperscript{39}

Careful management of the extensive wounds that often result from this radical form of surgery is critical. The wound must be reevaluated daily because repeated débridement is commonly required. The wound should be kept covered to protect against secondary infection, encourage the formation of granulation tissue, and absorb inflammatory exudates. Both alginate and hydrogel dressings have been used successfully. Topical negative pressure therapy can reduce edema and stimulate the formation of granulation tissue, and may reduce wound pain.

Once the infection has resolved and a bed of healthy granulation tissue is present, the wound may be closed with skin flaps or split-thickness grafts.\textsuperscript{7} Many cases will require reconstructive surgery with possible free-tissue transfer.\textsuperscript{13} Wound management techniques developed for burn victims may be applicable, and the successful use of the skin substitute Integra (Integra LifeSciences, Plainsboro, NJ) following surgical débridement for

---

**Figure 4**

Treatment algorithm for necrotizing fasciitis. IVIG = intravenous immunoglobulin G, MRI = magnetic resonance imaging

*Amputation may be indicated at the first surgical intervention if the infection is quickly progressing, necrosis has consumed most of the involved limb, and limb salvage is deemed impossible.*
necrotizing fasciitis has been reported. By providing early wound coverage, grafting can be delayed until the patient has recovered from the initial injury.40

Antibiotic Therapy

Antibiotics are an important complement to surgical therapy but cannot be the sole treatment because fascia is poorly vascularized, and the disease process further reduces its blood supply. This results in poor antibiotic delivery to the site of infection. Despite having little effect on the wound itself, antibiotics can reduce the bacterial load, terminate toxin production, and help prevent organ failure. Intravenous antibiotic therapy should be instituted at presentation after microbial cultures are obtained.

Initially, the infection should be treated empirically with broad-spectrum agents with activity against gram-positive, gram-negative, and anaerobic organisms until antimicrobial sensitivities are obtained (Table 4).

Acceptable regimens include imipenem, meropenem, ampicillin/subbactam, and piperacillin/tazobactam, all in combination with clindamycin. Gram stain of pus or deep wound tissue acquired during surgery should guide initial adjustments to antibiotic therapy. The presence of gram-positive cocci in clusters seen on Gram stain is adequate to support the use of vancomycin or linezolid because of the increased prevalence of MRSA.34,35,41 In contrast, Gram stain revealing gram-positive cocci in pairs or chains is treated with clindamycin and a β-lactam antibiotic. When the Gram stain reveals a polymicrobial flora, initial empiric therapy should be continued. As culture results and sensitivities become available, antibiotic therapy should be further adjusted if necessary.

Clindamycin is advised at the earliest suspicion of group A streptococcal or Staphylococcus aureus infection because it shuts down bacterial ribosome function, which inhibits both M protein and exotoxin production. Linezolid also possesses this ability. This inhibition facilitates phagocytosis and suppresses the synthesis of tumor necrosis factor-α, thereby reducing the overzealous immune response.9 The Eagle effect describes the limits of the efficacy of penicillin after a streptococcal infection has reached steady state,37 but the efficacy of clindamycin is not affected by the size of the bacterial inoculum or the stage of growth. Clindamycin has been shown to be more effective than penicillin against streptococcus species.24 One study found that use of clindamycin reduced the risk of hospital mortality by 89% among patients with necrotizing fasciitis.42

Supportive Care

Adequate fluid resuscitation and blood pressure support are essential because patients with necrotizing fasciitis often are septic at presentation or become so very quickly. Nutritional support is necessary because loss of fluid, protein, and electrolytes from the large surgical wounds are comparable to those observed in burn victims. In the acute phase, intake of twice the basal caloric requirement is appropriate. A patient who is unable to tolerate enteral feedings should receive total parenteral nutrition.13 Postoperative care must also address adequate pain management, such as use of patient-controlled analgesia.43

Adjunctive Therapies

Adjunctive therapies include intravenous immunoglobulin G (IVIG), hyperbaric oxygen (HBO), and recombinant human-activated protein C.44 The latter has been cited in a case report as a promising new therapy for sepsis that may reduce mortality in patients with group A streptococcus necrotizing fasciitis.44 In both in vivo and in vitro studies, IVIG has been found to inhibit the activation of T-cells by superantigens and

---

Table 4
Initial Antibiotic Management for Necrotizing Fasciitis

<table>
<thead>
<tr>
<th>Gram Stain Result</th>
<th>Initial Empiric Therapy*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gram-positive Cocci in Clusters</td>
<td>Clindamycin plus any of the following: imipenem, meropenem, ampicillin/subbactam, piperacillin/tazobactam</td>
</tr>
<tr>
<td>Gram-positive Cocci in Pairs or Chains</td>
<td>Clindamycin plus vancomycin, or monotherapy with linezolid</td>
</tr>
<tr>
<td>Polymicrobial With Gram-positive Cocci and Gram-negative Bacilli</td>
<td>Clindamycin plus any of the following: piperacillin/tazobactam, ampicillin/subbactam, or high-dose penicillin</td>
</tr>
</tbody>
</table>

* Before results of Gram stain are available
to inhibit the activity of streptococcal antigens to elicit cytokine production. Thus, it has been speculated to be of potential use in necrotizing fasciitis caused by group A streptococcus. The typical dosage is 1 to 2 g/kg of body weight per day for 2 days. Some studies have reported decreased mortality with the use of IVIG in patients with streptococcal toxic shock syndrome, but a recent review found no evidence that it improves clinical outcomes in necrotizing fasciitis.

HBO therapy has been proved to be efficacious for clostridial gangrene, but its use in necrotizing fasciitis is supported only by anecdotal and retrospective reports. Bacterial infection is associated with reduced tissue oxygen tension, and HBO may be able to reverse several of the pathophysiologic mechanisms potentiating the necrotizing process. It is thought that reestablishment of a normal or elevated partial oxygen pressure by administration of HBO can terminate the vicious cycle of infection, ischemia, and reduced host defense mechanisms. HBO therapy may also be helpful in identifying infection boundaries, potentially reducing unnecessary debridement. Standard therapy is 2.0 to 2.5 atm for 90 to 120 minutes twice daily until infection progression is halted.

Reported outcomes vary greatly. Escober et al found an 11-fold greater chance of survival with HBO treatment and a decrease in morbidity with fewer amputations in a retrospective study of 42 patients. Rizeman et al reported that HBO use reduced mortality from 66% to 23% in a retrospective study of 29 patients. Conversely, Brown et al concluded that there was no significant reduction in mortality rate associated with HBO therapy among 54 patients with necrotizing fasciitis.

HBO therapy carries risks. Among the potential complications are claus-trophobia, tympanic membrane rupture, seizures, and central nervous system oxygen toxicity. Because of the lack of randomized, prospective data regarding the utility of HBO therapy, currently it is best characterized as an adjunct therapy of potential benefit that should never delay or interfere with surgical intervention.

**Summary**

Necrotizing fasciitis is an uncommon infection of the superficial fascia and surrounding tissue. It is typically the result of a mixed infection with virulent, toxin-producing bacteria, or group A Streptococcus alone. MRSA appears to be emerging as an additional microbiologic factor. Early diagnosis is essential because the disease can rapidly progress to severe systemic toxicity and shock, organ failure, or acute respiratory distress syndrome. The single most effective management tool is a high degree of clinical suspicion followed by prompt surgical débridement of all necrotic tissue, with repeat débride-ment as needed. Broad-spectrum antibiotic therapy accompanies surgical management, and clindamycin is significantly beneficial when streptococcal or staphylococcal species are involved. Patients with necrotizing fasciitis are often critically ill, and intensive supportive care is necessary. Adjuvant therapies may be beneficial, but further study is needed to prove their efficacy.

**Acknowledgment**

The authors would like to thank Jeffrey S. Ross, MD, for his generous help preparing the pathology slides.

**References**

Evidence-based Medicine: Level II/III randomized, prospective studies include references 3, 7, 9, 11-15, 18-22, 24, 26, 28, 30-33, 36-39, 41, 42, and 47. Level III/IV case-control or cohort studies include references 8, 23, 25, 27, 29, 34, 35, 46, and 48-51. Reference 10 is a level V (expert opinion) report. The remainder are review articles, case reports, and textbook chapters.

Citation numbers printed in bold type indicate references published within the past 5 years.

Necrotizing Fasciitis


