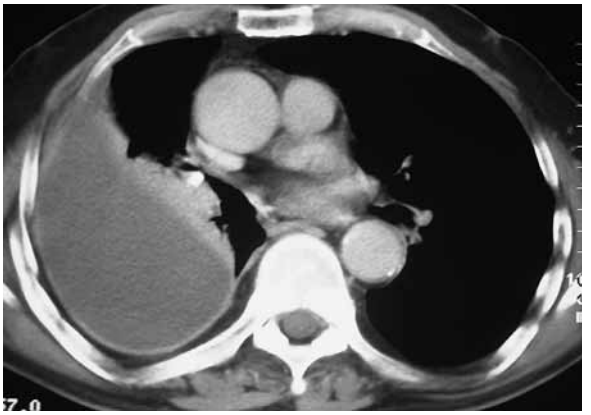


Fig. 3.**15** CT shows large right-sided empyema with "split pleura" sign.

Pulmonary Tuberculosis

Tuberculosis is an infectious disease which may affect any organ but shows a definite predilection for the lungs. In 95% of cases the causative organism is *Mycobacterium tuberculosis humanus*. A less common strain is *Mycobacterium bovis*. Atypical mycobacteria such as *M. kansasii* and *M. balnei* occur only sporadically. The incidence of another atypical mycobacterial infection, *mycobacterium avium complex* (MAC), increased markedly in the 1980s and 1990s mainly due to the increasing number of patients with acquired immunodeficiency syndrome (AIDS) and depleted helper T-lymphocyte CD4 counts (see p. 97).

In 1900, tuberculosis was still a worldwide epidemic with a mortality rate of approximately 250 per 100 000 per year. Effective antituberculous therapy and better socioeconomic conditions have substantially reduced the incidence and prevalence of tuberculosis, with a resulting decline in mortality rates. Mortality rates in Germany are less than 3 per 100 000 per year. In the United States, 1800 tuberculosis-related deaths are reported each year, corresponding to a mortality rate of less than 1 per 100 000 per year. There was a steady decline in the incidence of tuberculosis in industrialized countries during the second half of the 20th century until the mid 1980s. In Germany, the incidence had fallen from 174 new cases per 100 000 in 1942 to less than 32 new cases per 100 000 population in 1994 (1994 Report of the German Central Committee on Tuberculosis Control). However, in the late 1980s and early 1990s, this downward trend reversed, due mainly to the increasing numbers of cases in patients with AIDS (Im 1995). In the United States, there were approximately 20 000 new cases in 1985; this had increased to more than 25 000 new cases by 1990. More recently, the resurgence of tuberculosis in Western countries has also been attributed to immigration and the development of multidrug-resistant disease (MDR-TB; Faustini et al. 2006).

The incidence and prevalence of tuberculosis has always remained high in endemic regions and is the leading cause of death in patients with AIDS in developing countries.

The main factor determining whether tuberculous infection progresses to disease is the immune competence of the individual (Murray 1996). Today the disease most commonly is found in persons whose immune status is compromised by old age, alcohol abuse, diabetes mellitus, steroid therapy, or AIDS. There is also a relatively high incidence in certain ethnic groups, many of whom have recently immigrated to Western Europe and North America.

Tuberculosis is classically divided into primary and postprimary disease (Fig. 3.**16**). Some controversy exists as to whether the latter represents reactivation or reinfection (McAdams et al. 1995). Primary tuberculosis occurs in those not previously exposed to *M. tuberculosis*, is frequently asymptomatic, and therefore is not detected clinically. Postprimary or "cavitating" tuberculosis occurs in previously sensitized individuals; before the advent of antituberculosis chemotherapy, it was frequently fatal (*galloping consumption*). Today, fibrocirrhotic endstage disease with severe ventilatory impairment may lead to eventual death from decompensated cor pulmonale.

The frontal chest radiograph remains the initial imaging investigation in tuberculosis. Some studies have emphasized the role of high-resolution CT, particularly in the detection of endobronchial spread (Lee 1991, Im et al. 1993, Hatipoglu et al. 1996). Bacteriological diagnosis is made from detection of acid-fast bacilli (AFB) in sputum, gastric washings, pleural fluid and, in patients proceeding to bronchoscopy, from bronchoalveolar lavage (BAL) fluid. Newer immunologic and nucleic acid-based techniques are also emerging (Furin and Johnson 2005).

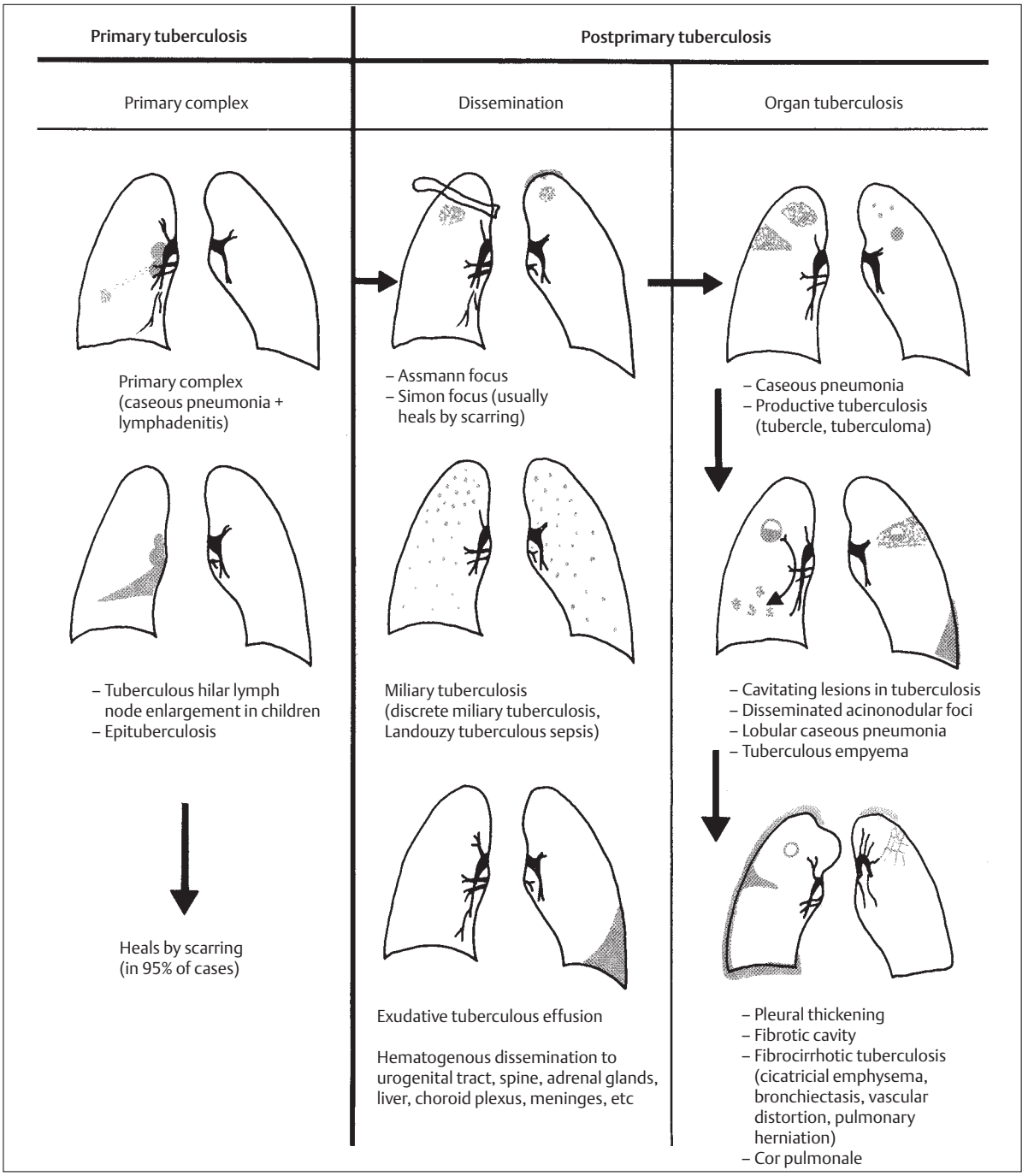


Fig. 3.**16** Pulmonary tuberculosis.

Goals of Diagnostic Imaging:
Adequate screening programs to detect early disease particularly in high risk groups. In Germany, 30% of new infections are detected in this way.

Accurate interpretation of radiographic abnormalities with a relatively low threshold for suggesting tuberculosis in the differential diagnosis.

Monitoring the response to therapy with serial imaging.

Detecting the sequelae of healed tuberculosis such as cicatricial emphysema, bronchiectasis, and cor pulmonale.

Pathology

The German pathologist K. E. Ranke identified three stages in the evolution of tuberculosis. In modern nomenclature, the last two Ranke stages are included in the postprimary phase (Table 3.**1** and Fig. 3.**16**).

Table 3.**1** Stages of pulmonary tuberculosis (from Doerr, Schmidt, Schmincke)

A. Primary stage
Primary pulmonary focus (Ghon focus) and regional lymphadenitis = primary complex (dumbbell-shaped consolidation as described by K. E. Ranke)

B. Postprimary stage
I. Dissemination
  1. Early dissemination
    a) Simon foci
    b) Miliary tuberculosis
    c) Rarely in immunocompromised hosts: acute tuberculous sepsis (Landouzy)
  2. Late dissemination
    a) Subapical acinonodular disseminated foci
    b) Coarse granular dissemination (Aschoff–Puhl focus)
    c) Early infraclavicular consolidation (Assmann-Redeker-Simon)

II. Isolated organ tuberculosis
  1. Nodulation, fibrocirrhosis, cavitation
  2. Reticular lymphangitis

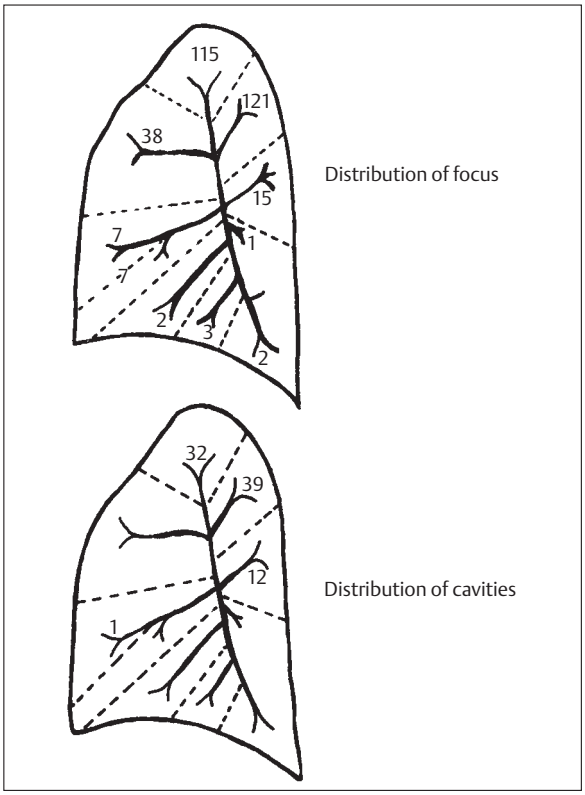


Fig. 3.**17** Frequency distribution of tuberculous lesions by lung segments (Doerr 1983).

Primary Complex

Inhaled tubercle bacilli initially evoke a focal, nonspecific subpleural alveolitis which converts to a tuberculosis-specific inflammatory focus in about 10 days (Ghon focus). The latter is characterized by central colliquative necrosis, also termed caseous necrosis due to its grayish-yellow appearance. There is surrounding granulation tissue rich in lymphocytes, epitheloid cells, and Langerhans giant cells. Spread of tubercle via the lymphatics leads to a specific hilar lymphadenitis. In the great majority of cases, this primary complex (Ghon focus + regional lymphadenitis) heals with fibrosis and may calcify. Large infected lymph nodes may compress the bronchi, particularly the right middle lobe bronchus, with resulting distal atelectasis; this occurs almost exclusively in children (epituberculosis). In the severely immunocompromised patient, caseous lymphadenitis may erode into an airway resulting in tuberculous dissemination through primary endobronchial spread.

Hematogenous Dissemination

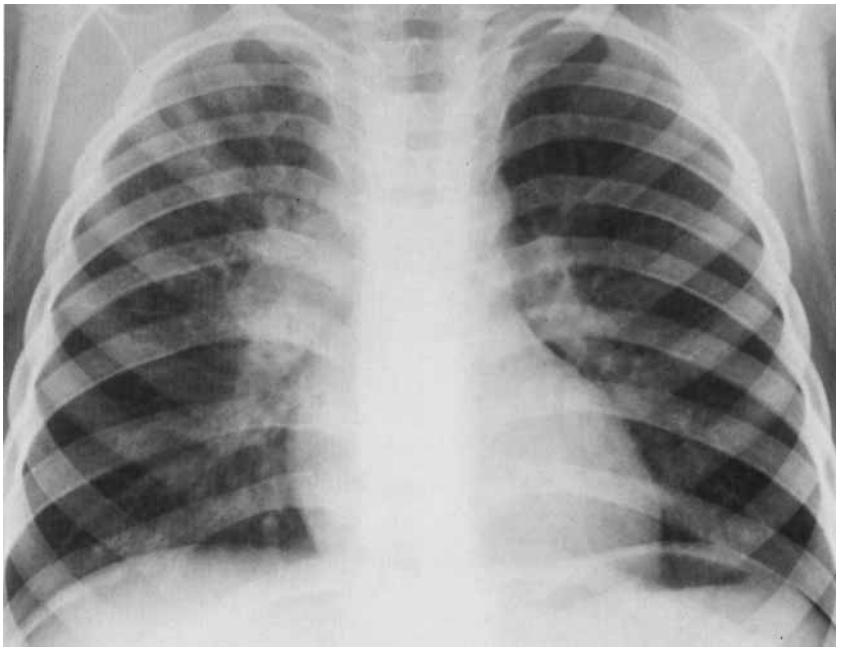
Mycobacteria entering the blood from the primary complex may become disseminated to numerous extrapulmonary sites (urogenital system, bones, meninges, adrenals, bowel, etc.).

- *Miliary tuberculosis*: Hematogenous dissemination appears as myriad small nodules (*millet seeds*) throughout the lung but displaying an upper zone predominance. These fine nodules are tubercles with a core of caseous necrosis and surrounding granulation tissue. Discrete miliary tuberculosis, characterized by fewer nodules, is associated with less severe degrees of immunocompromise. Disseminated tuberculosis with multiorgan involvement is associated with a high mortality rate.
- The most frequent pulmonary manifestation of hematogenous dissemination is the appearance of a *solitary tuberculous focus* at the lung apex (the Simon focus, Assmann infiltrate, subapical acinonodular focus). This predilection for the upper lobes is due to the higher tissue oxygen tension and relatively low perfusion in this region.
- *Exudative pleurisy*: Bacilli invade the pleura where they form tubercles; this is associated with development of a pleural effusion rich in lymphocytes.

Postprimary Organ Tuberculosis

This form of postprimary tuberculosis is characterized by cavitating lesions in the upper lobes or in the apical segments of the lower lobes (Fig. 3.**17**). Rupture of a parenchymal focus into an adjacent airway and subsequent endobronchial spread may lead to extensive pulmonary involvement.

- *Exudative tuberculosis* is characterized by a lobular, caseous pneumonia with relatively few epithelioid cells. Coalescence may occur to form larger foci of caseous pneumonia.
- *Productive tuberculosis* is characterized by well-defined solid nodules, 1–2 mm in diameter and rich in epithelioid cells; these correspond to the size of the primary lobule. If the immune response is weak,



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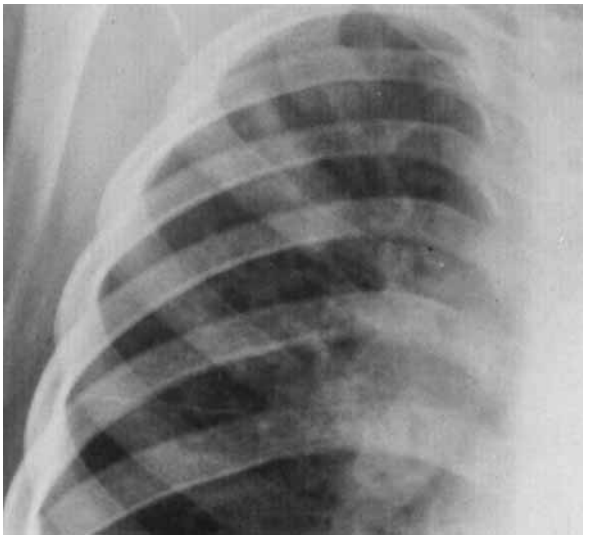


b

Fig. 3.**18 a, b** Acute primary complex. The hazy infiltrate in the right upper lobe, lymphatic stranding, and lymphadenitis form a dumbbell-shaped configuration.

larger foci may develop (acinonodular form). Tuberculomas measuring 1–3 cm in diameter and comprising a caseous core surrounded by a mantle of granulation tissue are also found.

- *Cavitating tuberculosis*: Cavitation results from erosion of enlarging tubercles into the airway and associated liquefaction of caseous material. In active tuberculosis, the wall of the cavity contains infectious caseous material. Eventually, the cavity becomes fibrosed and may even acquire an epithelial lining.
- *Fibrocirrhotic tuberculosis*, in which the tuberculous process heals by fibrosis, is associated with fibrous contraction and distortion of the lung architecture leading to cicatricial emphysema and traction bronchiectasis.

Clinical Features

Primary tuberculosis usually is asymptomatic. Occasionally, low-grade pyrexia with night sweats, coughing, anorexia, and erythema nodosum develop. With progressive postprimary tuberculosis, the above clinical manifestations are present together with hemoptysis and dyspnea. The tuberculin skin test is positive, and acid-fast bacilli may be detected in the sputum, gastric washings, pleural and bronchoalveolar lavage fluid.

Radiologic Findings

Chest Radiograph

Primary tuberculosis is rarely detected on the chest radiograph. Positive radiographic findings are present in only about 20% of children with a positive tuberculin skin test:

- The *Ghon focus* is a circumscribed, small, peripheral area of consolidation.
- *Hilar and mediastinal Lymphadenitis* presents as hilar enlargement and mediastinal widening. Occasionally, lymphangitic stranding connecting the primary focus with the hilar lymphadenitis forms a dumbbell-shaped opacity. This represents the primary complex (Fig. 3.**18**).
- *A segmental opacity* may be due to segmental atelectasis distal to bronchial compression by enlarged lymph nodes (epituberculosis).
- The *calcified healed primary complex* is a frequent incidental finding on chest radiographs and has no clinical significance (Fig. 3.**19**).

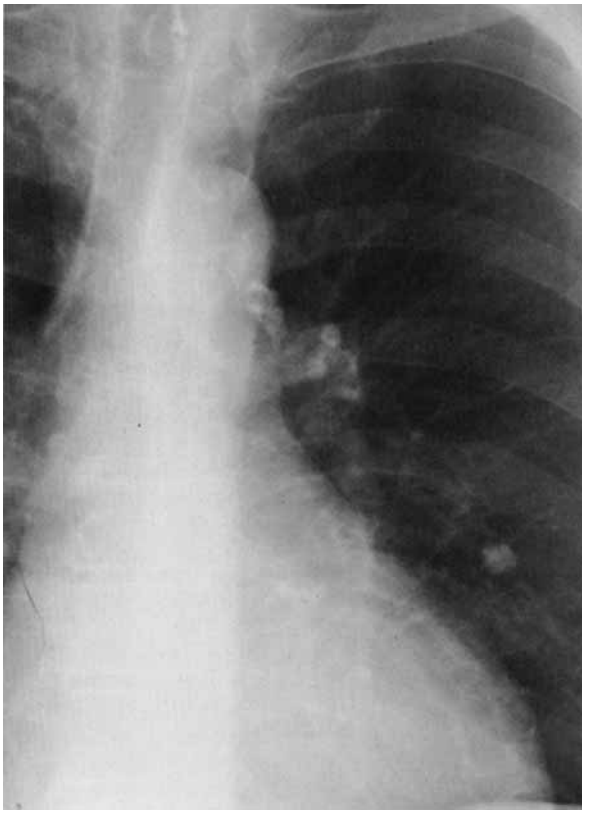


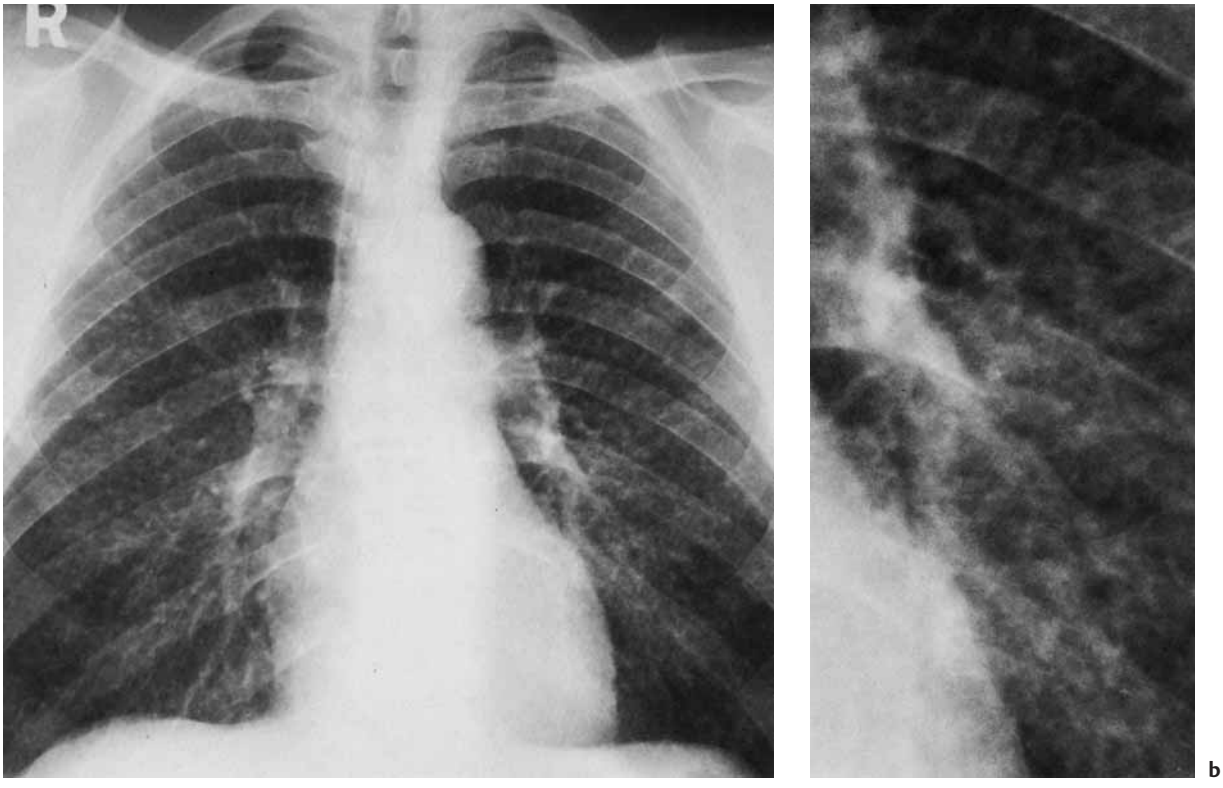
Fig. 3.**19** Calcified primary complex, considered a normal incidental finding.

Hematogenous Dissemination

- *Miliary tuberculosis* exhibits a finely mottled nodular pattern resulting from summation of individual nodules. The profusion of the mottling increases in an apicobasal direction. Occasionally, in advanced cases, miliary tuberculosis may produce a coarse granular or "snowstorm" pattern due to coalescence of the nodules (Fig. 3.**20**).
- *Exudative tuberculous pleuritis* radiographically resembles other effusions (Fig. 3.**21**).

Postprimary/secondary pulmonary tuberculosis produces a spectrum of radiographic manifestations; exudative, productive, cavitary, and fibrotic changes frequently occur simultaneously. Because of the predilection for the apical and posterior segments of the upper lobe and the apical segment of the lower lobe, parenchymal changes in these regions in the correct clinical setting should arouse suspicion of tuberculosis.

- *Exudative/productive tuberculosis* manifests as areas of confluent consolidation, or more discrete areas of nodular opacification may be seen. Fine nodular opacities may indicate bronchiolar involvement and endobronchial spread (Fig. 3.**22**).
- *Tuberculomas* form as pulmonary nodules or masses, 0.5–4 cm in diameter. They have smooth margins and a predilection for the upper zones (Fig. 3.**23**). In 80% of cases, conventional or computed tomography will show small satellite lesions and calcifications.



a **b**

Fig. 3.**20 a, b** Miliary tuberculosis with fine nodular shadowing throughout both lungs.

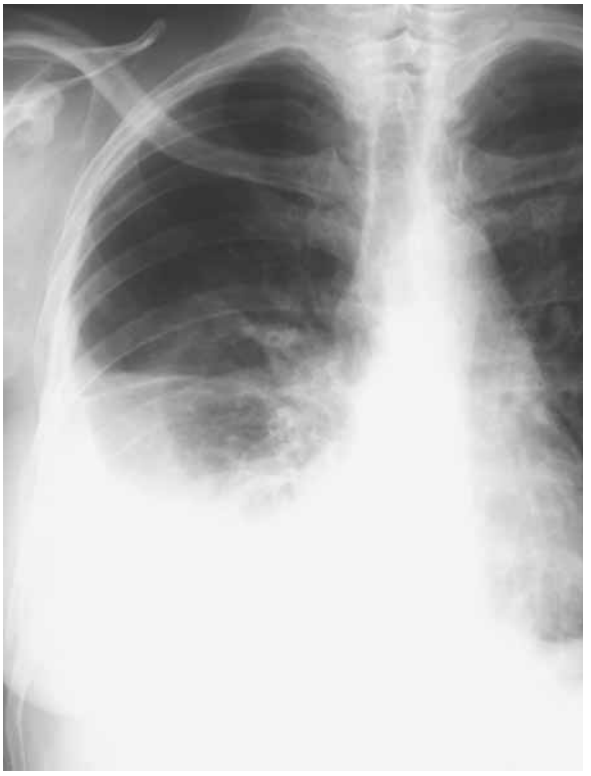
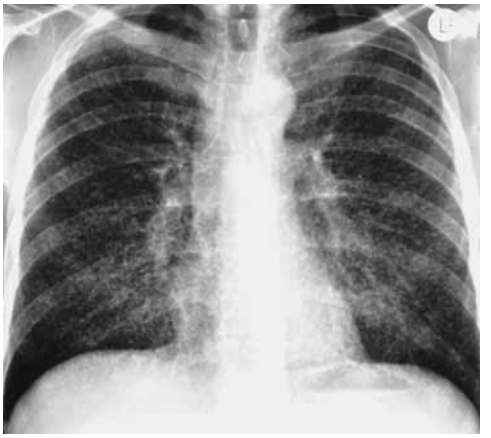


Fig. 3.**21** Tuberculous pleural effusion. Mycobacteria were cultured from the lymphocyte-rich pleural aspirate.



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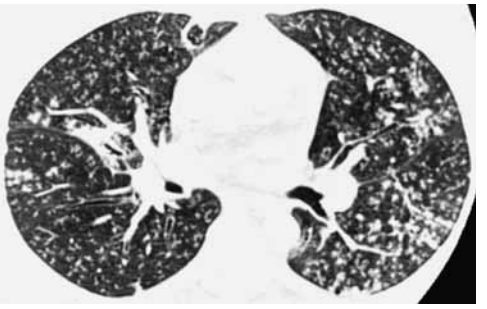


b

Fig. 3.**22 a, b** Tuberculosis. Plain radiograph (**a**) shows bilateral reticulonodular shadowing. CT (**b**) shows features of endobronchial spread.

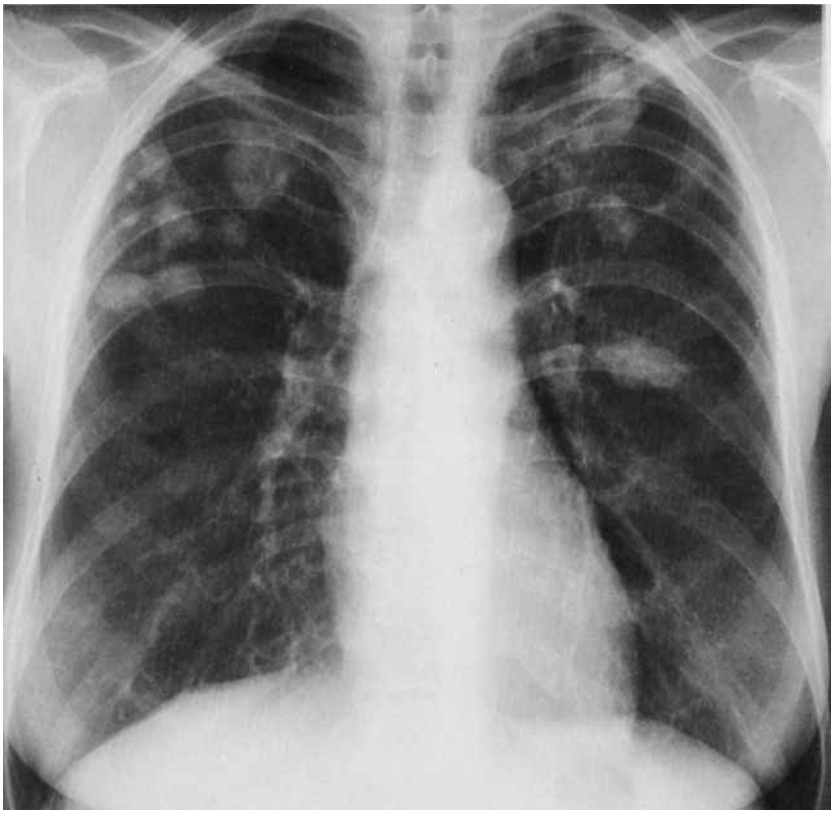


Fig. 3.**23** Multiple tuberculomas.

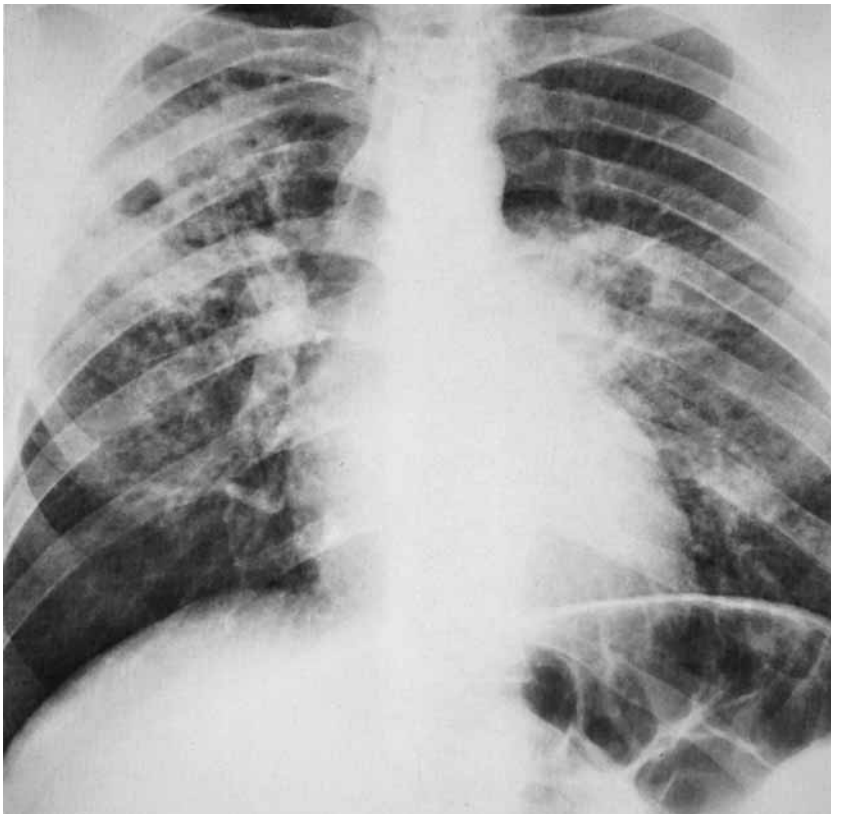


Fig. 3.**24** Exudative cavitating tuberculosis with areas of caseous pneumonia and liquefaction.

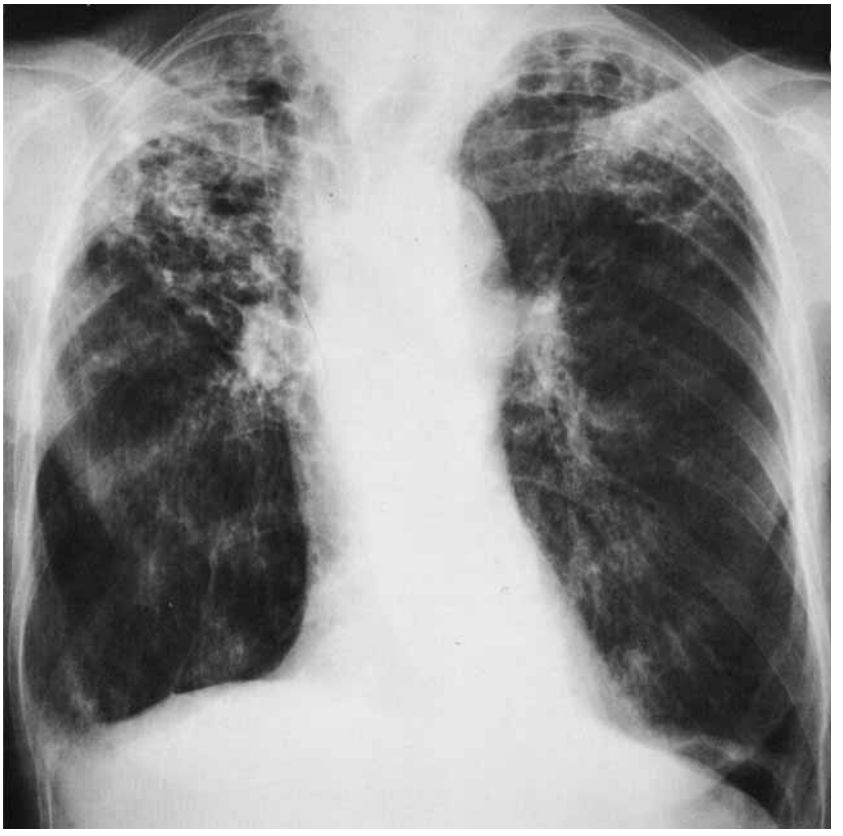


Fig. 3.**25** Fibrocirrhotic pulmonary tuberculosis. Upper lobe destruction and fibrosis are associated with elevation of the hila and compensatory emphysema in the lower zones.

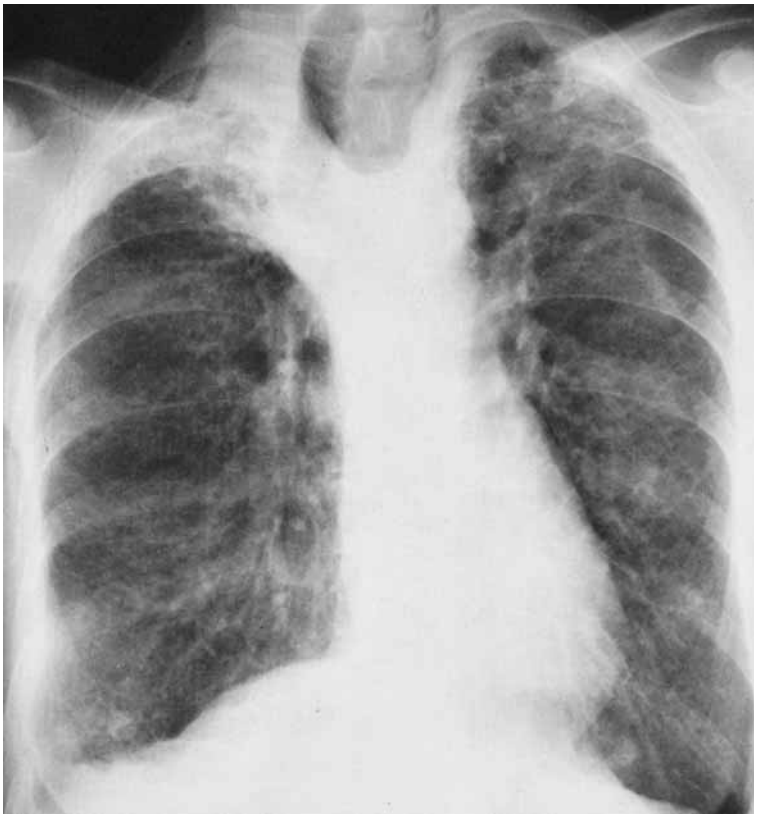


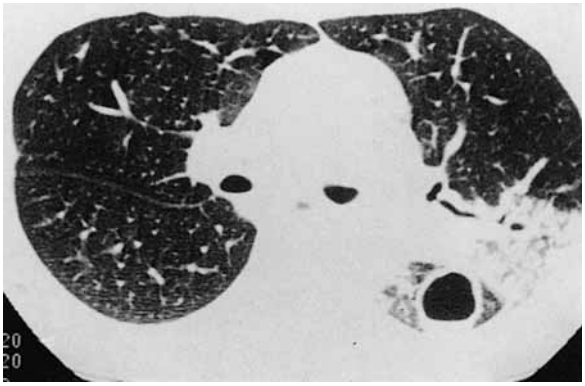
Fig. 3.**26** Tuberculous pleural thickening and fibrocirrhotic pulmonary change.

- *Tuberculous cavities* are 5–10 cm in diameter and result from caseous necrosis of tuberculous pneumonia with subsequent expectoration of the contents. Cavities frequently are combined with disseminated acinar shadows due to endobronchial spread. Coalescence of the latter may occur (Fig. 3.**24**).
- Radiologic manifestations of *fibrotic tuberculosis* include apical pleural thickening, parenchymal scarring, calcification, and fibrotic bands radiating from the hilum to the apex. Cranial migration/elevation of hilar structures indicates fibrous contraction; eventually paracicatricial emphysema, bronchiectasis, and bronchovascular distortion may ensue (Fig. 3.**25**). A thick pleural peel may encase the residual lung and lead to thoracic deformity with kyphoscoliosis (Fig. 3.**26**).

Computed Tomography

CT manifestations of tuberculosis include:

- *Cavitation*: HRCT has been shown to be superior to the chest radiograph in demonstrating cavitation, particularly in cases complicated by fibrosis and architectural distortion (Im 1993, Naidich et al. 1984). Cavitation is frequently, but not invariably, an indicator of active disease (Fig. 3.**27**) as "healed" cavities may persist after antituberculous therapy (Webb et al. 1992).
- *Endobronchial spread*: Features of endobronchial spread are detected by HRCT in up to 98 % of cases (Im 1993). These include centrilobular nodules or linear structures, "tree-in-bud" branching linear structures and poorly defined nodules (Fig. 3.**27**). Centrilobular nodules and tree-in-bud linear structures represent caseating material within the terminal and respiratory bronchioles (Im 1993). Poorly defined nodules probably represent peribronchiolar inflammation (Im 1993, Webb et al. 1992).
- *Miliary tuberculosis*: HRCT images show fine nodules that are distributed uniformly throughout the lungs (Fig. 3.**27**). These may be sharply or poorly defined and range in size from 1 to 4 mm in diameter (Oh 1994). These nodules are distributed randomly throughout the secondary lobule in contrast to the centrilobular nodules of endobronchial spread.
- *Fibrocirrhotic tuberculosis*: Findings indicating chronic parenchymal change include fibrotic bands, bronchovascular distortion, and cicatricial emphysema (Fig. 3.**27**).



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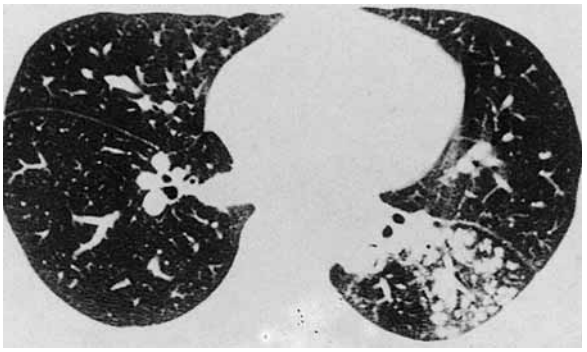

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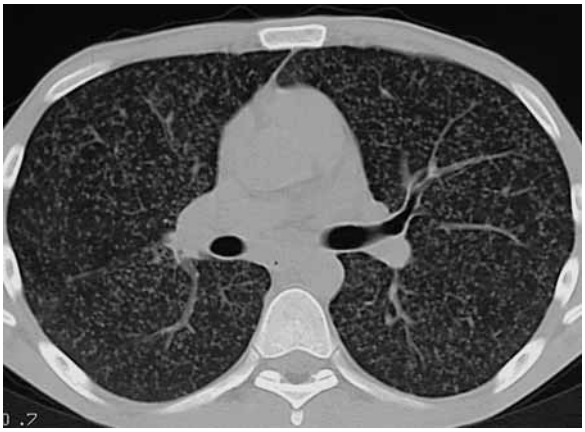

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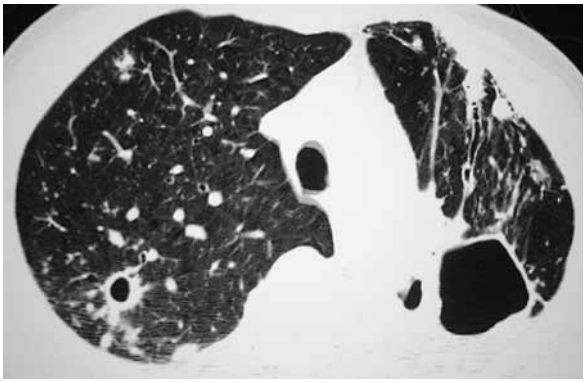


d

Fig. 3.**27 a–d** CT appearances of tuberculosis. **a** Features of active postprimary tuberculosis with cavitating lesion in apical segment of left lower lobe and adjacent nodular shadowing in apicoposterior segment of left upper lobe. **b** Tuberculosis with features of endobronchial spread including centrilobular nodules, branching linear structures (*tree-in-bud* appearance), and confluent poorly defined nodules. **c** CT shows bilateral fine nodular shadowing consistent with military TB. **d** CT shows "healed" cavity with fibrosis in left upper lobe but with evidence of reactivated disease with cavitation and features of endobronchial spread in right upper lobe.

Fungal Diseases of the Lung

Fungal disease of the lung may be classified as endemic or opportunistic.

Endemic fungal diseases are caused by pathogenic fungi in an immunocompetent individual. They include histoplasmosis, coccidioidomycosis, blastomycosis, and sporotrichosis. These infections are endemic in the U.S., Africa, and Asia and are seen sporadically in Europe as a result of foreign travel.

Opportunistic fungal infection (aspergillosis, candidiasis) is caused by saprophytic fungi, which usually are present in the oral mucosa and become pathogenic in the immunocompromised host. These pneumomycoses have been encountered more frequently since the advent of antibiotics and chemotherapy. However, the overall incidence of pulmonary fungal infections remains low.

The clinical symptoms and radiographic findings of these diseases usually resemble those of bacterial pneumonia. Thin section/high resolution CT, in some cases, may be helpful in suggesting the diagnosis. Definitive diagnosis, however, is dependent on identification of the fungus at microscopy.

Candidiasis

Clinical Features

Candida albicans is part of the normal human microbial flora of the oral cavity. Pulmonary candidiasis occurs only in the immunocompromised individual.

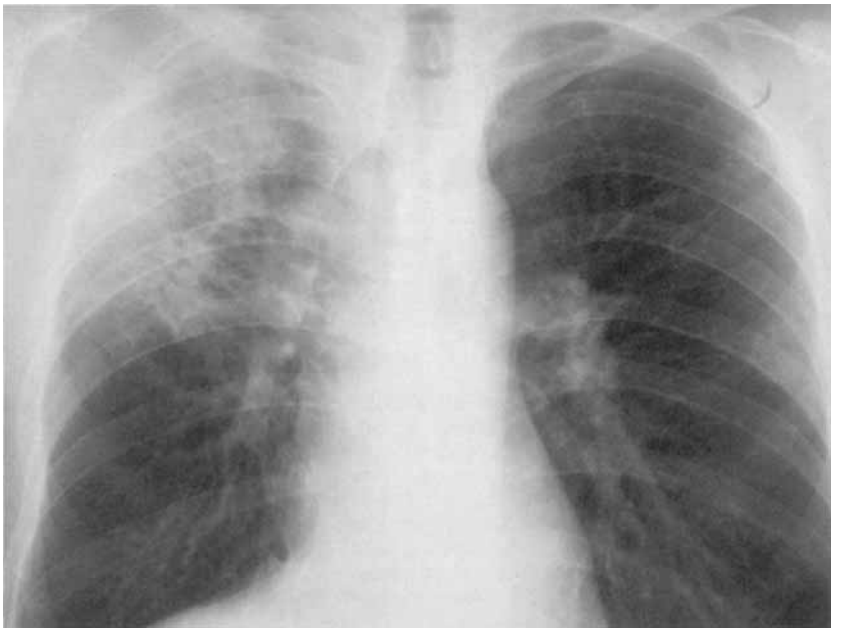


Fig. 3.**28** Candida pneumonia in a leukemic patient on chemotherapy, who presented with oral candidiasis. The right upper lobe continued to show patchy consolidation for several weeks, and two smaller cavitating foci developed on the left side.

Pulmonary candidiasis should be suspected in the presence of a pneumonia that is refractory to standard therapy or in the immunocompromised host with florid oral or esophageal candidiasis. The diagnosis may be established by demonstration of candida in transbronchial biopsy specimens. Sputum analysis is of no value because of the ubiquitous nature of the organism (Geary et al. 1980).

Radiologic Findings

A wide spectrum of radiographic findings has been described in candida pneumonia. Appearances may be indistinguishable from that of bacterial pneumonia with lobar or segmental consolidation (Fig. 3.**28**). Diffuse bilateral alveolar or mixed alveolar-interstitial shadowing may be seen (Buff et al. 1982; Fig. 3.**29**). Candida pneumonia may present as multiple small pulmonary abscesses; these may be randomly distributed if hematogenous spread has occurred or peribronchiolar when resulting from aspiration (Müller 1990). A miliary–nodular pattern has been described in pulmonary candidiasis (Pagani 1981), and diffuse pulmonary hemorrhage is also recognized as a manifestation (Müller 1991).

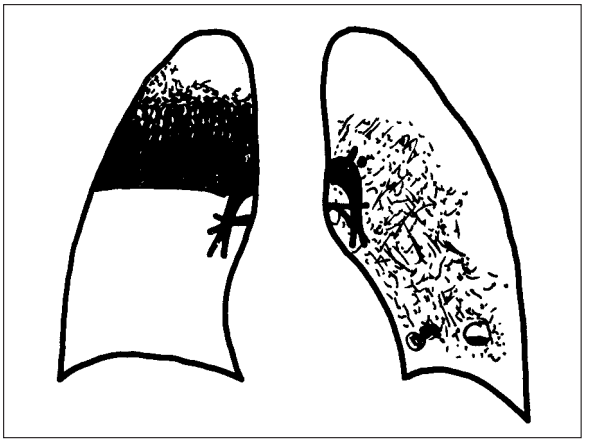


Fig. 3.**29** Pulmonary candidiasis may present as lobar pneumonia, interstitial pneumonia, or bronchopneumonia with cavitation.

Aspergillosis

Aspergillus fumigatus, *A. flavus*, and *A. niger* are ubiquitous and flourish in substances such as cereal grains. They also constitute part of the flora of the healthy oral cavity. The following are recognized manifestations of aspergillosis.

Primary invasive aspergillosis develops when massive amounts of fungal spores are inhaled, usually from cereal dust. The hosts have normal immunity.

Secondary angioinvasive aspergillosis occurs as an opportunistic infection in patients with severe debilitating illness, particularly leukemia and lymphoma, or in those undergoing prolonged therapy. Pathologically, this disease is characterized by mycotic vascular invasion, thrombosis, and hemorrhagic infarction with subsequent necrosis and cavitation. Invasive aspergillosis is associated with a mortality of 60 to 70%, and, in survivors, there is a 50% recurrence rate.

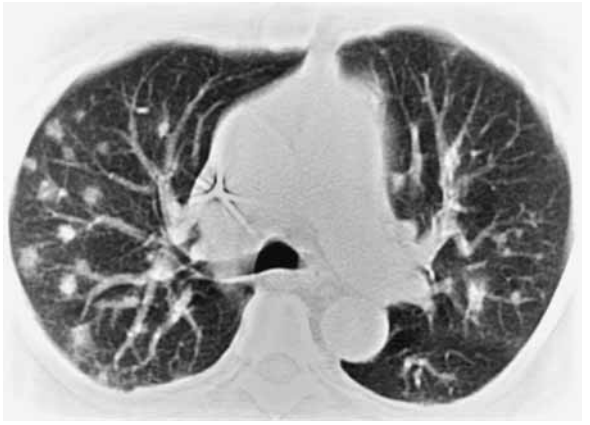


Fig. 3.**30** Angiocentric invasive aspergillosis, confirmed at autopsy.

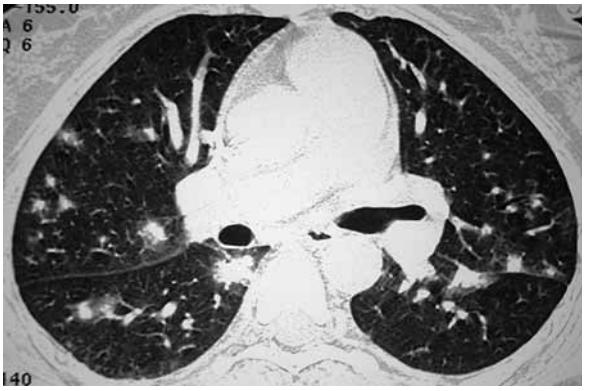


Fig. 3.**31** Angiocentric invasive aspergillosis—early-stage disease with multiple areas of nodular consolidation some of which have a rim of ground-glass opacification highly suggestive of invasive fungal infection.

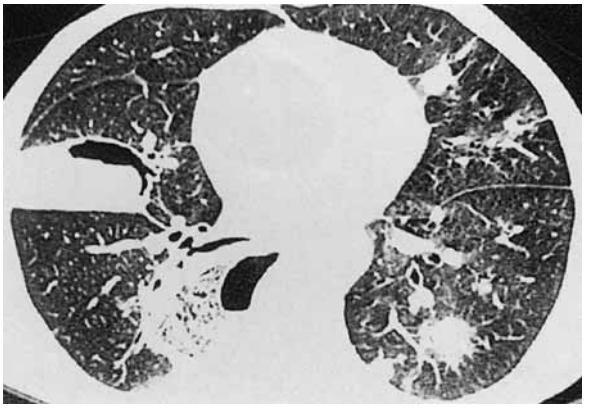


Fig. 3.**32** Angiocentric invasive aspergillosis—recovery phase. Bilateral multifocal rounded consolidation with "air crescents" visible on the right side.

The initial *chest radiograph* may be normal. Multiple foci of consolidation may be present; these are frequently rounded in shape and probably represent infarcted parenchyma (Hruban et al. 1987). The characteristic "air crescent" sign develops late in the course of the disease and usually is associated with a recovering neutrophil count. It is seen in approximately 40% of cases and is associated with improved survival rates.

Computed tomography shows characteristic findings that strongly suggest the diagnosis early in the course of the disease (Kuhlman et al. 1988 and 1987). In early invasive aspergillosis, a "halo" of ground-glass opacification surrounds dense parenchymal foci (Fig. 3.**30**, Fig. 3.**31**). This represents a rim of hemorrhage or coagulation necrosis surrounding an area of infarction (Hruban et al. 1987). The halo sign precedes the air crescent sign (Fig. 3.**32**) by up to 2 weeks (Kuhlman et al. 1987).

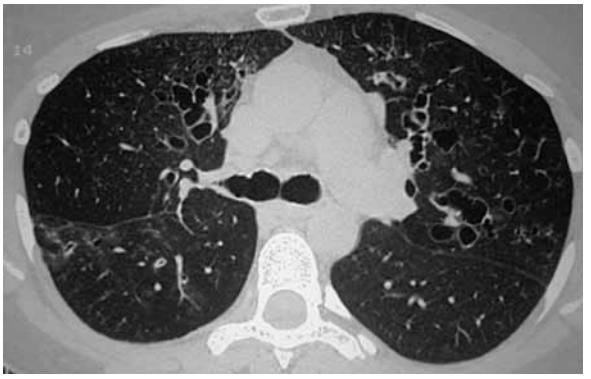
Magnetic resonance imaging may also be helpful in early diagnosis of invasive aspergillosis (Herold 1989). On standard T1-weighted spin echo sequences, rounded consolidations have a target appearance with a hypointense center and hyperintense rim; the rim enhances on administration of intravenous gadolinium.

Invasive aspergillosis of the airways: Aspergillosis centered on the airways accounts for 14–34% of cases (Orr 1978, Young 1970) and also occurs in immunocompromised patients. Diagnosis is based on the presence of organisms deep to the basement membrane. CT findings include lobar consolidation, bilateral peribronchial consolidation, ground-glass attenuation, and centrilobular nodules less than 5 mm in diameter (Logan 1994).

Allergic bronchopulmonary aspergillosis (ABPA) represents a hypersensitivity reaction, usually in asthmatics, and manifestations include asthma, blood eosinophilia, precipitating antibodies to *Aspergillus* and elevated IgE titers. Pathologically, mycelial plugs develop in the proximal airways (Gefter et al. 1981), but, in contrast to invasive aspergillosis of the airways, tissue invasion is minimal or absent (Glimp and Bayer 1981).

The chest radiograph shows transient infiltrates in a lobar, segmental, or subsegmental distribution which predominantly involve the upper lobes. Atelectasis is less common, occurring in 3–46% of cases (Gefter et al. 1981, Malo et al. 1977). Bronchoceles also are a frequent radiographic manifestation of ABPA; these vary in shape but classically present a "gloved-finger" appearance. The lung distal to the bronchocele is aerated by collateral air drift. Eventually, central bronchiectasis involving the inner two-thirds of the bronchial tree and showing an upper lobe predominance may develop (Fig. 3.**33 a**, **b**).

Aspergilloma is the most common form of aspergillosis. It occurs in hosts with normal immunity, and the fungus colonizes preexisting cavities (cysts, tuberculous cavities, cystic bronchiectasis) and forms a fungus ball. This may erode the cavity wall both mechanically and through enzymatic action and lead to hemoptysis; this occurs in 50–80% of cases and may occasionally be life



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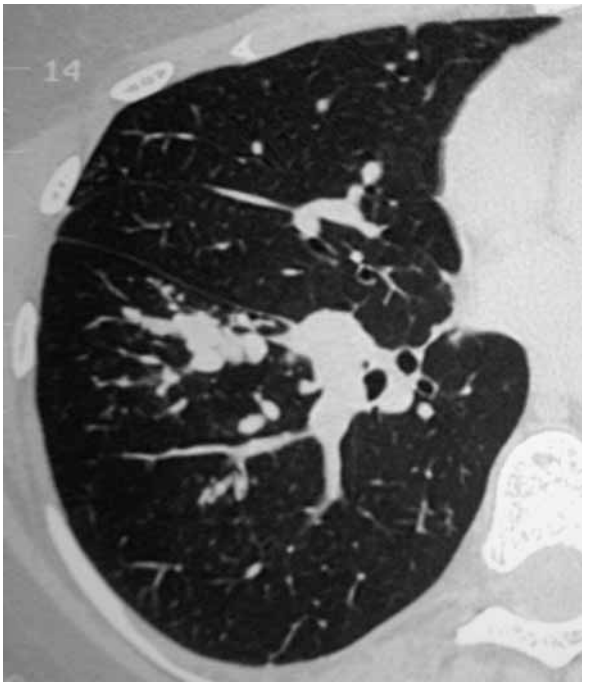


b

Fig. 3.**33 a, b** Allergic bronchopulmonary aspergillosis. Varicose bronchiectasis is seen in both upper lobes (**a**) with extensive mucoid impaction in the apical segment of the right lower lobe (**b**).

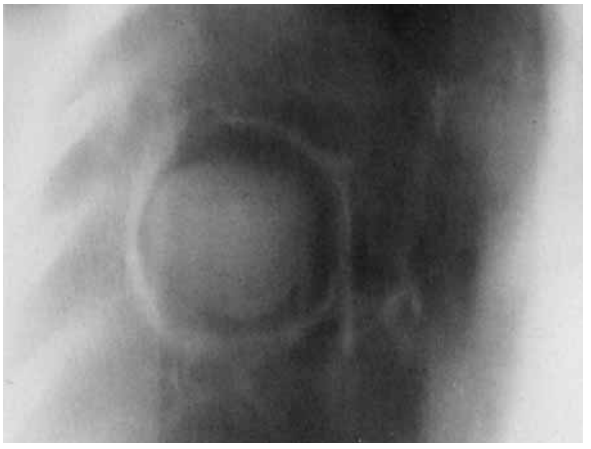


Fig. 3.**34** Conventional tomogram shows an aspergilloma occupying an old tuberculous cavity.

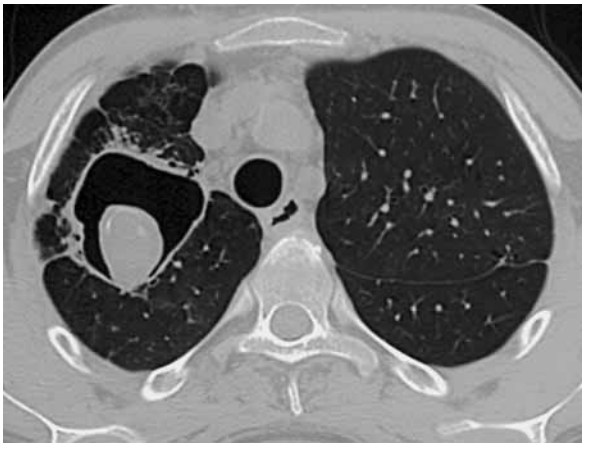


Fig. 3.**35** CT of aspergilloma in an emphysematous bulla. The wall of the cavity is thickened due to recurrent episodes of inflammation. The aspergilloma is partially calcified.

threatening (Faulkner et al. 1978, Freundlich and Israel 1973, Jewkes et al. 1983).

The *chest radiograph* shows a round, homogeneous opacity which is mobile within the cavity. A circular or crescent-shaped air space may be visible between the mycetoma and the cavity wall (Fig. 3.**34**). Localized pleural thickening may be seen adjacent to the cavity, indicating superimposed aspergillus infection (Libshitz 1974).

CT will show the mycetoma of inhomogeneous attenuation and the surrounding crescent of air within the cavity (Figs. 3.**35**, 3.**36**). The mobility of the intracavitary mass may be demonstrated by image acquisition in the prone position. The mycetoma has a characteristic sponge-like appearance and contains multiple foci of air (Armstrong et al. 1995, Roberts et al. 1987).

ABPA may be diagnosed by microscopic detection of *Aspergillus* mycelia in the bronchial aspirate. Aspergilloma may be largely a radiologic diagnosis. Both transbronchial and open lung biopsy may be hazardous in the immunocompromised with bone marrow suppression. Sputum analysis has no value because the sputum contains nonpathogenic fungi.

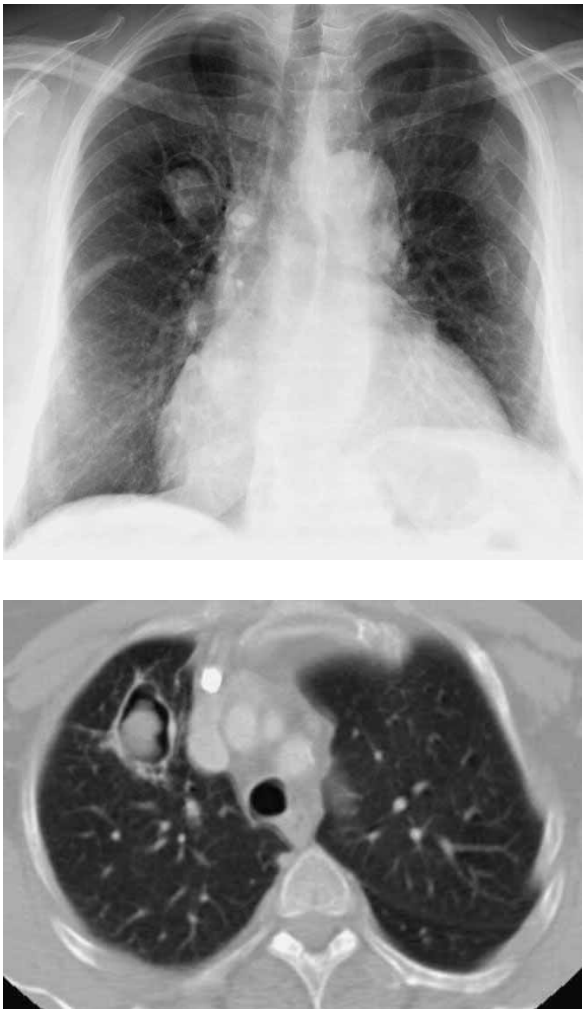


Fig. 3.**36 a, b** Aspergilloma.

Histoplasmosis

Clinical Features

Histoplasmosis is a fungal infection that occurs mainly in North America. Except for an endemic region in northern Italy, it occurs only sporadically in Europe. Pulmonary changes caused by *Histoplasma capsulatum* are comparable to tuberculosis in both primary and postprimary phases of development.

Acute histoplasmosis develops as a result of airborne primary infection. An incubation period of 2 weeks precedes the onset of pyrexia, malaise, dyspnea, productive cough, and hemoptysis; this infection may also run an asymptomatic course.

Radiologic Findings

- The *chest radiograph* shows multiple, ill-defined areas of consolidation throughout both lungs. There is accompanying hilar and mediastinal lymphadenopathy (Fig. 3.**37**). These pneumonic consolidations heal, leaving residual pulmonary granulomata that undergo central calcification to produce a target pattern (Connell and Muhm 1976; Fig. 3.**38**). When granulomata are multiple, calcification occurs in up to 75 % of cases, but only 25 % of solitary granulomata will calcify.
- *Chronic progressive histoplasmosis* is the consequence of reactivation and has a poor prognosis. Progressive cavitation with fibrosis may progress to complete lung destruction.

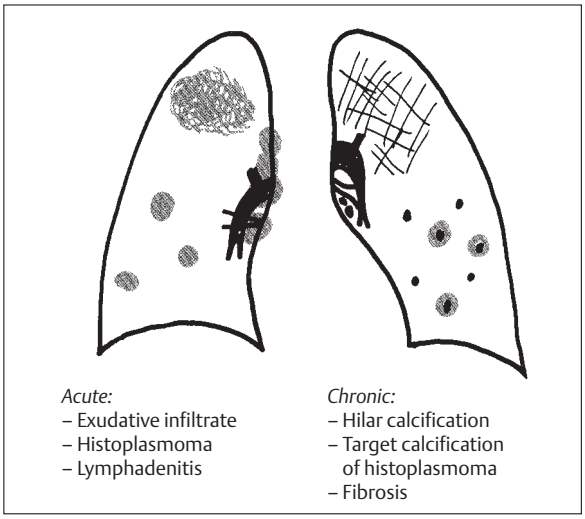


Fig. 3.**37** Histoplasmosis.

Coccidioidomycosis

Clinical Features

Coccidioidomycosis is endemic in the southwestern United States. It usually is asymptomatic and only the coccidioidin skin test is positive with elevated complement fixation. In those who become symptomatic, manifestations include severe pneumonia, pulmonary cavitation, pleurisy, and pericarditis. The development of pulmonary fibrosis represents end-stage disease (Bayer 1981, McGahan et al. 1981).

Radiologic Findings

The chest radiograph shows pneumonic consolidation and pulmonary nodules (coccidioidomas) that occasionally cavitate. In disseminated coccidioidomycosis, there is a generalized micronodular pattern (Fig. 3.**39**).

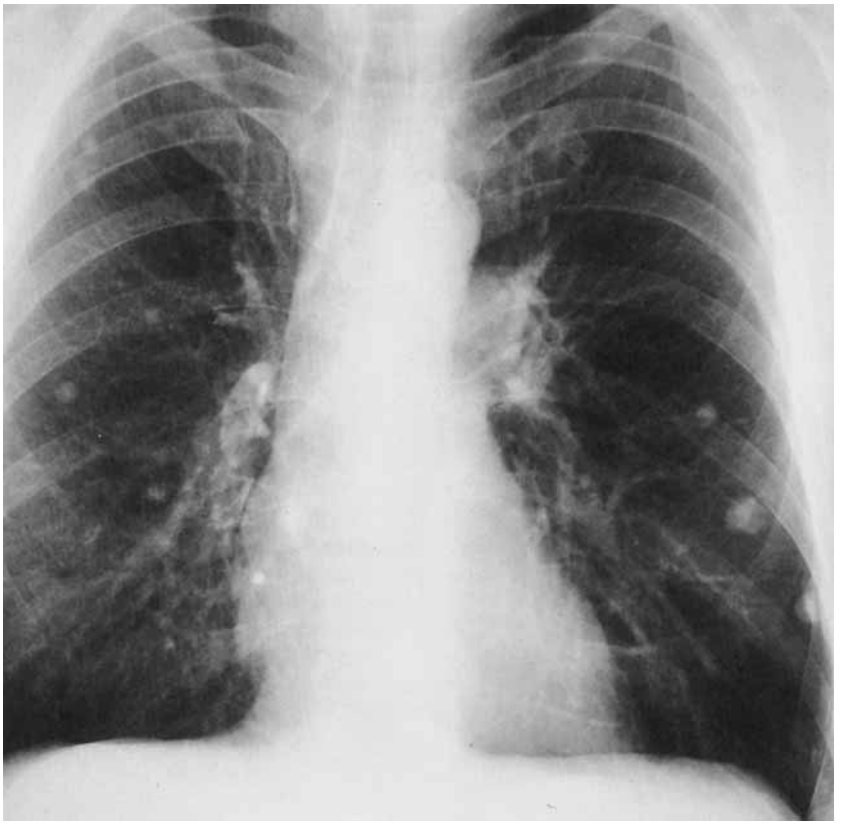


Fig. 3.**38** Histoplasmosis. Disseminated, calcified granulomas were found in a North American male who had a history of histoplasmosis 20 years earlier.

Changes of pulmonary fibrosis are associated with advanced disease.

Actinomycosis

Clinical Features

Actinomyces israelii is intermediate between mycelial fungi and bacteria and is a common saprophyte in the human mouth, especially in the presence of dental caries. This is a relatively rare disease entity and involves the cervicofacial region, the intestinal tract, and the lung. In the thorax, manifestations include chronic cavitating pneumonia, pleural empyema, and chest wall invasion (Fig. 3.**40**).

Radiologic Findings

The chest radiograph shows nonsegmental, predominantly peripheral consolidation that may cavitate. Consolidation typically crosses interlobar fissures. Pleuroesophageal and pleuropulmonary fistulae, pleural empyema, rib osteomyelitis, and inflammatory soft tissue masses of the chest wall may develop.

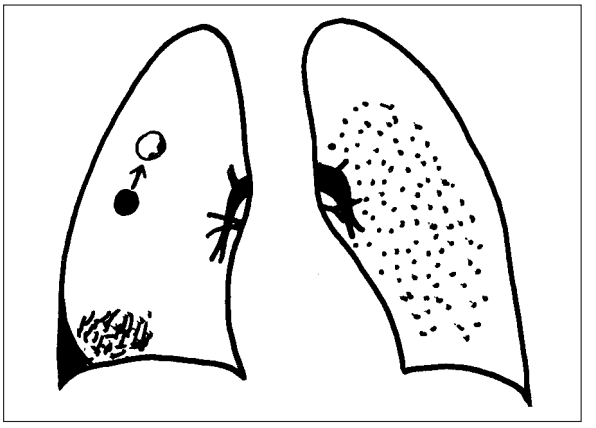


Fig. 3.**39** Coccidioidomycosis: coccidioidomas, cavitation, bronchopneumonia, pleural involvement, and widespread microgranulomas. Pulmonary fibrosis is present in end-stage disease.

Nocardiosis

Nocardia asteroides is a ubiquitous aerobic saprophyte found in the soil. It is a weakly acid-fast bacillus, which, after inhalation, may infect the lung sporadically. Pulmonary nocardiosis may be similar to actinomycosis in its radiographic appearance. Single or multiple

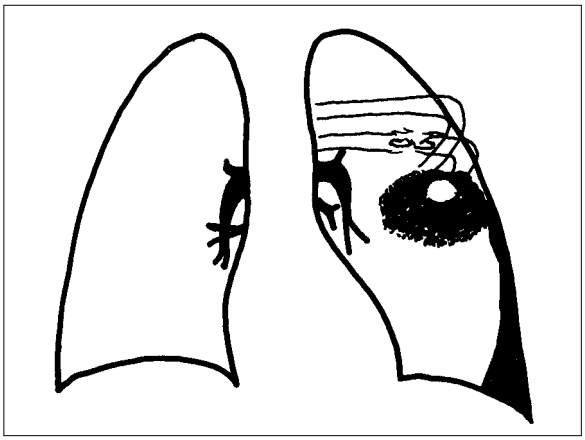


Fig. 3.**40** Actinomycosis: consolidation with abscess formation, pleural empyema, osteomyelitis, chest wall abscess.

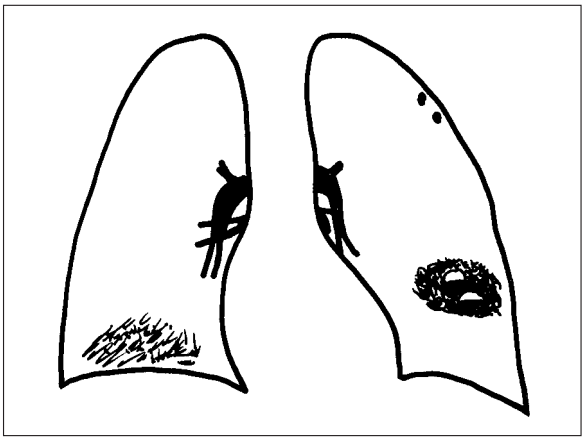


Fig. 3.**41** Cryptococcosis: tumor-like masses with liquefaction, subpleural granulomas, and bronchopneumonia.

parenchymal abscesses frequently are seen, and pleural involvement is also common. There is an increased incidence of nocardiosis in the immunocompromised, in AIDS, and in alveolar proteinosis.

Cryptococcosis (Torulosis)

Cryptococcosis results from inhalation; the spores of *Cryptococcus neoformans* are found in dust and excreta (e. g., pigeon droppings) and cause pulmonary infection in immunocompromised hosts. The chest radiograph shows small, subpleural granulomas, foci of bronchopneumonia, and round masses (torulomas), which may cavitate (Fig. 3.**41**).

Other mycoses like North and South American blastomycosis, sporotrichosis, and mucormycosis are extremely rare and present radiologically as nonspecific pneumonic infiltrates. Diagnosis is based on demonstration of the fungus in tissue, smear, or culture.

Parasitic Infections

Parasitic infections are most prevalent in Asia, Africa, South America, and the Mediterranean basin. The causative organisms are protozoa (ameba, toxoplasma,) and helminths (echinococcus, schistosomes, ascarids, etc.). They induce hypersensitivity reactions in the lungs with formation of an eosinophilic "Loeffler" infiltrate. Parasites may colonize the lungs and form cysts, granulomata, and abscesses. Radiographic abnormalities together with blood eosinophilia should raise the suspicion of a parasitic pulmonary infection. Diagnosis is confirmed by identification of parasites in the sputum, stool, and urine, and if necessary by biopsy and histological assessment.

Amebiasis

Clinical Features

Amebae are found worldwide but are endemic in the Mediterranean region. They are ingested in contaminated food and initially induce colitis (amebic dysentery). They reach the liver via the bloodstream and form hepatic abscesses, which may extend through the diaphragm to infect the lung. Direct hematogenous spread from the liver to the lung is rare. Clinical manifestations include cough, blood eosinophilia, and expectoration of bile when a hepatobronchial fistula is present (Meng 1994).

Radiologic Findings

In 95% of cases, the chest radiograph shows opacification of the right lower hemithorax due to pneumonic consolidation and an accompanying pleural effusion. The initially ill-defined infiltrate may form an abscess (Fig. 3.**42**).

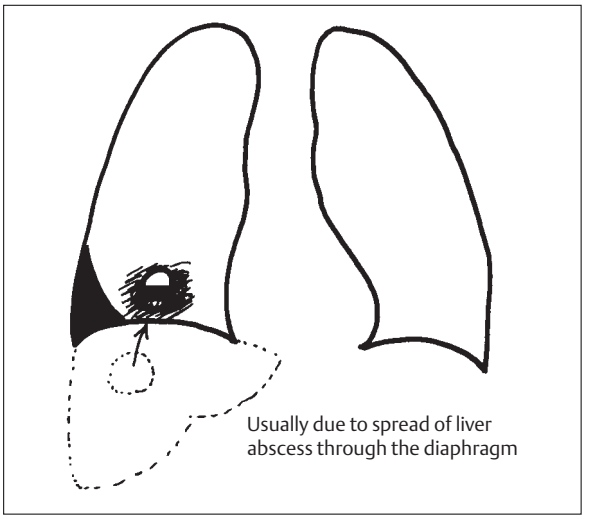


Fig. 3.**42** Amebiasis (amebic abscess): pleural effusion, basal pneumonia with cavitation.

Toxoplasmosis

Clinical Features

Infestation with *Toxoplasma gondii* is common but rarely leads to disease. Congenital toxoplasmosis due to transplacental infection is the most important form and presents with encephalitis and chorioretinitis. Adult toxoplasmosis is relatively uncommon except in patients with AIDS, in whom it is the most common cause of focal central nervous system lesions. In the HIV-negative population, it manifests as lymphadenitis and occasionally as interstitial pneumonia.

Radiologic Findings

Radiographs show focal reticular, linear, and ill-defined opacities resembling acute viral pneumonia. Associated hilar lymph node enlargement is frequent (Müller and Fraser 2001; Fig. 3.**43**).

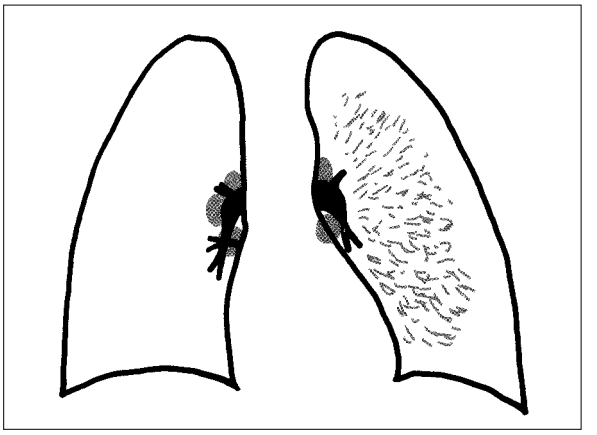


Fig. 3.**43** Toxoplasmosis: interstitial pneumonia, hilar lymphadenopathy.

Pneumocystis Jiroveci Pneumonia (Previously Known as Pneumocystis Carinii Pneumonia—PCP)

Clinical Features

Pneumocystis jiroveci/carinii pneumonia was originally described in premature infants. In adults, it is a frequent pathogen in the immunocompromised. The marked increase in the incidence of PCP has largely resulted from the acquired immunodeficiency syndrome epidemic; 60–70% of patients with AIDS will develop PCP, and in the 1990s, it was the most frequent index disease for AIDS in industrialized countries (40%) (Kuhlman 1996, Safrin 1993, Naidich and McGuinness 1991). It continues to be a significant cause of morbidity in HIV/AIDS and more recent studies have indicated that it is second only to bacterial pneumonia in the etiology of pulmonary complications (Benito Hernandez et al. 2005).

Initial growth of pneumocystis is in the alveoli where it becomes attached to Type 1 pneumocytes. Damage and death of these cells destroys the integrity of the alveolar-capillary membrane, and filling of the alveoli with eosinophilic exudate occurs. Concomitant activation of macrophages and plasma cells in the interstitium results in an interstitial pneumonitis (Kuhlman 1996).

In HIV-positive patients, the preferred method of diagnosis is induced sputum samples proceeding to bronchoalveolar lavage when necessary. Transbronchial biopsy is avoided because of the associated high mortality and risk of pneumothorax in these patients. In patients without AIDS, transbronchial or open lung biopsy is appropriate (Geary et al. 1980, Kuhlman 1996).

Radiologic Findings

Chest Radiograph

The initial chest radiograph may be normal, but in 80% of cases it shows diffuse, bilateral, granular, or reticular infiltrates (Kuhlman 1996, Safrin 1993, Naidich et al. 1991, Goodman 1991, DeLorenzo et al. 1987). These may involve the perihilar and lower zones or have an upper lobe distribution. Progression to diffuse air space consolidation may occur. Hilar lymph node enlargement and pleural effusions are unusual.

Computed Tomography

In acute PCP, the commonest HRCT finding is bilateral ground-glass opacification. Less commonly, a mosaic pattern with scattered foci of parenchymal involvement interspersed with normal lung is found (Fig. 3.**44**). In a proportion of cases, thickened interlobular septa are found in association with ground-glass opacification (Bergin et al. 1990). Progression to diffuse homogeneous ground-glass opacification with sparing of the subpleural lung may occur (Kuhlman 1990, Scott 1991).

Recent years have seen a change in the pulmonary manifestations of pneumocystis infection. Cystic lung disease, spontaneous pneumothorax, and an upper lobe distribution of opacification are now seen more frequently (Figs. 3.**45**, 3.**46**). In the past, these were usually associated with aerosolized pentamidine prophylaxis but this has now been largely replaced by more effective chemoprophylaxis (Boiselle et al. 1999).

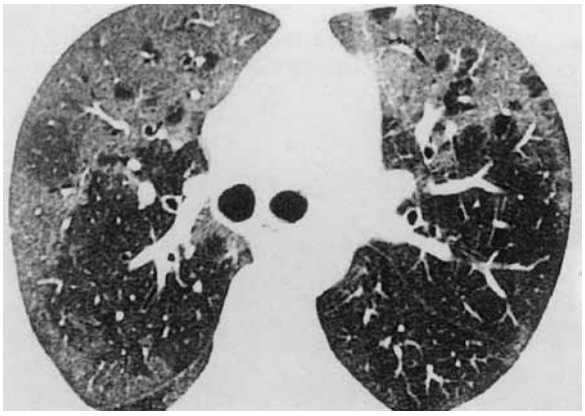


Fig. 3.**44** Pneumocystis pneumonia. Ground-glass opacification involving both upper lobes. Some inhomogeneity is seen anteriorly with sparing of scattered secondary pulmonary lobules.

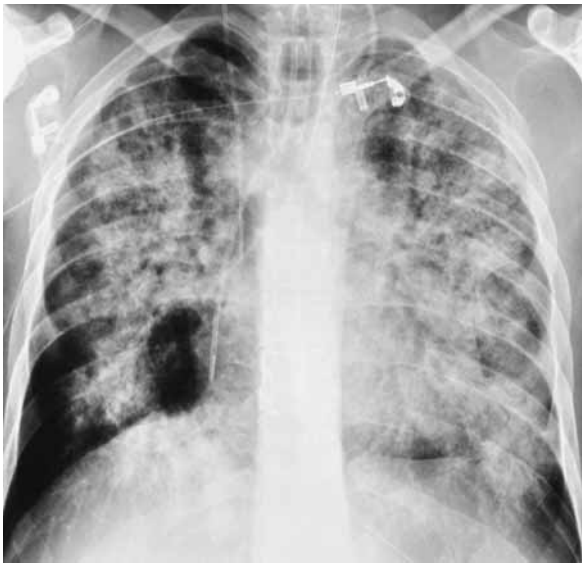
Schistosomiasis

Clinical Features

Schistosomiasis hematobium is endemic in North Africa, *Schistosoma mansoni* in South America and the Caribbean, and *Schistosoma japonicum* in Japan. Cercariae, the infective larvae, penetrate the skin, enter the capillaries, and migrate through the systemic venous system to the right heart. From there they enter the pulmonary circulation and subsequently the systemic arterial system to reach the liver, kidneys, and urinary bladder. Diagnosis is based on identification of *Schistosoma* eggs in the stool and urine.

Radiologic Findings

The chest radiograph shows transient pulmonary infiltrates representing an eosinophilic Loeffler-type pneumonia which is associated with passage of the larvae



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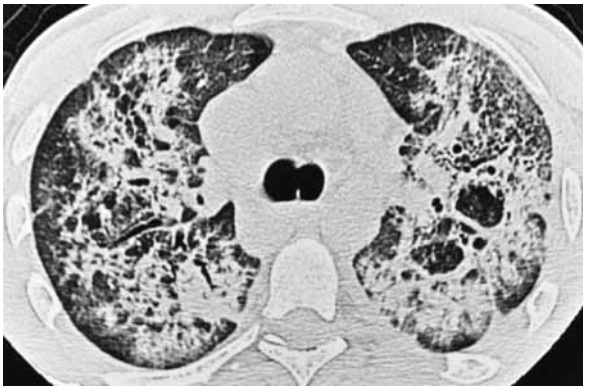

b

Fig. 3.**45 a, b** Pneumocystis infection in a renal transplant patient. Extensive ground-glass opacification/consolidation, interstitial thickening, and some cystic change are seen.