7 Chest Radiology in AIDS

Linda B. Haramati and Elizabeth R. Jenny-Avital

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L.B. Haramati, MD
Department of Radiology, Albert Einstein College of Medicine and Montefiore Medical Center, 111 East 210th Street, Bronx, New York 10467, USA
E.R. Jenny-Avital, MD
Department of Medicine, Division of Infectious Diseases, Albert Einstein College of Medicine, Jacobi Medical Center, 1400 Pelham Parkway South, Bronx, New York 10461, USA

7.1 Introduction

Human immune deficiency virus (HIV) infection is a heterogeneous condition whose most advanced expression, AIDS, is associated with a number of well-characterized opportunistic infections and neoplasms. However, clinically asymptomatic HIV infection accounts for a longer duration of HIV infection than does symptomatic AIDS, except for a minority of patients who progress rapidly to AIDS. Although asymptomatic HIV-infected patients do not have AIDS-related opportunistic infections or CD4 cell counts of less than 200 cells/mm^3, they do have demonstrable immunologic abnormalities. Another interesting group of HIV-infected patients have experienced substantial immune reconstitution as a result of highly active antiretroviral therapy (HAART). These patients generally have very low CD4 cell counts, which rise during treatment with HAART (Palella et al. 1998). The newly reconstituted CD4 cell count seems to protect against the opportunistic infections that occur with lower CD4 cell counts (Schneider et al. 1999). Paradoxically, some patients develop localized inflammatory reactions soon after HAART is begun, as the viral load is abruptly decreasing. These "immune reconstitution phenomena" are often targeted against a preexisting infection such as tuberculosis (Narita et al. 1998) or *Mycobacterium avium* complex infection (Race et al. 1998).

HIV-infected patients without AIDS have subtle immunologic derangements which coexist alongside the usual risk factors for lung disease. The interplay between the patient's perturbed immune system and various infectious agents and environmental factors likely accounts for the variety of cardiopulmonary manifestations increasingly linked to HIV infection. Bacterial pneumonia, tuberculosis, cardiomyopathy (Barbaro et al. 1998), pulmonary hypertension (Opravil et al. 1997), lymphocytic interstitial pneumonitis (Travis et al. 1992), and emphysema (Diaz et al. 2000) are all linked to HIV but do not occur at a predictable level of immune dysfunction.

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Table 7.1. Intrathoracic infections and neoplasms occurring with increased prevalence in HIV infection

Clinical setting	Condition	Radiographic findings	Epidemiology and associations
	Common		
First AIDS-defining opportunistic infection (CD4 <200 cells/mm^3)	*Pneumocystis carinii* pneumonia	Bilateral symmetric, perihilar granular, reticular or airspace opacities Air cysts, pneumothorax Pleural effusions and lymphadenopathy uncommon	Very common in USA Prevented by antibiotic prophylaxis
	Cryptococcosis	Single/multiple nodules, consolidation, +/- cavitation	Common in USA and developing countries Meningitis far more common
	Histoplasmosis[a]	Normal or diffuse, small, ≤3 mm lung nodules	Endemic: Ohio, Mississippi River valleys, Caribbean, Central America
	Coccidioidomycosis[a]	Diffuse nodular or reticulonodular parenchymal opacities Focal consolidation, hilar adenopathy, pleural effusions	Desert of southwest USA, parts of South America
	Penicilliosis[a]	Localized diffuse reticular opacities, consolidation	Southeast Africa, associated with characteristic skin lesions
	Less common		
	Nocardiosis	Large consolidation, diffuse inter stitial pattern, mass, +/- cavitation	Southern USA, rural areas
	Uncommon		
	Toxoplasmosis	Bilateral coarse nodular and reticulonodular opacities	Europe, cat exposure
	Rhodococcosis	Dense consolidation, cavitation, pleural effusion, empyema	Present in soil, causes disease in farm animals
	Bacillary angiomatosis	Endobronchial lesions, parenchymal nodules, lymphadenoma	Reservoir in cats
	Blastomycosis	Focal airspace opacities or masses Diffuse nodular opacities	Midwest, South Central USA
Subsequent opportunistic infection seen in long standing advanced HIV	Aspergillosis	Chronic necrotizing: thick-walled cavity Disseminated disease: bilateral nodules/masses	Preexisting lung disease Steroids, neutropenia, antibiotics
	Cytomegalovirus	Reticular, reticulonodular opacities	
	Pseudomonas, other gram-negative pneumonia	Consolidation, cavitation	
	Kaposi's sarcoma	Coarsening bronchovascular bundles Ill-defined nodules with perihilar predominance	Seropositive for HHV-8 Male homosexual, Africa
	Strongyloidiasis	Hyperinfection syndrome: bilateral miliary nodules, reticular interstitial opacities	Tropical, subtropical regions
	Mycobacterium avium complex	Lymphadenopathy, parenchymal consolidation, small or large pulmonary nodules, +/- cavitation	
	Nontuberculous mycobacteria	Similar to tuberculosis	
Any CD4	Tuberculosis	Consolidation, nodules, lymphadenopathy, cavitation infrequent at low CD4, +/- pleural effusions	Prior exposure Developing countries
	Bacterial pneumonia	Lobar consolidation	Smoking, intravenous drug use
	Cardiomyopathy[a]	- cardiac silhouette Pulmonary venous congestion	Infection with cardiotropic viruses
	Pulmonary hypertension	Enlarged central pulmonary arteries	
	Emphysema (precocious)	Severe emphysema	Smoking
	Lymphoma	Lung nodules and masses Pleural effusions	Epstein-Barr virus infection

[a]Occurs in persons who have resided in endemic areas

Among patients with AIDS, opportunistic infections occur largely as a result of reactivation of infections acquired remotely. Thus they vary with the patients' geographic and exposure history (Table 7.1). The most overt example of this is the frequent occurrence of *Pneumocystis carinii* pneumonia in North America and Europe in contrast to its rarity in Africa, presumably due to differences in environmental distribution of *Pneumocystis carinii*. The endemic mycoses with geographically limited distributions cause fungal disease in patients who live or have lived in the region of endemicity. This is true for coccidioidomycosis, histoplasmosis, and penicilliosis. A history of past exposure to tuberculosis and to certain animals (e.g., cats – bacillary angiomatosis) is relevant to the occurrence of disease due to those agents. Infections due to agents that reside in soil such as *Nocardia* and *Rhodococcus* are seen more often in patients who live or have lived in rural locales. Sometimes laboratory tests can also be used to determine past exposure to relevant infectious agents: PPD for tuberculosis or *Toxoplasma* serology for *T. gondii*. The HIV risk factor may be relevant in predicting the types of opportunistic conditions that complicate AIDS. For example, human herpes virus 8 (HHV-8), the virus associated with Kaposi's sarcoma, is sexually transmitted, more common in gay men, and endemic in Africa.

7.2 Bacterial Infections

HIV-infected patients are prone to developing bacterial infections in the chest, including both tracheobronchitis (McGuinness et al. 1997) and pneumonia (Hirschtick et al. 1995). At CD4 cell counts above 200 cells/mm^3, the infections are usually caused by common pathogens (Shah et al. 1997). Two or more episodes of bacterial pneumonia in a single year are AIDS-defining in an HIV-infected person regardless of CD4 cell count.

7.2.1 Tracheobronchitis

Tracheobronchitis is common in HIV infection, especially in cigarette smokers. A specific microbial etiology is often not identified with either acute or chronic tracheobronchitis. However, usual respiratory viruses, *Chlamydia*, and *Mycoplasma* are often implicated.

Tracheobronchitis is usually not evident on chest radiographs. Subtle findings such as peribronchial thickening and "increased markings" may, however, be present. On computed tomography (CT), bronchial wall thickening, mucoid impaction, and small centrilobular nodules are typical features (McGuinness et al. 1997) (Fig. 7.1). Bronchiectasis occurs with increasing frequency in AIDS patients and may be re-

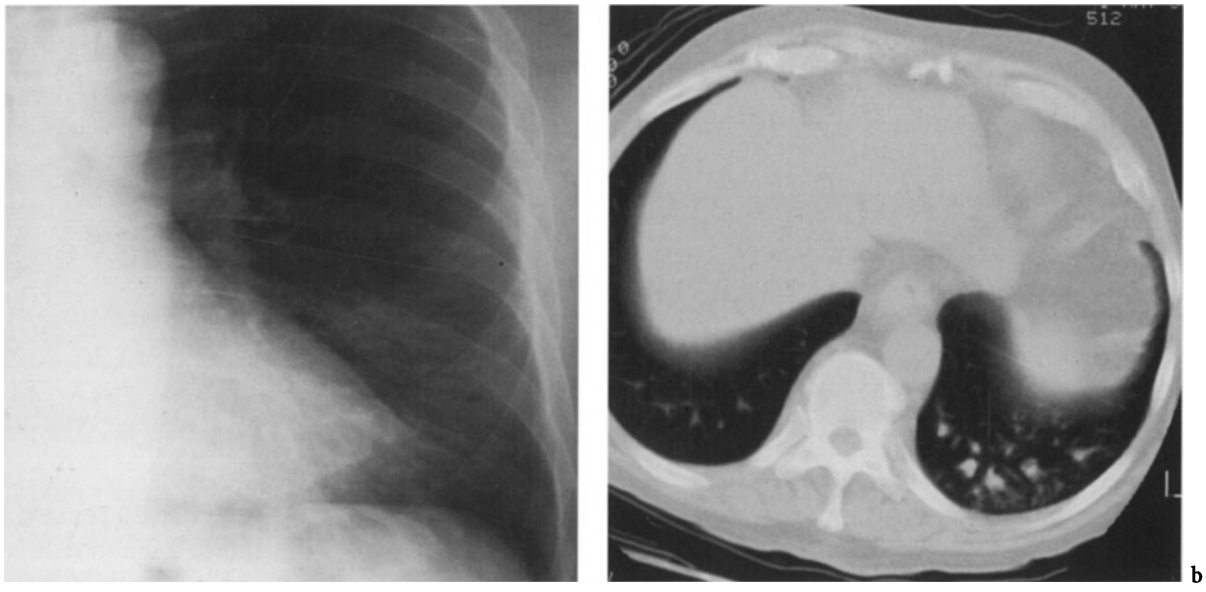


Fig. 7.1a, b. A 55-year-old man with AIDS and persistent cough and bronchitis. **a** Cone-down view from a PA chest radiograph demonstrates tubular opacity in the left lower lobe. **b** Chest CT at the lung basis demonstrates tubular "v" and "y" shaped opacities in the left lower lobe consistent with mucoid impaction

lated to repeated episodes of bronchitis and pneumonia, although it has been described in AIDS without previously documented infections and may, at times, be a more direct effect of HIV on the bronchi.

7.2.2 Pneumonia

Bacterial pneumonia caused by the usual pathogens is common at all stages of HIV infection but more so as immunodeficiency progresses. Additional risk factors for the occurrence of bacterial pneumonia such as cigarette smoking, intranasal drug use, periodic unconsciousness due to drugs, alcohol, or seizures, depressed sensorium due to neurologic disease, and poor dentition are commonly operative in addition to HIV infection.

Pneumococcal pneumonia is still the most important cause of acute bacterial pneumonia and presents as a "typical pneumonia." Bacteremic pneumococcal pneumonia is more common with HIV infection than in those without immunosuppression, although bacteremic pneumococcal pneumonia is not uncommon in normal hosts. In general, when AIDS patients develop bacterial pneumonia, it is more frequently multilobar and is more frequently associated with bacteremia than in the non-AIDS population.

Certain bacterial pathogens are associated with pneumonias occurring at low CD4 counts. This includes *Pseudomonas aeruginosa*, enteric gram-negative rods, *Rhodococcus equi*, and *Nocardia* species (Furman et al. 1996). Pneumonias due to these pathogens are often associated with cavitation (Fig. 7.2).

Septic emboli, caused by bacterial endocarditis and usually due to *Staphylococcus aureus*, occur in bacteremic intravenous drug abusers. Such patients are acutely ill, complain of marked pleuritic chest pain, and have fever and leukocytosis. Chest radiographic and CT findings include bilateral focal pulmonary parenchymal opacities with a peripheral predominance (Fig. 7.3). The opacities are often wedge shaped and develop cavitation. Pleural effusions and cardiomegaly may be present.

Routine antimicrobial prophylaxis against *Pneumocystis carinii* with trimethoprim/sulfamethoxazole in patients with a CD4 cell count of less than 200 cells/mm^3 and against *Mycobacterium avium* complex with a macrolide antibiotic in patients with a CD4 cell count of less than 50 cells/mm^3 reduce the occurrence of bacterial pneumonia. Such antibiotics also change the spectrum and antimicrobial susceptibility of the patient's flora.

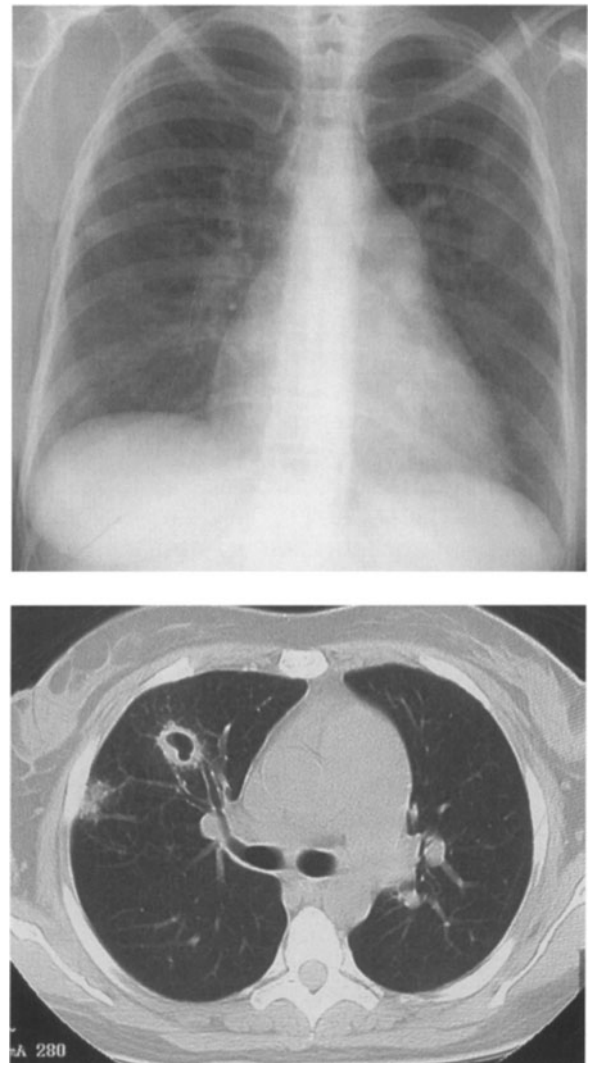
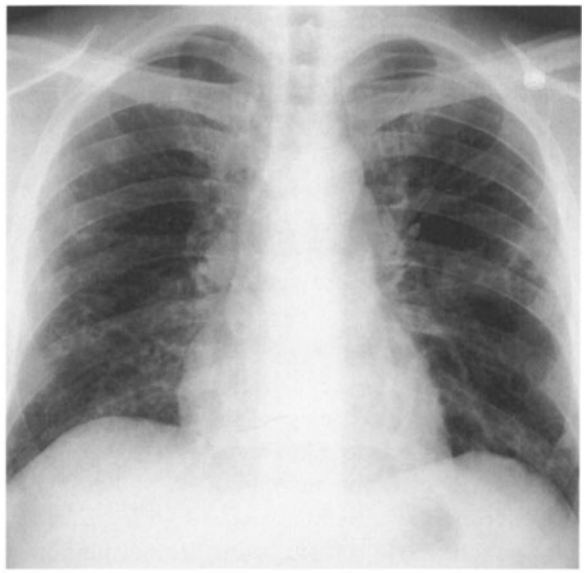


Fig. 7.2a, b. A 37-year-old woman with AIDS, CD4=5/mm^3, presenting with cough and fever. She was diagnosed with *Pseudomonas* pneumonia. **a** PA chest radiograph demonstrates vague bilateral upper lobe opacities. Additionally, there is cardiomegaly. The central pulmonary arteries are prominent, consistent with pulmonary arterial hypertension. **b** Chest CT at the level of the right upper lobe bronchus 1 week later demonstrates a 2-cm-thick walled cavity in the anterior segment of the right upper lobe. There is a focal area of consolidation posterolateral to the cavity

7.2.3 Nocardiosis

Nocardiosis is caused by a soil-borne aerobic actinomycete, a higher bacterium, usually acquired by inhalation. *Nocardia* causes disseminated infection in a variety of immunocompromised hosts such as those with lymphoreticular malignancies, those with organ transplants, and those receiving immunosuppressive treatments. *Nocardia*, especially *N. brasiliensis*, can



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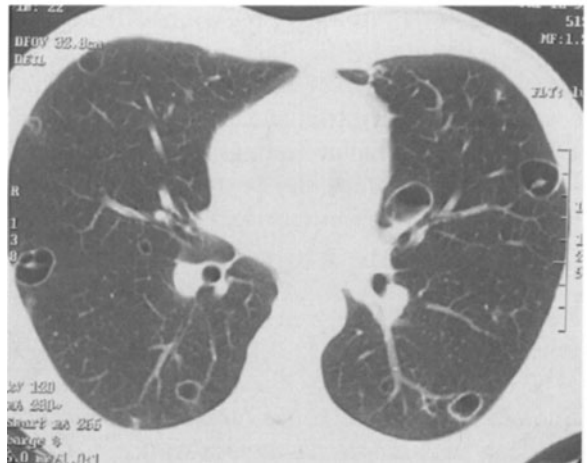

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Fig. 7.3a, b. A 39-year-old HIV-infected man, an intravenous drug user, with a CD4 count of 59/mm^3. Blood cultures were positive for *Staphylococcus aureus*. **a** PA chest radiograph demonstrates multiple bilateral lung nodules, many cavitaries, consistent with septic emboli. Splenomegaly is present. **b** Chest CT demonstrates multiple bilateral cavities with a dramatic peripheral predominance, typical for septic emboli

cause skin and soft tissue infections in immunocompromised hosts. *Nocardia* is not common in AIDS. When it does occur, it is more commonly reported from southern and rural regions of the United States, possibly reflecting differential exposure to soil-borne pathogens compared to urban areas. In AIDS patients, *Nocardia* infection, usually *N. asteroides*, commonly involves the lungs; it may concomitantly involve the CNS or soft tissues. Less frequently, only extrapulmonary sites are involved. Most of the *Nocardia* species which cause disseminated disease respond clinically to trimethoprim/sulfamethoxazole, so that routine prophylaxis against *Pneumocystis carinii* pneumonia prevents nocardiosis. Nocardiosis in AIDS typically presents in patients with a low CD4 cell count and it may be a first opportunistic infection heralding HIV infection (Uttamchandani et al. 1994).

Radiographically, *Nocardia* presents with large areas of consolidation involving several lobes, a diffuse interstitial pattern, or a solitary well-defined mass. Cavitation within a consolidated area is also common. Pleural or pericardial effusion and mediastinal lymphadenopathy may occur (Kramer and Uttamchandani 1990) (Fig. 7.4). An upper lobe predominance is described and the radiographic findings may be mistaken for tuberculosis (Bani-Sadr et al. 1995).

7.2.4 Rhodococcosis

Rhodococcosis is caused by infection with *Rhodococcus equi*, a gram-positive rod which resides in soil and is known to cause pneumonia in horses, pigs, and other farm animals. Human infection is uncommon and occurs exclusively in immunocompromised hosts, including AIDS patients with CD4 cell counts of less than 200 cells/mm^3 (Harvey and Sunstrum 1991). Patients typically have a history of animal or soil exposure. The clinical presentation is usually subacute with symptoms developing over weeks. The radiographic manifestations of rhodococcal pneumonia

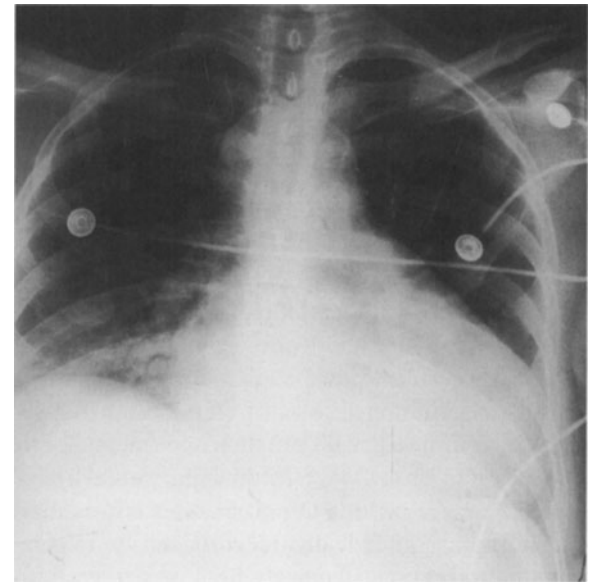


Fig. 7.4. A 39-year-old woman with late-stage AIDS diagnosed with pericarditis due to *Nocardia asteroides*. AP chest radiograph demonstrates a globularly enlarged cardiac silhouette consistent with pericardial effusion

include parenchymal consolidation, usually accompanied by cavitation (Shapiro et al. 1992). Pleural effusions and empyema frequently complicate rhodococcal pneumonia. Lymphadenopathy may be present (Muntaner et al. 1997).

Diagnosis is by identification of the organism in culture from respiratory specimens and, in many cases, blood. This pathogen resembles nonpathogenic diphtheroids and so it may be spuriously dismissed as normal flora. In some cases, calcified phagolysosomes, the pathognomonic finding of malakoplakia, are evident (Kwon and Colby 1994; Supparatpinyo et al. 1994). Prolonged treatment with appropriate antibiotics can be successful.

7.3 Zoonoses

7.3.1 Bacillary Angiomatosis

Bartonella henselae and *Bartonella quintana* are both causes of the clinical syndromes of bacillary angiomatosis and peliosis in HIV-infected patients. Bacillary peliosis also occurs in other immunocompromised patients. *B. henselae* is the organism almost exclusively associated with cat scratch disease and it is capable of causing high-grade bacteremia in cats without causing illness in the animal. *B. quintana* is the agent of trench fever and is transmitted by the human body louse (Koehler et al. 1997).

Bacillary angiomatosis is a neovascular proliferative disorder that was initially described to involve skin and lymph nodes in HIV-infected patients but has since been described to involve visceral organs including the liver, spleen, and lungs. The most common manifestation of bacillary angiomatosis is skin disease – subcutaneous or dermal purplish nodules that superficially resemble Kaposi's sarcoma. *B. quintana* can cause lytic bone lesions.

In the chest, bacillary angiomatosis can have a varied presentation. Violaceous endobronchial lesions can mimic Kaposi's sarcoma on bronchoscopic inspection of the airways. In the lung, parenchymal nodules are the most common manifestation. Additional findings include pleural effusions, lymphadenopathy and chest wall masses. Because of the vascular nature of these lesions, the lymphadenopathy and chest wall masses frequently densely enhance with intravenous contrast administration when imaged by CT (Moore et al. 1995).

Because bacillary angiomatosis is a bacterial infection that responds well to antibiotic therapy, familiarity with its varied manifestations is important in leading to an early diagnosis. Diagnosis by blood culture using special techniques can be accomplished when clinical biopsy specimens cannot be obtained.

7.3.2 *Pasteurella*

Pasteurella multocida is a gram-negative bacterium that is a frequent colonizer of the upper respiratory tract of dogs and cats. Skin and soft tissue infection is the most common manifestation of *Pasteurella* infection and is usually associated with an animal bite or scratch. In patients with normal immunity and underlying lung disease, *Pasteurella* may cause upper and lower respiratory infections often related to a dog or cat bite. In immunocompromised patients, *Pasteurella* can cause pneumonia even in the absence of underlying lung disease (Drabick et al. 1993). The radiographic findings include focal areas of consolidation which may be complicated by cavitation and pleural effusion (Fig. 7.5).

7.3.3 *Bordetella*

Another zoonosis that can occur in AIDS patients is infection with *Bordetella bronchiseptica*, a gram-

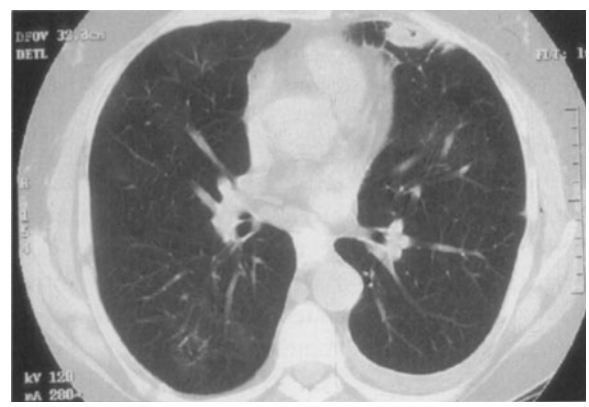


Fig. 7.5. A 50-year-old HIV-infected man, CD4=22/mm^3, with leg edema and ulcers. He had a dog at home who licked his legs. He presented with cough and fever and was diagnosed with *Pasteurella multocida* pneumonia. Chest CT of the mid lungs demonstrates a cavitary ovoid nodule in the inferior lingula and patchy bilateral areas of ground glass opacity. There is a small left pleural effusion

negative coccobacillus. *Bordetella* causes respiratory diseases in dogs, cats, and pigs, but rarely in humans with normal immunity. A broad spectrum of illness has been described in AIDS, including upper and lower respiratory infection and disseminated disease. Generally, the patients have advanced stage AIDS and CD4 cell counts of less than 50 cells/mm^3. A history of contact with sick dogs or cats can sometimes be elicited (Dworkin et al. 1999). Chest radiographic findings include interstitial infiltrates which may be bilateral and focal areas of consolidation (Fig. 7.6). Cavitation has occasionally been described. Diagnosis is accomplished by culture of respiratory specimens.

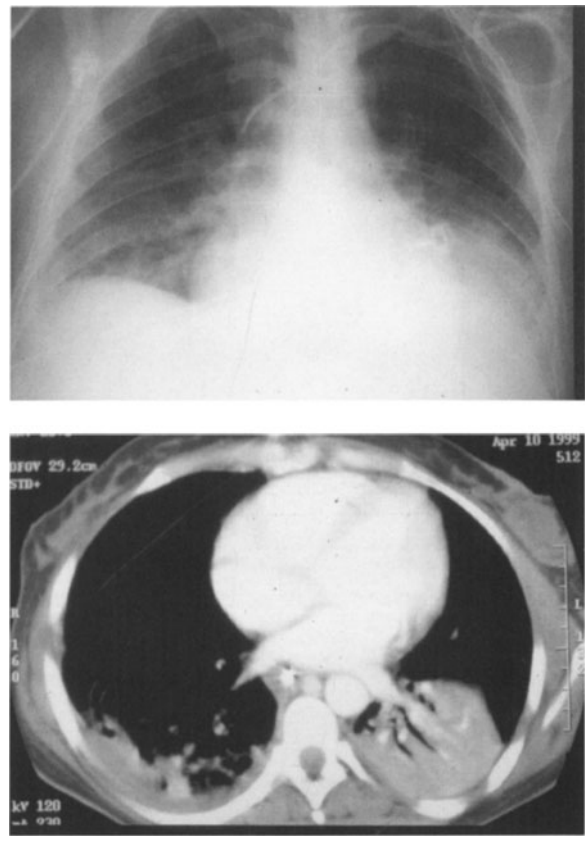


Fig. 7.6a, b. A 36-year-old woman with AIDS and pneumonia due to *Bordetella bronchiseptica*. **a** AP chest radiograph demonstrates dense consolidation of the left lower lobe and patchy opacity in the right lower lobe. **b** Chest CT demonstrates the left greater than right lower lobe lung parenchymal disease. In addition, a small right pleural effusion is evident

7.4 Mycobacterial Infections

7.4.1 Tuberculosis

HIV infection is a potent risk factor for the development of tuberculosis (Barnes et al. 1991) throughout its course (Jones et al. 1993). In fact, development of tuberculosis is considered AIDS defining in an HIV-infected person regardless of the CD4 cell count. Histologically, the inflammatory response to tuberculosis varies with the CD4 cell count, with well-formed granulomata and few bacilli seen in patients with preserved CD4 cell counts in contrast to poorly formed granulomata with abundant organisms in patients with low CD4 cell counts (Di Perri et al. 1996). Similarly, the radiographic appearance of tuberculosis varies with the CD4 cell count (Keiper et al. 1995). In HIV-infected patients with tuberculosis and CD4 cell counts of more than 200 cells/mm^3, the chest radiograph demonstrates the typical pattern of reactivation tuberculosis (Greenberg et al. 1994). Airspace disease and consolidation usually involve the apical and posterior segments of the upper lobes and/or the superior segments of the lower lobes. Cavitation is frequent and lymphadenopathy infrequent. As the CD4 count decreases, the radiographic appearance changes. Lung parenchymal disease is more randomly distributed. Lung nodules may be the dominant parenchymal finding in some cases. Cavitation occurs infrequently. Lymphadenopathy is often present and may be unilateral or bilateral (Haramati et al. 1997). On contrast-enhanced chest CT, tuberculous lymphadenopathy is usually low attenuation and may be peripherally enhancing (Pastores et al. 1993). Disseminated tuberculosis is more common in severely immunocompromised patients (Fig. 7.7). Pleural effusions are slightly more prevalent in AIDS-associated tuberculosis as the CD4 count decreases. More significantly, AIDS patients with low CD4 cell counts typically have a higher mycobacterial organism burden, and therefore their tuberculous effusions are more likely to stain positive for acid-fast organisms and culture positive for *Mycobacterium tuberculosis* (Relkin et al. 1994). Despite the differences in radiographic appearance and increased frequency of disseminated disease as the CD4 cell count decreases, AIDS patients with tuberculosis respond well to antituberculous therapy at all levels of CD4.

Multidrug-resistant tuberculosis can be prevalent in certain populations with AIDS, including noncompliant patients and those from countries where drug-resistant strains are endemic. The radiographic findings are indistinguishable from those of sensitive

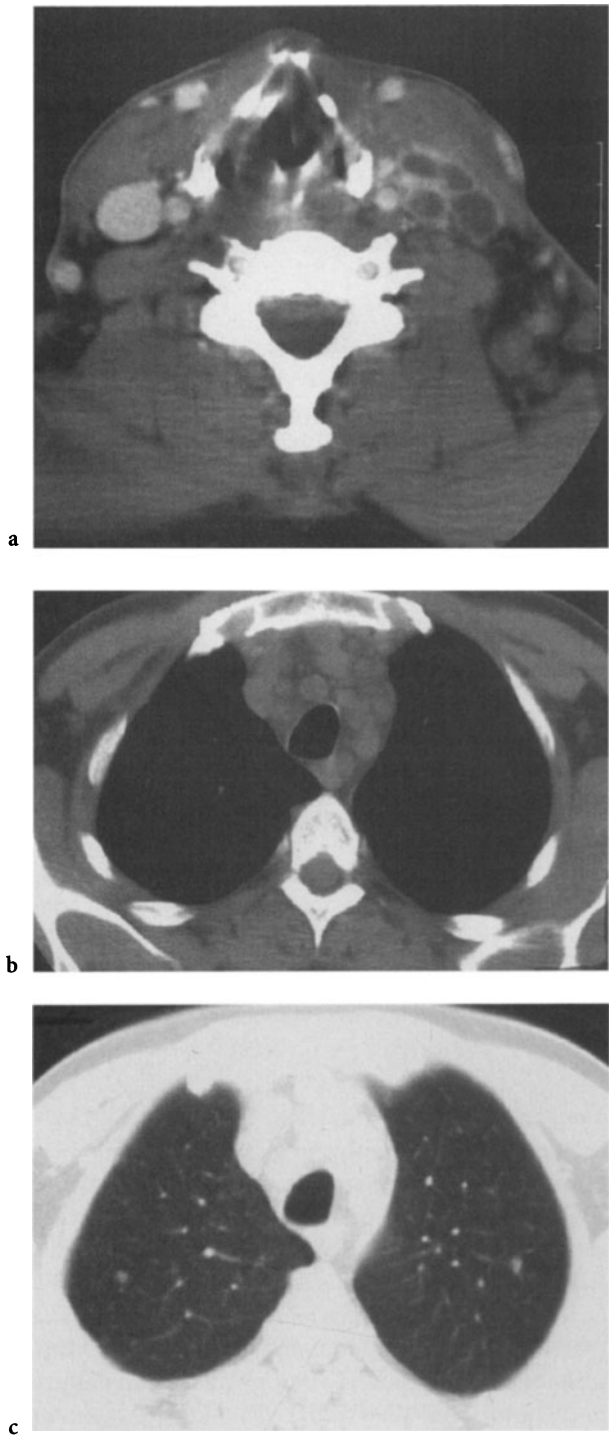


Fig. 7.7a–c. A 43-year-old man with AIDS, CD4=127/mm^3, presenting with fever, cough, and a left neck mass. He was diagnosed with disseminated tuberculosis. The portable chest radiograph was nearly normal. **a** Contrast-enhanced neck CT demonstrates multiple enlarged lymph nodes in the left neck. The lymph nodes are low attenuation with peripheral enhancement. **b** Chest CT without intravenous contrast demonstrates numerous mediastinal lymph nodes bilaterally. **c** Lung windows from the chest CT demonstrate numerous bilateral lung nodules ranging in size from 2 to 5 mm

strains of tuberculosis, but there is continued radiographic progression beyond 2 weeks after initiation of conventional antituberculous therapy (Fig. 7.8).

Some AIDS patients with tuberculosis experience a transient worsening of their radiographic findings and symptomatology related to the initiation of HAART. Such paradoxical reactions had been described in the pre-HIV era in association with the initiation of tuberculosis therapy. In both instances, it is believed that the paradoxical reaction is due to an augmented immune response against mycobacte-

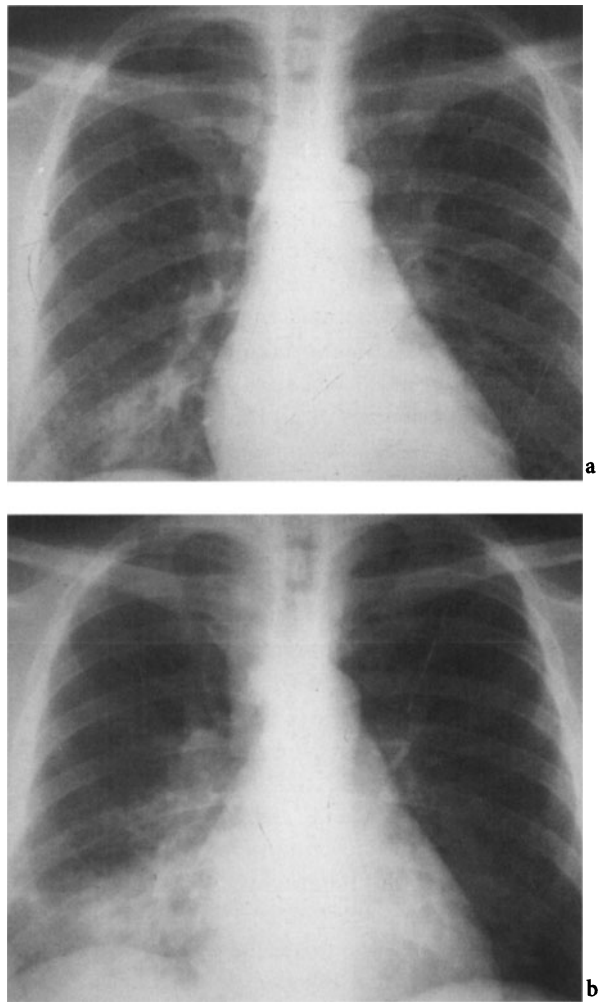


Fig. 7.8a, b. A 30-year-old man with AIDS, fever, and sweats. Acid-fast bacilli were identified in his sputum and standard antituberculous therapy was initiated. The patient's disease continued to progress and he was ultimately diagnosed with multidrug-resistant tuberculosis. **a** PA chest radiograph at the time of clinical presentation demonstrates a patchy infiltrate at the right lung base. **b** Follow-up PA chest radiograph 4 weeks later demonstrates worsening consolidation in the right middle and lower lobes. There is new lymphadenopathy in the right hilum and right paratracheal regions

rial antigens. In the chest, such reactions are manifested as increasing lymphadenopathy, worsening infiltrates, pleural effusion, and the appearance of miliary infiltrates (Fishman et al. 2000). This may be confused with drug-resistant tuberculosis. The reaction is self-limited, although steroids may be required to control symptoms.

7.4.2 *Mycobacterium avium* Complex

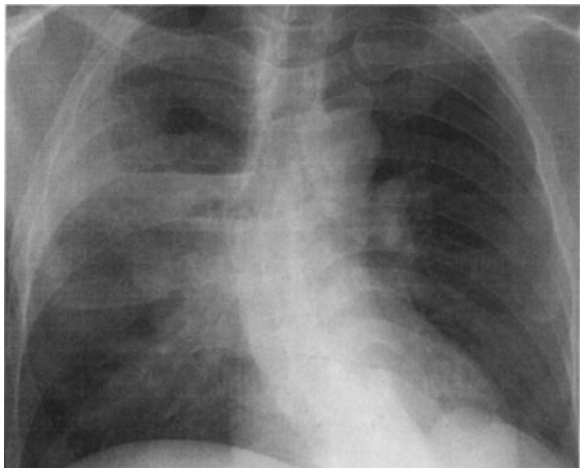
Mycobacterium avium complex (MAC) infection in AIDS patients is often a disseminated disease affecting patients with CD4 counts of less than 50 cells/mm^3. The portal of entry is usually the respiratory or gastrointestinal tract. In disseminated disease the organism can be cultured from blood and bone marrow. Patients usually have subacute constitutional symptoms (Horsburgh 1991). Patients with disseminated MAC will have intrathoracic disease in about 10% of cases. Radiographic findings include lymphadenopathy, parenchymal consolidation, and small or large pulmonary nodules with or without cavitation (Kalayjian et al. 1995) (Fig. 7.9).

MAC can also cause pulmonary disease in AIDS patients who do not have disseminated disease. On average, those patients have less advanced AIDS with CD4 cell counts usually >50 cells/mm^3. The usual radiographic findings include areas of consolidation which may be cavitary (Hocqueloux et al. 1998). However, the majority of patients with MAC in their sputum do not have disseminated MAC and do not have radiographic abnormalities attributable to MAC.

An immune reconstitution reaction akin to the paradoxical reaction described for tuberculosis can occur with MAC, usually resulting in lymphadenitis. Patients with very low CD4 cell counts develop fever and lymphadenitis in the neck, chest (Fig. 7.10), or abdomen soon after the initiation of HAART as their CD4 cell count rises. This is believed to be an augmented inflammatory response to a MAC infection (Race et al. 1998).

7.4.3 Other Mycobacterial Infections

HIV-infected patients are generally at risk for developing a variety of mycobacterial infections. In addition to tuberculosis and MAC, uncommon mycobacterial infections occur with some frequency in AIDS patients. The disease may be localized to the lungs, gastrointestinal tract, or other sites, or may be disseminated (Aronchick and Miller 1993).



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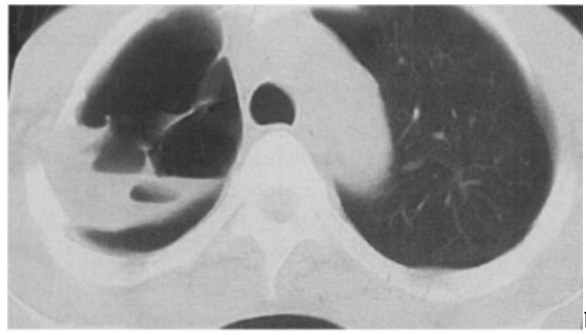

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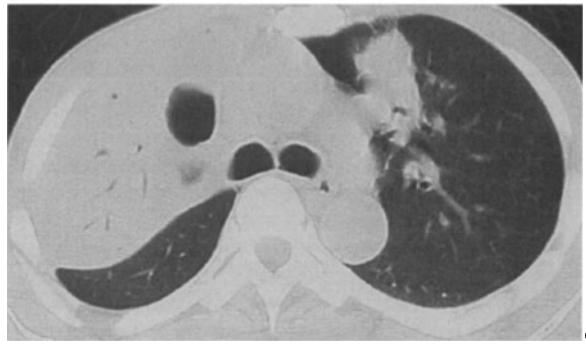

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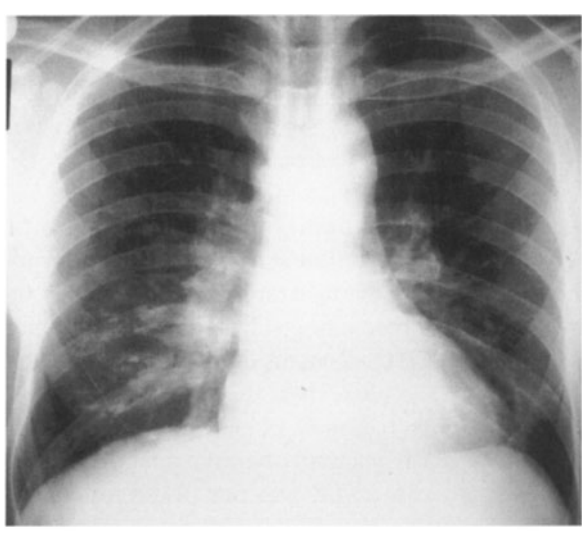
Fig. 7.9a–c. A 52-year-old man with late-stage AIDS, fever, and productive cough. Sputum and needle aspirate yielded the diagnosis of MAC pneumonia. **a** PA chest radiograph demonstrates a large septated cavity with multiple air fluid levels in the right upper lobe. There is adjacent consolidation. **b** Chest CT at the level of the aortic arch demonstrates a septated cavity in the right upper lobe containing multiple air fluid levels. **c** Chest CT at the level of the carina demonstrates consolidation with air bronchograms in the right upper lobe. There is also a cavity medially in the right upper lobe and a focal area of consolidation in the anterior left upper lobe



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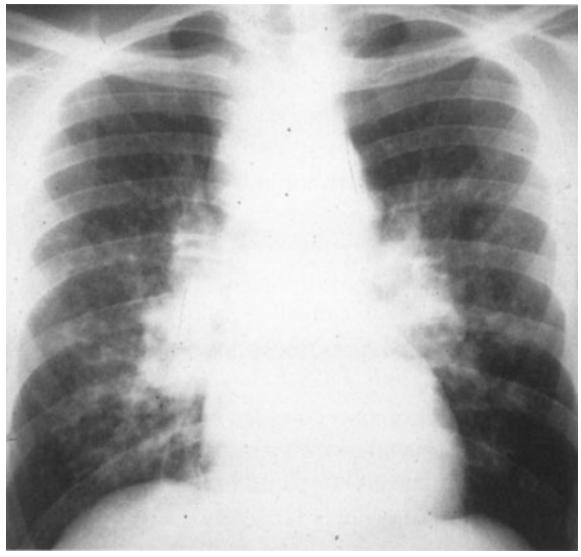

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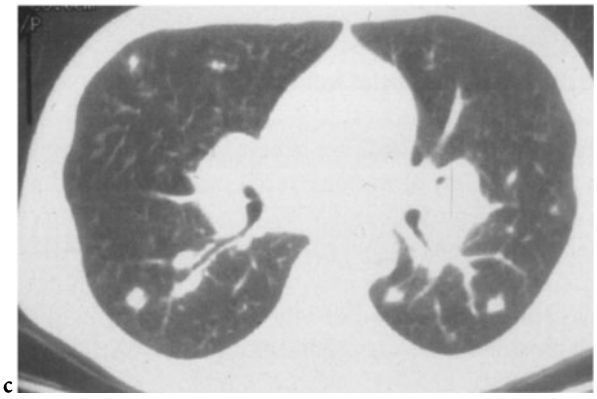

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Fig. 7.10a–c. A 34-year-old man with AIDS diagnosed with pulmonary and disseminated MAC disease. **a** PA chest radiograph at the time of diagnosis when the CD4 was 28/mm^3 demonstrates bilateral hilar and mediastinal lymphadenopathy associated with perihilar nodules. **b** Chest CT at the same time as **a** demonstrates bilateral hilar lymphadenopathy and scattered bilateral lung nodules. **c** The patient was started on HAART, leading to a rise in his CD4 from 28/mm^3 to 221/mm^3 in 5 weeks. His symptoms worsened. Follow-up PA chest radiograph demonstrates marked worsening of the lymphadenopathy and lung parenchymal disease. Further evaluation only demonstrated MAC, with the worsening symptoms and radiographic findings consistent with reconstitution lymphadenitis

Organisms that are described in association with HIV infection include *M. kansasii* (Fishman et al. 1997), *M. gordonae*, *M. fortuitum*, and *M. xenopi*, among others. These infections predominate in patients with very low CD4 cell counts. The radiographic picture overlaps with that of tuberculosis (Laissy et al. 1997) (Fig. 7.11). The growth rate and drug sensitivities vary with the specific mycobacterial organism and must be determined at culture for appropriate therapy.

7.5 *Pneumocystis carinii* Pneumonia

Widespread effective prophylaxis and HAART have markedly reduced the rate of *Pneumocystis carinii* (PCP) infection in AIDS patients. Despite this decline, PCP remains the most common opportunistic pulmonary infection in patients with AIDS (Gatell et al. 1996). The organism is ubiquitous, but cannot be grown in culture, and its taxonomy is controversial. It was originally considered to be a protozoan, but based on genetic analysis is now thought to more closely resemble a fungus (Kuhlman 1996). Presenting symptoms are dyspnea and dry cough. Patients with PCP are usually hypoxic and have an elevated serum lactate dehydrogenase (LDH). The typical radiographic features of PCP are bilateral, often symmetric, perihilar, granular, reticular, or airspace opacities (Fig. 7.12) (Amorosa et al. 1990). Lymphadenopathy and pleural effusions are uncommon radiographic findings in PCP. Lung parenchymal disease may occasionally be focal or nodular, or have an upper lobe predominance. Kerley B lines are uncommon. Air cysts develop in 10%–34% of patients, usually due to pneumatocele formation, and less frequently necrotizing pneumonitis (Sandhu and Goodman 1989). The air cysts tend to develop during the course of active infection and decrease in size or even resolve completely after successful treatment. However, pneumothorax complicates the course of infection in about one-third of patients with PCP and air cysts (Chow et al. 1993).

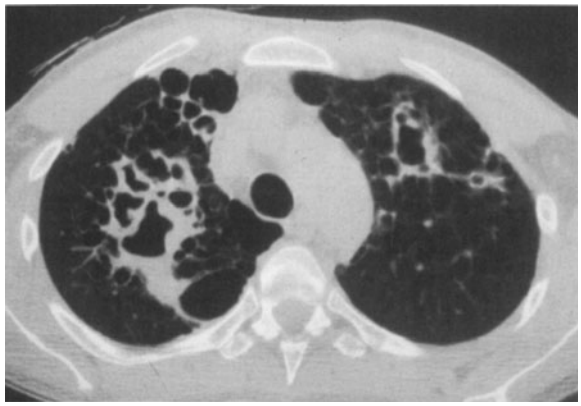


Fig. 7.11. A 45-year-old man with AIDS and *Mycobacterium fortuitum* pneumonia. Chest CT at the level of the aortic arch demonstrates bilateral upper lobe thick-walled cavities. One in the right upper lobe contains a shallow air-fluid level

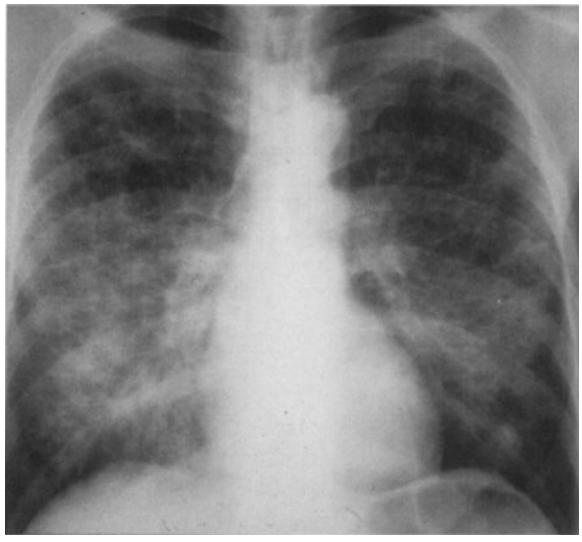


Fig. 7.12. A 41-year-old man with AIDS and *Pneumocystis carinii* pneumonia. AP chest radiograph demonstrates bilateral perihilar hazy granular and reticular opacities. There are bilateral upper lung cysts

The pneumothoraces may be unilateral or less frequently bilateral. On CT, PCP typically appears as ground glass opacity, often with a patchy distribution, although it may be homogeneous (Gruden et al. 1997). Air cysts are more readily detected on CT (Kuhlman et al. 1990).

PCP can, in rare cases, be a disseminated disease. This usually occurs in patients receiving aerosolized pentamidine prophylaxis. The drug has very little systemic absorption and any organ can become infected. Radiographic and CT findings include enlarged lymph nodes, liver, and spleen containing punctate calcifications or areas of low attenuation.

7.6 Fungal Infections

Cellular immunity plays an important role in host defense mechanisms against many fungal infections. Therefore, both endemic (Conces 1999) and opportunistic fungal infections increase in prevalence in AIDS patients with CD4 counts below 100 cells/mm^3 (Connolly et al. 1999). In patients with fungal infections on HAART, a paradoxical worsening of their clinical and radiographic findings may occur as part of the "immune reconstitution syndrome" as the viral load decreases and CD4 cell count increases (see Fig. 7.14).

7.6.1 Endemic Fungal Infections

7.6.1.1 Histoplasmosis

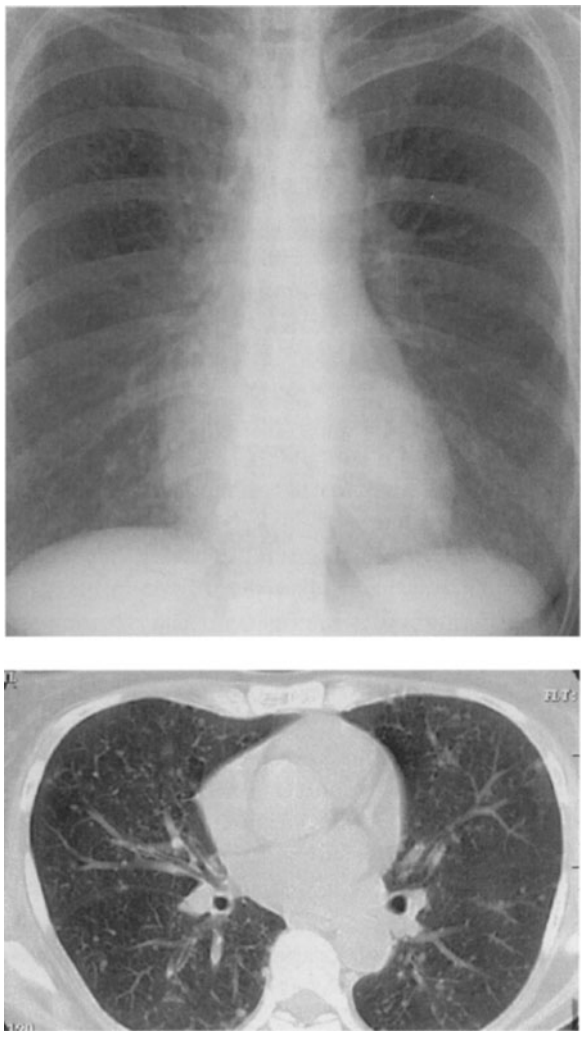
Histoplasmosis is the most common endemic fungal infection in the United States. It is caused by *Histoplasma capsulatum*, a soil-borne fungus which thrives in regions with moderate temperature and humidity. Infection is caused by inhalation of the organism. In the United States, exposure to *Histoplasma* is greatest in the Ohio and Mississippi River valleys. In immunocompetent adults, histoplasmosis is either asymptomatic or causes a mild flu-like illness, a self-limited febrile pulmonary illness. Disseminated histoplasmosis typically occurs in immunocompromised hosts or children less than 1 year old. In patients who live or have lived in endemic areas, disseminated histoplasmosis is a common initial AIDS-defining opportunistic infection occurring at a CD4 count of less than 200 cells/mm^3 (Conces 1999). In New York City, histoplasmosis is seen in AIDS patients from Puerto Rico, the Dominican Republic, and Central America. Clinically, patients present with fever, weight loss, and hematologic abnormalities, and about half develop pulmonary involvement. Elevated LDH often causes diagnostic confusion with PCP. Diagnosis is accomplished by culture or histology. A rapid polysaccharide antigen test is also extremely useful.

The chest radiographic findings in AIDS patients with disseminated histoplasmosis may be normal in about half of the cases. When the chest radiograph is abnormal, the most common finding is diffuse small lung parenchymal nodules (Fig. 7.13). Other parenchymal findings include linear, irregular, and airspace opacities. Pleural effusions and lymphadenopathy are uncommon (Conces et al. 1993).

7.6.1.2 Coccidioidomycosis

Coccidioidomycosis is caused by the dimorphic fungus *Coccidioides immitis*. *C. immitis* is endemic in the soil in locales with arid climate, hot summers, and low rainfall. This includes the desert areas of the southwest United States, northern Mexico, and discrete areas in South and Central America. AIDS patients are at an increased risk for developing disseminated coccidioidomycosis either on the basis of progressive primary infection or on the basis of reactivation of a previously acquired latent infection (Fish et al. 1990).

In immunocompetent hosts, the majority of infections are inapparent or mild, while a minority develop a febrile respiratory illness. Fungemia, diffuse pulmonary involvement, and extrathoracic infections occur in patients with deficient cell-mediated immunity, including AIDS patients who live or have lived in areas endemic for coccidioidomycosis. In endemic areas, coccidioidomycosis is a common initial AIDS-defining opportunistic infection (Singh et al. 1996). The usual symptoms are fever, weight loss, and cough, occurring at a mean CD4 count of 100 cells/mm^3. The lung is commonly involved, in 80% of cases in one large study (Bronnimann et al. 1987). Meninges, skin, and other extrapulmonary sites may be involved, with or without lung involvement. Diagnosis is accomplished by culture, histology, or, in an appropriate clinical setting, serology.



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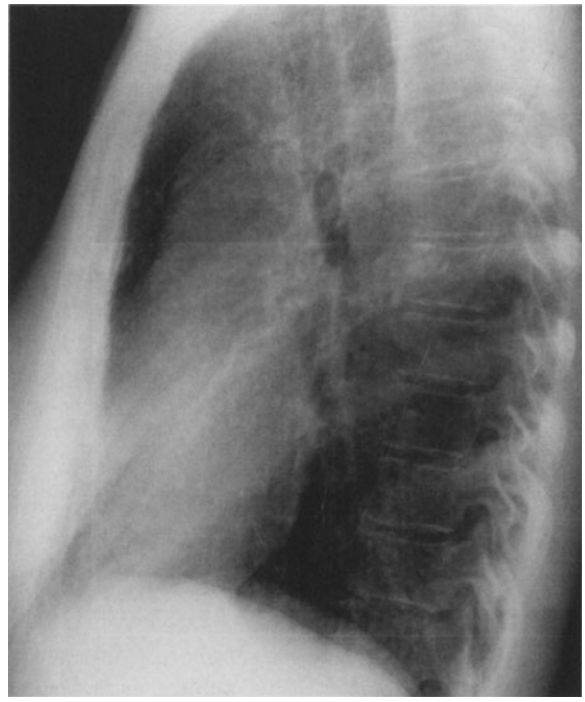


c

Fig. 7.13a–c. A 54-year-old woman with AIDS and disseminated histoplasmosis. **a, b** PA and lateral chest radiographs demonstrate innumerable small bilateral lung nodules. **c** Chest CT through the mid lungs demonstrates innumerable bilateral lung nodules ranging in size from 2 to 5 mm

The most frequent chest radiographic finding in AIDS patients with coccidioidomycosis is diffuse nodular or reticulonodular lung parenchymal opacities. Focal alveolar infiltrate, discrete nodules, hilar lymphadenopathy, pulmonary cavity, and pleural effusion occur. Those patients with diffuse pulmonary disease usually have lower CD4 cell counts and a high mortality.

7.6.1.3 Blastomycosis

Blastomycosis is the least prevalent of the endemic fungal infections in the United States. Its endemic areas are the southeastern United States and the Ohio and Mississippi River Valley regions. Infection with blastomycosis is not common in AIDS patients (Conces 1999). When it occurs, it may be localized to the lung or disseminated. The chest radiographic findings of localized disease include focal airspace opacities or masses. The most common chest radiographic finding in disseminated disease is diffuse nodular opacities. Cavitation, lymphadenopathy, and pleural effusions occur less commonly. Disseminated disease often has a fulminant clinical course.

7.6.1.4 Penicilliosis

Penicillium marneffei is a dimorphic fungus endemic to Southeast Asia and the southern part of China. In some parts of Thailand, penicilliosis is the third most common AIDS-related opportunistic infection after extrapulmonary tuberculosis and cryptococcal meningitis. It occurs commonly as an initial AIDS-defining infection. Usual presenting symptoms are fever, weight loss, anemia, and skin lesions – most commonly generalized papules with central umbilication. In one large series (Supparatpinyo et al. 1994), chest radiographs were abnormal in 30/80 cases. Predominant abnormalities were diffuse reticular infiltrates and localized alveolar infiltrates. Diffuse alveolar infiltrates, localized interstitial infiltrates, and pleural effusion also occurred.

Diagnosis is accomplished by isolation of the organism in culture or visualization on tissue biopsy or touch preps, almost invariably from extrapulmonary sites. Blood, lymph node, bone marrow, and skin lesions have the highest diagnostic yield.

Importantly, HIV-infected patients who have traveled to or resided in an endemic area for *P. marneffei* (Rabaud et al. 1996) present with penicilliosis in a non-endemic area due to reactivation of remotely acquired disease (Jones and See 1992).

7.6.2 Opportunistic Fungal Infections

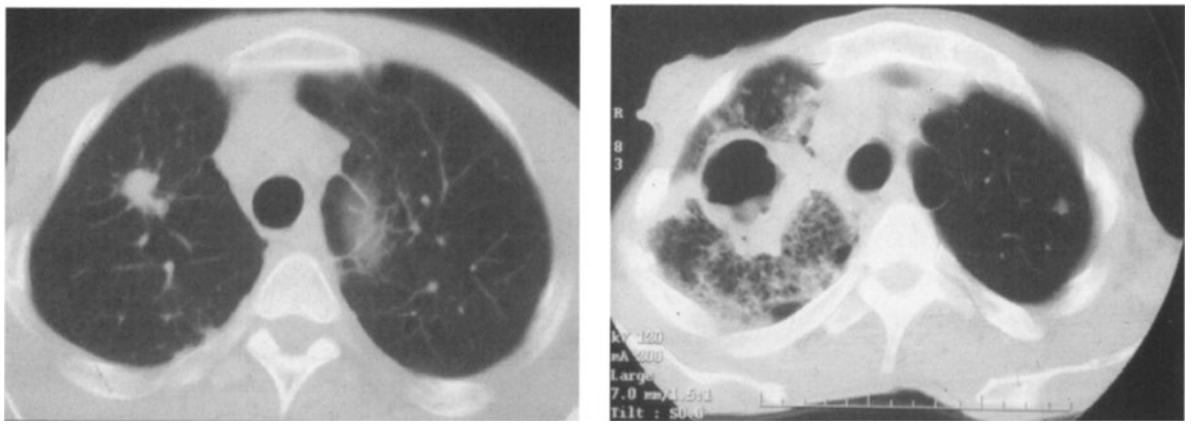
7.6.2.1 Cryptococcosis

Cryptococcosis is caused by the fungus *Cryptococcus neoformans*. The organism is usually found in soil contaminated by pigeon excreta. In immunocompetent patients, *Cryptococcus neoformans* is minimally pathogenic and occasionally will cause localized pulmonary disease. In AIDS patients, although the initial portal of entry of infection is the lungs, the presenting clinical illness is usually cryptococcal meningitis. The reported rate of cryptococcal pulmonary disease in AIDS patients with cryptococcal meningitis varies, but may be as high as 50% and is often a manifestation of disseminated disease (Wasser and Talavera 1987). The pulmonary disease is often clinically inapparent. Chest radiographic findings in AIDS patients with pulmonary cryptococcal disease include single or multiple lung nodules, areas of consolidation with or without cavitation and diffuse reticular opacities (Fig. 7.14). Pleural effusions and lymphadenopathy may be present (Miller et al. 1990). Definitive diagnosis is by biopsy and culture of a pulmonary lesion. A presumptive diagnosis can be made when a compatible lesion is associated with a positive serum cryptococcal antigen or a positive blood culture.

7.6.2.2 Aspergillosis

Aspergillosis, due to soil-borne ubiquitous *Aspergillus* – usually *A. fumigatus*, causes pulmonary disease in patients with long-standing advanced AIDS. Risk factors associated with aspergillosis in AIDS include: corticosteroid use, antecedent broad-spectrum antibiotics, neutropenia, and longstanding depressed CD4 count. Localized pulmonary disease is more frequent than widespread hematogenous dissemination. Aspergillosis in AIDS patients takes three major forms: invasive disease, chronic necrotizing aspergillosis, and tracheobronchial aspergillosis. Infection typically occurs in patients with preexisting cavitary lung disease or neutropenia and occurs less frequently in AIDS patients than in organ transplant patients (Denning et al. 1991).

Invasive pulmonary aspergillosis affects patients with advanced AIDS and is fatal in more than half. Although it is diagnosed in only 1%–2% of AIDS patients, autopsy series show a 5% prevalence of disease. Infection occurs by inhalation and the symptoms are



a b

Fig. 7.14a, b. A 65-year-old man with AIDS and a lung nodule diagnosed as cryptococcal disease. **a** Chest CT through the upper lungs demonstrates an irregularly marginated nodule in the right upper lobe associated with subpleural nodularity. There is a left apical bulla. **b** Chest CT 2 months later after initiation of HAART and marked decrease in viral load demonstrates enlargement and cavitation of the right upper lobe nodule associated with surrounding reticular and small nodular opacities and a new left upper lobe nodule. Biopsy revealed granulomatous inflammation and cryptococcosis

usually nonspecific. Diagnosis of invasive aspergillosis requires tissue for diagnosis because the significance of *Aspergillus* in the sputum is uncertain. Some believe that upper airway colonization is frequent while others have demonstrated that identification of *Aspergillus* in respiratory secretions is highly predictive of active disease. The availability of itraconazole, an oral drug, may improve the outcome since early empiric therapy is practical in cases of suspected aspergillosis.

Radiographic findings in invasive pulmonary aspergillosis in AIDS usually include ill-defined nodules or masses, or areas of consolidation. Cavitation or the "air crescent sign" which is often seen in non-AIDS patients with invasive pulmonary aspergillosis, is less common in AIDS. Pleural effusions and lymphadenopathy are not common (Staples et al. 1995); systemic dissemination occurs in about one-third of cases.

Chronic necrotizing aspergillosis is the most common form of aspergillosis in AIDS patients. It resembles chronic necrotizing aspergillosis in the non-AIDS population. Patients usually have preexisting upper lobe cavitary disease from *Pneumocystis carinii* pneumonia or from mycobacterial infection. They often develop intracavitary masses ("air crescent sign") which may be mobile (Fig. 7.15). Although cavitary aspergillosis may mimic tuberculosis, cavitary tuberculosis is unusual in markedly immunocompromised patients. Compared to non-AIDS patients, rapid local progression of the cavity is more common. The main complication of chronic necrotizing aspergillosis is hemoptysis, which occurs in almost half the patients and is fatal in 50% of those (Miller et al. 1994). Systemic dissemination complicates chronic necrotizing aspergillosis in 10%–20% of cases.

Tracheobronchial aspergillosis is uncommon but was initially described in patients with AIDS and takes two forms: obstructing bronchial aspergillosis, which has a more indolent course and presents with cough, fever, and wheezing, and pseudomembranous necrotizing aspergillosis, which is similar but which is associated with tissue invasion. The chest radiograph in patients with tracheobronchial aspergillosis may be normal or only subtly abnormal. Mucoid impaction in lower lobe airways and patchy parenchymal opacities are the main findings.

7.6.3 Miscellaneous Fungal Infections

Candida species are mucosal pathogens which cause thrush and esophagitis but rarely cause invasive disease. Unusual opportunistic fungal infections on occasion cause pulmonary disease in severely immunocompromised AIDS patients. *Torulopsis glabrata* is one such species. Chest radiographic findings include areas of consolidation or lung masses which may be cavitary and may be accompanied by lymphadenopathy.

7.7 Cytomegalovirus Infection

Cytomegalovirus (CMV) is the most common viral pathogen in AIDS patients. It is present in 80% of AIDS patients on autopsy series. CMV disease is usu-

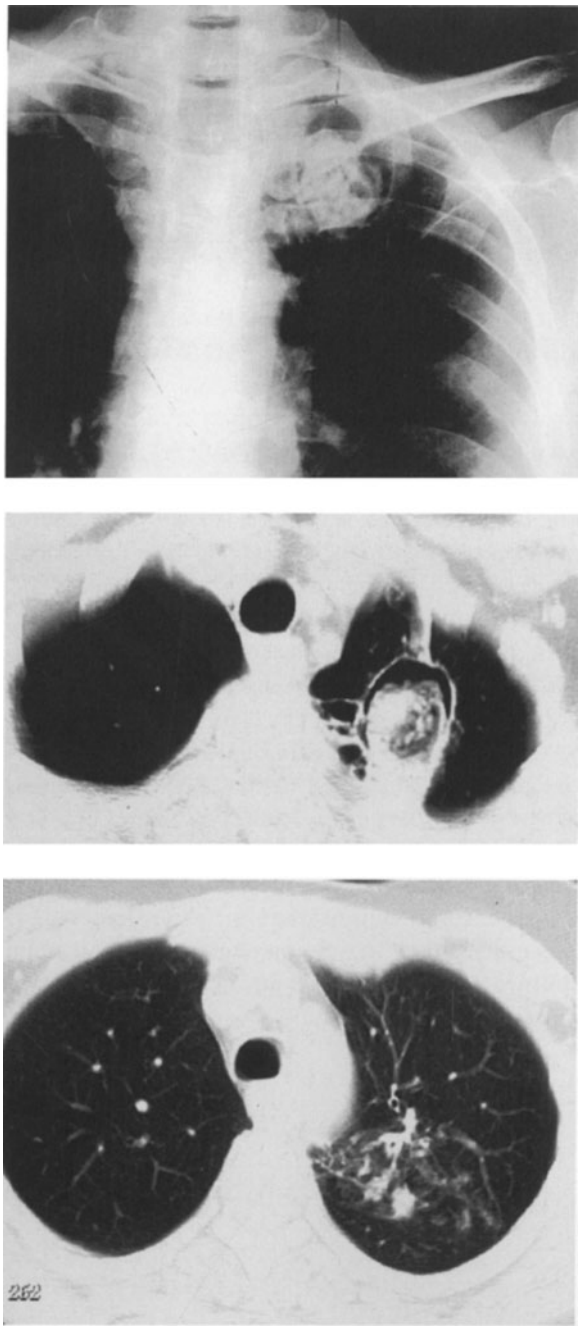


Fig. 7.15a–c. A 38-year-old woman with late-stage AIDS and dementia presenting with hemoptysis. She was diagnosed with chronic necrotizing aspergillosis. **a** Cone-down view from an AP chest radiograph demonstrates a sponge-like mass within a left apical cavity. **b** Chest CT at the apices demonstrates a large dependent sponge-like mass within a left apical cavity associated with an "air crescent" sign. There are band-like linear opacities extending from the cavity to the pleural surface anteriorly, medially, and posteriorly. **c** Chest CT 3 cm lower than in **b** demonstrates irregularly marginated nodules below the inferior aspect of the cavity

ally recognized as retinitis or enteritis, with pulmonary disease diagnosed less commonly. CMV pneumonia is usually a manifestation of disseminated disease occurring in AIDS patients with CD4 cell counts of less than 50 cells/mm^3. Symptoms are nonspecific and include cough, fever, and dyspnea. The diagnosis relies on identification of cytomegalic cells with intranuclear and intracytoplasmic inclusions in biopsy specimens. CMV pneumonia is preceded by the diagnosis of extrapulmonary CMV disease in two-thirds of AIDS patients (Waxman et al. 1997). Radiographic findings of CMV pneumonia are often subtle and include reticular or reticulonodular opacities and peribronchial thickening. Widespread granular opacities, areas of consolidation, and lung nodules or masses are more conspicuous radiographic findings of CMV pneumonia (Fig. 7.16). CT findings include bronchiectasis and a "tree in bud" pattern of branching small centrilobular nodules as well as areas of ground glass attenuation, reticular opacities, lung nodules, and masses (McGuinness et al. 1994).

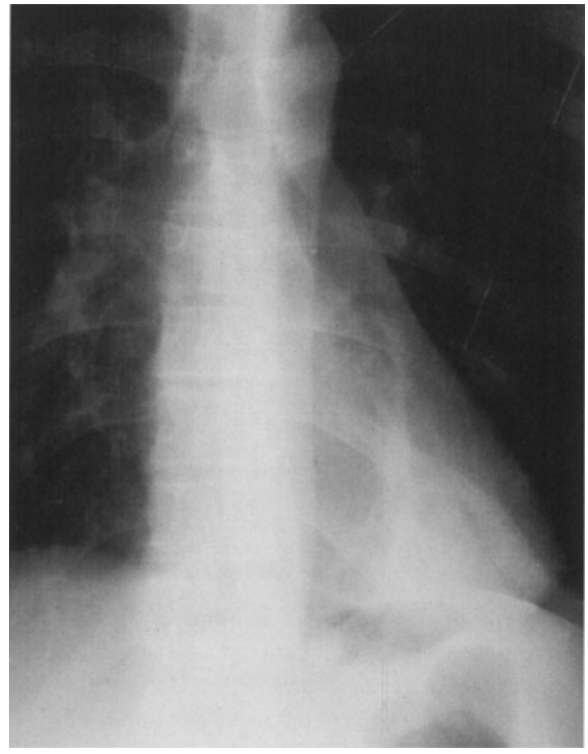


Fig. 7.16. A 31-year-old man with AIDS and persistent left lower lobe consolidation. Biopsy of the left lower lobe was diagnostic of CMV pneumonia

7.8 Parasitic Diseases

7.8.1 Toxoplasmosis

Toxoplasmosis is caused by the intracellular protozoan *Toxoplasma gondii*. Cats are the definitive hosts. People become infected with toxoplasmosis by ingesting either inadequately cooked meat containing tissue cysts or sporulated oocytes shed in cat feces. Prior exposure to toxoplasmosis is evident in up to 90% of Europeans, but is less common in the United States. This variation in prevalence is probably related to differences in diet and the presence of cat waste in the environment. AIDS patients who develop toxoplasmosis usually have reactivation of a previously acquired infection as evidenced by prior seropositivity for *Toxoplasma* IgG. The central nervous system is by far the most frequently affected organ system, but the lung is second in frequency. *Toxoplasma* pneumonia may occur in isolation or concomitant with disease in the central nervous system. The clinical signs are nonspecific, although the patients are usually acutely ill with fever, cough with rales, and a CD4 cell count of less than 200 cells/mm^3. Elevated LDH is common, causing diagnostic confusion with PCP. Diagnosis is made by identifying the organism in fluid obtained by bronchoalveolar lavage or in tissue (Rabaud et al. 1996).

Chest radiographic findings in *Toxoplasma* pneumonia consist predominantly of bilateral lung parenchymal disease. Coarse nodular opacities and reticulonodular opacities are most common. Confluent consolidation may also be seen. Pleural effusion is occasionally present. Lymphadenopathy is not typical (Goodman and Schnapp 1992).

7.8.2 Strongyloidiasis

Strongyloides stercoralis is a roundworm which is endemic in tropical and subtropical regions. In the United States, *Strongyloides* is prevalent in the Appalachian region. Humans are the principal hosts. Infection occurs percutaneously by infectious filariform larvae due to contact with fecally contaminated soil or due to unhygienic conditions resulting in other exposure to feces. Dissemination of the filariform larvae from the skin to the lungs occurs first and may be accompanied by a Löffler syndrome of patchy pneumonitis, respiratory symptoms, and eosinophilia. Expectorated larvae are ultimately swallowed so that the adult worms may develop in the upper small intestine, where they persist for many years. The adult worms usually produce noninfectious rhabditiform larvae which pass out of the stool. *Strongyloides* infection may be asymptomatic or may produce symptoms related to the skin, the lungs, or, most commonly, the gastrointestinal tract (Genta 1989).

In the hyperinfection syndrome, the rhabditiform larvae transform into infectious filariform larvae which penetrate the intestinal wall and widely disseminate in the body, often tracking enteric bacteria into the bloodstream, with resultant bacteremia and meningitis (Igra-Siegman et al. 1981). The hyperinfection syndrome is seen in immunocompromised patients who live or have lived in areas endemic for *Strongyloides* (Guerin et al. 1995).

Chest radiographic findings in pulmonary strongyloidiasis vary with the severity of infection. Chest radiographs may be normal. More commonly, bilateral miliary nodules or reticular interstitial opacities are seen (Fig. 7.17). Patchy alveolar opacities and lobar consolidation may also be present. In patients with hyperinfection syndrome, bilateral alveolar opacities and even adult respiratory distress syndrome (ARDS) may develop. Diffuse involvement may be mistaken for PCP. Diagnosis is accomplished by wet mount or Papanicolaou stain, sputum examination, or bronchoalveolar lavage (Nomura and Rekrut 1996).

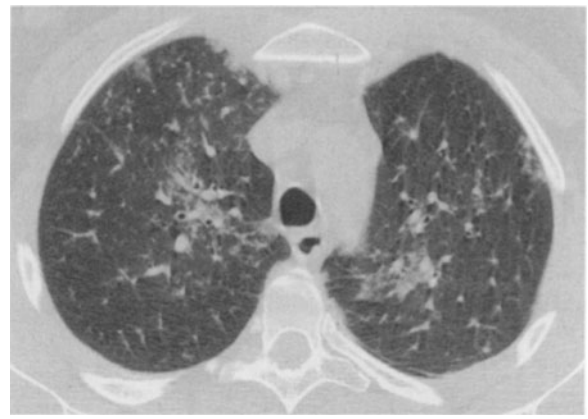


Fig. 7.17. A 42-year-old woman with AIDS presenting with "asthma" and diarrhea. *Strongyloides stercoralis* was present in sputum and stool. Chest CT at the level of the aortic arch demonstrates small nodules and patchy areas of ground glass opacity bilaterally

7.9 Sarcoidosis

Sarcoidosis rarely develops in HIV-infected patients. Case reports from the last decade have described only a few cases of new-onset sarcoidosis in HIV-infected patients, usually in patients with relatively preserved immune systems. Recently, we have observed a number of cases of newly diagnosed sarcoidosis in HIV-infected patients, with the sarcoidosis developing after initiation of HAART and coincident with partial immune reconstitution. Several additional cases have been described in the recent literature (Naccache et al. 1999; Mirmirani et al. 1999). Most of these patients had radiographic findings typical for sarcoidosis, including hilar and mediastinal lymphadenopathy and pulmonary nodules (Fig. 7.18). These cases suggest that sarcoidosis may fall into the spectrum of immune reconstitution phenomena in AIDS patients on HAART.

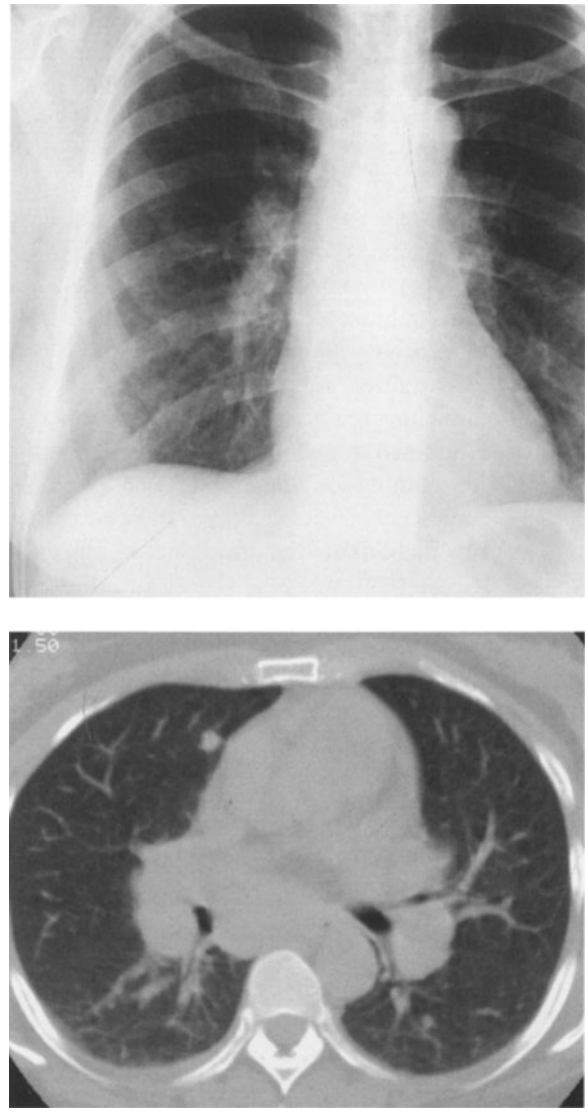


Fig. 7.18a, b. A 43-year-old woman with AIDS and a CD4 count of less than 50/mm^3 was begun on HAART. This led to a rise in the CD4 count and a decreased viral load. However, she developed new lymphadenopathy and lung nodules. On biopsy there were noncaseating granulomas. All cultures and special stains were unrevealing and she was diagnosed with sarcoidosis. **a** PA chest radiograph demonstrates mild bilateral hilar and mediastinal lymphadenopathy which had not been present on a chest radiograph 3 months earlier. There is also a poorly defined right lower lobe nodule. **b** Chest CT 1 month later demonstrates a 6-mm nodule in the anterior right upper lobe. There is also extensive bilateral hilar and subcarinal lymphadenopathy that had progressed from the chest radiograph obtained 1 month earlier

7.10 Malignancies and Associated Conditions

Proliferation of oncogenic viruses and diminished immune surveillance potentially explain the observations that HIV-infected patients have a higher than expected incidence of several malignancies (Broder and Karp 1992). Kaposi's sarcoma was the disease that initially prompted recognition of the AIDS epidemic in 1981. Shortly thereafter, AIDS-related lymphoma was recognized. Cervical carcinoma, anorectal carcinoma (usually in gay men), and bronchogenic carcinoma have also been linked to HIV infection. Each of these malignancies can have intrathoracic manifestations (White 1996). Cohort studies have revealed declining rates of AIDS-associated malignancies coincident with the widespread use of HAART (Jones et al. 1999).

7.10.1 Kaposi's Sarcoma

Human herpesvirus 8 (HHV-8) has been recently recognized as the sexually transmitted etiology of Kaposi's sarcoma in both classical, endemic and AIDS-related Kaposi's sarcoma. In the United States, HHV-8 and consequently Kaposi's sarcoma occur predominantly in the male homosexual population and only occasionally in women (Kedes et al. 1997; Haramati and Wong 2000). In Africa, HHV-8 is endemic and Kaposi's sarcoma occurs in both men and women (Martin et al. 1998).

Kaposi's sarcoma presents either as indolent cutaneous disease characterized by typical violaceous

skin lesions or as aggressive visceral disease involving the skin, mucous membranes, gastrointestinal tract, and upper and lower respiratory tract. Indolent cutaneous disease has a minimal effect on survival while visceral disease is much more aggressive and is often fatal. Both may respond favorably to HAART. Patients with intrathoracic Kaposi's sarcoma typically have concomitant cutaneous disease (Cadranel and Mayaud 1995). Generally, intrathoracic disease progresses in a sequential fashion, with radiographic extent correlating with bronchoscopic extent of disease.

Early chest radiographic findings include coarsening of bronchovascular bundles and peribronchial cuffing. Ill-defined nodules occur later, often with a perihilar predominance. The nodules may be small, large, or coalescent (Davis et al. 1987). Kerley B lines and pleural effusions are manifestations of more advanced disease (Gruden et al. 1995). Intrathoracic lymphadenopathy is not a prominent feature of Kaposi's sarcoma. On chest CT, nodules and masses are the most common feature of intrathoracic Kaposi's sarcoma. They are usually distributed along the bronchovascular bundles and are often ill-defined (Traill et al. 1996; Khalil et al. 1995) (Fig. 7.19). Pleural effusions and thickened interlobular septa are a manifestation of more advanced disease.

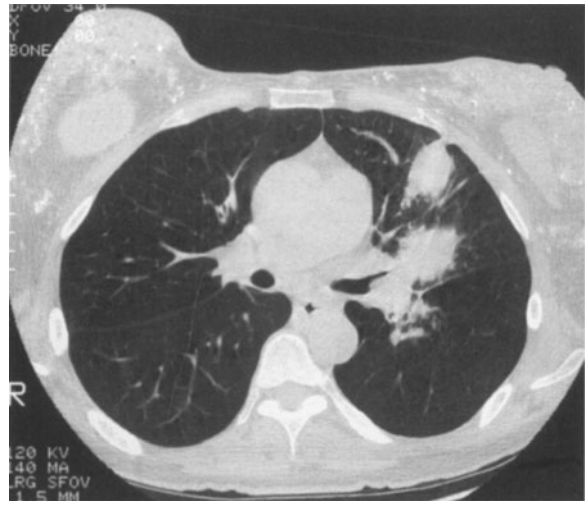


Fig. 7.19. A 52-year-old man with a history of smoking and increasing dyspnea. His HIV risk factor was homosexual contact. Transbronchial biopsy diagnosed Kaposi's sarcoma. Chest CT through the mid lungs demonstrates irregular marginated masses and nodules in the left upper and lower lobes. Note the presence of breast implants and free silicone in both breasts

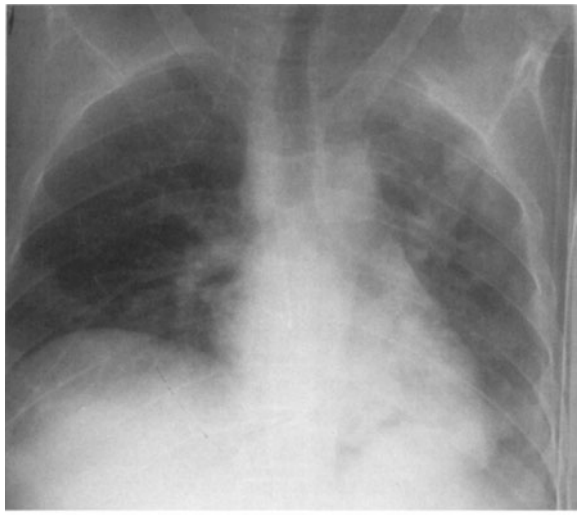
7.10.2 Lymphoma

Lymphoma is a neoplasm that occurs with increased prevalence in HIV-infected patients and is a cause of significant morbidity and morality. AIDS-related lymphoma is frequently a high-grade B cell non-Hodgkin's lymphoma. However, HIV-infected patients are also at increased risk for developing T cell lymphomas, Hodgkin's disease, and polyclonal lymphoma (Broder and Karp 1992). While the Epstein-Barr virus genome has been identified in most AIDS-related primary brain lymphomas, it is less consistently identified in other AIDS-related lymphomas. A rare type of lymphoma called body cavity lymphoma is associated with infection with HHV-8 (Cesarman et al. 1995). The incidence of non-central nervous system AIDS-related lymphoma has shown a smaller decline with HAART compared with opportunistic infections, Kaposi's sarcoma, and primary brain lymphoma (Grulich 1999).

Intrathoracic disease occurs in 10%–50% of patients with AIDS-related lymphoma, depending on the population studied. Extranodal disease predominates in the chest (Bazot et al. 1999). The most frequent intrathoracic manifestations of AIDS-related lymphoma are lung parenchymal consolidation, masses, and nodules, which may progress rapidly and may be cavitary (Sider et al. 1989). Pleural effusions or masses can be seen in two-thirds of cases (Eisner et al. 1996) (Fig. 7.20). Although the disease is predominantly extranodal, lymphadenopathy is present in slightly more than half of cases. Body cavity lymphoma in the chest is manifested as pleural and/or pericardial effusion without associated lymphadenopathy or parenchymal abnormality and with slight thickening of the parietal surface (Morassut et al. 1997).

7.10.3 Lymphocytic Interstitial Pneumonia

Lymphocytic interstitial pneumonia (LIP) was described early in the AIDS epidemic in children. It occurs occasionally in adults with AIDS. It is characterized by a polyclonal proliferation of lymphocytes mixed with plasma cells and histiocytes. It is felt to be an immune response to infection with HIV or perhaps the Epstein-Barr virus, in some cases. AIDS-related LIP is not premalignant. The radiographic findings in LIP include reticular and nodular interstitial opacities (Fig. 7.21). Rarely, alveolar opacities may be seen (Oldham et al. 1989). On CT, small nodules are the dominant finding, often with a pre-



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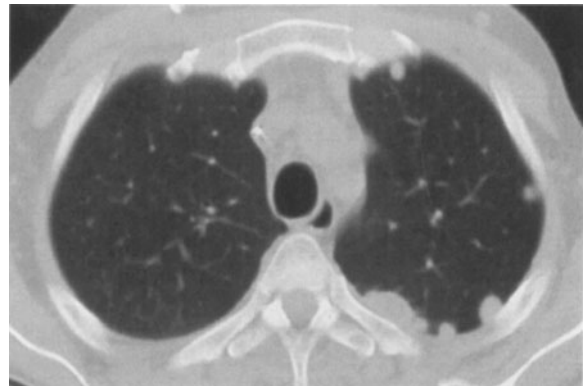

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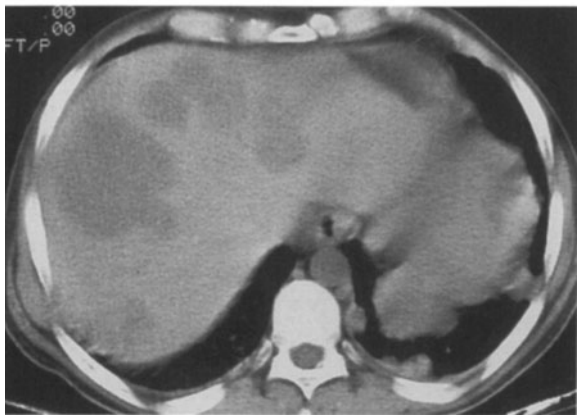
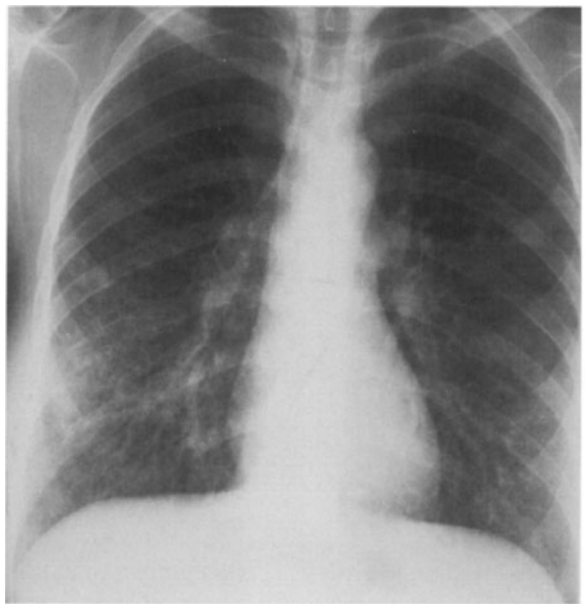


c

Fig. 7.20a–c. A 41-year-old man with AIDS-related lymphoma. **a** AP chest radiograph demonstrates multiple bilateral nodules, more numerous on the left. **b** Chest CT just above the level of the aortic arch demonstrates multiple left-sided pleural nodules. **c** CT at the level of the diaphragm demonstrates left-sided pleural nodules and masses as well as multiple liver masses



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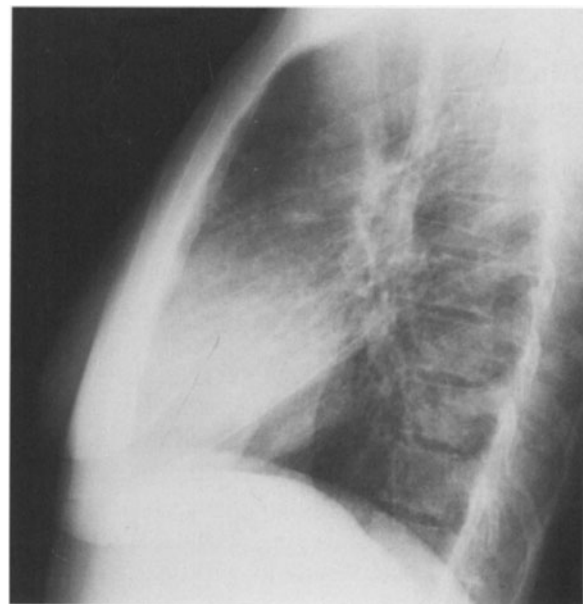

b

Fig. 7.21a, b. A 25-year-old woman with AIDS, CD4=85/mm^3, who complained of dyspnea on exertion. Transbronchial biopsy was diagnostic of lymphocytic interstitial pneumonitis. PA and lateral chest radiographs demonstrate bilateral reticulonodular interstitial opacities

dominance in the peribronchovascular distribution. Areas of ground glass opacity may also be present.

7.10.4 Emphysema

In the late 1980s, precocious emphysema and bullous lung damage associated with advanced immunosuppression and the prior occurrence of pulmonary infections, especially PCP, was described (Kuhlman et al. 1989). More recent studies, however, have identified a substantial occurrence of precocious emphysema with high-resolution CT among HIV-infected patients without prior pulmonary infections and without advanced HIV infection (Diaz et al. 2000). The occurrence of precocious emphysema is greatest in those who have smoked the most. Impaired pulmonary diffusion has been correlated with HIV immunosuppression in a variety of studies. Structurally, the impaired diffusion was found to be related to the presence of emphysema rather than to interstitial disease on high-resolution CT (Diaz et al. 1999). HIV-infected smokers were disproportionately affected compared to HIV-negative smokers and HIV-infected nonsmokers. While it is often emphasized that PCP may present with a normal chest radiograph, it is especially important to consider chronic lung disease as a potential etiology of dyspnea and hypoxia in an afebrile HIV-infected smoker with a relatively preserved CD4 count.

7.10.5 Lung Cancer

There has been some debate about whether or not lung cancer occurs with increased frequency in patients with AIDS (Chan et al. 1993). The preponderance of evidence does point to such an increase (Parker et al. 1998). Because lung cancer is most often caused by exposure to cigarette smoking and not by an oncogenic virus, the reason for its increased prevalence in HIV infection is unclear. Some authors speculate that the diminished immune surveillance associated with HIV infection increases the risk of all malignancies. Others suggest that because intravenous drug use is associated with smoking, AIDS patients with that risk factor have high rates of lung cancer.

HIV-infected patients with lung cancer generally present with late-stage, unresectable disease. The dominant cancer cell type is adenocarcinoma. The radiographic appearance of lung cancer in AIDS patients is usually a peripheral or central nodule or mass, similar to the radiographic appearance of lung cancer in the general population (Fig. 7.22). The second most common radiographic appearance is a pleural effusion; in these cases chest CT will often reveal an underlying lung mass that has extended to the pleura at the time of presentation (White et al. 1995).

7.10.6 Cervical Carcinoma and Anal Carcinoma

Cervical carcinoma and anal carcinoma are caused by infection with specific subtypes of the human papilloma virus (HPV) in virtually all cases (Broder and Karp 1992). Women and homosexual men with AIDS often acquire HIV infection by sexual contact, which is a common route of infection with HPV. Infection with HIV also seems to increase the risk that a woman infected with HPV will develop cervical dysplasia or carcinoma. In some series, cervical dysplasia will be present in up to 50% of HIV-infected women. When cervical carcinoma develops it will often metastasize widely to many sites, including the chest.

Radiologic findings in metastatic cervical and anal carcinoma to the chest include small or large lung nodules, lymphangitic disease, and lymphadenopathy (Fig. 7.23). On contrast-enhanced CT, the lymphadenopathy may be low attenuation and peripherally enhancing. This appearance can also be seen in tuberculosis.

7.11 Cardiovascular Diseases

HIV infection can have a direct effect on the cardiovascular system. It is specifically implicated in AIDS-related cardiomyopathy and pulmonary hypertension. Pericardial effusions in AIDS patients can be due to a variety of etiologies.

7.11.1 Cardiomyopathy

Dilated cardiomyopathy usually occurs as a complication of HIV infection at a moderate stage of disease. In a large prospective clinical and echocardiographic study of asymptomatic patients (CD4 >400/mm^3), cardiomyopathy developed in 8% over 5 years, all

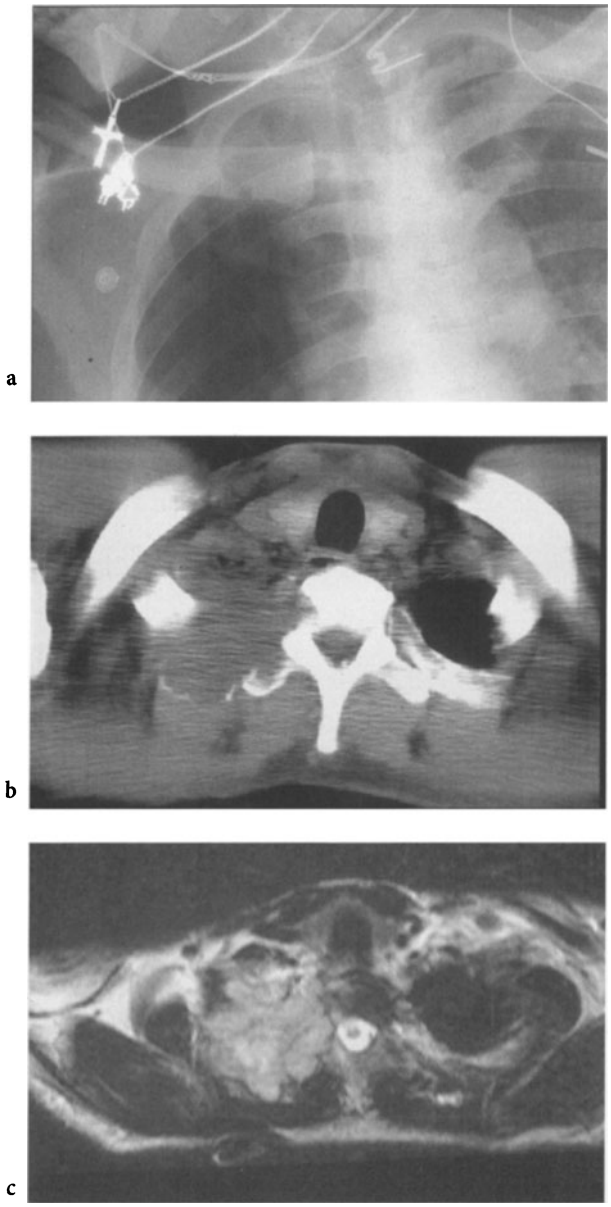

Fig. 7.22a–c. A 47-year-old HIV-infected man presented with right-sided back pain. He was diagnosed with poorly differentiated non-small cell lung cancer. **a** Cone-down view from an AP chest radiograph demonstrates a right apical opacity associated with destruction of the right second posterior rib. **b** Chest CT at the level of the thoracic inlet demonstrates the right apical mass extending through the posterior chest wall with destruction of the second posterior rib and right T2 transverse process. **c** T2-weighted magnetic resonance image of the chest shows the right apical mass to invade the adjacent posterior chest wall musculature

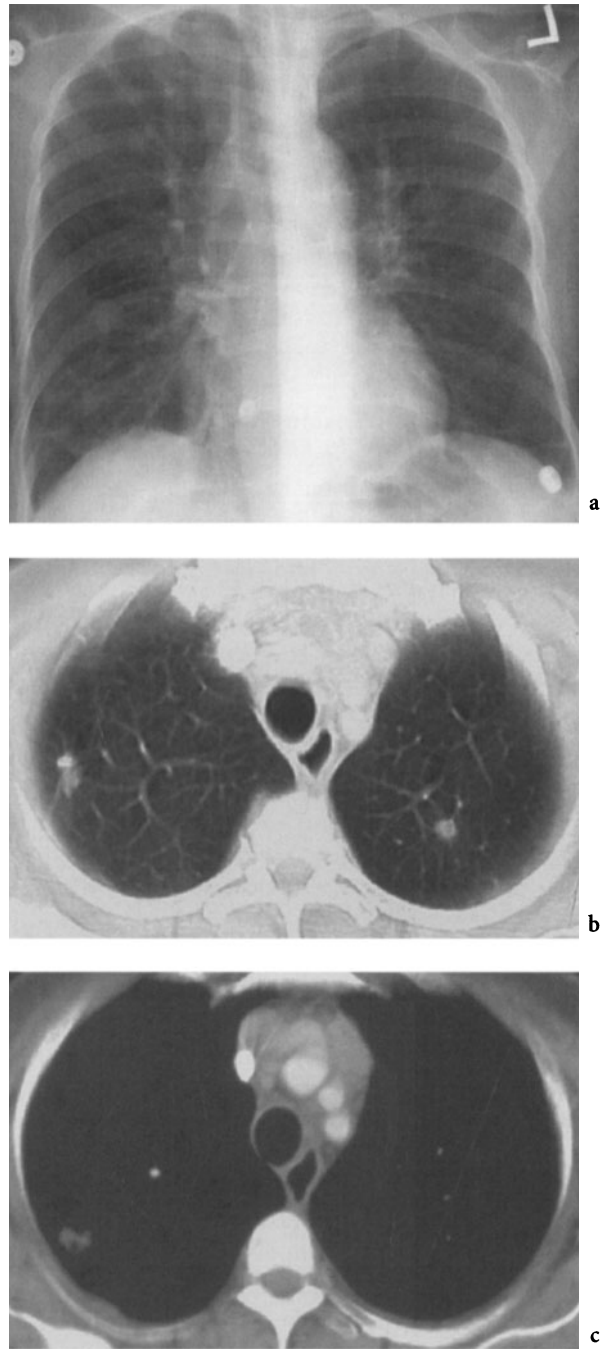

Fig. 7.23a–c. A 56-year-old HIV-infected woman with metastatic cervical carcinoma. **a** PA chest radiograph demonstrates bilateral lung nodules. **b** CT through the upper lungs demonstrates bilateral nodules. **c** Contrast-enhanced CT just above the level of the aortic arch demonstrates anterior mediastinal lymphadenopathy

with CD4 >200/mm^3 (Barbaro et al. 1998). It occurs with increasing frequency as the CD4 cell count diminishes. On histologic analysis, myocarditis is usually present (Anderson et al. 1988). HIV nucleic acid can be found in the myocardium of up to 76% of patients with HIV-associated cardiomyopathy. Other cardiotropic viruses, including Coxsackie virus group B, cytomegalovirus, and Epstein-Barr virus, are occasionally present as well. The patients clinically usually have symptomatic congestive heart fail-

ure. On echocardiography, these patients have four-chamber enlargement associated with left and right ventricular hypokinesia. The chest radiographic findings are those of dilated cardiomyopathy with an enlarged cardiac silhouette, with or without pulmonary venous congestion and edema (Corboy et al. 1987). Abrupt cardiac decompensation with cardiomegaly may be seen in patients with a rapid drop in hematocrit, usually due to zidovudine.

7.11.2 Pulmonary Hypertension

Pulmonary hypertension occasionally complicates the course of HIV infection. In some patients, the pulmonary hypertension is secondary to interstitial fibrosis which may complicate PCP. In patients who use intravenous drugs, pulmonary hypertension may occur as a consequence of foreign body granulomatous vasculitis. However, in some HIV-infected patients, pulmonary hypertension develops in the absence of other predisposing conditions. In this group, infection with HIV itself is the presumed cause of pulmonary hypertension. HIV-associated pulmonary hypertension can occur at any CD4 cell count (Opravil et al. 1997). On chest radiography, the central pulmonary arteries are enlarged and markedly decreased in caliber peripherally (Fig. 7.24). Right ventricular enlargement may be present.

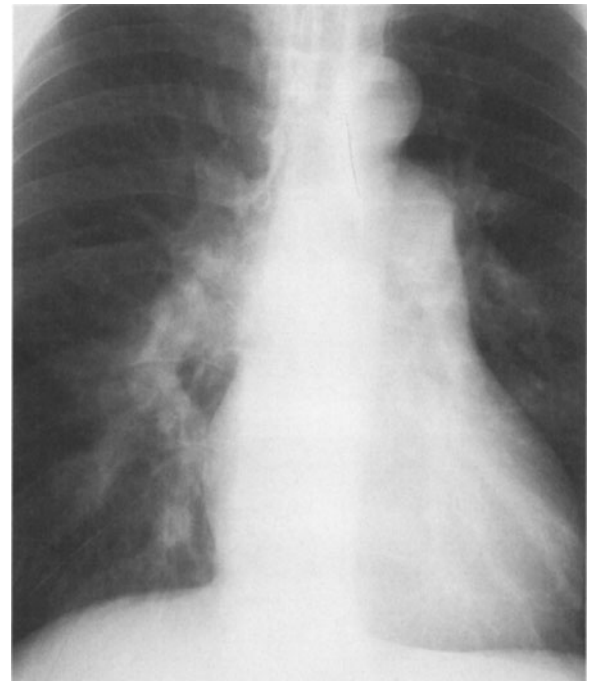

Fig. 7.24. A 48-year-old HIV-infected man with pulmonary hypertension. His pulmonary arterial pressure was 90 mmHg. PA chest radiograph demonstrates markedly enlarged central pulmonary arteries consistent with pulmonary arterial hypertension

7.11.3 Pericardial Effusion

Pericardial effusions occur in 10%–15% of HIV-infected patients (Silva-Cardoso et al. 1999). Most are clinically unimportant and are identified incidentally on echocardiography or CT. In 5%–10% of cases, they are hemodynamically significant with diastolic compression of the right atrium seen on echocardiography. Some present with pericardial tamponade (Reynolds et al. 1992). The effusions are often idiopathic or related to viral infection or congestive heart failure. However, 30% of patients with tuberculosis (Fig. 7.25) and 40% of those with Kaposi's sarcoma had moderate to severe pericardial effusions in one series. Purulent bacterial pericarditis, fungal pericarditis, and involvement with lymphoma and metastatic carcinoma occasionally will cause AIDS-associated pericardial effusion.

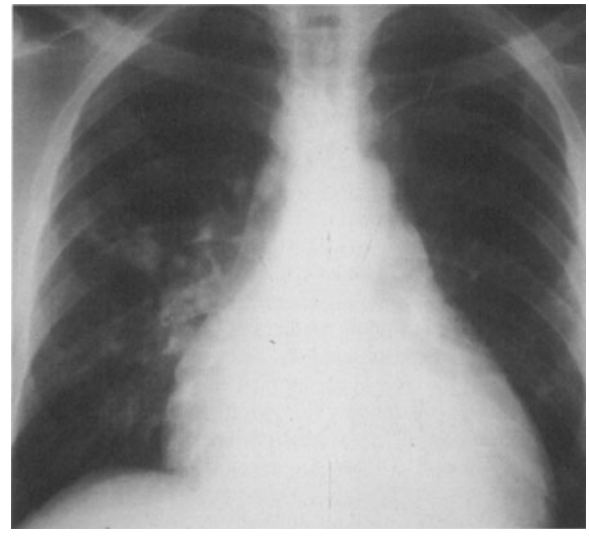

Fig. 7.25. A 25-year-old man with AIDS diagnosed with tuberculosis involving the right lung and pericardium. PA chest radiograph demonstrates a globularly enlarged cardiac silhouette consistent with pericardial effusion. There is also a right perihilar nodular lung parenchymal opacity

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