Necrotizing fasciitis

A report of 3 cases

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Necrotizing fasciitis is a serious infectious disease characterized by a necrotizing cellulitis of the subcutaneous tissue. The mortality has not changed over the last 50 years, probably because the condition is not recognized early enough to permit effective treatment by debridement. We report 3 cases illustrating different clinical courses.

Case reports

Case 1. A 74-year-old woman developed a suppurating ulceration on her leg 3 weeks before admission. Edema and discoloration of the foot had gradually developed. There had been no trauma. On admission the patient had pain and fever (39.5°C). The right ankle and dorsum of the foot showed severe edema, erythema, and a hemorrhagic bulla. Culture disclosed hemolytic streptococci and *Staphylococcus aureus*. Excision of all the necrotic tissue and drainage was performed. It was noted by finger dissection that undermining of the apparently normal skin was by far more extensive than assumed by the size of the ulcer. Thrombosis was found in the superficial vessels, and the nerves were necrotic. Intravenous administration of penicillin 3 MIE four times daily and meticillin 1 g four times daily was started. The infection subsided and the skin defect was covered with split-thickness grafts. The recovery was uneventful.

Case 2. A 74-year-old woman was admitted with a painful red swelling of the left ankle and a large bulla on the anterior part of the left leg. Nine days earlier, she had sustained a sprain of her left ankle. Radiography showed no fracture. The condition was diagnosed as erysipelas and thrombophlebitis and treatment with antibiotics was started. Fourteen days after the trauma, the patient developed septicemia. She had pain in the leg, fever (38.2°C), audiovisual hallucinations, severe electrolyte disturbances, and life-threatening cardiac arrhythmias. Cultures from blood and the bulla were negative. Treatment with broad spectrum antibiotics and surgical revision of the bulla had no effect on the patient's general condition and the necrosis now involved the entire circumference of the lower left leg (Figure 1), which was amputated, leaving the wound open. The patient recovered and 2 weeks later a secondary closure was done. An increased antistreptococcal hyaluronidase titer was found during the critical phase, but 2 weeks later it was normal.

Case 3. A 65-year-old man was admitted after having sustained a contusion of his left arm 2 days earlier. The patient suffered from a mild chronic bronchitis. At examination the patient was conscious and alert, had some difficulty in breathing, and the temperature was slightly elevated (38°C). There was marked swelling of the entire left arm, with dusky discoloration, bullae, and patchy necrosis of the skin. The bullae were aspirated and the arm was elevated. The following hours the patient's condition deteriorated rapidly with respiratory and hemodynamic insufficiency and mental sluggishness. The condition was diagnosed as an exacerbation of the bronchitis causing the respiratory distress. Treatment with terbutaline,

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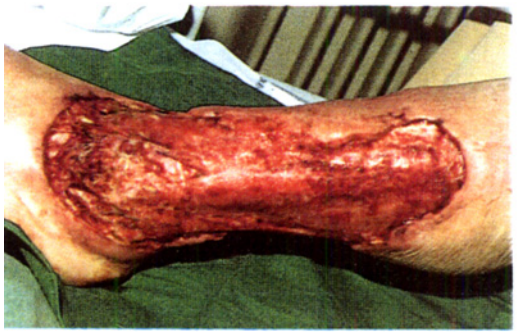


Figure 1

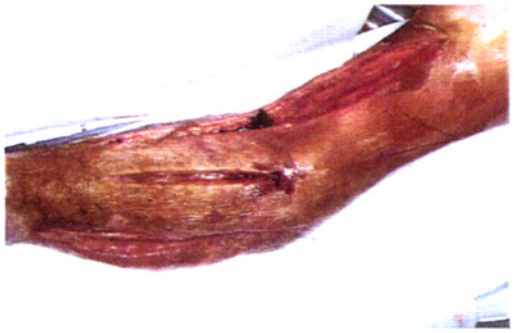
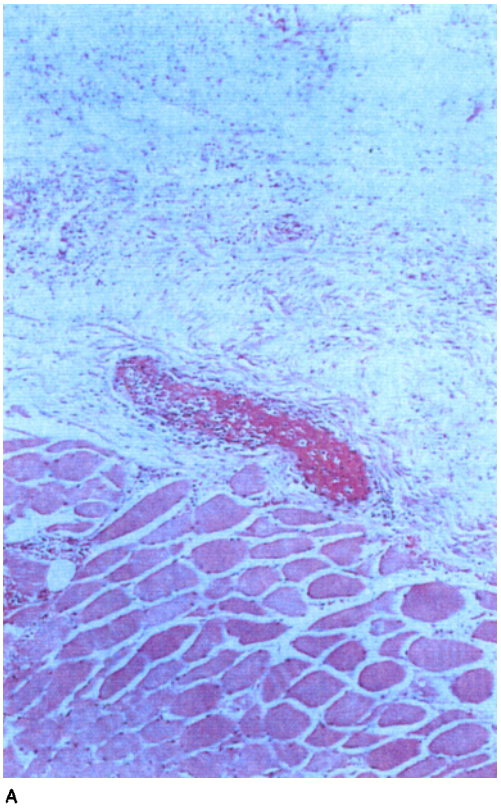


Figure 2

Figure 1. Case 2. A 74-year-old woman with necrotizing fasciitis on the left leg. Note apparently normal skin on the foot and proximally on the leg in spite of extensive undermining.

Figure 2. Case 3. A 65-year-old man with necrotizing fasciitis on the left arm. Necrotic subcutaneous tissue is seen through the incisions, as well as several collapsed bullae in the skin.



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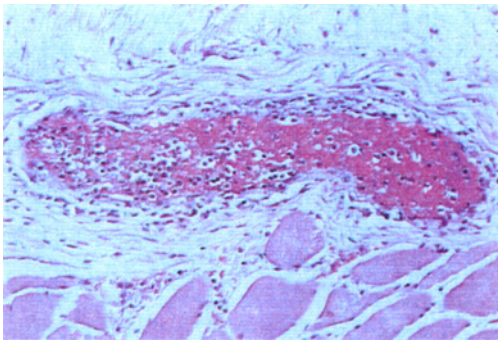

B

Figure 3. A. Typical picture of necrotizing fasciitis with subcutaneous necrosis, polymorphonuclear infiltration, and the only slightly involved muscle. HE, ×100.
B. Same section as A. A thrombosed vessel in the deep fascia is clearly demonstrated. HE, ×400.

treatment had no effect on the patient's condition, and 16 hours after admission the patient died.

At autopsy, there were signs of slight bronchitis only. Incision of the left arm showed gross undermining of the skin with subcutaneous necrosis down to the fascia (Figure 2). There was no muscle involvement. Microscopy showed necrosis of the subcutis and fascia, marked polymorphonuclear infiltration, fibrinous thrombi of arteries and veins, and only slight involvement of the muscle (Figures 3, 4). Cultures from bullae and subcutis obtained both before and after death were positive for hemolytic streptococci. Re-examination of the radiographs revealed small streaks of gas in the area between muscle and subcutis.

Discussion

Necrotizing fasciitis may develop after only minor trauma. The affected area becomes red, swollen, painful, and warm. Thrombotic occlusions of both venules and arterioles follow, causing patchy necrosis and gangrene of the skin. The necrosis of the subcutaneous tissue develops rapidly leading to the pathognomonic undermining of the skin, which is far more disseminated in the subcutis than one would believe by examining the skin. Simultaneously, generalized symptoms may appear. Death occurs as a result of septicemia with respiratory and cardiac failure. Remarkably, the prognosis of this infectious disease has not changed since the first report of Meleney in 1924 (Ovesen and Stoklund 1986). The introduction of antibiotics has not improved the prognosis. Initially, many cases of necrotizing fasciitis are interpreted as more benign conditions, and conservative treatment with antibiotics has no effect on the course of the disease (Case 2). Our 3 cases illustrate three different courses. Death in Case 3 was caused by septicemia.

Beta-hemolytic streptococci are the most frequently isolated bacteria associated with necrotizing fasciitis. Other species that have been isolated are *Staphylococcus aureus, Escherichia coli, Klebsiella pneumoniae, Proteus mirabilis, Pseudomonas aeruginosa,* anerobic bacteria – especially *Bacteroides* species and petrostreptococci. There is no clear relationship between the causative agents and the clinical manifestations. The pathogenesis is uncertain. It has been speculated that bacterial toxins and enzymes promote the necrotic process. Thrombosed vessels can be demonstrated histologically, and Hammar et al. (1985) found evidence of disturbed coagulation and fibrinolysis in necrotizing fasciitis; they concluded that increased fibrin deposition and vascular occlusions in the skin cause most complications. Possible local ischemia facilitates the spread of anaerobic bacteria, which probably play an important role in the progression of the infection. This may have been a contributing factor in Case 3.

If necrotizing fasciitis is suspected, exploratory incisions should be made (Ledingham and Tehrani 1975, Tehrani and Ledingham 1977, Freeman et al. 1981). The diagnosis is confirmed by the presence of necrotic undermining of the skin. Aerobic and anaerobic bacterial cultures, together with Gram-stained smears, should be performed. A Gram-stained smear from the ulceration can be helpful in the differential diagnosis of gas gangrene. The absence of inflammatory reaction with the occurrence of Gram-positive rods can lead to early diagnosis of a clostridial infection. In contrast to the latter, necrotizing fasciitis is characterized by massive infiltration of leucocytes. Because necrotizing fasciitis can progress rapidly at any stage, radical debridement and drainage of the undermined viable skin should be performed immediately. In a few cases amputation is imperative. Antibiotics should, as in other cases of septicemia with unknown bacterial etiology, cover a wide spectrum. Parenteral penicillin in large doses in combination with an aminoglycoside and metronidazole is recommended.

References

Freeman H P, Oluwole S F, Ganepola G A P, Dy E. Necrotizing fasciitis. Am J Surg 1981;142(3):377–83.

Hammar H, Sverdrup B, Borglund E, Blombäck M. Coagulation and fibrinolytic systems during the course of erysipelas and necrotizing fasciitis and the effect of heparin. Acta Derm Venereol (Stockh) 1985;65(6):495–503.

Ledingham I M, Tehrani M A. Diagnosis, clinical course and treatment of acute dermal gangrene. Br J Surg 1975;62(5):364–72.

Meleney F L. Hemolytic streptococcus gangrene. Arch Surg 1924;9:317–64.

Ovesen O C, Stoklund F K. Fasciitis necroticans. En alvorlig nekrotiserende bløddelsinfektion. Ugeskr Laeger 1986;148(30):1875–7.

Tehrani M A, Ledingham I M. Necrotizing fasciitis. Postgrad Med J 1977;53(619):237–42.