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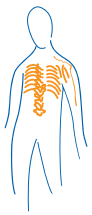

Skin and Soft Tissue Infections

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DEFINITION

Skin and soft tissue infections (SSTIs) comprise infections of skin, subcutaneous tissue, fascia, and the muscle by a multitude of organisms.

EPIDEMIOLOGY

SSTIs are among the most common infections found in all age groups, although the exact incidence is unknown. Several factors predispose to development of SSTIs:

- Epidermal breaks caused by trauma, surgical wounds, human or animal bites, or dry and irritated skin with concomitant tinea infection
- Immunosuppressed states caused by malnutrition, diabetes mellitus, or acquired immunodeficiency syndrome (AIDS)
- Chronic venous or lymphatic insufficiency

PATHOLOGY

Infectious Mechanisms

Microbes penetrate the integument after entering through a cut, bite, or hair follicle. Components of the host's defense system, including oxygen radicals, complement, immunoglobulins, macrophages, lymphocytes, and granulocytes, are recruited to the site of invasion through a vast plexus of dermal capillaries.

Bacteria contain proteins whose *N*-terminal amino acid sequence begins with an *N*-formyl-methionine group that is chemoattractive to phagocytes, including macrophages and granulocytes. Other microbial cell wall components, such as the zymosan of yeast, endotoxins of gram-negative bacteria, and the peptidoglycans of gram-positive bacteria, activate the alternative complement pathways, producing serum-derived chemotactic factors. Efflux of phagocytes occurs from the capillary through endothelial cell interstices and follows the gradient of chemotactic factors derived from bacteria and serum to the site of active infection.

Activated endothelial cells also produce chemotactic cytokines such as interleukin-8 (IL-8). Activated granulocytes synthesize leukotriene B_4 from arachidonic acid, a potent chemoattractant for leukocytes. Production of proinflammatory cytokines such as IL-1, IL-6, and tumor necrosis factor augments immune function, inducing fever, priming neutrophils, and increasing antibody production and synthesis of acute phase reactants such as C-reactive protein.

Cytokine-driven stimulation of endothelial cells generates nitric oxide and prostaglandins, both of which cause vasodilatation. The net physiologic effect is greater blood flow to the tissue, causing acute inflammation. As described by Celsus (30 BC-38 AD), acute inflammation is characterized by rubor (i.e., redness), calor (i.e., increased heat), tumor (i.e., swelling), dolor (i.e., pain), and, as added by Virchow in the 19th century, function laesa (i.e., loss of function). Chapter 86 discusses host defenses against infection in more depth.

Pathologic Manifestations

Impetigo is characterized by thick, crusted lesions with rounded or irregular margins that typically occur on the face. Most cases are caused by *Staphylococcus aureus*, including methicillin-resistant *S. aureus* (MRSA), or by group A streptococci (e.g., *Streptococcus pyogenes*). Certain strains of streptococci causing impetigo have been implicated in the development of poststreptococcal glomerulonephritis.

Folliculitis is a superficial bacterial infection of the hair follicles. Purulent material is found in the epidermis. It manifests as clusters of multiple, small, raised, pruritic, erythematous lesions that are typically less than 5 mm in diameter.

Furuncles (i.e., boils) are infections of the hair follicle. Purulent material extends through the dermis into the subcutaneous tissue, where small abscesses may form. A carbuncle is coalescence of several inflamed follicles into a single inflammatory mass. Purulent drainage exudes from multiple follicles.

Cellulitis is superficial inflammation of the skin and underlying tissues. It is characterized by erythema, warmth, and tenderness of the involved area (Fig. 94-1). Erysipelas is a variant of cellulitis that is predominantly caused by toxin-producing *S. pyogenes*. It manifests as a superficial, spreading, warm, erythematous (fiery red) lesion distinguished by its indurated and elevated margin. Lymphatic involvement and vesicle formation are common. Groups B, C, and D streptococci may also be implicated (Fig. 94-2).

Necrotizing fasciitis is a progressive and rapidly spreading inflammatory reaction deep in the fascia that is associated with secondary necrosis of the subcutaneous tissues. Thrombosis of the dermal vessels is responsible for tissue necrosis. Necrotizing fasciitis may be polymicrobial (type I), involving aerobic microbes (e.g., streptococci, staphylococci, gram-negative bacilli) and anaerobes (e.g., *Peptostreptococcus, Bacteroides, Clostridium* species), or it may be monomicrobial (type II) and caused by *S. pyogenes* (Fig. 94-3). When involving the scrotum and perineal area, it is known as *Fournier's gangrene*.

Pyomyositis is a less serious infection involving the musculature that results from direct inoculation of bacteria. For example,