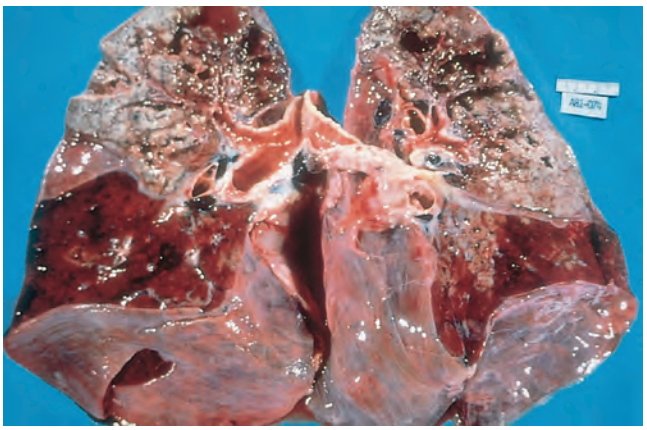

Figure 8-28 Secondary pulmonary tuberculosis. The upper parts of both lungs are riddled with gray-white areas of caseation and multiple areas of softening and cavitation.

firm and gray-white to yellow in color and have variable degrees of central caseation and peripheral fibrosis (Fig. 8-28). In immunocompetent individuals, the initial parenchymal focus undergoes progressive fibrous encapsulation, leaving only fibrocalcific scars. Histologically, the active lesions show characteristic coalescent tubercles with central caseation. Tubercle bacilli can often be identified with acid-fast stains in early exudative and caseous phases of granuloma formation but are usually too few to be found in the late, fibrocalcific stages. Localized, apical, secondary pulmonary tuberculosis may heal with fibrosis either spontaneously or after therapy, or the disease may progress and extend along several different pathways.

Progressive pulmonary tuberculosis may ensue in older adults and immunosuppressed people. The apical lesion expands into adjacent lung and eventually erodes into bronchi and vessels. This evacuates the caseous center, creating a ragged, irregular cavity that is poorly walled off by fibrous tissue. Erosion of blood vessels results in hemoptysis. With adequate treatment the process may be arrested, although healing by fibrosis often distorts the pulmonary architecture. The cavities, now free of inflammation, may persist or become fibrotic. If the treatment is inadequate or if host defenses are impaired, the infection may spread via airways, lymphatic channels, or the vascular system. **Miliary pulmonary disease** occurs when organisms draining through lymphatics enter the venous blood and circulate back to the lung. Individual lesions are either microscopic or small, visible (2-mm) foci of yellow-white consolidation scattered through the lung parenchyma (the adjective "miliary" is derived from the resemblance of these foci to millet seeds). Miliary lesions may expand and coalesce, resulting in consolidation of large regions or even whole lobes of the lung. With progressive pulmonary tuberculosis, the pleural cavity is invariably involved, and serous **pleural effusions**, **tuberculous empyema**, or **obliterative fibrous pleuritis** may develop. Progressive primary tuberculosis that occurs in immunosuppressed individuals spreads in a similar manner.

Endobronchial, **endotracheal**, and **laryngeal tuberculosis** may develop by spread through lymphatic channels or from expectorated infectious material. The mucosal lining may be studded with minute granulomatous lesions that may only be apparent microscopically.

Systemic miliary tuberculosis occurs when bacteria disseminate through the systemic arterial system. Miliary tuberculosis is most prominent in the liver, bone marrow, spleen, adrenals, meninges, kidneys, fallopian tubes, and epididymis, but could involve any organ (Fig. 8-29).

Isolated tuberculosis may appear in any of the organs or tissues seeded hematogenously and may be the presenting manifestation. Organs that are commonly involved include the meninges (tuberculous meningitis), kidneys (renal tuberculosis), adrenals (formerly an important cause of Addison disease), bones (osteomyelitis), and fallopian tubes (salpingitis). When the vertebrae are affected, the disease is referred to as **Pott disease**. Paraspinal "cold" abscesses in these patients may track along tissue planes and present as an abdominal or pelvic mass.

Lymphadenitis is the most frequent presentation of extrapulmonary tuberculosis, usually occurring in the cervical region ("scrofula"). In HIV-negative individuals, lymphadenitis tends to be unifocal and localized. HIV-positive people, on the other hand, almost always have multifocal disease, systemic symptoms, and either pulmonary or other organ involvement by active tuberculosis.

As previously mentioned, **intestinal tuberculosis** contracted by the drinking of contaminated milk is common in countries where bovine tuberculosis is present and milk is not pasteurized. In countries where milk is pasteurized, intestinal tuberculosis is more often caused by the swallowing of coughed-up infective material in patients with advanced pulmonary disease. Typically the organisms are seeded to mucosal lymphoid aggregates of the small and large bowel, which then undergo granulomatous inflammation that can lead to ulceration of the overlying mucosa, particularly in the ileum. Healing creates strictures.

Mycobacterium avium *Complex*

Mycobacterium avium and *M. intracellulare* are separate species, but the infections they cause are so similar that they are simply referred to as *M. avium* complex, or MAC. MAC is common in soil, water, dust, and domestic animals. **Clinically significant infection with MAC is uncommon except among people with T-cell immunodeficiency due to AIDS, and immunosuppression resulting from treatment for transplant rejection or autoimmune diseases.** In patients with marked T-cell immunodeficiency, MAC

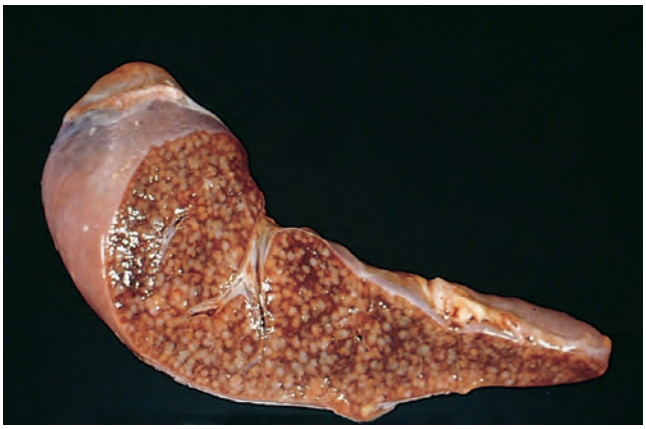

Figure 8-29 Miliary tuberculosis of the spleen. The cut surface shows numerous gray-white tubercles.