

1108 by molecular evidence from TB-endemic areas indicating that a large percentage of cases of adult pulmonary TB result from recent infection (either primary infection or reinfection) and not from reactivation.

Primary Disease Primary pulmonary TB occurs soon after the initial infection with tubercle bacilli. It may be asymptomatic or may present with fever and occasionally pleuritic chest pain. In areas of high TB transmission, this form of disease is often seen in children. Because most inspired air is distributed to the middle and lower lung zones, these areas are most commonly involved in primary TB. The lesion forming after initial infection (*Ghon focus*) is usually peripheral and accompanied by transient hilar or paratracheal lymphadenopathy, which may or may not be visible on standard chest radiography (Fig. 202-4). Some patients develop erythema nodosum on the legs (see Fig. 25e-40) or phlyctenular conjunctivitis. In the majority of cases, the lesion heals spontaneously and becomes evident only as a small calcified nodule. Pleural reaction overlying a subpleural focus is also common. The Ghon focus, with or without overlying pleural reaction, thickening, and regional lymphadenopathy, is referred to as the *Ghon complex*.

In young children with immature CMI and in persons with impaired immunity (e.g., those with malnutrition or HIV infection), primary pulmonary TB may progress rapidly to clinical illness. The initial lesion increases in size and can evolve in different ways. Pleural effusion, which is found in up to two-thirds of cases, results from the penetration of bacilli into the pleural space from an adjacent subpleural focus. In severe cases, the primary site rapidly enlarges, its central portion undergoes necrosis, and cavitation develops (*progressive primary TB*). TB in young children is almost invariably accompanied by hilar or paratracheal lymphadenopathy due to the spread of bacilli from the lung parenchyma through lymphatic vessels. Enlarged lymph nodes may compress bronchi, causing total obstruction with distal collapse, partial obstruction with large-airway wheezing, or a ball-valve effect with segmental/lobar hyperinflation. Lymph nodes may also rupture into the airway with development of pneumonia, often including areas of necrosis and cavitation, distal to the obstruction. Bronchiectasis (Chap. 312) may develop in any segment/lobe damaged by progressive caseating pneumonia. Occult hematogenous dissemination commonly follows primary infection. However, in the absence of a sufficient acquired immune response, which usually contains the infection, disseminated or miliary disease may result (Fig. 202-5). Small granulomatous lesions develop in multiple organs and may cause



FIGURE 202-4 Chest radiograph showing right hilar lymph node enlargement with infiltration into the surrounding lung tissue in a child with primary tuberculosis. (Courtesy of Prof. Robert Gie, Department of Paediatrics and Child Health, Stellenbosch University, South Africa; with permission.)



FIGURE 202-5 Chest radiograph showing bilateral miliary (millet-sized) infiltrates in a child. (Courtesy of Prof. Robert Gie, Department of Paediatrics and Child Health, Stellenbosch University, South Africa; with permission.)

locally progressive disease or result in tuberculous meningitis; this is a particular concern in very young children and immunocompromised persons (e.g., patients with HIV infection).

Postprimary (Adult-Type) Disease Also referred to as *reactivation* or *secondary TB*, postprimary TB is probably most accurately termed *adult-type TB* because it may result from endogenous reactivation of distant LTBI or recent infection (primary infection or reinfection). It is usually localized to the apical and posterior segments of the upper lobes, where the substantially higher mean oxygen tension (compared with that in the lower zones) favors mycobacterial growth. The superior segments of the lower lobes are also more frequently involved. The extent of lung parenchymal involvement varies greatly, from small infiltrates to extensive cavitory disease. With cavity formation, liquefied necrotic contents are ultimately discharged into the airways and may undergo bronchogenic spread, resulting in satellite lesions within the lungs that may in turn undergo cavitation (Figs. 202-6 and 202-7). Massive involvement of pulmonary segments or lobes, with coalescence of lesions, produces caseating pneumonia. While up to one-third of untreated patients reportedly succumb to severe pulmonary TB within a few months after onset (the classic “galloping consumption” of the past), others may undergo a process of spontaneous remission or proceed along a chronic, progressively debilitating course (“consumption” or *phthisis*). Under these circumstances, some pulmonary lesions become fibrotic and may later calcify, but cavities persist in other parts of the lungs. Individuals with such chronic disease continue to discharge tubercle bacilli into the environment. Most patients respond to treatment, with defervescence, decreasing cough, weight gain, and a general improvement in well-being within several weeks.

Early in the course of disease, symptoms and signs are often non-specific and insidious, consisting mainly of diurnal fever and night sweats due to defervescence, weight loss, anorexia, general malaise, and weakness. However, in up to 90% of cases, cough eventually develops—often initially nonproductive and limited to the morning and subsequently accompanied by the production of purulent sputum, sometimes with blood streaking. Hemoptysis develops in 20–30% of cases, and massive hemoptysis may ensue as a consequence of the erosion of a blood vessel in the wall of a cavity. Hemoptysis, however, may also result from rupture of a dilated vessel in a cavity (*Rasmussen’s aneurysm*) or from aspergilloma formation in an old cavity. Pleuritic chest pain sometimes develops in patients with subpleural parenchymal lesions or pleural disease. Extensive disease may produce dyspnea