

# Imaging of Parasitic Diseases of the Thorax

Santiago Martinez, Carlos S. Restrepo,  
Jorge A. Carrillo

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### 3.1 Introduction

A broad spectrum of parasitic infections frequently affects the lungs, mediastinum, and thoracic wall, manifesting with abnormal imaging findings that often make diagnosis challenging. Although most of these infections result in nonspecific abnormalities, familiarity with their imaging features and the diagnostic pathways help the radiologist to formulate an adequate differential diagnosis and to guide diagnosticians in reaching a definitive diagnosis.

### 3.2 Protozoa

#### 3.2.1 Amebiasis

*Entamoeba histolytica* is responsible for amebiasis, a parasitosis with a worldwide distribution and an elevated mortality rate, possibly the highest after malaria and schistosomiasis. As much as 1% of the world's population is thought to be infected and up to 100,000 related deaths are reported annually (Mandell et al. 2005; Murray et al. 2005; World Health Organization 1997). The infection is more prevalent among the lower socioeconomic classes in tropical and subtropical climates; other risk factors include immigrants from endemic areas, institutionalized populations (e.g., those with mental retardation), communal living, and promiscuous homosexual males (Ravdin 1988). The most severe cases are often identified among neonates, during pregnancy and postpartum, corticosteroid use, malignancy, and malnutrition (Ravdin 1988).

Cysts remain viable for long periods of time in the environment after being excreted in the feces of human hosts. Humans can become infected by ingestion of these forms that ultimately become trophozoites in the small bowel, constituting the invasive form of the parasite (Barrett-Connor 1982; Botero 2003; Binford and Connor 1976; Hasleton 1996; Travis et al. 2002; Murray et al. 2002; Fishman 1998). The majority of cases develop non-invasive infection in keeping with the natural life cycle of

the parasite; in these patients, trophozoites become encysted and excreted again via the feces into the environment (Mandell et al. 2005). While invasive disease may develop by direct invasion of the colonic epithelium, the factor that determines pathogenicity remains unknown (Mandell et al. 2005).

Thoracic disease is the second most common manifestation of extraintestinal amebiasis after liver abscesses (Le Roux 1969; Shamsuzzaman and Hashiguchi 2002). It complicates 7–20% of all cases of hepatic disease and about 2% patients with intestinal disease (Fishman 1998). Thoracic infection may result from several routes: direct extension from a liver abscess to the thorax, which occurs in 6–40% of patients with hepatic compromise; hematogenous spread; and rarely, aspiration (Shamsuzzaman and Hashiguchi 2002). Pericardial compromise is present in less than 2% of all thoracic complications related to amebic hepatic abscesses and is more common in the setting of left hepatic lobe disease. Pain, cardiac tamponade, and sepsis are the most common clinical findings (Le Roux 1969; Ibarra-Perez et al. 1981; Landay et al. 1980).

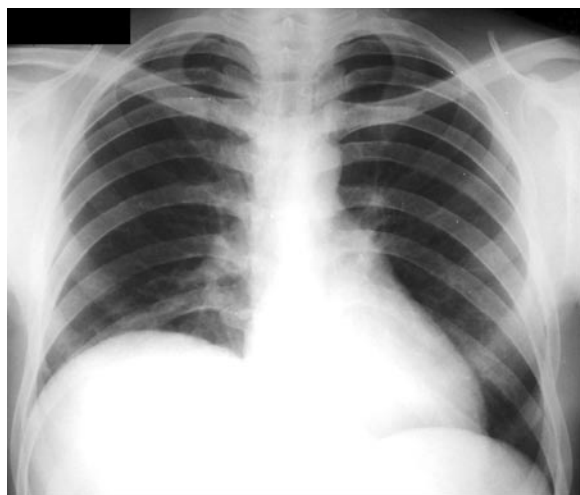
Thoracic amebiasis is more commonly suspected in patients from endemic areas with a liver abscess. Stool examination is of limited value because cysts or trophozoites are seen in a small proportion of patients with extraintestinal amebiasis (Shamsuzzaman and Hashiguchi 2002), and many pleuropulmonary manifestations may be unrelated to amebiasis, even if parasitic forms are identified in the feces. While “anchovy sauce” content can be obtained from an amebic hepatic abscess or from expectorate from a hepatobronchial fistula, the isolation of parasites in these samples is erratic. Cultures can be

obtained only in feces, since the protozoan does not grow in pus. Serologic analysis is helpful for the diagnosis of invasive disease in non-endemic populations (Barrett-Connor 1982; Shamsuzzaman and Hashiguchi 2002).

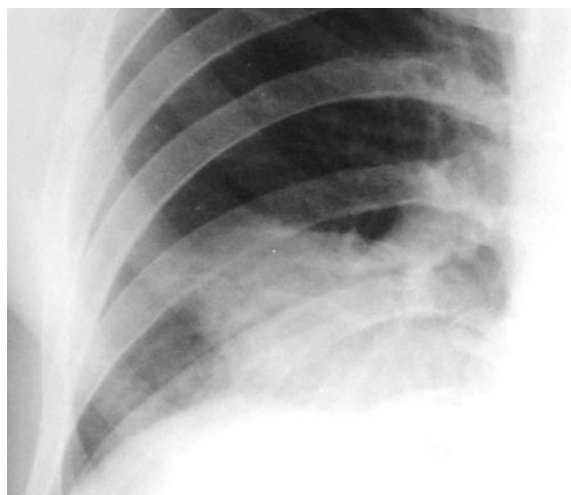
Pleural effusion is a common finding in thoracic amebiasis. It can be either sterile, as in inflammatory pleural reactions, or a frank empyema if the hepatic abscess happened to rupture into the pleural cavity. Typically, pleuropulmonary findings are preceded by elevation of the right hemidiaphragm (Fig. 3.1). Common parenchymal findings include consolidation (Fig. 3.2) and cavitation (Figs. 3.3, 3.4). When the infection is drained into the airway, a hepatobronchial or bronchobiliary fistula can result. Hematogenous dissemination results in consolidation further away from the diaphragm (Fig. 3.5). Rarely, invasion of the inferior vena cava occurs, eventually resulting in pulmonary thromboembolism. As in pleural disease, pericarditis and pericardial effusion can result from an inflammatory reaction or drainage of a liver abscess into the pericardium or pleura, usually located in the left hepatic lobe (Figs. 3.6, 3.7) (Le Roux 1969; Shamsuzzaman and Hashiguchi 2002; Ibarra-Perez et al. 1981; Landay et al. 1980).

### 3.2.2 Malaria

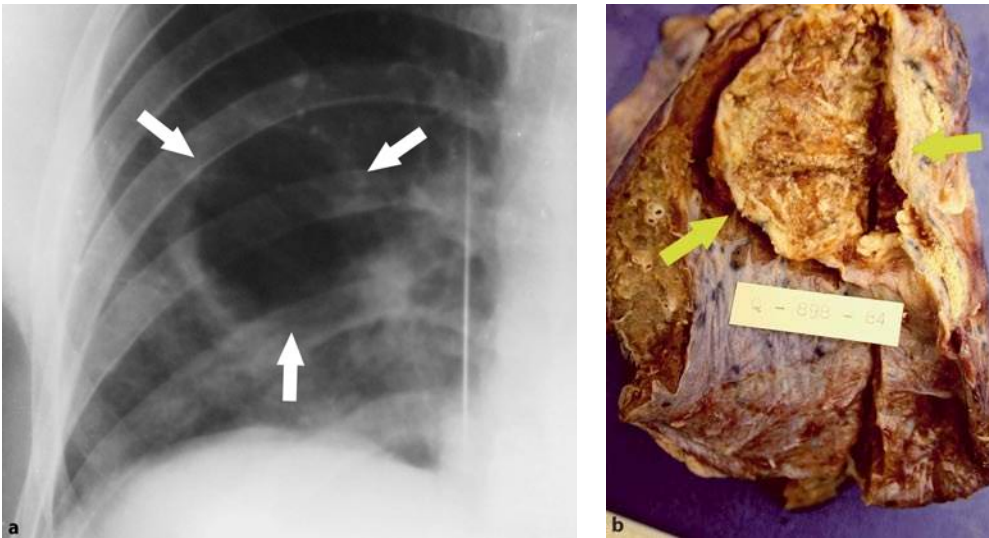
The *Anopheles* mosquito is the efficient vector that transmits *Plasmodium* species (*P. falciparum*, *P. vivax*, *P. ovale*, and *P. malariae*) to humans, the result being malaria, the most devastating parasitic disease in the world. The 2005 World Malaria Report from the World Health Organiza-



**Fig. 3.1** Thoracic amebiasis. Posteroanterior (PA) chest radiograph demonstrates elevation of the right hemidiaphragm, a common finding in the early phase of the thoracic infection



**Fig. 3.2** Pulmonary amebiasis. PA chest radiograph, cone view, shows area of consolidation in the right lower lobe

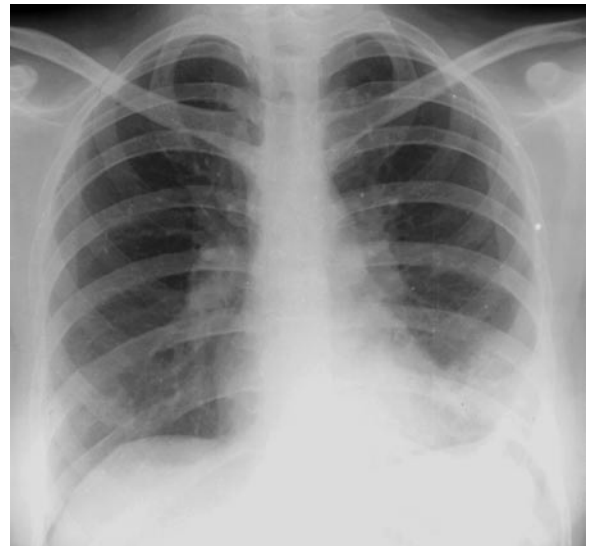


**Fig. 3.3** Cavitory pulmonary amebiasis. **a** PA chest radiograph, close-up view, demonstrates cavitation (*arrows*) in the right lower lobe. **b** Macroscopic specimen after autopsy shows an ab-

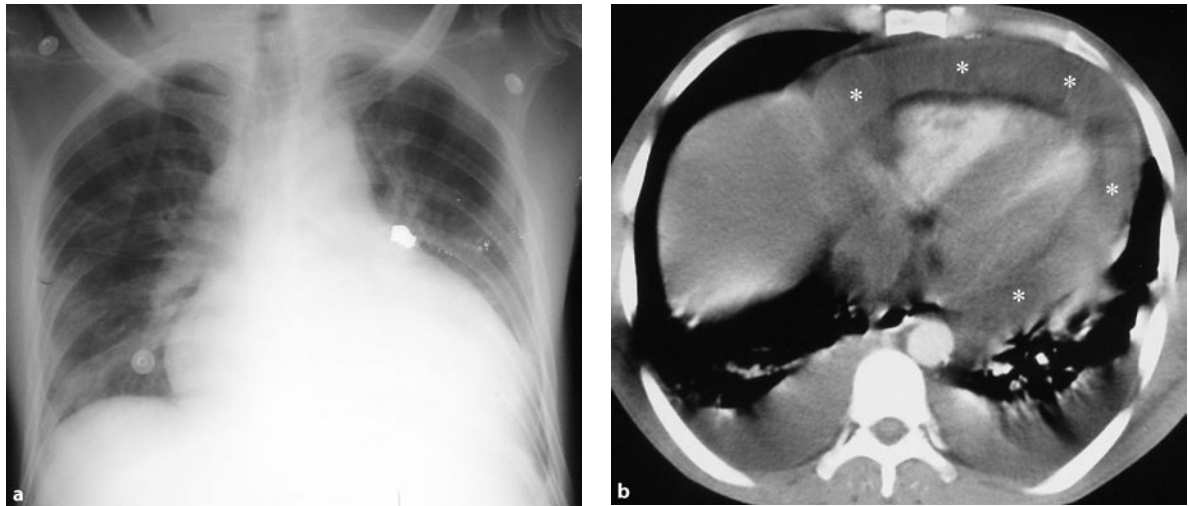
sscess in the right lower lobe (*arrows*) (Reproduced with permission from Radiographics, Martinez et al. 2005)



**Fig. 3.4** Cavitory pulmonary amebiasis. Close-up view of an axial CT scan of the chest (soft tissue window) shows cavitating consolidation in the right lower lobe (*white arrows*). Note air fluid level (*black arrows*)

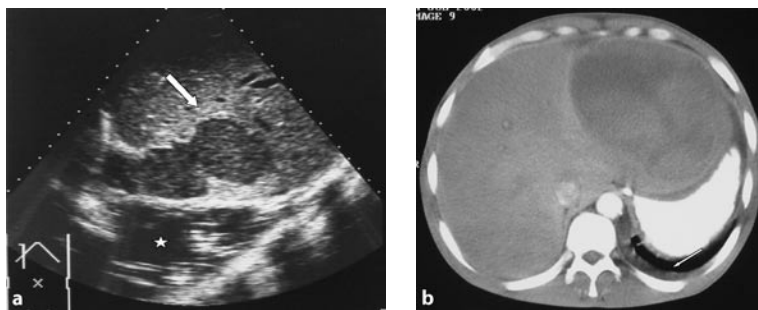


**Fig. 3.5** Pulmonary amebiasis. PA chest radiograph demonstrates left lower lobe consolidation and ipsilateral pleural fluid collection. Compromise of areas different from the right lower lobe usually results from hematogenous dissemination



**Fig. 3.6** Pericardial amebiasis presenting with cardiac tamponade. **a** PA chest radiograph demonstrates marked enlargement of the cardiomeastinal silhouette. A bullet fragment identified overlying the left cardiac silhouette is located in the paravertebral soft tissues and is unrelated. **b** Axial CT scan of the chest

(soft tissue window) shows marked pericardial effusion (*asterisks*). Additionally, there are bilateral pleural effusions. Pericardial fluid resulted from rupture of a left-sided liver lobe abscess (Reproduced with permission from Radiographics, Martinez et al. 2005)



**Fig. 3.7** Amebic liver abscess with a pleural reaction. **a** Ultrasound image of the liver showing an amebic liver abscess (*arrow*) and right pleural effusion (*star*). **b** Contrast-enhanced CT scan of the liver demonstrating a left hepatic lobe amebic abscess and left pleural effusion (*arrow*)

tion (WHO) estimated that up to 500 million people had been infected in the previous year (World Health Organization 2005). Consequently, several million deaths in endemic areas such as the tropical areas of South America, Africa, and Southern Asia result every year (Mandell et al. 2005; Murray et al. 2005; Botero 2003). Over 3.2 billion people in 107 countries are at risk of infection (World Health Organization 2005). Children younger than 5 years old, pregnant women and HIV-infected patients are at special risk (Mandell et al. 2005; World Health Organization/Unicef 2005). Of particular concern is *P. falciparum*, which is responsible for the majority of cases of severe malaria, causes more than 1 million deaths each year and exhibits an increasing occurrence of resistance to chloroquine and other antimalarial drugs (World Health Organization 2000a).

Parasites are inoculated into the human blood by the bite of the *Anopheles* mosquito. They mature inside the

erythrocytes and are released by hemolysis. The process occurs at certain time intervals that coincide with the periodicity of episodic fever (tertian or quartan) (Murray et al. 2005; World Health Organization 1997; Ravdin 1988; Barrett-Connor 1982; Botero 2003; Murray et al. 2002) and it is specific for each species (Murray et al. 2005; Botero 2003; Binford and Connor 1976; Hasleton 1996; Travis et al. 2002; Fishman et al. 1998; Taylor and White 2002). Thus, malignant tertian fever (*P. falciparum*) manifests with irregular fever spikes, whereas benign tertian fever (*P. vivax*, *P. ovale*) and quartan fever (*P. malariae*) manifest with fever spikes every 48 and 72 h respectively (Botero 2003).

Clinical findings of malaria include fever with chills, sweating, anemia, leucopenia, and splenomegaly (Murray et al. 2005; Botero 2003; Binford and Connor 1976; Hasleton 1996; Travis et al. 2002; Fishman et al. 1998; Taylor and White 2002). Diagnosis is usually made by



identifying trophozoites or other parasitic forms within the erythrocytes in a thin blood smear or parasites in a thick smear. Serologic and nucleic acid amplification tests are also available (Murray et al. 2005; Binford and Connor; Hasleton 1998; Travis et al. 2002; Taylor and White 2002).

The primary thoracic manifestation of malaria is adult respiratory distress syndrome (ARDS). As of 2005, the WHO continued to include ARDS as a criterion for the definition of severe *P. falciparum* malaria (World Health Organization 2000a). In the setting of malaria, ARDS secondary to *P. falciparum* is associated with high mortality of 80% (World Health Organization 2000a). Although the pathophysiologic events related to the development of ARDS remain unclear, available data support the fact that pulmonary edema is rather a high permeability edema secondary to microvascular dysfunction (Cosgriff 1990). Infected erythrocytes sequestered in the lungs lead to a release of local cytokines that eventually results in pulmonary edema, dyspnea, hypoxic acute lung injury, and ARDS (Mandell et al. 2005; Taylor and White 2002). Although the majority of cases of ARDS are related to the *P. falciparum* infection, association with *P. vivax* and *P. ovale* is also documented in the literature (Lee and Maguire 1999; Munteis et al. 1997; Tanios et al. 2001; Curlin et al. 1999; Carlini et al. 1999). Radiographic and CT findings are consistent with ARDS, i.e., pleural effusions, diffuse interstitial edema, and lobar consolidation (Figs. 3.8, 3.9) (Taylor and White 2002; Cosgriff 1990; Lee and Maguire 1999; Munteis et al. 1997; Tanios et al. 2001; Curlin et al. 1999; Carlini et al. 1981; Cayea et al. 1981; Gachot et al. 1995; Torres et al. 1997). Eosinophilic pneumonia with bilateral heterogeneous opacities has been described in association with the use of the anti-malarial drug pyrimethamine (Davidson et al. 1988).

### 3.2.3 Trypanosomiasis

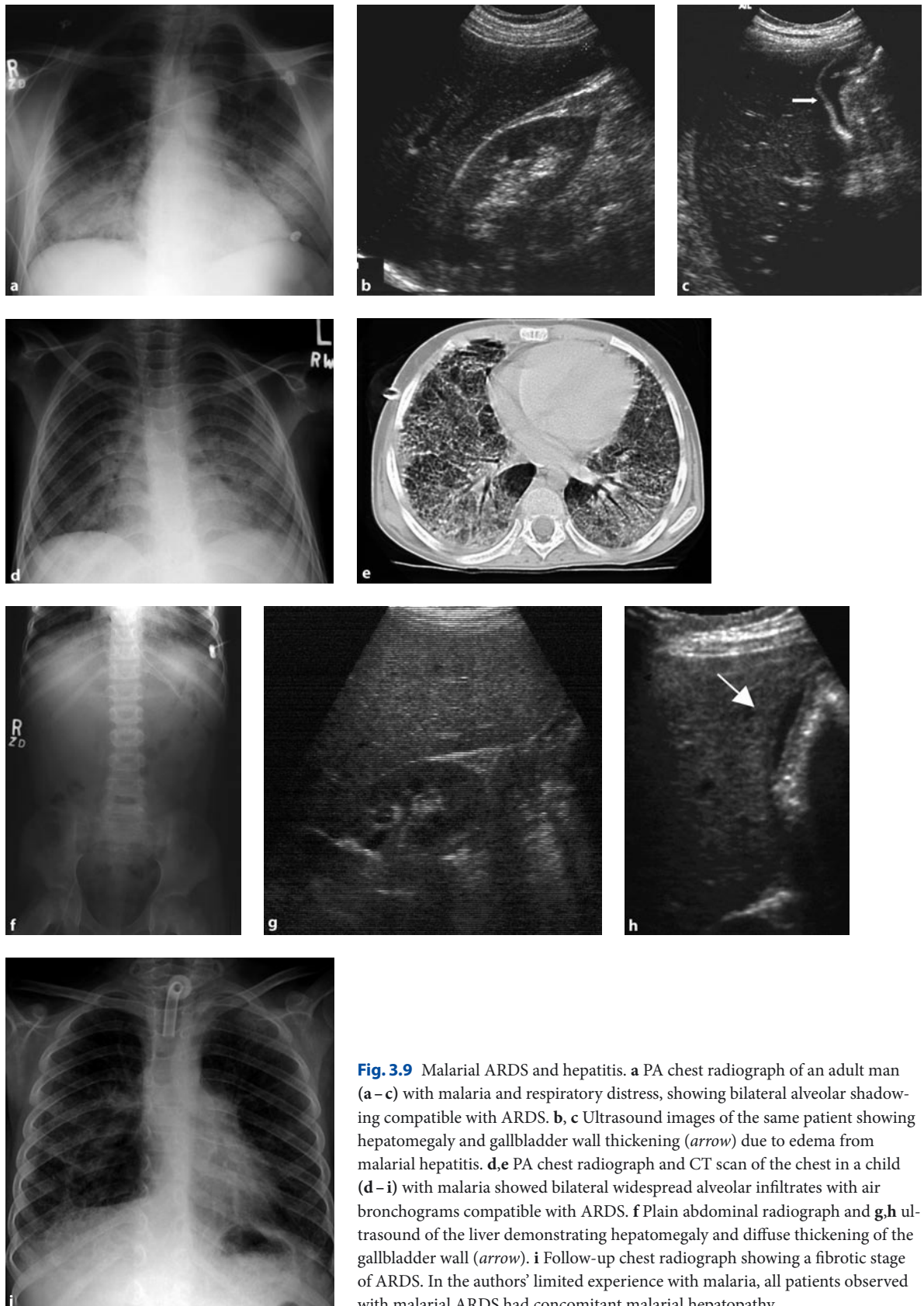
*Trypanosoma cruzi* is the etiologic agent of American trypanosomiasis or Chagas' disease. The infection is acquired through the bite of an insect from the *Reduviidae* family (genera *Triatoma*, *Rhodnius*, and *Panstrongylus*), by the inoculation of trypomastigotes into the human body. The insect usually defecates near to the bite and the inoculation may occur from rubbing the reduviid bug feces over the bite or some other skin defect (Murray et al. 2005). The parasites multiply within the macrophages, which eventually rupture, releasing amastigotes capable of invading diverse organs hematogenously, including the heart and the gastrointestinal tract, but namely the esophagus and colon (Botero 2003; Binford and Connor 1976; Hasleton 1996; Travis et al. 2002; Murray et al. 2002; Fishman et al. 1998).

Chagas' disease is endemic to areas of Central and South America, especially Argentina, Brazil, Bolivia, Chile, Paraguay, and Uruguay. By 2000, the WHO estimated that 16–18 million people were infected in the American countries (World Health Organization 2000b). As many as 45,000 people die yearly due to the disease (Mandell et al. 2005). Starting in 1997 with Uruguay, several countries (i.e., Venezuela, Chile, and Brazil) have reported interruption of vectorial and transfusional transmission of Chagas' disease, with a significant decrease in the incidence of the disease of up to 95% (World Health Organization 1999, 2000b, 2000c). Chagas' disease has also been described in the south-western United States among immigrants from endemic areas (Murray et al. 2002; Hagar and Rahimtoola 1991; Kirchhoff and Neva 1985).

Acute manifestations of trypanosomiasis are rarely clinically detected. Roman's sign is a classic manifestation that includes fever and facial or unilateral



**Fig. 3.8** Acute respiratory distress syndrome (ARDS) in a young patient with *P. falciparum* malaria. PA chest radiograph demonstrates bilateral and diffuse heterogeneous opacities in all four quadrants of the lungs. Further clinical criteria for ARDS were positive. Parasitic forms of *P. falciparum* were identified in the thin peripheral blood smear



**Fig. 3.9** Malarial ARDS and hepatitis. **a** PA chest radiograph of an adult man (**a**–**c**) with malaria and respiratory distress, showing bilateral alveolar shadowing compatible with ARDS. **b**, **c** Ultrasound images of the same patient showing hepatomegaly and gallbladder wall thickening (*arrow*) due to edema from malarial hepatitis. **d**, **e** PA chest radiograph and CT scan of the chest in a child (**d**–**i**) with malaria showed bilateral widespread alveolar infiltrates with air bronchograms compatible with ARDS. **f** Plain abdominal radiograph and **g**, **h** ultrasound of the liver demonstrating hepatomegaly and diffuse thickening of the gallbladder wall (*arrow*). **i** Follow-up chest radiograph showing a fibrotic stage of ARDS. In the authors' limited experience with malaria, all patients observed with malarial ARDS had concomitant malarial hepatopathy

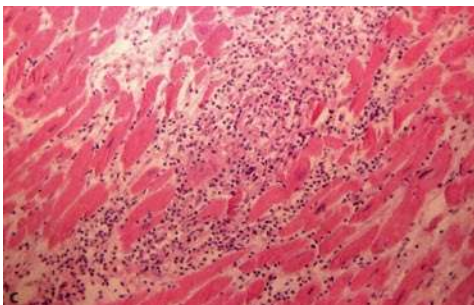
palpebral edema. Signs and symptoms of acute myocarditis and meningoencephalitis have sometimes been described (Mandell et al. 2005). Although detection of trypomastigotes in the blood smear, culture, and xenodiagnosis are the most common methods used to establish the diagnosis, their sensitivity is not greater than 50% (Mandell et al. 2005). Polymerase chain reaction (PCR) techniques appear to be promising, especially in persons with borderline serology, infected patients who have received specific treatment, and acute or congenital disease not detected in the blood smear (Mandell et al. 2005). Radiographically, acute myocarditis can present with acute heart failure (Fig. 3.10).

Patients are more commonly diagnosed in the chronic phase. Late cardiac manifestations include chronic myocarditis with focal and diffuse loss of myocytes, fibrosis, and focal atrophy, as well as involvement of the conduction system with a bundle branch block, which can progress to a complete atrioventricular block. Late gastrointestinal compromise is related to neuronal damage of the myenteric plexus, with development of achalasia and megacolon (Botero 2003; Hagar and Rahimtoola 1991; Kirchhoff and Neva 1985; Camara et al. 1993; Prata 1994; Umezawa et al. 2001). Although bronchopathy has rarely been described due to denervation of the bronchial walls, it appears not to have significant clinical impact (Lemle

1999). Serologic tests are preferred for the diagnosis of chronic forms.

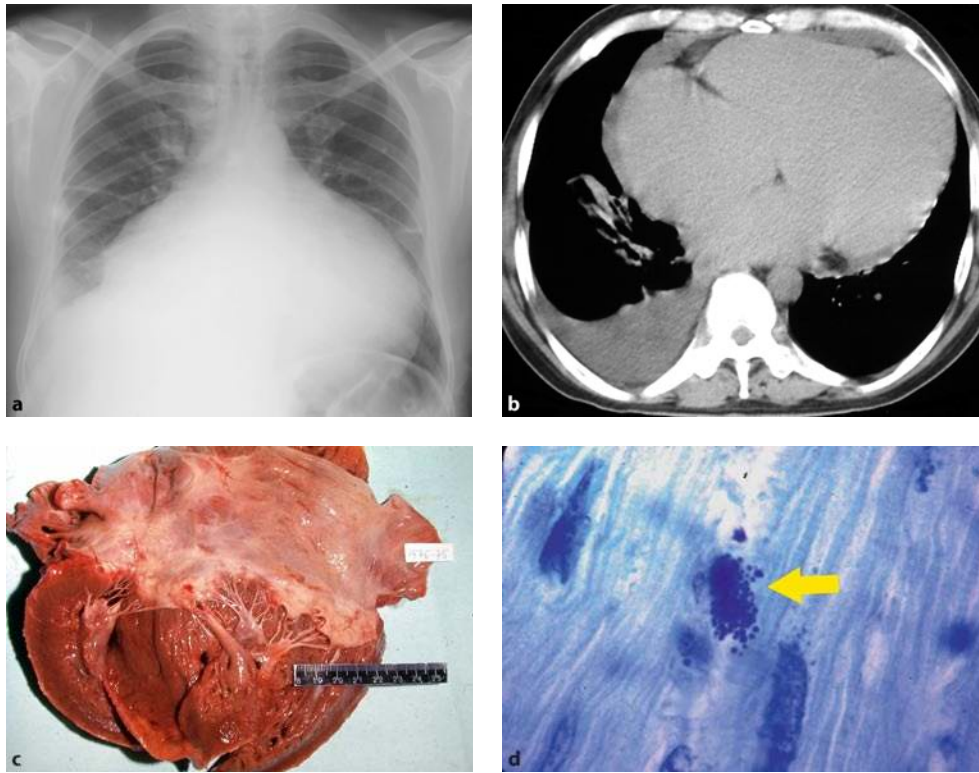
Radiologic findings reflect the aforementioned clinical features. Severe cardiomegaly, usually with evidence of venous hypertension (e.g., septal lines, pulmonary edema, pleural effusion), is often seen in chronic dilated cardiomyopathy (Fig. 3.11) (Hagar and Rahimtoola 1991; Kirchhoff and Neva 1985; Camara et al. 1993; Prata 1994; Umezawa et al. 2001). Achalasia (Fig. 3.12) and megacolon are usually confirmed with barium studies (Botero 2003; Camara et al. 1993).

The role of MR in cardiac Chagas' disease has recently been described. Areas of myocardial fibrosis are identified as myocardial delayed enhancement in inversion recovery gradient-echo sequences 10–20 min after the intravenous injection of gadolinium-based contrast (Fig. 3.13). These hyperintense areas, which can be transmural, subendocardial, along the midwall, and subepicardial, are more prone to being identified toward the apex, inferior, and inferolateral walls. Although often indistinguishable from myocardial fibrosis due to coronary artery disease, the finding could be a helpful marker of disease severity among seropositive patients, especially those who are asymptomatic (Society for Cardiovascular Magnetic Resonance 2003; Mahrholdt et al. 2005; Rochitte et al. 2005).



**Fig. 3.10** Acute trypanosomiasis. **a** PA chest radiograph essentially normal in a patient with diffuse adenopathy, fever, and weakness. **b** PA chest radiograph 48 h later shows interval development of diffuse parenchymal opacities compatible with pulmonary edema. The patient eventually developed ventricular tachyarrhythmia and died. **c** Photomicrograph revealed acute myocarditis due to *T. cruzi* (**b** and **c** are reproduced with permission from Radiographics, Martinez et al. 2005)



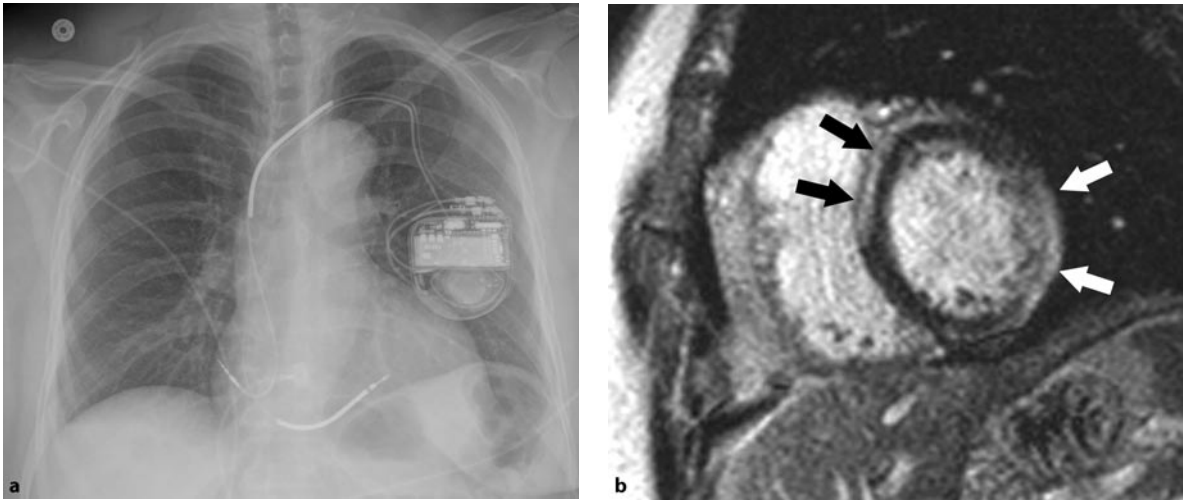


**Fig. 3.11** Chronic myocarditis due to Chagas' disease. **a** PA chest radiograph shows severe and diffuse enlargement of the cardiac silhouette, vascular redistribution in the upper lobes, and right-sided pleural effusion. **b** Axial CT of the chest (mediastinal window) demonstrates marked dilatation of all cardiac chambers and right-sided pleural effusion. **c** Macroscopic specimen exhibits marked cardiomegaly and thickening of the ventricular wall. **d** Photomicrograph (Giemsa stain,  $\times 10$ ) shows *T. cruzi* amastigotes within a myocardial fiber (arrow) (**a**, **c**, and **d** are reproduced with permission from Radiographics, Martinez et al. 2005)



**Fig. 3.12** Achalasia secondary to Chagas' disease. **a** PA chest radiograph demonstrates a right-sided tubular paramediastinal soft tissue mass (arrows). **b** Barium swallow shows massive dilatation of the esophagus compatible with a Chagasic megaesophagus





**Fig. 3.13** Cardiac Chagas' disease. **a** PA chest radiograph demonstrates mild enlargement of the cardiac silhouette. There is a dual-lead automatic implantable cardioverter defibrillator in the correct position. **b** Double inversion recovery MR image obtained 10 min after intravenous injection of gadolinium. In the

anterior and anteroseptal wall, there is epicardial enhancement with sparing of the endocardium and myocardium (*black arrows*). An additional focus of transmural enhancement is present in the lateral wall (*white arrows*)

### 3.2.4 Toxoplasmosis

Human toxoplasmosis results from infection with *Toxoplasma gondii*, an intracellular protozoan found in a variety of animals such as birds and humans, whose ultimate reservoir is felines (Mariuz et al. 1997; Wallace 1973). The infection has worldwide distribution and although it may infect immunocompetent patients, most affected individuals have a cell-mediated immunodeficiency such as AIDS (Mariuz et al. 1997), usually with a CD4 count below 200/mm<sup>3</sup> (Crowe et al. 1991). Infection is acquired through the ingestion of either contaminated food/beverages or meat with tissular forms from intermediate hosts. Transplacental infection is also a well-known route resulting in congenital toxoplasmosis (Mariuz et al. 1997).

The lungs and heart are the most frequently affected organ after the central nervous system (Hofman et al. 1993a; Roldan et al. 1987; Hofman et al. 1993b; Holliman 1988). The lung can be affected either primarily or as the result of dissemination from a CNS infection. Clinical manifestations of *Toxoplasma* pneumonia include fever, coughing, and dyspnea (Rabaud et al. 1996); however, it can be asymptomatic or minimally symptomatic. Myocardial involvement includes arrhythmias (atrial and ventricular), sudden death, AV block, pericarditis, and heart failure (Hofman et al. 1993b; Mariani et al. 2006; Duffield et al. 1996; Sahasrabudhe et al. 2003). Diagnosis is usually made by identifying parasites in the bronchoalveolar lavage and lung biopsy (Bonilla and Rosa 1994; Oksenhendler et al. 1990), or in cardiac tissues of patients with acute myocarditis (Hofman et al. 1993b). Serologic and PCR techniques are also available (Mariuz et al.

1997; Lavrard et al. 1995). Despite appropriate treatment, relapse and mortality remain high among HIV patients (Rabaud et al. 1996; Pomeroy and Filice 1992).

Radiographic manifestations are nonspecific and rather similar to other opportunistic diseases in the HIV patient (Schnapp et al. 1992). The most common radiological manifestations include diffuse and bilateral reticulonodular or nodular opacities (Fig. 3.14) (Goodman and Schnapp 1992). Other less common findings include consolidation with or without air bronchogram formation (Figs. 3.15, 3.16), micronodules, cavitations (Fig. 3.17), pleural effusion, and pneumothorax (Rabaud et al. 1996; Pomeroy and Filice 1992; Mendelson et al. 1987; Tawney et al. 1986; Libanore et al. 1991). Radiographic signs of pulmonary edema can be seen in the setting of acute myocarditis (Fig. 3.18).

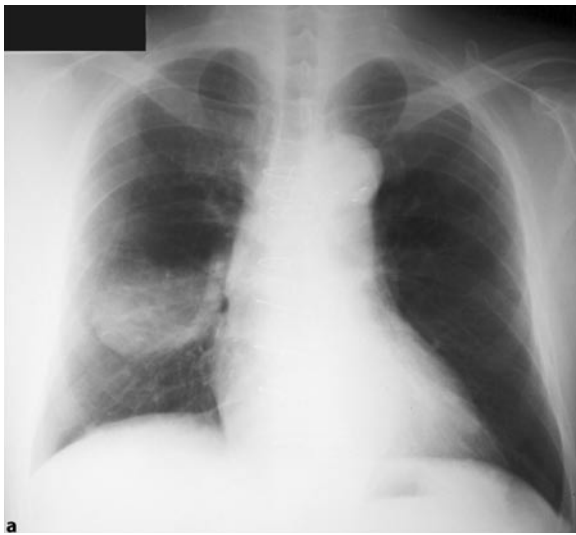
## 3.3 Nematodes (Roundworms)

### 3.3.1 Ascariasis

Infection with *Ascaris lumbricoides* (Fig. 3.19) is among the most common worldwide parasitic infections. Human infection is thought to be present in one-fourth of the world's population (World Health Organization 2002) with an estimated mortality between 20,000 and 60,000 per year (Mandell et al. 2005; Sarinas and Chitkara 1997), primarily as a consequence of intestinal obstruction (Sarinas and Chitkara 1997). The parasite has worldwide distribution; however, tropical and subtropical climates in Southeast Asia, Africa, and Central/South America

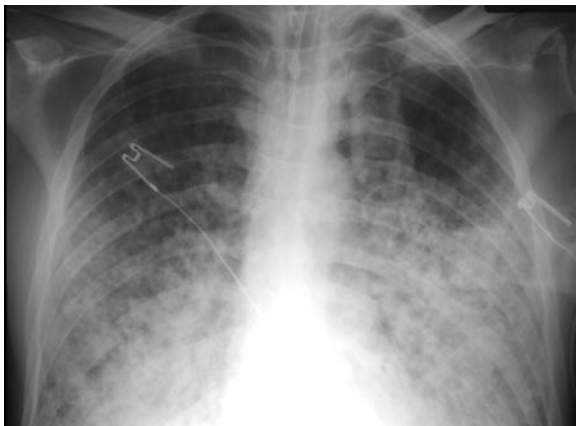


**Fig. 3.14** Pulmonary toxoplasmosis in a patient with AIDS. PA chest radiograph demonstrates diffuse and bilateral reticulo-nodular opacities. Parasites were isolated in the bronchoalveolar lavage (Courtesy of Dr. Philip Goodman – Duke University Medical Center, Durham, NC, USA)

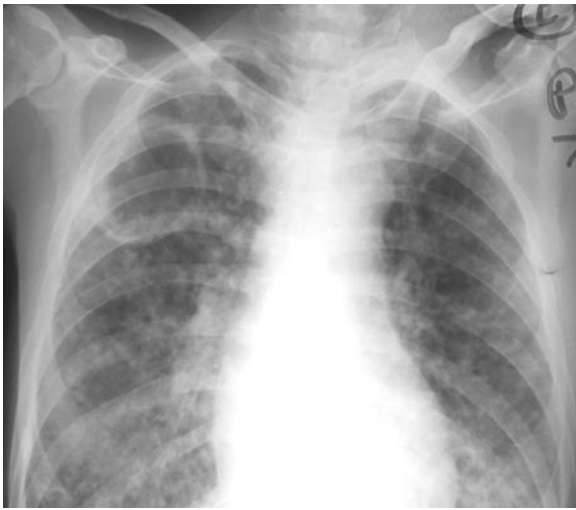


**Fig. 3.15** Pulmonary toxoplasmosis in a patient with AIDS. **a** PA chest radiograph shows a round homogenous consolidation in the right lower lung. **b** Lateral chest radiograph helps fur-

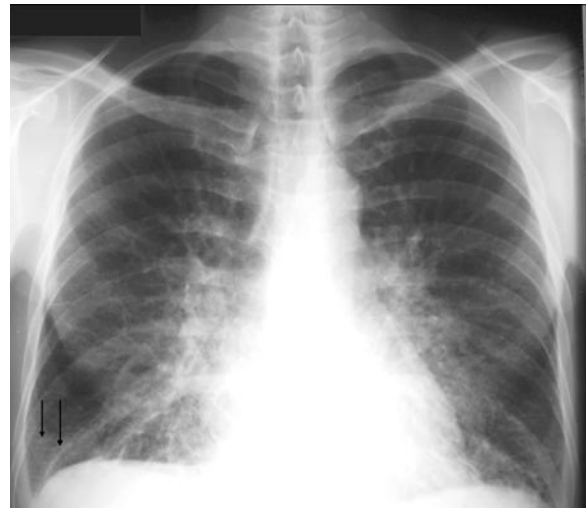
ther localization of the infection in the right lower lobe. Diagnosis was proven pathologically (Courtesy of Dr. Philip Goodman – Duke University Medical Center, Durham, NC, USA)



**Fig. 3.16** Pulmonary toxoplasmosis in a patient with AIDS. PA chest radiograph demonstrates extensive consolidation and nodular opacities throughout the lungs. Diagnosis was proven pathologically. (Courtesy of Dr. Philip Goodman – Duke University Medical Center, Durham, NC, USA)



**Fig. 3.17** Cavitary pulmonary toxoplasmosis in a patient with AIDS. PA chest radiograph demonstrates bilateral diffuse nodular opacities. In addition, there is a cavitary lesion in the right upper lobe. Diagnosis after resection demonstrated the presence of *T. gondii* as the causative agent (Courtesy of Dr. Philip Goodman – Duke University Medical Center, Durham, NC, USA)



**Fig. 3.18** Pulmonary edema in the setting of acute myocarditis due to *T. gondii*. PA chest radiograph showing a normal heart size, bilateral and symmetrical perihilar shadowing with basal septal B lines of Kerley (arrows), and bilateral small pleural effusions consistent with cardiogenic edema (Courtesy of Dr. Philip Goodman – Duke University Medical Center, Durham, NC, USA)

account for the majority of people infected (Sarinas and Chitkara 1997). Under certain unusual circumstances, *Ascaris suum*, a large roundworm endemic to pigs, can infect humans (see visceral larva migrans) (Maruyama et al. 1996; Sakakibara et al. 2002).

The parasite is acquired by ingestion of embryonated eggs, usually present in contaminated foods or fluids (Fishman 1998). For completing their natural cycle, parasitic larvae migrate from the small intestine to the pulmonary arterial circulation 4 days after the ingestion of the eggs (Mandell et al. 2005). After 6–10 days of growing, larvae break into the alveoli and eventually are ingested; once in the intestine and after 2 months, mature worms produce eggs that are excreted in human feces, completing the cycle. Larvae in the lungs produce an inflammatory reaction with destruction of capillaries and alveolar walls, subsequent edema, hemorrhage, and epithelial cell desquamation, causing chemotaxis of neutrophils and eosinophils (Botero 2003; Binford and Connor 1976; Hasleton 1996; Travis et al. 2002; Murray et al. 2002; Fishman et al. 1998).

Although ascariasis is usually asymptomatic, up to 15% of infected patients may experience clinical symptoms (Mandell et al. 2005). Lung disease is usually asymptomatic, but as many as 20% of patients (Spillmann 1975) will present with fever, coughing, expectoration, and peripheral blood eosinophilia (Mandell et al. 2005; Sarinas and Chitkara 1997), sometimes recognized as Loeffler's-like syndrome (Fishman 1998). Asthma has also been described in the setting of ascariasis (Cromp-

ton 2001). Eosinophils and Charcot-Leyden crystals are often identified in the sputum (Sarinas and Chitkara 1997). Confirmation requires identification of larvae in the sputum or gastric aspirates (Murray et al. 2005; Fishman 1998; Proffitt and Walton 1962). In patients from non-endemic areas who present with pulmonary symptoms, the diagnosis is suggested when eggs are present in the stool (Botero 2003; Zumba and James 2002).

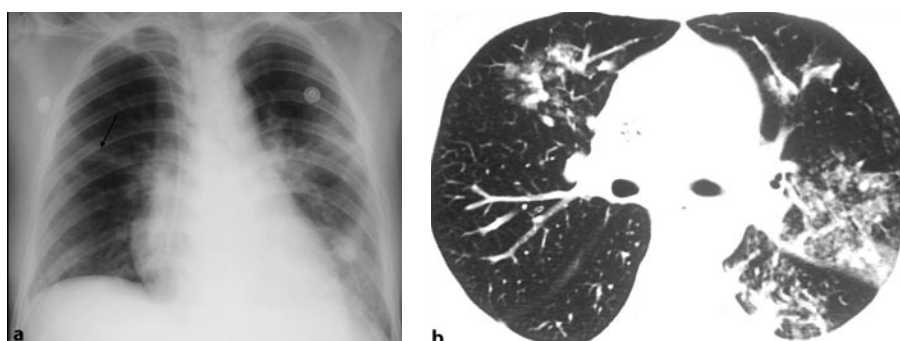
Chest radiography and CT may demonstrate migratory, ground-glass, and alveolar opacities that characteristically clear within days to weeks (Figs. 3.20, 3.21). Lobar consolidation and alveolar hemorrhage have also been described (Barrett-Connor 1982; Botero 2003; Proffitt and Walton 1962; Zumla and James 2002).

### 3.3.2 Strongyloidiasis

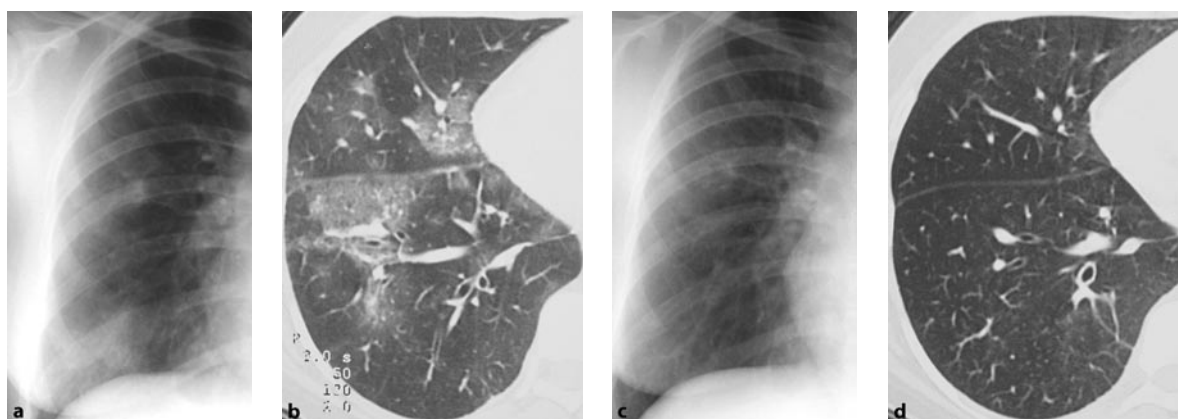
Humans are the primary host of the microscopic nematode *Strongyloides stercoralis* (Barrett-Connor 1982; Botero 2003; Binford and Connor 1976; Hasleton 1996; Travis et al. 2002; Murray et al. 2002; Fishman et al. 1998). Although reports on the global prevalence of the disease are highly dissimilar (ranging from 1 to 100 million people worldwide), it appears clear that strongyloidiasis is not as frequent as infection with other nematodes (e.g., *Ascaris lumbricoides* or *Trichuris trichiura*) (Mandell et al. 2005; Murray et al. 2002). The parasite is found in all tropical and subtropical regions (Mandell et al. 2005; Murray et al. 2002).



**Fig. 3.19** Photograph of an *Ascaris lumbricoides* worm



**Fig. 3.20** Pulmonary ascariasis. **a** PA chest radiograph demonstrates heterogeneous opacities in the left lung and to a lesser extent in the right lung (*arrow*). **b** Axial chest CT (lung window) shows ill-defined areas of ground-glass opacity and consolidation in both lungs



**Fig. 3.21** Pulmonary ascariasis. **a** PA chest radiograph shows subtle opacities in the right mid and lower zones. **b** A close-up view of an axial CT scan of the chest (lung window) demonstrates ground-glass opacities in the right middle and lower

lobes. **c,d** Follow-up examinations demonstrating complete resolution of the abnormalities after a period of 2 weeks (**a** and **b** are reproduced with permission from Radiographics, Martinez et al. 2005)



Infective filariform larvae are capable of invading the skin from the contaminated soil (Murray et al. 2005). Larvae migrate hematogenously to the lungs where they mature and are eventually coughed up and swallowed. In the small intestine adults produce eggs, which generate rhabditiform larvae that can be either excreted into the environment in human feces, completing the cycle, or become filariform larvae (Murray et al. 2005; Barrett-Connor 1982; Botero 2003; Binford and Connor 1976; Travis et al. 2002; Murray et al. 2002; Fishman 1998) capable of autoinfection to perpetuate the cycle. This chronic pathway of continuous autoinfection can lead to a massive and life-threatening parasitic infestation (the so-called hyperinfection syndrome), especially in AIDS patients and in patients who are receiving glucocorticoid therapy (Chu et al. 1990). Hyperinfection syndrome is associated with a mortality rate close to 100% without treatment and perhaps higher than 25% when treated (Mandell et al. 2005).

Diagnosis can be suggested clinically in patients from – or those who have traveled to – endemic areas, who present with peripheral blood eosinophilia, pulmonary symptoms, and abdominal pain/diarrhea. On the other hand, hyperinfection syndrome usually presents with systemic inflammatory response syndrome in the setting of an immunocompromised patient. Eosinophilia is often absent. Definitive diagnosis is made by identifying larvae in the sputum, bronchoalveolar lavage or lung biopsy (Mandell et al. 2005; Barrett-Connor 1982; Botero 2003; Binford and Connor 1976; Chu et al. 1990).

Radiologic findings include ill-defined and migratory opacities that typically resolve in 1–2 weeks. Hyperinfection syndrome can manifest with extensive consolidation, which can be infectious, hemorrhagic (Fig. 3.22), due to noncardiogenic edema, or a mixture (Woodring et al. 1994). A miliary pattern and reticular opacities can be better depicted on the CT scan (Fig. 3.23) (Woodring et al. 1994; Krysl et al. 1991; Reeder and Palmer 1980).



**Fig. 3.22** Pulmonary strongyloidiasis. **a** PA chest radiograph demonstrates bilateral widespread opacities throughout the lungs with perihilar and basilar predominance. **b** Axial CT scan of the chest (lung windows) shows bilateral apical ground-glass opacities. **c** Axial CT scan of the chest (lung windows) section

at the level of the lung bases showing more coalescent opacities. **d** Plain radiograph of the chest of another patient with pulmonary strongyloidiasis, showing bilateral perihilar and basal confluent infiltrates. (**a** and **c** are reproduced with permission from Radiographics, Martinez et al. 2005)

Superimposed bacterial infection with cavitation and abscess formation is not an uncommon finding (Chu et al. 1990; Woodring et al. 1994; Berk et al. 1987; Cook et al. 1987; Davidson 1992; Makris et al. 1993; Simpson et al. 1993; Ford et al. 1981; Ali-Munive et al. 2002). Rarely, larvae can migrate to the pleura and pericardium causing pleural and pericardial effusion (Barrett-Connor 1982; Woodring et al. 1994; Reeder and Palmer 1980; Davidson 1992; Bruno et al. 1982; Froes 1930).

### 3.3.3 Hookworms

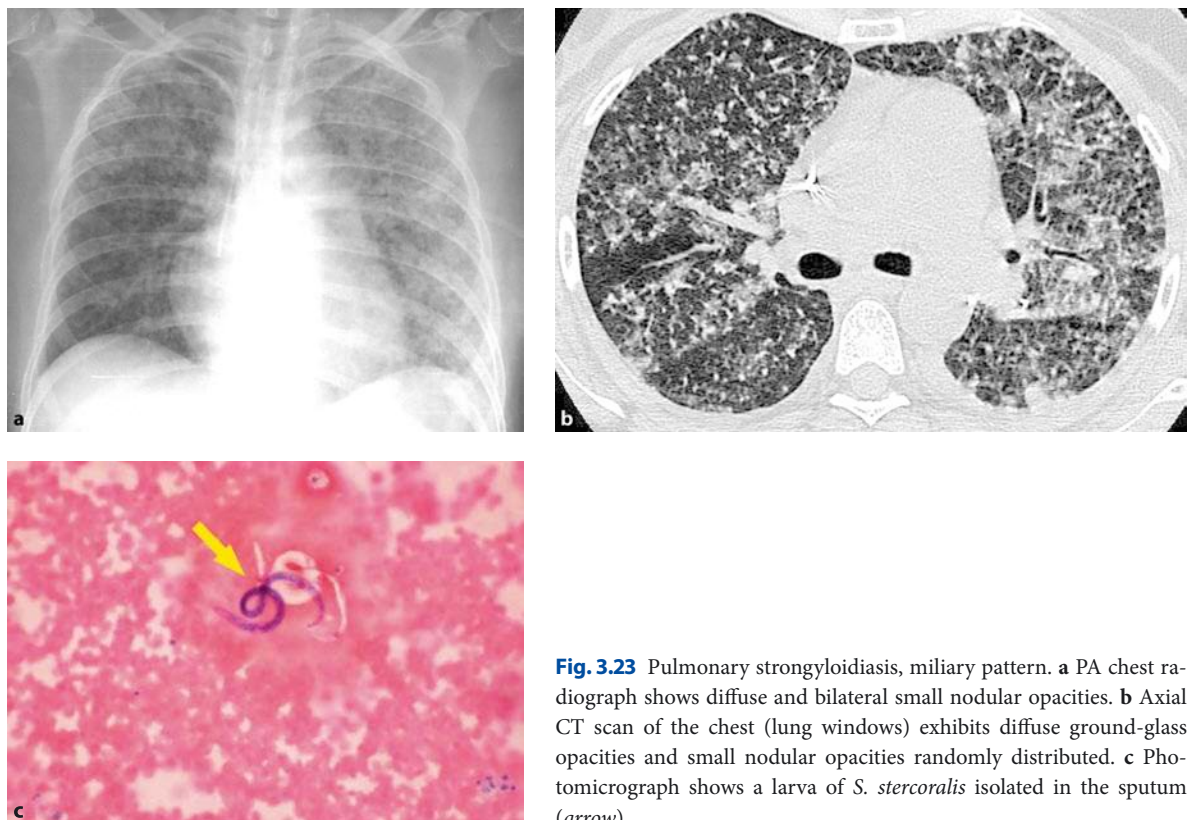
Hookworm infection is caused by *Ancylostoma duodenale* and *Necator Americanus*. Hookworms share several characteristics with *A. lumbricoides* including worldwide distribution, especially in the tropical and subtropical regions, and the necessity of a maturation phase in the lungs. The infection is thought to occur in about 25% of the world's population (Murray et al. 2005; Sarinas and Chitkara 1997; Hotez and Pritchard 1995). As opposed to ascariasis, hookworm infection is acquired by the percutaneous penetration of larvae from the soil. Parasites migrate to the lungs where they ultimately penetrate the alveolar walls, ascend through the trachea and eventually are swallowed up into the small intestine (Mandell et al. 2005).

Pulmonary disease is related to the migration of parasites to the lungs, which causes eosinophilic pleural effu-

sion (Yassin et al. 2007) or transient pneumonitis (Loeffler's-like syndrome), pathophysiologically and clinically similar to ascariasis (Sarinas and Chitkara 1997), though less frequent and severe (Mandell et al. 2005). Peripheral blood eosinophilia is common (Sarinas and Chitkara 1997). Radiographic manifestations are thought to be similar to those of ascariasis (Sarinas and Chitkara 1997); however, there are not enough data available in the radiologic literature. Diagnosis is confirmed by demonstrating the parasite in respiratory, gastric or duodenal secretions (Sarinas and Chitkara 1997). The presence of eggs is not helpful during pulmonary disease, since this event is expected to occur 2 months after the dermal penetration (Sarinas and Chitkara 1997), i.e., after the parasite's pulmonary phase (Mandell et al. 2005).

### 3.3.4 Visceral Larva Migrans/Loeffler's Syndrome

Visceral larva migrans (VLM), or Loeffler's syndrome, is an infection classically caused by *Toxocara canis* and *Toxocara cati* (Beaver et al. 1952; Dent et al. 1956); however, association with *Ascaris suum* has also been described (Maruyama et al. 1996; Sakakibara et al. 2002; Sakai et al. 2006; Van Knapen et al. 1992). In all three species, the infection is usually acquired by ingestion of contaminated products (Mandell et al. 2005; Chitkara and Sarinas 1997; Phills et al. 1972). Once in the intestinal mucosa, larvae



**Fig. 3.23** Pulmonary strongyloidiasis, miliary pattern. **a** PA chest radiograph shows diffuse and bilateral small nodular opacities. **b** Axial CT scan of the chest (lung windows) exhibits diffuse ground-glass opacities and small nodular opacities randomly distributed. **c** Photomicrograph shows a larva of *S. stercoralis* isolated in the sputum (arrow)

can migrate to a variety of organs including lymphatics, liver, lungs, and less frequently the heart, central nervous system, eye, and other organs (Chitkara and Sarinas 1997). *T. canis* and *T. cati* have worldwide distribution wherever dogs and cats, respectively, are present (Murray et al. 2005). Children are at higher risk due to pica, proximity with pets, and outdoor play (Chitkara and Sarinas 1997). Infection with *A. suum* was reproduced experimentally in humans in the 1970s (Phills et al. 1972); however, outbreaks have been described in Japan since then (Maruyama et al. 1996; Sakakibara et al. 2002; Sakai et al. 2006).

Larvae of these species are unable to grow into mature adults in humans and thus eggs or worms are not detected in feces (Sakai et al. 2006). VLM due to *T. canis* and *T. cati* can cause pulmonary disease in 25–85%. Clinical manifestations include mild forms with coughing and wheezing, severe bronchiolitis, asthma, and acute/chronic eosinophilic pneumonia with respiratory failure (Chitkara and Sarinas 1997; Bartelink et al. 1993; Inoue et al. 2002), usually with associated peripheral blood eosinophilia (Fishman 1998; Chitkara and Sarinas 1997). The pulmonary migration of *A. suum* triggers an inflammatory reaction similar to that elicited by *A. lumbricoides* (Phills et al. 1972). Clinical forms of infection with *A. suum* are similar to other presentations of VLM (Sakai et al. 2006; Phills et al. 1972). Diagnosis of VLM is achieved by the detection of antibodies by enzyme-linked immunosorbent assay (ELISA) (Sakai et al. 2006; Chitkara and Sarinas 1997). Larvae of *A. suum* can be isolated in respiratory secretions and gastric aspirates during the pulmonary phase (Phills et al. 1972).

Scant information regarding radiologic manifestations of VLM due to *T. canis* and *T. cati* is available in the literature. Among children, abnormal chest radiograph is identified in approximately 40% of cases, most commonly perihilar reticular opacities (Snyder 1961); some data support that abnormalities can resolve within days (Beshear and Hendley 1973). Isolated case reports have shown other characteristics like mediastinal and retroperitoneal adenopathy evident on the CT scan (Wolach et al. 1995). In adults, radiographic manifestations include scattered bilateral reticular or heterogeneous opacities that can be migratory (Chitkara and Sarinas 1997; Bartelink et al. 1993) and less commonly, nodules and segmental consolidations that can resolve within days (Chitkara and Sarinas 1997; Inoue et al. 2002; Feldman and Parker 1992; Sane and Barber 1997). Bacterial superimposed infection has been documented in immunosuppressed patients (Kremery et al. 1992). Limited information is available regarding CT findings (Fig. 3.24). Isolated case reports in adults demonstrated peripheral alveolar and ground-glass opacities (Inoue et al. 2002) and small ill-defined ground-glass and solid nodules (Sane and Barber 1997; Roig et al. 1992). Radiologic characteristics of infection due to *A. suum* appear to be similar to other forms of VLM, including bilateral reticulonodular opacities on chest radiographs and



**Fig. 3.24** Computed tomography scan of the chest showing bilateral patches of peripheral pulmonary infiltrates in a patient with eosinophilia and proven toxocariasis by positive blood serology test. The pulmonary changes healed completely following antiparasitic drug treatment

bilateral nodules with halo or ill-defined ground-glass opacities on CT scans, which may be associated with multiple low attenuation lesions in the liver (Maruyama et al. 1996; Sakakibara et al. 2002; Sakai et al. 2006).

### 3.3.5 Dirofilariasis

As the prevalence of the dog heartworm among canines appears to be rising, human dirofilariasis is increasingly being reported (Chitkara and Sarinas 1997; Asimacopoulos et al. 1992; Richert and Krakaur 1959; Roy et al. 1993). The disease has been sporadically reported worldwide, with many cases emanating from the East Coast and South of the United States (Mandell et al. 2005; Asimacopoulos et al. 1992). It is caused by *Dirofilaria immitis*, a filarial nematode that is transmitted from dogs to humans by mosquitoes. An immature adult worm, unable to mature in the accidental human host, can reach a peripheral vein and travel in the bloodstream until it lodges in the arterial pulmonary circulation where it dies, causing local vasculitis and eventually an infarct (Mandell et al. 2005; Botero 2003; Murray et al. 2002).

The infection is frequently asymptomatic and becomes evident due to the incidental detection of a solitary pulmonary nodule (Mandell et al. 2005; Murray et al. 2005; Fishman 1998). Patients can sometimes present with coughing, pain, and hemoptysis, most likely due to pulmonary infarct (Mandell et al. 2005; Fishman 1998). Eosinophilia is present in less than 15% of cases (Mandell et al. 2005).

Although a solitary pulmonary nodule, 3 cm or less in diameter (the so-called coin-shaped lesion), represents the classic radiologic presentation of the infection (Figs. 3.25, 3.26) (Asimacopoulos et al. 1992; Levinson

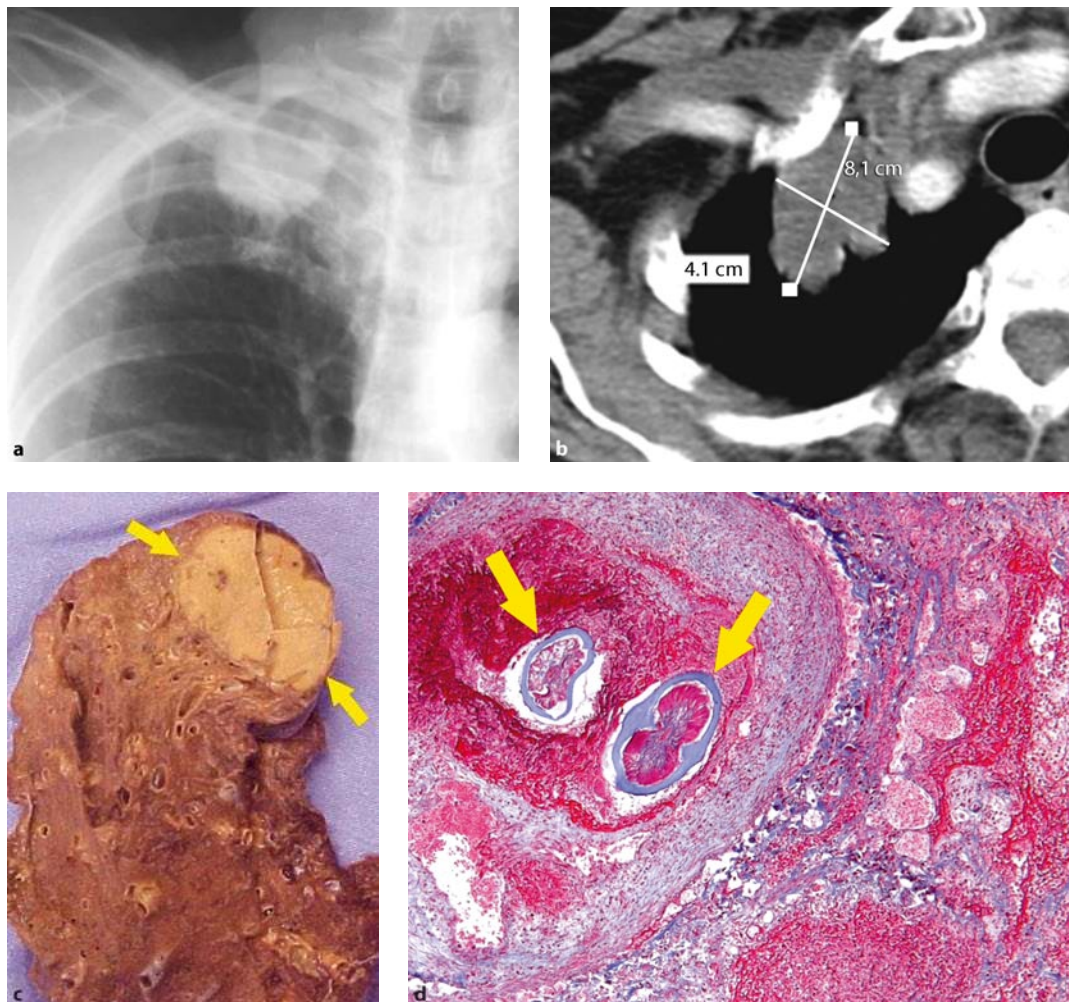


et al. 1979; Shah 1999), other findings such as multifocal heterogeneous opacities that resolve after several weeks and evolve into multiple nodules have also been described (Mandell et al. 2005; Kochar 1985). Despite serologic tests being available, they are not considered to be helpful because these lesions are worrisome for malignancy and thus usually excised (Mandell et al. 2005). Like other infectious nodules, pulmonary nodules in dirofilariasis can show increased uptake on PET scan (Moore and Franceschi 2005), so they cannot be differentiated from malignant pulmonary nodules. Definitive diagnosis is usually made based on the histopathology of the excised nodule and, sporadically, with needle aspiration biopsy of the nodule and demonstration of fragments of the parasite (Fishman et al. 1998; Asimacopoulos et al. 1992).

### 3.3.6 Microfilariae (Tropical Pulmonary Eosinophilia)

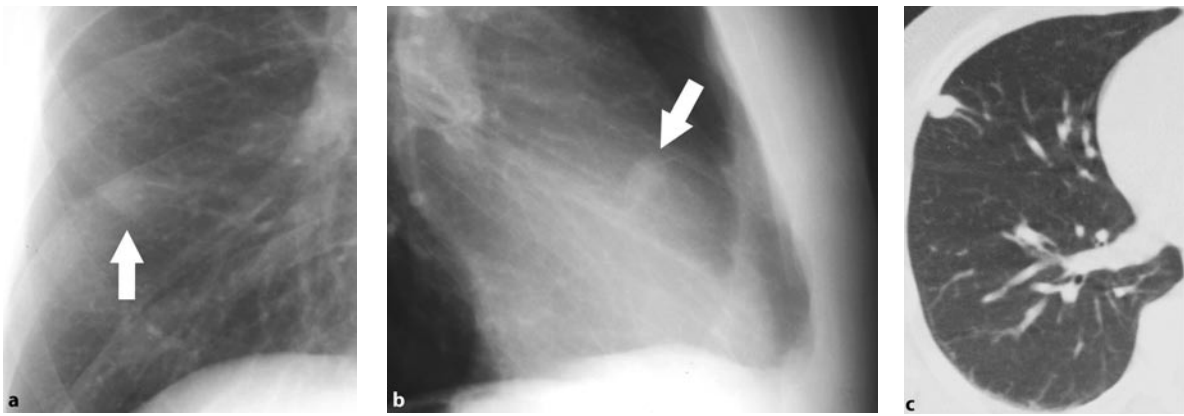
Tropical pulmonary eosinophilia (TPE) is a rare parasitic infection caused by microfilariae of the lymphatic-dwelling organisms *Wuchereria bancrofti* and *Brugia malayi*, affecting people living in India and Southeast Asia. Humans contract the disease when they are bitten by mosquitoes that carry infective larvae.

Clinically, patients present with an asthma-like syndrome. Peripheral blood eosinophilia and elevated serum IgE levels are present. Radiographically, reticulonodular infiltrates or a miliary pattern are seen on chest radiographs. The differential diagnosis includes sarcoidosis, miliary tuberculosis, and bronchial asthma. A dilated



**Fig. 3.25** Pulmonary dirofilariasis presenting as a lung mass. **a** Close-up PA chest radiograph demonstrates a round mass in the right apical area, the so-called coin-shaped lesion. **b** Axial CT of the chest (soft tissue window) further characterizes the mass, showing well-defined borders. The patient underwent lobectomy. **c** Macroscopic specimen demonstrates a round mass in the right upper lobe (arrows). **d** Photomicrograph shows arterial thrombosis with parasitic fragments inside the pulmonary artery (arrows) as well as intense inflammatory reaction. (Courtesy of Dr. Fortunato Juarez, INER, Mexico DF, Mexico)





**Fig. 3.26** *Dirofilariasis* presenting as a solitary pulmonary nodule. **a,b** PA and lateral chest radiographs, cone views, demonstrate a small soft tissue nodule in the right middle lobe (arrows). **c** Axial CT scan of the chest (lung window) shows a small

nodule in the periphery of the right middle lobe (Courtesy of Dr. Edward Patz – Duke University Medical Center, Durham, NC, USA)

thoracic duct in the chest and diffuse lymphangiectasia in the abdomen and pelvis were reported by Ahn et al. (2005) on a CT scan of the body; this finding gives a clue to the diagnosis of filariasis. Diagnosis is confirmed by identification of microfilaria in lymph nodes or pulmonary nodules, and by specific serology tests. However, often microfilariae cannot be identified on microscopic examination; then a favorable response to treatment with diethylcarbamazine is diagnostic (Kuznen 2006; Lahiri 1993).

### 3.3.7 Gnathostomiasis

Gnathostomiasis, caused by *Gnathostoma spinigerum*, is a food-borne parasitic zoonosis caused by ingestion of the uncooked or undercooked flesh of freshwater fishes, with predominantly cutaneous manifestations. Although four distinct species of the genus *Gnathostoma* were identified as the causative agents for human gnathostomiasis, human infections with *G. doloresi* have been found in Japan, concentrated in the Miyazaki Prefecture. Lung involvement is very rare, and few case reports have been reported in the medical literature (Charoenratanakul 1997; Priyjanonda et al. 1955).

## 3.4 Cestodes (Tapeworms)

At least four species of *Echinococcus* can infect humans: *E. granulosus*, *E. multilocularis*, *E. vogeli* and *E. oligarthrus*. Infection is acquired by ingesting food/beverages contaminated with eggs. Larvae penetrate the bowel wall and migrate primarily to the liver. *E. granulosus*, the most common *Echinococcus* to cause disease, is seen the Mediterranean region, Eastern Europe, Africa, South Amer-

ica, the Middle East, Australia, and New Zealand (Murray et al. 2005; Botero 2003; Binford and Connor 1976; Hasleton 1996; Travis et al. 2002; Fishman et al. 1998). *E. multilocularis* has a holo-arctic distribution (Alaska, Canada, the entire tundra region) and in some regions of Asia (Russia, China, Japan) and northern Europe (central and eastern France, Switzerland, Austria, Germany) (Gottstein and Reichen 2002). *E. vogeli* and *E. oligarthrus* are endemic in the tropical regions of South America (Botero 2003; D'Alessandro 1997; Rodrigues-Silva et al. 2002). An estimated 65 million people in endemic areas are infected (Murray et al. 2005).

### 3.4.1 Unilocular Cystic Echinococcosis

Unilocular cystic echinococcosis is caused by *E. granulosus*. Overall, the liver is the most commonly affected organ; however, thoracic disease is a very common presentation among adults (Beggs 1985; Bonakdarpour 1967; Polat et al. 2003) and perhaps the most common among children (Slim et al. 1971; Bloomfield 1980). Diverse organs in the chest can be affected by the disease either by growing cysts from the liver that traverse the diaphragm or by hematogenous spread (Gottstein and Reichen 2002). Although the lungs are affected in most cases of thoracic hydatid disease, other thoracic organs can be affected (pleural fissures, parietal pleura, chest wall, mediastinum, diaphragm and heart) (Kilic et al. 2006; Oguzkaya et al. 1997; Qian 1988).

Pulmonary hydatid disease can remain asymptomatic for long periods of time (months to years) or manifest with diverse symptoms including coughing, hemoptysis, biliptysis, pneumothorax, pleuritis, lung abscess, parasitic pulmonary embolism, anaphylaxis secondary to cyst rupture, or cyst superinfection (Gottstein and Reichen

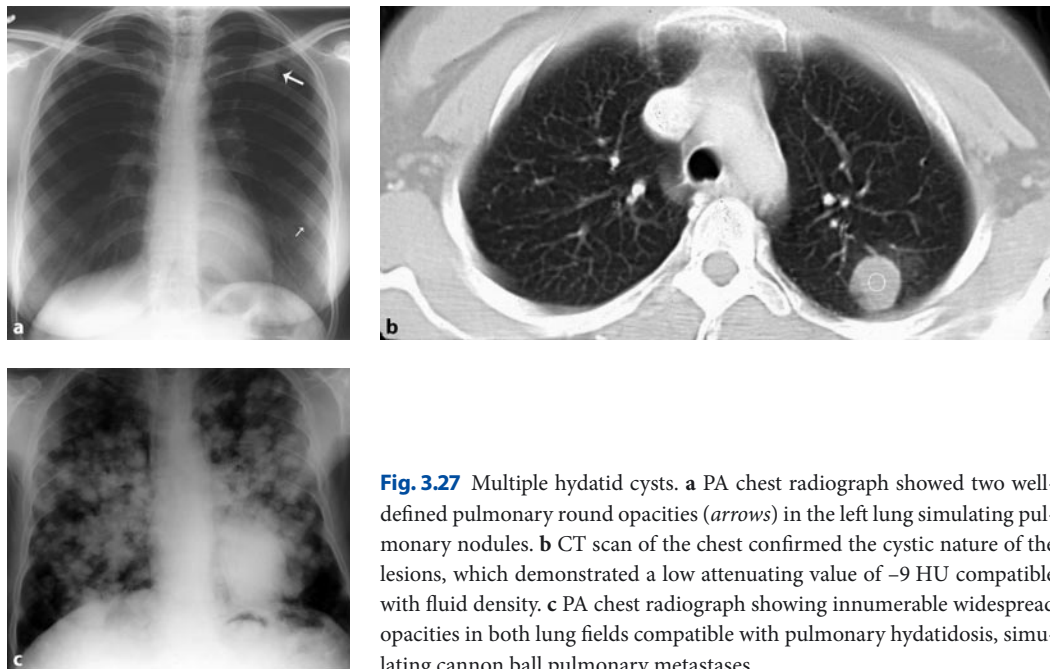
2002; Polat et al. 2003). Pathologically, cysts consist of three layers:

1. The pericyst, composed of fibroblasts, giant cells, and eosinophils, which form a rigid layer
2. An acellular, middle laminated membrane with nutrient functions
3. A thin, translucent inner germinal layer, containing various scolices and generating daughter cysts (Pedrosa et al. 2000)

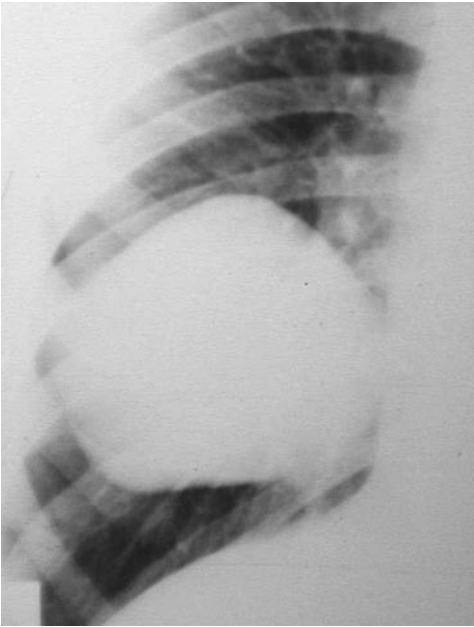
Eosinophilia is present in less than 15% of cases and generally occurs in the setting of leakage of antigenic material, i.e., cyst rupture. Although a variety of serologic studies are available, high false-negative and false-positive results have been described (Morar and Feldman 2003). Indirect hemagglutination and ELISA tests in association with abdominal ultrasonography can be used as screening tests in high-risk populations (Gottstein and Reichen 2002; Shambesh et al. 1999; MacPherson et al. 1987). Definitive diagnosis relies on the histopathology (Gottstein and Reichen 2002).

Radiographic and CT findings demonstrate cystic lesions, which can be solitary (60% of cases) or multiple (Fig. 3.27), unilateral or bilateral (20–50%). Lesions are predominantly identified in the lower lobes (60%) and can exhibit diameters between 1 and 20 cm. The coexistence of liver and lung disease is present in only 6% of patients (Polat et al. 2003). Uncomplicated cysts, the most common presentation (Saksouk et al. 1986), may be seen as round or oval masses that have well-defined borders (Fig. 3.28), do not enhance after contrast material injection, whilst the pericyst enhances if infected, and have a hypoattenuating content relative to the cap-

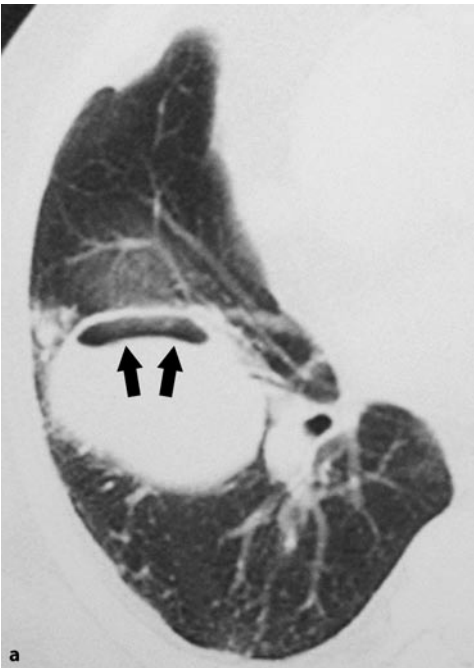
sule. On MRI, the cyst content appears characteristically hypointense on T1- and hyperintense on T2-weighted images with a well-defined wall that is isointense and hypointense respectively (Von Sinner et al. 1990, 1991). The development of daughter cysts inside the mother cyst is an early sign of degeneration. On MRI the contents of the mother cyst tend to be more intense with hypointense, viable daughter cysts on T1- and both the mother and the daughter cysts are isointense on T2-weighted images (Von Sinner et al. 1990, 1991). The “meniscus sign” or “crescent sign,” which is characterized by the presence of air between the pericyst and the laminated membrane, appears as growth continues and the cysts erode adjacent bronchioles (Figs. 3.29, 3.30). It is considered to be a sign of impending rupture. Cystic rupture may result in different radiologic signs. The “double arch,” “cumbo sign,” or “onion peel sign,” defined as the presence of the meniscus sign and an air–fluid level within the endocyst. The “water lily sign” represents an endocyst floating in a partially fluid-filled cyst (Fig. 3.31), whereas an endocyst floating in a cyst with no fluid is recognized as the “mass within the cavity” appearance (Beggs 1985; Bonakdarpour 1967; Polat et al. 2003; Pedrosa et al. 2000; Balikian and Mudarris 1974). Rupture can be associated with consolidation adjacent to the cyst (Saksouk et al. 1986). Atypical manifestations of cysts are less frequent, but have certainly been described (Koul et al. 2000). Radiographic and CT findings in transdiaphragmatic dissemination (Fig. 3.32) include pleural effusion, hemidiaphragm elevation, pulmonary consolidation, laminar basal atelectasis, pleural cysts, hydropneumothorax, bronchobiliary fistula and empyema (Balikian and Mudarris 1974; Koul et al. 2000; Bhatia 1997; Gouliamos et al. 1991; Ramos



**Fig. 3.27** Multiple hydatid cysts. **a** PA chest radiograph showed two well-defined pulmonary round opacities (arrows) in the left lung simulating pulmonary nodules. **b** CT scan of the chest confirmed the cystic nature of the lesions, which demonstrated a low attenuating value of  $-9$  HU compatible with fluid density. **c** PA chest radiograph showing innumerable widespread opacities in both lung fields compatible with pulmonary hydatidosis, simulating cannon ball pulmonary metastases



**Fig. 3.28** Hydatid disease (*E. granulosus*). PA chest radiograph, close-up view, shows a large cyst in the right lower lung. (Reproduced with permission from Radiographics, Martinez et al. 2005)



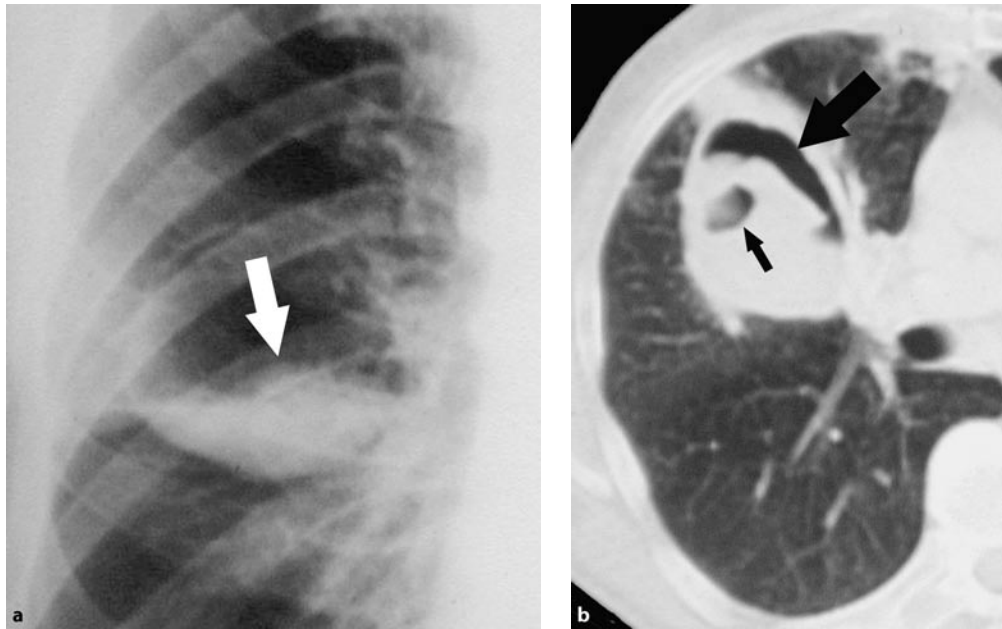
**Fig. 3.29** Pulmonary hydatid disease (*E. granulosus*). **a** Axial CT scan of the chest (lung window) shows a hypoattenuating crescent sign, the so-called meniscus sign (arrows). **b** Photomicrograph (HE,  $\times 40$ ) obtained after surgical resection demonstrates the inner germinal layer, to which several daughter protoscolices of *E. granulosus* are attached (Reproduced with permission from Radiographics, Martinez et al. 2005)



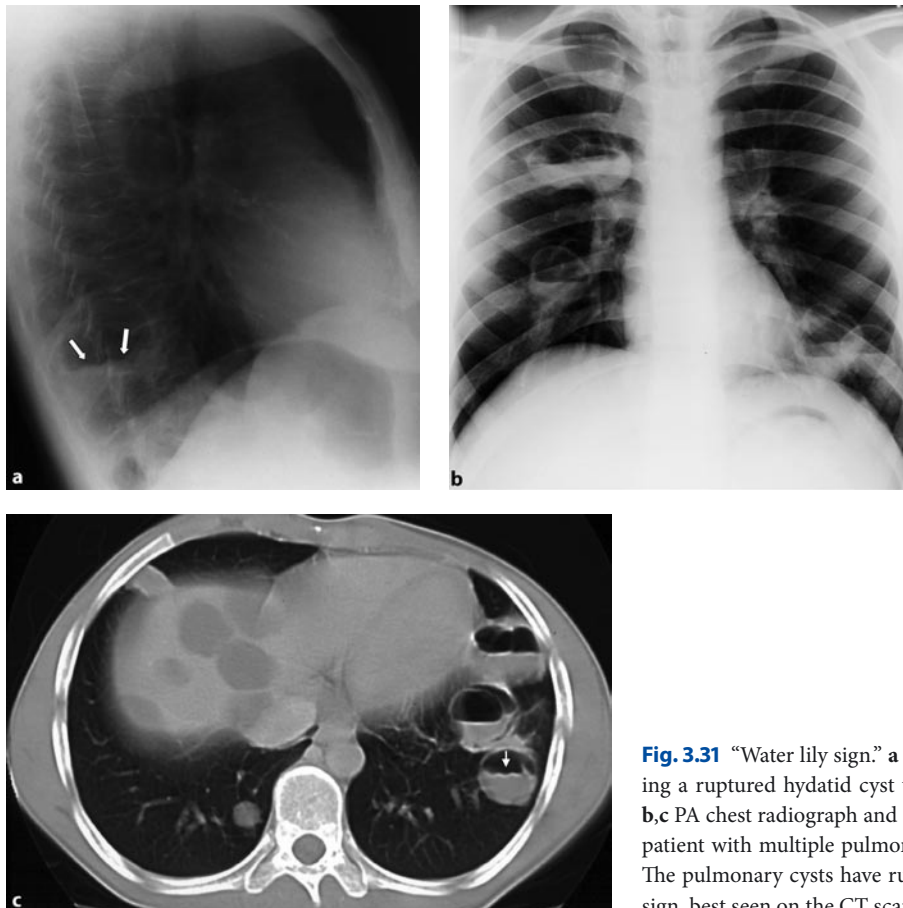
et al. 1975; Von Sinner 1990a; Kurkcuoglu et al. 2002a; Kabiri et al. 2001; Sahin et al. 2006; Nadir et al. 2005). Pulmonary cysts do not or very rarely calcify (Bonakdar-pour 1967; Polat et al. 2003). Similar radiologic findings have been reported in other intrathoracic extrapulmonary cysts, e.g., mediastinum, pericardium, hila, chest wall (Nadir et al. 2005; Cantoni et al. 1993; Dahniya et al. 2001; Marcos et al. 1969; Ozdemir et al. 1994; Tuzun and Hekimoglu 2001), cardiovascular system (Fig. 3.33) (Cantoni et al. 1993; Miralles et al. 1994; Malouf et al.

1985; Tercan et al. 2005; Odev et al. 2002; Franquet et al. 1999). In contrast to pulmonary hydatid cysts, mediastinal hydatid cysts do calcify at the periphery, i.e., they show pericystic calcifications (Fig. 3.33) (Zidi et al. 2006). In some patients with cardiac hydatid disease, cyst rupture may occur, leading to hydatid embolism to the pulmonary arteries (Fig. 3.34), or to the brain, causing a stroke.

In patients with peripheral intrapulmonary solitary or multiple coin lesions adjacent to the pleura and chest



**Fig. 3.30** Pulmonary hydatid disease (*E. granulosus*). **a** PA chest radiograph, cone view, shows cavitation with dependent floating soft tissue (arrow). **b** Axial CT scan of the chest (lung window) better characterizes the findings. There is air surrounding the pericyst (thick arrow) and inside the endocyst (thin arrow)

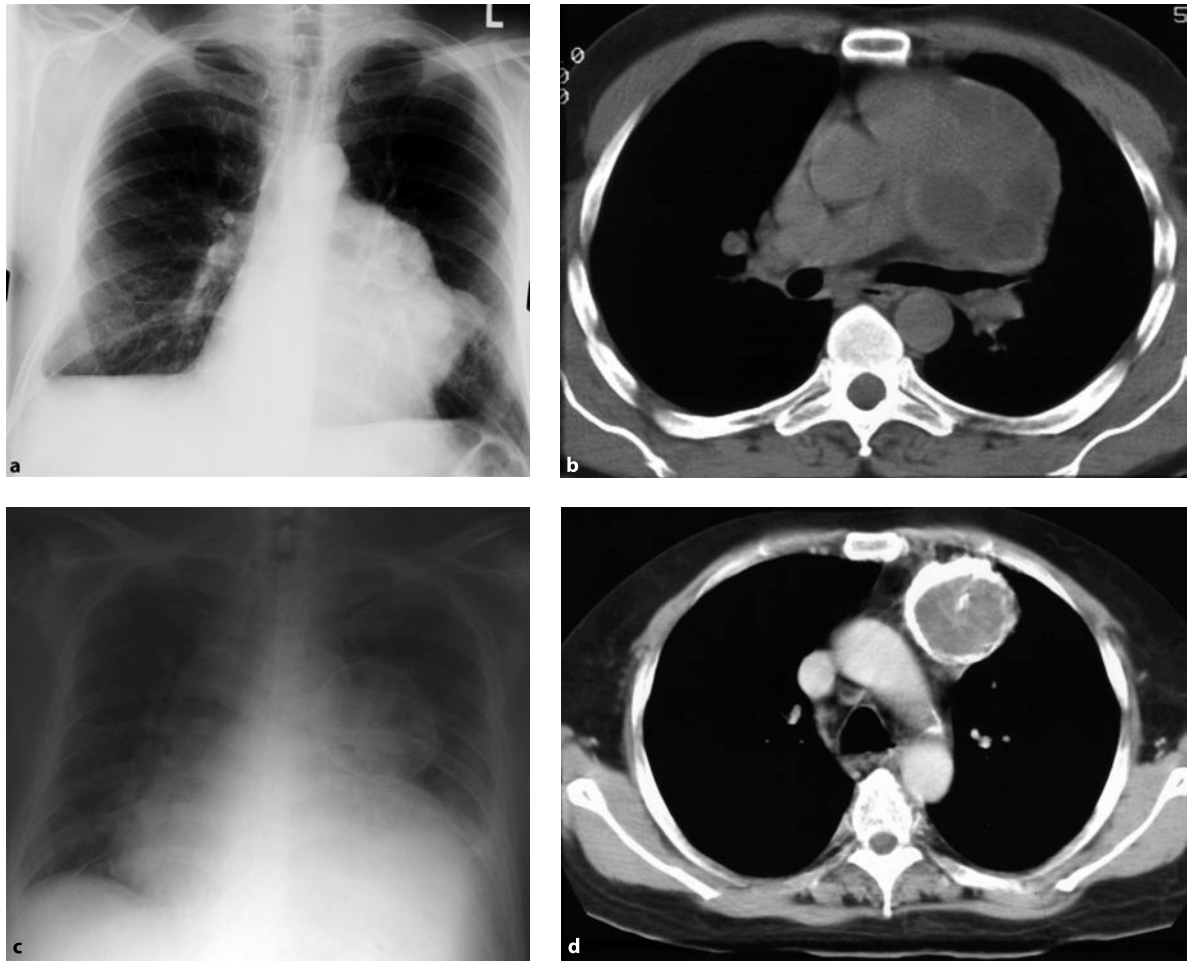


**Fig. 3.31** “Water lily sign.” **a** Lateral chest radiograph showing a ruptured hydatid cyst with a water lily sign (arrows). **b,c** PA chest radiograph and CT scan of the chest of another patient with multiple pulmonary and hepatic hydatid cysts. The pulmonary cysts have ruptured and exhibit a water lily sign, best seen on the CT scan (arrow)





**Fig. 3.32** Transdiaphragmatic dissemination of a hepatic hydatid cyst. **a** PA chest radiograph, cone view, showing right lower lobe consolidation and egg-shell calcification in the liver (*arrows*). **b–d** Ultrasound and CT images confirming the presence of a hepatic hydatid cyst that has ruptured through the diaphragm into the right pleural cavity (*star*) (Courtesy of Dr. Nadim Kanj, Department of Internal Medicine, American University of Beirut Medical Center, Beirut, Lebanon)

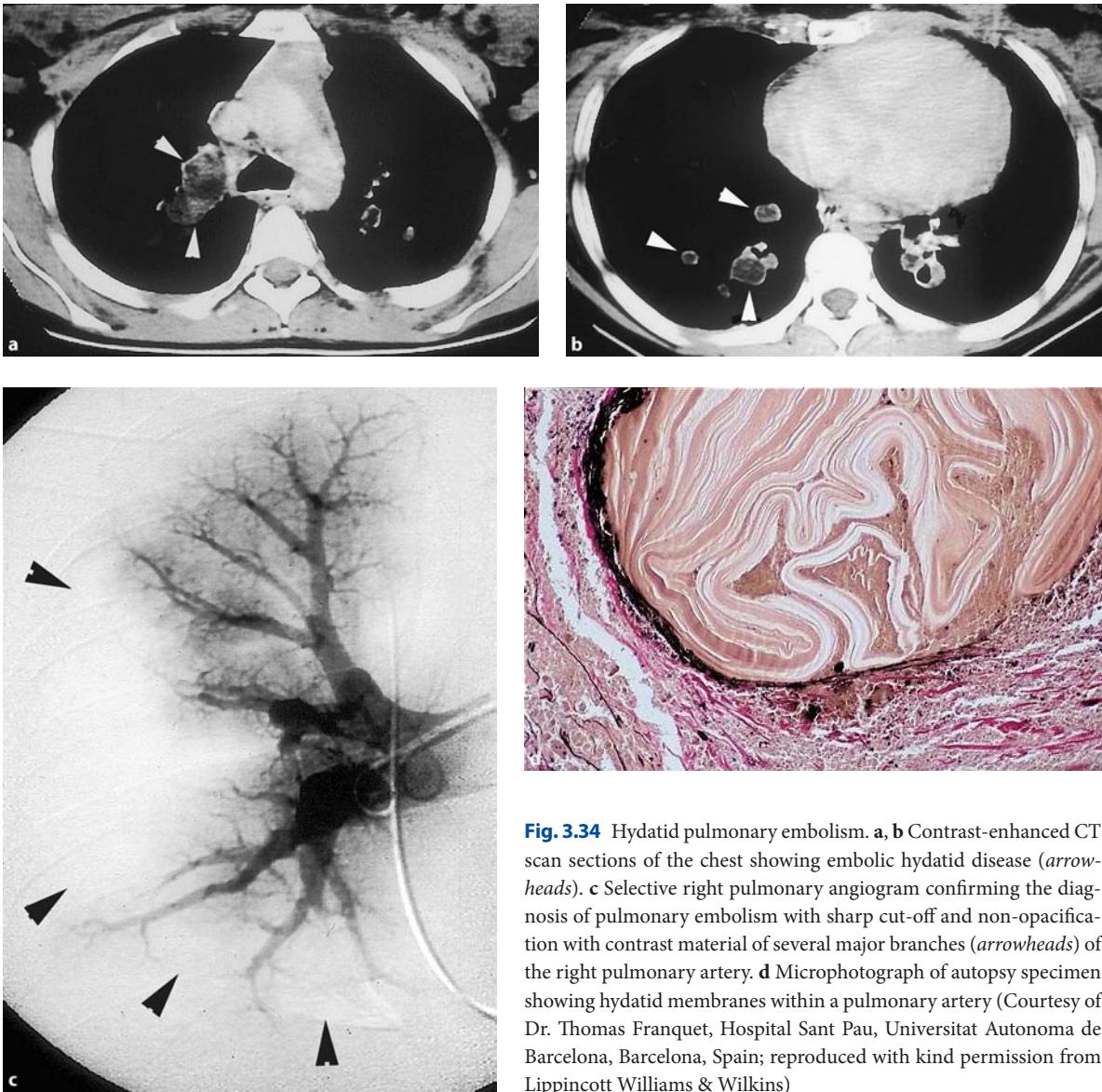


**Fig. 3.33** Pericardial and mediastinal hydatid cysts. **a** PA chest radiograph showing a left paracardiac lobulated mass. **b** Non-enhanced CT scan of the chest showed a pericardial hydatid cyst containing daughter cysts. **c,d** Portable chest radiograph and CT scan of the chest demonstrate a calcified inactive hydatid cyst in the anterior mediastinum

wall, ultrasound is helpful in demonstrating the cystic nature of the lesion (Fig. 3.35), thus suspecting hydatid disease. MRI and CT have been used to evaluate response to medical treatment (i.e., albendazole and praziquantel, etc.) in hepatic disease (Von Sinner et al. 1991; Von Sinner 1990b). The use of medical treatment in thoracic disease is controversial; however, it has been attempted with variable success [i.e., a cure rate of up to 30% (Fig. 3.36), persistence of disease in about 50%, and recurrence in up to 20% of all cases] in advanced disease, inoperable cases or patients who do not accept surgical treatment. Improvement is identified as disappearance or decrease in size of cysts as well as a decrease in surrounding inflammatory changes (Morar and Feldman 2003; Kurkcuoglu et al. 2002b; Morris et al. 1985; Horton 1989; Teggi et al. 1993; Kuzucu et al. 2004).

### 3.4.2 Alveolar Echinococcosis

This parasitosis of unknown incidence, caused by *E. multilocularis*, is less frequent than *E. granulosus* (Morar and Feldman 2003), but more aggressive. It is prevalent in Central Europe, Russia, western China, northern Japan, North America, particularly Alaska and Canada, and North Africa (Morar and Feldman 2003). Intermediate hosts include foxes and small rodents. Definitive hosts are dogs and cats. Humans become infected by accidentally ingesting the eggs of the tapeworm, excreted by the final hosts or during normal feeding by a variety of rodents and small lagomorphs (Vuitton et al. 2003). The infection behaves like a malignant liver tumor, invading and metastasizing to the brain, lungs and other organs (Mandell et al. 2005; Morar and Feldman 2003). If left



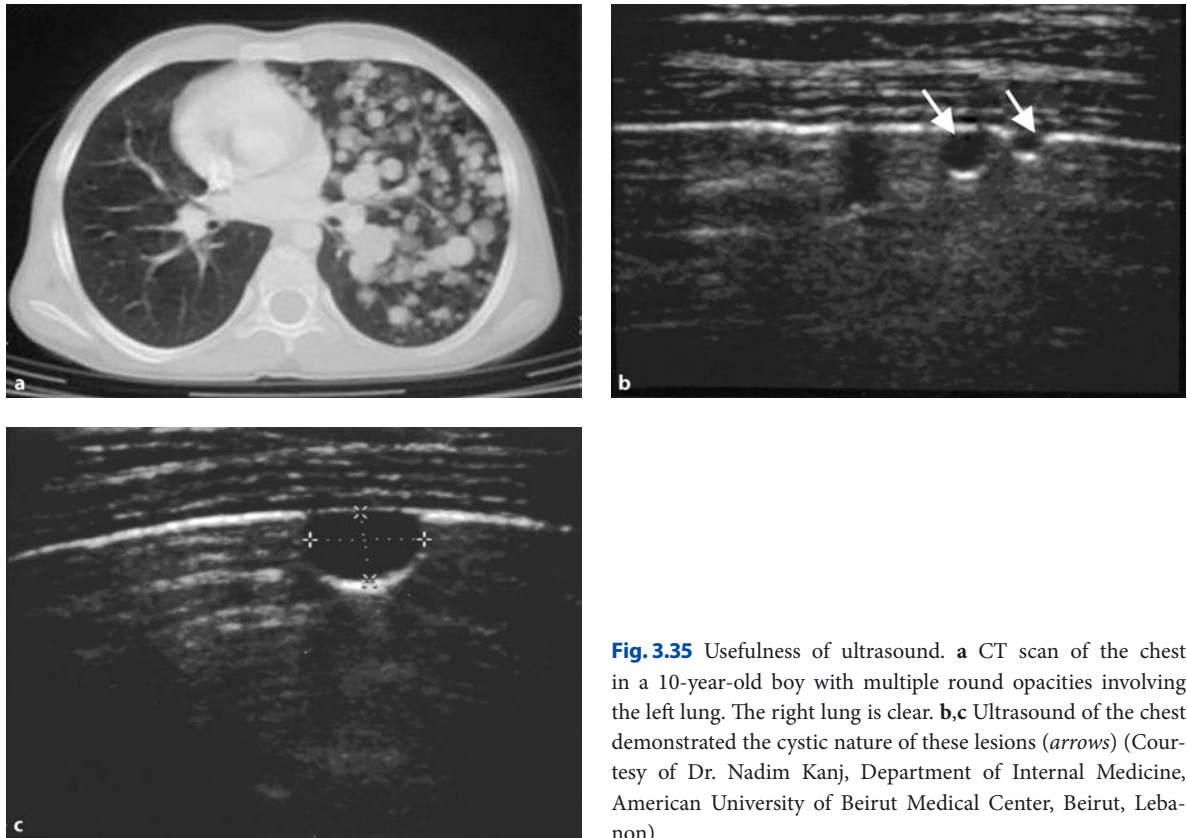
**Fig. 3.34** Hydatid pulmonary embolism. **a, b** Contrast-enhanced CT scan sections of the chest showing embolic hydatid disease (*arrowheads*). **c** Selective right pulmonary angiogram confirming the diagnosis of pulmonary embolism with sharp cut-off and non-opacification with contrast material of several major branches (*arrowheads*) of the right pulmonary artery. **d** Microphotograph of autopsy specimen showing hydatid membranes within a pulmonary artery (Courtesy of Dr. Thomas Franquet, Hospital Sant Pau, Universitat Autònoma de Barcelona, Barcelona, Spain; reproduced with kind permission from Lippincott Williams & Wilkins)

untreated, virtually 100% of patients will ultimately die within 15 years (Morar and Feldman 2003; Wilson et al. 1995; Ammann and Eckert 1996).

Clinical manifestations include fatigue, weight loss, coughing, and hemoptysis. According to the European Echinococcosis Registry, thoracic compromise, very often associated with liver disease, occurs by direct extension to the diaphragm, lungs, and pleura (12% of all cases) or metastatic spread to the lungs (~6% of all cases) (Ammann and Eckert 1996; Akinoglu et al. 1991; Kern et al. 2003). Metastasis to the right atrium (Etievent et al. 1986) and thoracic spine have also been described (Reuter et al. 2000). Definitive diagnosis can be achieved

by immunohistochemical and histological analysis. Serologic tests, also available, are important in the early detection of asymptomatic cases (Gottstein and Reichen 2002).

Metastases to the lungs appear as multiple, peripherally located, small, ill-defined, irregular lesions, less than 3 cm in diameter, which may or may not exhibit stippled or peripheral calcifications (Tuzan and Hekimoglu 2001; Jiang 2002; Treugut et al. 1980; Reittner et al. 1996). Variable changes in the right lung base occur when a liver mass invades the diaphragm and extends into the chest, including pleural effusion, empyema, and right lower lobe parenchymal nodules or masses due to cysts (Treugut et



**Fig. 3.35** Usefulness of ultrasound. **a** CT scan of the chest in a 10-year-old boy with multiple round opacities involving the left lung. The right lung is clear. **b,c** Ultrasound of the chest demonstrated the cystic nature of these lesions (arrows) (Courtesy of Dr. Nadim Kanj, Department of Internal Medicine, American University of Beirut Medical Center, Beirut, Lebanon)

al. 1980). Bronchobiliary fistula is a complication of direct extension. It can be suspected on CT examination by the coexistence of a liver mass extending through the diaphragm with clustered calcifications and air, and associated continuity with air bronchograms in the right lower lobe. The fistulous tracts can be identified on MR cholangiographic sequences. The diagnosis can be confirmed by demonstrating passage of the radiotracer or contrast material to the tracheobronchial tree on a Tc-HIDA hepatobiliary scintigraphy scan or endoscopic retrograde cholangiography respectively (Senturk et al. 1998).

Response to medical treatment with benzimidazoles, particularly in liver disease, has been demonstrated by imaging techniques (e.g., conventional radiographs, ultrasound, and CT scanning) in animal models (Taylor et al. 1989) and humans (Senturk et al. 1998; Wilson et al. 1992; Reuter et al. 2000; Ammann et al. 1994).

### 3.4.3 Polycystic Echinococcosis

*Echinococcus vogeli* causes polycystic echinococcosis, a relatively recently described echinococcosis endemic in

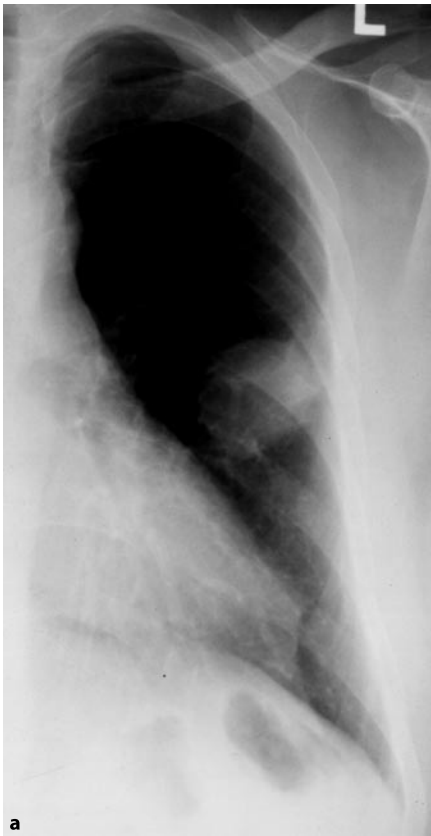
the tropical areas of Central and South America. Humans become infected after a complex cycle by ingestion of fluid/food contaminated with parasite eggs shed in bush dogs' feces. Polycystic echinococcosis shares several similarities with alveolar echinococcosis:

1. The liver is the target organ and thus the most commonly affected
2. It can affect the thoracic organs either by direct extension or hematogenous spread (metastasis) (D'Alessandro 1997; Martinez et al. 2005)

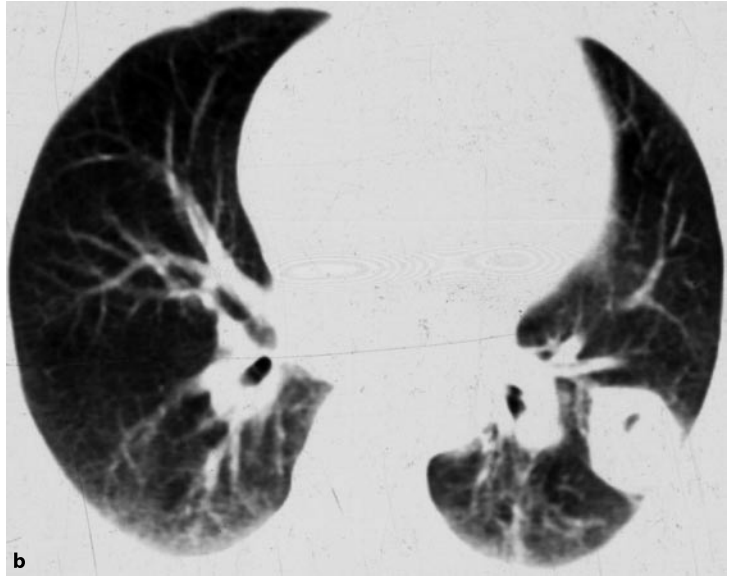
By the late 1990s, at least 86 cases had been recognized in the literature (D'Alessandro 1997), and today around 200 cases have been identified, but are thought to be only the tip of the iceberg (Dr. A. D'Alessandro, personal communication).

In hematogenous spread, solitary or multiple soft tissue nodules, rarely calcified, can be identified on the chest radiograph (Fig. 3.37). Mediastinal cysts, which can calcify, can manifest as mediastinal widening, and are better evaluated by CT scanning. Direct extension from liver disease usually demonstrates pleural effusion, pleural cysts, and right lower lobe parenchymal cysts. Con-





**Fig. 3.36** Successful medical treatment of a small pulmonary hydatid cyst. **a** Initial PA chest radiograph, cone view, at presentation showing a round lesion in the left mid lung field. **b** CT scan of the chest showed a small crescent of air within the lesion compatible with a hydatid cyst. **c** Follow-up chest radiograph, cone view, obtained after medical therapy, showed a larger crescent of air compatible with degeneration of the cyst. **d** Further follow-up chest radiograph, cone view, obtained after a 2-year interval showed complete healing with a residual linear scar at the site of the cyst



solidation surrounding parenchymal cysts is sometimes identified (Fig. 3.38). Infection of the thoracic wall demonstrates cystic expansion of the ribs and surrounding soft tissues (Fig. 3.39) (D'Alessandro 1997; Rodrigues-Silva et al. 2002; Martinez et al. 2005; Meneghelli et al. 1992a, 1986; D'Alessandro et al. 1979).

Awareness of this particular type of echinococcosis is important in endemic regions, since it shows an effective response to albendazole and, in contrast to hydatid disease, surgical treatment is considered as a second line option (D'Alessandro 1997; Meneghelli et al. 1986, 1992b). Response of thoracic disease to medical treatment has been verified by CT scanning, either as the disappearance of or a reduction in the size of the cysts (Meneghelli et al. 1992b).

A fourth type of *Echinococcus* (*E. oligarthrus*) has been isolated in wild and domestic cats and its larvae are found in agouties and other rodents. The distribution is similar to that of *E. vogeli*. Fortunately, only a few cases have been documented in humans (D'Alessandro 1997; Basset

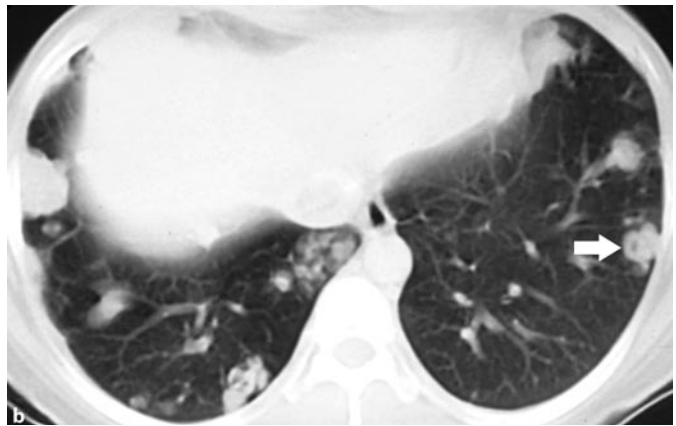
et al. 1998; D'Alessandro et al. 1995; Lopera et al. 1989; Salinas-Lopez et al. 1996).

#### 3.4.4 Sparganosis

*Spirometra* species occasionally induce eosinophilic pleuritis, or eosinophilic pulmonary granuloma simulating a malignant pulmonary nodule, in a few patients identified in Japan (Ishi et al. 2001; Iwatani et al. 2006).

#### 3.4.5 Cysticercosis

The central nervous system and muscles are the major site of involvement with cysticercosis; a case of disseminated cysticercosis with pulmonary involvement was reported from Brazil (Mamere et al. 2004). Pulmonary and chest wall musculature nodular lesions were detected on imaging by CT and MRI in the brain and thorax. Calci-

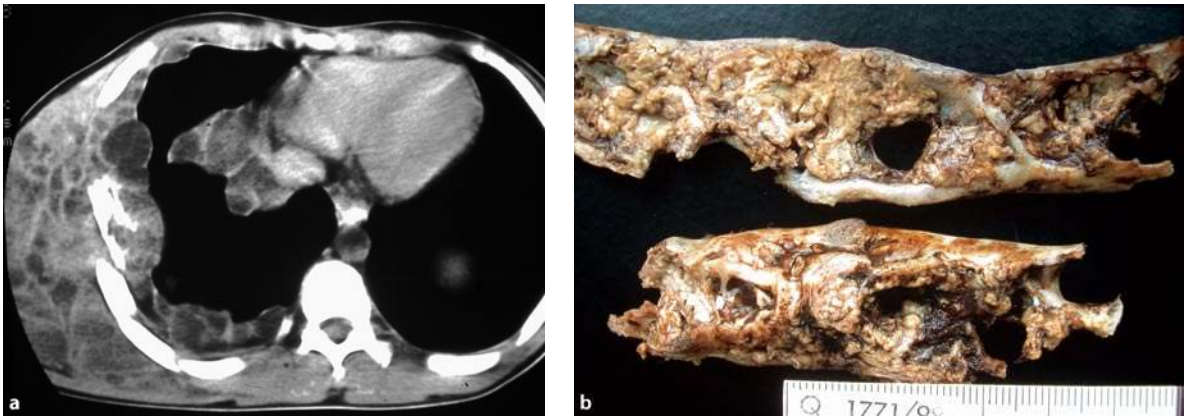


**Fig. 3.37** Polycystic echinococcosis (*E. vogeli*). **a** PA chest radiograph shows multiple bilateral soft tissue pulmonary nodules. **b** Axial CT scan of the chest (lung window) depicts multiple

pulmonary nodules. A cavitated nodule in the left lower lobe (arrow) reveals the cystic nature of nodules. *E. vogeli* was proven to be the causative agent



**Fig. 3.38** Polycystic echinococcosis (*E. vogeli*). Axial CT scan of the chest (soft tissue window) shows multiple cystic lesions in the liver. There is a pulmonary cyst in the left lower lobe (asterisk) with surrounding consolidation (arrows) reflecting the inflammatory process



**Fig. 3.39** Polycystic echinococcosis (*E. vogeli*) of the chest wall. **a** CT scan of the chest shows cystic thickening of the pleura with chest wall involvement. **b** Photograph of the surgically removed gross specimen demonstrates osseous expansion secondary to rib invasion (Reproduced with permission from Radiographics, Martinez et al. 2005)

fied burned-out lesions can be visualized by plain radiography (Fig. 3.40) in the chest wall muscles.

### 3.5 Trematodes (Flukes)

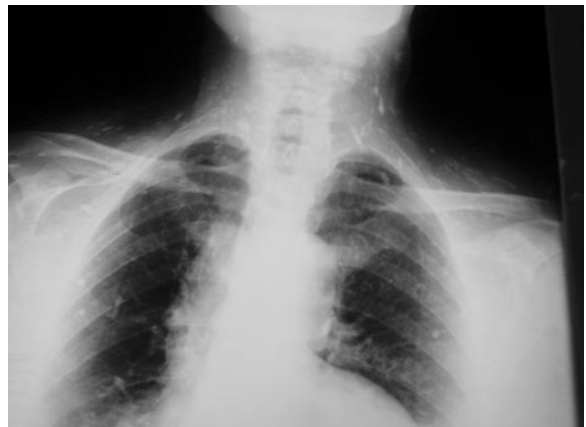
#### 3.5.1 Schistosomiasis

Schistosomiasis continues to be a major worldwide public health problem. It is estimated that 200 million people are infected around the world, mostly in Africa. One hundred and twenty million are symptomatic and 20 million have severe disease. Although the infection is endemic in more than 70 countries in all continents, 85% of affected patients live in Africa where Angola, the Central African Republic, Chad, Tunisia, Egypt, Morocco, Ghana, Madagascar, Malawi, Mozambique, Nigeria, Senegal, Sudan, the United Republic of Tanzania, Zambia, Mali, Uganda, and Zimbabwe seem to be the most commonly affected areas. Other affected countries include the Caribbean islands, Japan, Mauritius, Brazil, Cambodia, China, Laos, the Philippines, Botswana, Iran, and Iraq. Although progress has been achieved regarding control of schistosomiasis in the last 2 years, the infection continues to be a devastating and disabling disease, only surpassed by malaria. Mortality is calculated to be near 300,000 deaths per year (Chitsulo et al. 2000; World Health Organization 2006).

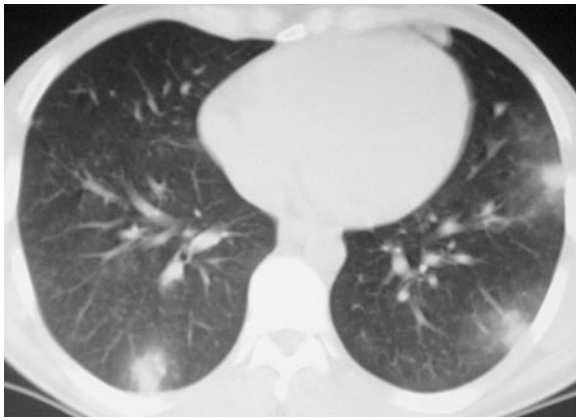
There are three important *Schistosoma* species: *S. hematobium*, *S. mansoni*, and *S. japonicum*. Although all three species can cause pulmonary disease (Morris and Knauer 1997), *S. mansoni* and *S. japonicum* are those most frequently observed (Nash et al. 1982). *S. mansoni* is endemic to Africa, Saudi Arabia, Egypt, Sudan, Madagascar, Brazil, Surinam, Venezuela, and Puerto Rico, whereas *S. japonicum* is more frequently seen in East

Asia. Infection is acquired through exposure of the skin to water contaminated with cercariae excreted by snails. This parasitic form has the ability to penetrate the skin or the intestinal wall, migrate to the lung and ultimately the liver, where it continues its life cycle (Murray et al. 2005; Botero 2003; Morris and Knauer 1997).

Acute pulmonary schistosomiasis (3–8 weeks after parasitic skin penetration), which results from type 3 immunologic reaction in which eosinophils are sequestered in the lungs, usually resolves within a few weeks. Clinical manifestations often include shortness of breath, wheezing, and dry coughing. The diagnosis is suggested in patients who live in or have traveled to endemic areas and who present with eosinophilia (Morris and Knauer 1997). Patients may have both clinical and radiologic manifestations after the onset of treatment. Katayama fever, more commonly described with *S. mansoni* and *S. japonicum*,



**Fig. 3.40** Cysticercosis. Plain radiograph of the chest showing calcified cysticerci of the chest wall and neck muscles



**Fig. 3.41** Early pulmonary schistosomiasis. Axial CT scan of the chest shows multiple peripheral subpleural pulmonary nodules surrounded by a halo of ground-glass opacity or attenuation in the lower lobes bilaterally. *S. mansoni* was the proven etiologic factor (Reproduced with permission from Radiographics, Martinez et al. 2005)

refers to pulmonary symptoms that coincide with febrile illness, and is thought to represent an immunologic reaction to the parasite's eggs. Associated symptoms include urticaria, arthralgia, hepatosplenomegaly, hepatitis, eosinophilia, and pulmonary disease. The acute infection is more commonly seen in individuals traveling to endemic regions (Morris and Knauer 1997). Chest radiograph can be normal (Lambertucci et al. 1997, 2005) or show multiple, bilateral, ill-defined, nodular opacities (Lambertucci et al. 1997; Nguyen et al. 2006; Waldman et al. 2001; Cooke et al. 1999; Schwartz 2002; Schwartz et al. 2000) and less frequently, consolidation (Lambertucci et al. 1997; Fatureto et al. 2003) or reticulonodular opacities (Schwartz 2002). These abnormalities are usually transitory and resolve within weeks (Cooke et al. 1999). CT scanning can show solitary (Fatureto et al. 2003) or multiple subpleural soft tissue nodules that may exhibit surrounding ground-glass opacities (the so-called "halo" sign) (Fig. 3.41). Sensitivity is low for the examination of stool and urine for eggs at this stage of infection, although rectal biopsy may help improve the diagnosis. ELISA is a better initial study, but positive results must be confirmed with enzyme-linked immunoelectrotransfer blot. Because serologic tests remain positive for years, even after treatment, they are not helpful for the diagnosis of reinfection or for determining the success of treatment (Schwartz 2002; Schwartz et al. 2000).

Chronic pulmonary disease occurs in patients with hepatosplenic disease in whom portal hypertension and collateral circulation develop. Ultimately, eggs can shunt from the portal system and reach the arterial pulmonary circulation (Schwartz 2002; Andrade and Andrade 1970).

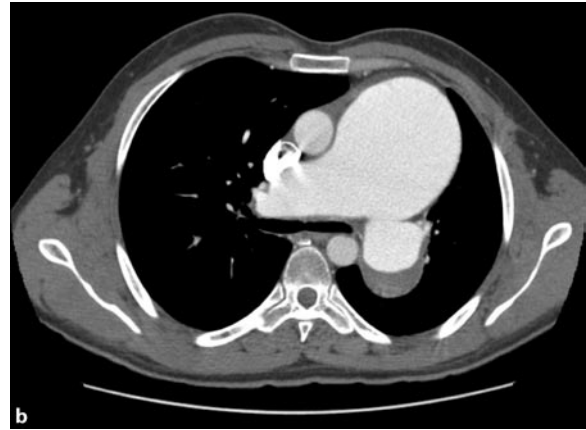
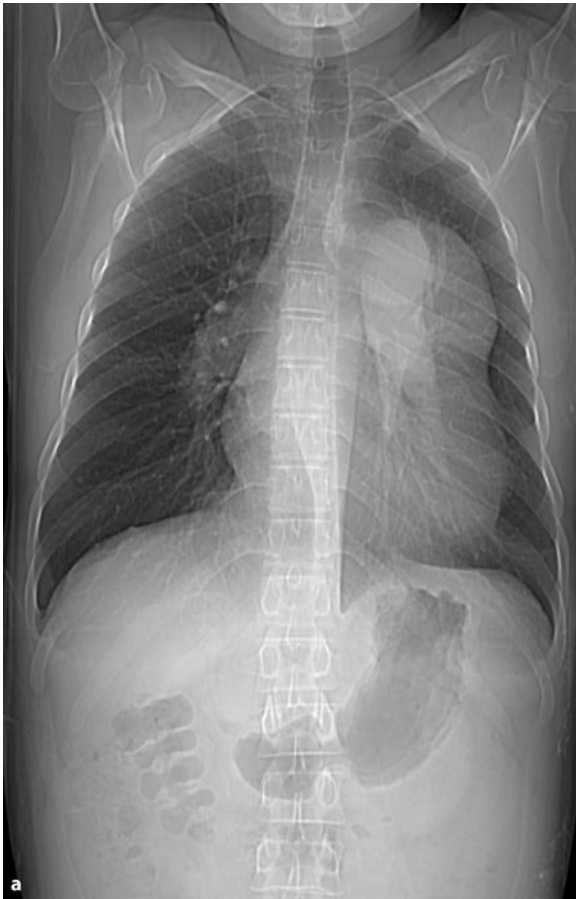
A granulomatous reaction to eggs deposited in the pulmonary vasculature leads to intimal fibrosis, pulmonary hypertension, and cor pulmonale. The clinical picture includes dyspnea, chest pain, fatigue, palpitations, coughing, and finally right-sided heart failure. Radiographic and CT findings (Fig. 3.42) include right ventricular enlargement, pulmonary hypertension, dilatation of the azygous vein (due to abundant collaterals draining a hypertensive portal system), and scattered calcified nodules i.e., granulomas throughout the lungs (Phillips et al. 1975; Pagan Saez 1985). Atypical presentation simulating tuberculosis and neoplasm has been described (El Mallah and Hashem 1953; Schaberg et al. 1991; Tizes et al. 1967). Diagnosis is made by identifying eggs in stool or urine samples or by rectal biopsy; sometimes lung biopsy has been proved effective. Serologic tests are not very helpful because they cannot help differentiate between a former infection and current disease (Richert and Krakaur 1959; Schwartz 2002; Schwartz et al. 2000; Phillips et al. 1975).

### 3.5.2 Paragonimiasis

Ingestion of raw or partially cooked freshwater crabs or crawfish, containing *Paragonimus westermani* (lung fluke) or other *Paragonimus* species, is the cause of human paragonimiasis. Alternatively, undercooked meat of infected pigs and wild boars are also sources (Nakamura-Uchiyama et al. 2002). The infection is endemic in areas of East Asia, Southeast Asia, Latin America (primarily Peru), and Africa (primarily Nigeria) (Barrett-Connor 1982; Botero 2003; Binford and Connor 1976; Hasleton 1996; Travis et al. 2002; Murray et al. 2002; Fishman et al. 1998). Many cases have been reported in the United States among Indo-Chinese and Latin American immigrants (Nakamura-Uchiyama et al. 2002; Johnson and Johnson 1983; Velez et al. 2002). It is believed that 195 million people are at risk, and 20.7 million are infected in endemic areas (Murray et al. 2002).

After ingestion, the infective larvae of *P. westermani* penetrate the duodenum and migrate to the peritoneal cavity and abdominal wall. Ultimately, parasitic forms travel through the diaphragm to the pleura and lungs (Velez et al. 2002). Although paragonimiasis affects primarily the lungs, skin and central nervous system compromise is also well described. Patients present with fever, chest pain, and respiratory symptoms such as chronic coughing and hemoptysis. Peripheral blood eosinophilia is present in more than 80% of patients (Nakamura-Uchiyama et al. 2002). Diagnosis is confirmed by detecting parasite eggs in the sputum, pleural fluid, or feces; in addition, larvae can often be found at bronchial brushing and washing. Intradermal and serologic tests are also available (Nakamura-Uchiyama et al. 2002).





**Fig. 3.42** Pulmonary hypertension due to chronic cardiopulmonary schistosomiasis. **a** Scout image of a CT scan of the thorax showing marked enlargement of the main pulmonary trunk, prominent pulmonary arteries demonstrating peripheral pruning, and hypertrophy of the right ventricle. **b** Contrast-enhanced CT scan of the chest showing marked enlargement of the main pulmonary artery trunk and the pulmonary arteries. (Courtesy of Dr. Dany Jasinowodolinski, Fleury-Sao Paulo, Brazil)

Radiologic findings correlate well with the stage of the disease. The penetration of juvenile worms through the diaphragm into the pleural cavity can cause pleural effusion, pneumothorax (Fig. 3.43) and potentially empyema. Migration of worms through the lungs usually manifests as consolidation (Fig. 3.44). Band-like opacities extending from the pleural surface can be identified at this stage, usually representing worm migration tracts (Fig. 3.45). As parasites grow, they tend to settle down. At this time, consolidation resolves and thin-walled cysts appear. When cysts are fluid-filled they manifest on the chest radiograph as round soft tissue nodules (Fig. 3.46), 0.5 to 4 cm in diameter. CT can better identify cysts, even when they are smaller (i.e., 5–15 mm in diameter) (Fig. 3.46) or in the presence of surrounding consolidation due to their hypodense content. If bronchial communication develops, air inside the cyst is better demonstrated on both the plain radiograph and CT (Fig. 3.47). A characteristic appearance at this stage is the ring shadow, which is characterized by a thin-walled cavity with a crescent-shaped opacity inside representing the parasite, usually less than 3 mm thick, and a crescent-shaped area of increased

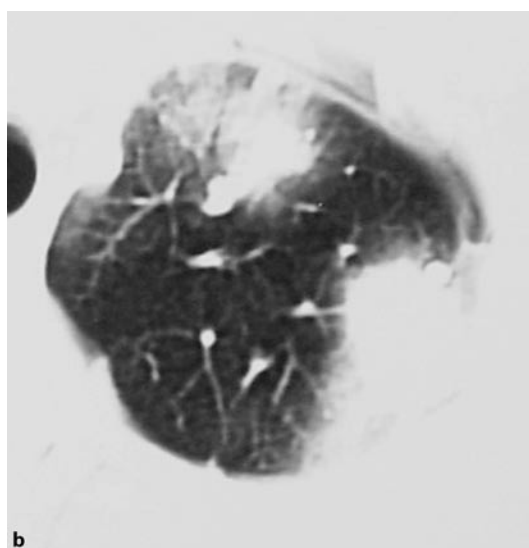
opacity or hyperattenuation within the cyst that represents worms attached to the wall (Fig. 3.48) (Nakamura-Uchiyama et al. 2002; Johnson and Johnson 1983; Velez et al. 2002; Im et al. 1992, 1993, 1997; Mukae et al. 2001; Sharma 1989). Loeffler's-like syndrome with transitory and/or migratory consolidative or ground-glass opacities has also been described (Bahk 1962; Suwanik and Harinsuta 1959). During and after treatment, fibronodular changes can develop in areas previously affected. Calcifications can be present; however, their presence should raise the question of possible coexistent tuberculosis. Fibrosis and cicatricial emphysema, frequent in tuberculosis, are virtually absent in paragonimiasis (Im et al. 1993, 1997).

### 3.6 Arthropods – Pentastomiasis

Pentastomiasis or porocephalosis is a zoonosis caused by *Armillifer armillatus* that affects snake-eating Africans (Guardia et al. 1991). The parasite penetrates the bowel wall, and migrates into the peritoneum to reach the liver and occasionally the lungs (Fig. 3.49).



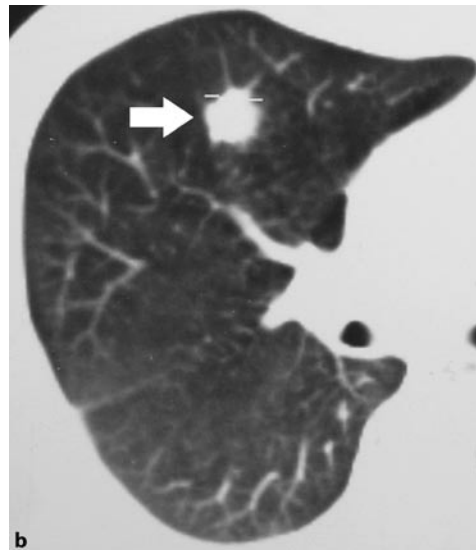
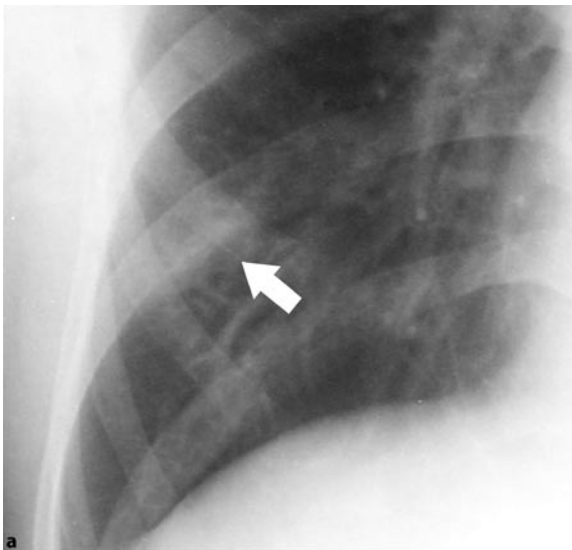
**Fig. 3.43** Thoracic paragonimiasis. Axial CT scan of the chest, close-up image, demonstrates left-sided pleural effusion (*arrows*) and pneumothorax (*asterisk*). A small area of consolidation is identified in the lingula (*arrowhead*)



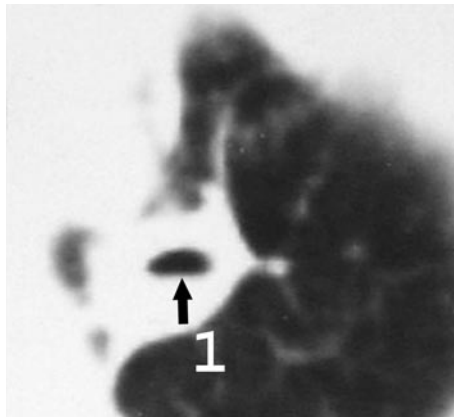
**Fig. 3.44** Thoracic paragonimiasis. **a** PA chest radiograph, cone view, shows heterogeneous opacities throughout the left hemithorax. **b** Axial CT scan of the chest shows subsegmental areas of consolidation (Reproduced with permission from Radiographics, Martinez et al. 2005)



**Fig. 3.45** Thoracic paragonimiasis. Axial CT scan of the chest, close-up image, shows band-like opacity in the right middle lobe (*arrow*) that represents worm migration tracts



**Fig. 3.46** Thoracic paragonimiasis presenting as a solitary pulmonary nodule. **a** PA chest radiograph, cone view, shows an ill-defined opacity (*arrow*) in the right middle lobe. **b** Axial CT scan of the chest, close-up image, shows to better advantage a round soft tissue nodule (*arrow*) in the right middle lobe. Histopathologic analysis performed after surgical resection demonstrated *P. westermani*. (Reproduced with permission from Radiographics, Martinez et al. 2005)



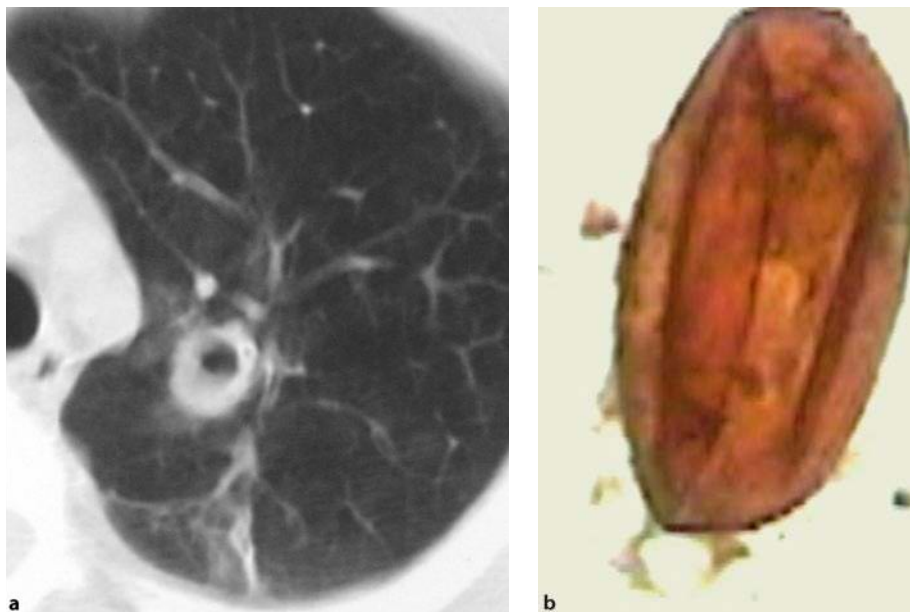
**Fig. 3.47** Thoracic paragonimiasis. Axial CT scan of the chest, close-up image, depicts a cavitated nodule in the left upper lobe with thick walls. A small air–fluid level is also identified (arrow)

### 3.7 Eosinophilic Pneumonias/Pulmonary Infiltrates with Eosinophilia/Eosinophilic Lung Diseases

Eosinophilic pneumonias (EP) or lung diseases are rare, and are characterized by the common finding of increased eosinophilia in blood and or tissue, i.e., pulmo-

nary infiltrates, the so-called pulmonary infiltrates with eosinophilia (PIE), a term coined in 1952 which covers a wide spectrum of diseases. It is therefore important to differentiate between parasitic and nonparasitic causes of PIE.

Parasitic EP occurs in almost all metazoan helminthic infections during larval migration of the parasite through the lungs, producing VLM, so-called Loeffler's syndrome described in 1932, and presumably caused by the *Ascaris* larvae. In patients who have not lived in tropical or subtropical regions *Toxocara* and *Ascaris* species should be considered, while for patients who have lived in tropical or subtropical regions *Paragonimus* species, *Strongyloides* species, *Ancylostoma* species, *Schistosoma* species, *Necator americanus*, *Wuchereria bancrofti*, and *Brugia malayi* should be taken into consideration. Tropical pulmonary eosinophilia (TPE) caused by filarial worms *W. bancrofti* and *B. malayi* is the most serious parasitic eosinophilic lung disease, in which cases have typically been reported to masquerade as acute or refractory bronchial asthma. Eosinophils are recruited to the lung to kill the parasite. Despite larval migration through the lungs, there is usually no permanent lung damage (Chitkara and Krishna 2006). Patients present with respiratory symptoms with or without fever. Chest radiographs and CT scans of the chest show migratory peripheral pulmonary infiltrates or consolidation or ground-glass opacities. Blood eosinophilia may or may not be present. The diagnosis is confirmed by the identification of eggs or larvae



**Fig. 3.48** Thoracic paragonimiasis. **a** Axial CT scan of the chest, close-up image, demonstrates a relatively thin-walled cavity with a crescent-dependent opacity representing the actual parasite. **b** Photomicrograph shows an egg of *P. westermani* obtained after the bronchoalveolar lavage





**Fig. 3.49** PA chest radiograph showing numerous calcified encysted *Armillifer armillatus* nymphs scattered in the lung fields. (Courtesy of Dr. Gordon Gamsu, Weill-Cornell Medical Center, New York, NY, USA)

in stool, sputum, pleural fluid, bronchoalveolar lavage, or in tissue specimens from transbronchial or thoracoscopic biopsies of the diseased lung segments. Serologic testing is also confirmatory. If all these tests are not diagnostic, empirical treatment with antiparasitic drugs for 2 weeks with resolution of the pulmonary symptoms and radiographic changes is diagnostic.

The nonparasitic causes of PIE include allergic conditions, auto-immune diseases and vasculitides, malignancies, and lastly idiopathic causes. For a detailed description of these pathologies, the reader is referred to the excellent review articles on this subject by Cottin and Alberts (Cottin and Cordier 2005; Alberts 2004).

For clinical understanding, eosinophilic lung diseases (ELD) may be also classified into two groups: intrinsic (primary, cryptogenic, or idiopathic) in which the cause is not known and representing approximately 20% of ELD, and extrinsic (secondary) in which the cause is known. ELD of unknown cause include: simple pulmonary eosinophilia (SPE), acute eosinophilic pneumonia (AEP), chronic eosinophilic pneumonia (CEP), bronchiolitis obliterans pneumonitis (BOOP) or organizing pneumonia, and idiopathic hypereosinophilic syndrome (IHS). ELD of known cause include:

1. Infectious diseases: fungal infections and hypersensitivity (coccidioidomycosis, histoplasmosis), bacterial and mycobacterial infections (chronic brucellosis, tuberculosis), and parasitic infections (almost all metazoan infestations are accompanied by eosinophilia, mainly during parasitic migration, citing *Strongyloides stercoralis*, *Ascaris lumbricoides*, *Schistosoma* and filarial species, *Paragonimus westermani*, *Ancylostoma duodenale*, *Toxocara* species, *Clonorchis sinensis*, *Echinococcus* species).

2. Allergic conditions: allergic bronchopulmonary aspergillosis (ABPA), bronchocentric granulomatosis (BCG), drug reactions or drug-induced pulmonary eosinophilia.
3. Auto-immune diseases and eosinophilic vasculitides: allergic granulomatosis and angiitis (Churg-Strauss syndrome), Wegener's granulomatosis, polyarteritis nodosa (PAN), Langerhan's cell granulomatosis or histiocytosis X, and sarcoidosis.
4. Neoplasm-related eosinophilia: systemic mastocytosis, eosinophilic leukemia, Hodgkin's and B-cell non-Hodgkin's lymphoma, T-cell lymphoblastic leukemia/lymphoma, large cell tumors of the lung, squamous cell carcinomas of the cervix, vagina, penis, skin, and nasopharynx, adenocarcinomas of the stomach, colon, and uterus, and transitional cell bladder carcinoma (Jeong et al. 2007; Savani and Sharma 2002).

### 3.8 Conclusion

Parasite-induced pulmonary diseases result in various imaging patterns that are nonspecific and mimic other inflammatory and neoplastic diseases. The pulmonary parasitic diseases are diagnosed by identification of eggs or larvae in stool, sputum, bronchoalveolar lavage, pleural fluid, tissue specimens from lung biopsy, and serology tests.

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