Antibiotic resistance is a growing problem in treatment of *S. aureus* infections. Methicillin-resistant *S. aureus* (MRSA) are resistant to nearly all penicillin and cephalosporin antibiotics. Until recently, MRSA was mainly found in healthcare facilities, but community-acquired MRSA infections are now common in many areas. As a result, empirical treatment of staphylococcal infections with penicillin and cephalosporin antibiotics is less likely to be effective.

MORPHOLOGY

Whether the lesion is located in the skin, lungs, bones, or heart valves, *S. aureus* causes pyogenic inflammation that is distinctive for its local destruction of host tissue.

Excluding impetigo, which is restricted to the superficial epidermis, staphylococcal skin infections are centered around the hair follicles where they begin. A **furuncle**, or **boil**, is a focal suppurative inflammation of the skin and subcutaneous tissue. They may be solitary or multiple or recur in successive crops. Furuncles are most frequent in moist, hairy areas, such as the face, axillae, groin, legs, and submammary folds. Beginning in a single hair follicle, a boil develops into a growing and deepening abscess that eventually "comes to a head" by thinning and rupturing the overlying skin. A **carbuncle** is a deeper suppurative infection that spreads laterally beneath the deep subcutaneous fascia and then burrows superficially to erupt in multiple adjacent skin sinuses. Carbuncles typically appear beneath the skin of the upper back and posterior neck, where fascial planes favor their spread. **Hidradenitis** is chronic suppurative infection of apocrine glands, most often in the axilla. Infections of the nail bed (**paronychia**) or on the palmar side of the fingertips (**felons**) are exquisitely painful. They may follow trauma or embedded splinters and, if deep enough, destroy the bone of the terminal phalanx or detach the fingernail.

S. aureus lung infections (Fig. 8-16) have a polymorphonuclear infiltrate similar to that of *S. pneumoniae* infections (Fig. 8-4), but cause much more tissue destruction. Lung infections usually arise from a hematogenous source, such as an infected thrombus, or in the setting of a predisposing condition such as influenza.

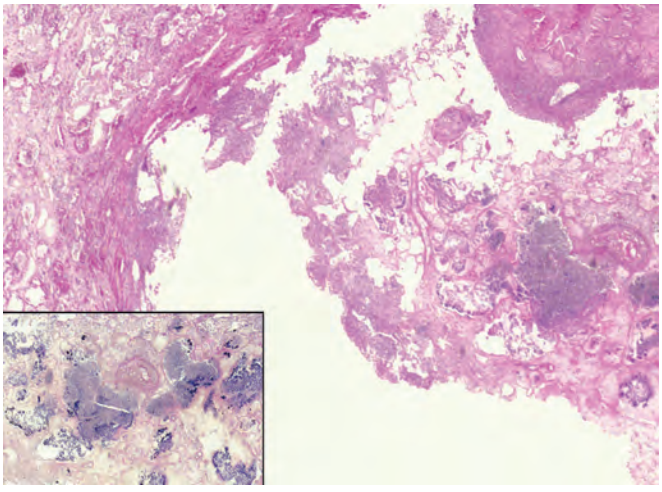

Figure 8-16 Staphylococcal abscess of the lung with extensive neutrophilic infiltrate and destruction of the alveoli (contrast with Fig. 8-4). The inset shows the same area on Gram stain highlighting clusters of bacteria.

Staphylococcal scalded-skin syndrome, also called Ritter disease, most frequently occurs in children with *S. aureus* infection of the nasopharynx or skin. There is a sunburn-like rash that spreads over the entire body and evolves into fragile bullae that lead to partial or total skin loss. The desquamation of the epidermis in staphylococcal scalded-skin syndrome occurs at the level of the granulosa layer, distinguishing it from toxic epidermal necrolysis, or Lyell disease, which is secondary to drug hypersensitivity and causes desquamation at the level of the epidermal-dermal junction (Chapter 25).

Streptococcal and Enterococcal Infections

Streptococci cause suppurative infections of the skin, oropharynx, lungs, and heart valves. They are also responsible for a number of postinfectious syndromes, including rheumatic fever (Chapter 12), immune complex glomerulonephritis (Chapter 20), and erythema nodosum (Chapter 25). These bacteria are gram-positive cocci that grow in pairs or chains. β-hemolytic streptococci are typed according to their surface carbohydrate (Lancefield) antigens. *S. pyogenes* (group A) causes pharyngitis, scarlet fever, erysipelas, impetigo, rheumatic fever, toxic shock syndrome, and glomerulonephritis. *S. agalactiae* (group B) colonizes the female genital tract and causes sepsis and meningitis in neonates and chorioamnionitis in pregnancy. *S. pneumoniae*, the most important α-hemolytic streptococcus, is a common cause of community-acquired pneumonia in older adults and meningitis in children and adults. The viridans-group streptococci include α-hemolytic and nonhemolytic streptococci found in normal oral flora that are a common cause of endocarditis. *S. mutans* is the major cause of dental caries. Streptococcal infections are diagnosed by culture, and, in those with pharyngitis, by the rapid streptococcal antigen test.

Enterococci are gram-positive cocci that grow in chains. They are often resistant to commonly used antibiotics and are a significant cause of endocarditis and urinary tract infections.

Pathogenesis. The different species of streptococci produce many virulence factors and toxins. *S. pyogenes, S. agalactiae,* and *S. pneumoniae* have capsules that resist phagocytosis. *S. pyogenes* also expresses M protein, a surface protein that prevents bacteria from being phagocytosed, and a complement C5a peptidase that degrades this chemotactic peptide. *S. pyogenes* secretes a phage-encoded pyrogenic exotoxin that causes fever and rash in scarlet fever. Poststreptococcal acute rheumatic fever is probably caused by antistreptococcal M protein antibodies and T cells that cross-react with cardiac proteins. Virulent *S. pyogenes* have been referred to as flesh-eating bacteria because they cause a rapidly progressive necrotizing fasciitis. Although the antiphagocytic capsule is the most important virulence factor of *S. pneumoniae,* it also produces pneumolysin, a toxin that inserts into host cell membranes and lyses cells, greatly increasing tissue damage. *S. mutans* produces caries by metabolizing sucrose to lactic acid (which causes demineralization of tooth enamel) and by secreting high-molecular-weight glucans that promote aggregation of bacteria and plaque formation.

Enterococci are low-virulence bacteria, although they do have an antiphagocytic capsule and produce enzymes that