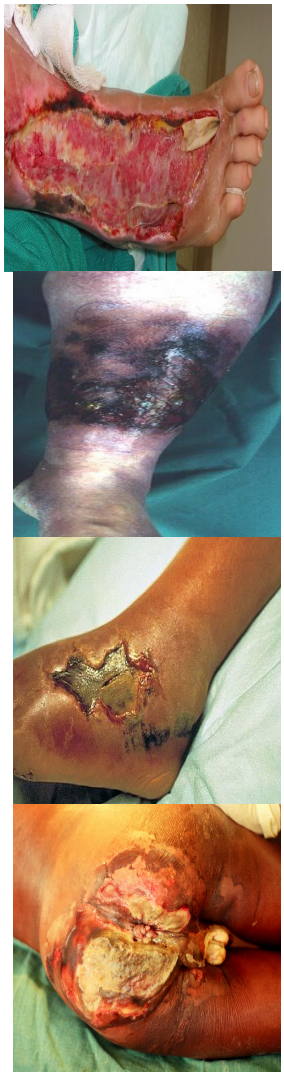

Necrotizing Fasciitis

.

Streptococcal Gangrene, Including Type II

Necrotizing Fasciitis.

In streptococcal gangrene, a monomicrobial infection, the pathogen is almost always a GAS, although groups C and G isolates, as well as other microorganisms, are infrequently identified. Occasionally, in newborns but also in patients early postpartum, group B streptococci have been recovered. Streptococcus pneumoniae necrotizing fasciitis is rarely reported and behaves like GAS infections. In addition, monomicrobial necrotizing fasciitis caused by both health care-acquired MRSA (HA-MRSA) and CA-MRSA, have been reported (see the section Methicillin-Resistant Staphylococcus aureus). Recently, 14 cases of CA-MRSA in patients with intraoperative findings consistent with necrotizing fasciitis, myonecrosis, or both, have been reported.

Patients may be immunocompromised by age or illness such as diabetes, alcoholism, or cirrhosis, but are usually healthy individuals. The location of the necrotizing lesion is most often an extremity and rarely, the face. A small area of involvement may underestimate the deep extent of the infection. Pain, erythema, and edema with rapid formation of bullae, constitutional symptoms with high fever, and toxicity are characteristic of early progression. There is bacteremia in approximately two-thirds of patients and evolution of necrosis of the overlying skin can

be rapid and dramatic, revealing deeper structures, including tendon sheaths and muscle. Though lymphangitis is rare, metastatic abscesses can occur. A streptococcal toxic shock-like syndrome often accompanies this infection, referred to in the lay press as a flesh-eating bacterial process. With bulla formation, the diagnosis is apparent from a Gram stain of an aspirate. As the process evolves, necrosis and eschar formation precede sloughing of tissues. Early symptoms of local pain eventually evolve to anesthetic areas and progressive toxicity is often accompanied by mental depression. Treatment with penicillin and clindamycin as well as surgical débridement and supportive therapy can be lifesaving, but the prognosis remains poor. Chapter 178 provides a detailed discussion of the streptococcal toxic shock-like syndrome, its prognosis, and therapy.

Findings in Gangrenous Cellulitis and Infectious Gangrene

FACTOR

PROGRESSIVE BACTERIAL SYNERGISTIC

SYNERGISTIC NECROTIZING CELLULITIS

STREPTOCOCCAL GANGRENE

CLOSTRIDIAL MYONECROSIS (GAS GANGRENE)

NECROTIZING INFECTIONS IN IMMUNOSUPPRESSION

Etiology

Streptococci, *Staphylococcus aureus*

Mixture of organisms: *Bacteroides,* peptosreptococci, or *Escherichia coli*

Group A streptococci or enteric bacteria

Clostridium perfringens

Rhizopus, Mucor, Absidia, Pseudomonas aeruginosa

Predisposing conditions

Surgery or draining sinus

Diabetes

Diabetes or abdominal surgery

Trauma

Diabetes, corticosteroid use, immunosuppression, burns

Fever

Minimal

Moderate

High

Moderate to high

Low in fungal, high in pseudomonal

Pain

Prominent

Prominent

Prominent

Prominent

Mild

Anesthesia

Absent

Absent

May occur

Absent

May occur

Crepitus

Absent

May occur

Absent

Present

Absent

Course

Slow

Rapid

Very rapid

Extremely rapid

Rapid

Modified with permission from Swartz MN … *Principles and Practice of Infectious Diseases* … Elsevier, Philadelphia, 200…

Type I Necrotizing Fasciitis.

Type I necrotizing fasciitis, a polymicrobial infection, is caused by a mix of facultative and anaerobic microbes, often delivered into the subcutaneous tissues after surgery, bowel perforation secondary to neoplasm or diverticulitis, trauma, or parenteral drug abuse via skin-popping. It often occurs in patients compromised by diabetes or malnutrition. Organisms include at least one anaerobic organism recovered in a mix of facultative microbes: nongroupable streptococci, enterococci, anaerobic streptococci and staphylococci, Bacteroides sp., and Enterobacteriaceae including E. coli, as well as various aquatic bacteria. Often, three to five species contribute to the infection, which may have a slower pace than GAS in evolving to a full-blown process with cutaneous manifestations.

Type I necrotizing fasciitis most commonly occurs on an extremity, abdominal wall, perineum, or about operative wounds . Clinically, it is indistinguishable from streptococcal gangrene. Although the initial pace of the illness may be slower, it is important to recognize that when this infection presents in the thigh (dissection along the psoas muscle) or abdominal wall, it may be secondary to an intestinal source (occult diverticulitis, rectosigmoid neoplasm). The involved area may be painful at first and then evolve with objective findings: swelling, erythema, warmth, and tenderness. The process is often more extensive than the overlying skin changes would suggest. Within several days, the skin color becomes purple, bullae develop, and frank cutaneous gangrene ensues . At this stage, the involved area is no longer tender but has become anesthetic as a result of occlusion of small blood vessels and destruction of superficial

nerves in the subcutaneous tissues. Crepitus is often present, particularly in patients with diabetes mellitus or if gas-forming anaerobes, such as Bacteroides sp., are causative. Though not universally used, the Laboratory Risk Indicator for Necrotizing Fasciitis has been devised from retrospective analysis of cases and tested in a secondary analysis of small clinical trials, with reported negative predictive value of 95 percent and high specificity.