Parasitic diseases of the respiratory tract

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Purpose of review

The human pulmonary system can be affected by a variety of parasites. This review focuses on the most common parasitic infestations involving the lung, except for protozoon disease.

Recent findings

In many parasitic lung conditions, the clinical manifestations and the imaging findings are non-specific and can make diagnosis challenging. Hydatid disease and paragonimiasis involve the lung directly. Chronic schistosomiasis can lead to pulmonary hypertension. *Strongyloides stercoralis* infestation is capable of transforming into a fulminant fatal disease. In many types of nematode infestations, the pulmonary phase can cause acute eosinophilic pneumonia. Chest radiographs of patients with paragonimiasis and dirofilariasis can cause diagnostic confusion. Cases of tropical pulmonary eosinophilia typically present with refractory bronchial asthma. Most of these diseases are initially diagnosed by detecting eggs or larvae in stool, sputum, pleural fluid or tissue, and are confirmed by serologic testing. Cystic hydatid disease generally requires surgical treatment, whereas almost all other parasitic lung conditions can be treated medically.

Summary

Although most parasites that affect the lung are endemic to tropical and subtropical regions, immigration and travel practices have resulted in transfer of these diseases to other areas. It is important for physicians to know the epidemiologic characteristics, clinical presentations, and treatments of choice for these conditions.

Keywords

eosinophilic pneumonia, hydatid disease, lung, parasitic disease

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Abbreviations

CT computed tomography
TPE tropical pulmonary eosinophilia

1070-5287

Introduction

Parasitic disease is a major cause of morbidity and mortality worldwide. The human pulmonary system can be affected by a variety of parasites. These organisms can enter the lungs during the migration phase of their life cycle before reaching their target destinations. They can also travel there by embolic spread or direct invasion, and can be a primary infestation or a feature of more generalized disease. The most important parasitic conditions that affect the lung are hydatid disease, paragonimiasis, schistosomiasis, ascariasis, hookworm infestations, dirofilariasis, tropical pulmonary eosinophilia, toxocariasis, amebiasis and malignant tertian malaria. This review focuses on the most common helminthic infestations of the lung (Table 1). Protozoon diseases, which include amebiasis and malignant tertian malaria, are not discussed.

Hydatid disease

Hydatidosis is one of the most geographically widespread zoonoses in the world, and treatment remains controversial. In light of this, this review deals with hydatid disease in particular depth. The condition is caused by the tapeworm *Echinococcus granulosus*. Four species of *Echinococcus* are recognized, but the vast majority of human infestations with these cestodes are caused by *E. granulosus* [1]. This organism is transmitted to humans in settings where other animals involved in its life cycle (such as dogs or sheep) are present. *E. granulosus* is concentrated in sheep-raising areas, such as the Mediterranean region, Eastern Europe, Africa, South America, the Middle East, Australia, New Zealand and China [2,3]. Humans may accidentally ingest *E. granulosus* eggs via direct contact with one of this worm's final hosts (usually a dog) or may ingest food or fluids contaminated with faeces that contain the eggs [4•]. There is less human exposure to *E. multilocularis*, but the real extent of the disease is unknown [1]. Its life cycle involves wild canines, usually foxes and wolves, as definitive hosts and mainly rodents as intermediate hosts. Domestic dogs and cats may also become infected and can transmit the infection to humans [1,5•]. *E. multilocularis* is more common in colder areas, such as the Arctic and some regions of Asia and west-central Europe [1–3,4•,5•,6–9]. The primary location of the alveolar hydatid is the liver. Primary lung affection is not described [3]. *E. multilocularis* may, however, initiate the formation of distant metastasis in the lung and other organs [3,4•,9–11]. The species *E. vogeli* and *E. oligarthus* are endemic to

Table 1 Epidemiology, mode of transmission, clinical features, diagnosis and treatment of parasitic diseases of the respiratory tract

Parasites and diseases	Geographic distribution	Mode of transmission	Clinical presentation	Diagnosis	Treatment
Cestodes					
Hydatid disease					
Echinococcus granulosus	Worldwide (particularly sheep-raising areas)	Oral (ingestion of contaminated food or fluids)	Chest pain, cough, expectoration of cyst contents, haemoptysis, hypersensitivity reactions, pleural lesions	Radiological findings, serology	Surgery, albendazole, mebendazole
Trematodes					
Paragonimiasis					
Paragonimus spp	South-east Asia, South America, Africa	Ingestion of raw or undercooked crabs or crayfish	Fever, cough, hemoptysis, chest pain, pleural lesions	Demonstration of eggs in bronchial secretions or lung tissue, serology	Praziquantel, bithionol
Schistosomiasis					
Shistosoma japonicum *Shistosoma mansoni* *Shistosoma haematobium*	Far-east Asia, sub-Saharan Africa and South America	Skin penetration (in water)	Katayama fever, pulmonary hypertension, cor pulmonale	Demonstration of eggs in stool or urine, serology	Praziquantel
Nematodes					
Strongyloidiasis					
Strongyloides stercoralis	Tropical and sub-tropical areas	Skin penetration (in soil)	Loeffler-like syndrome, hyperinfection syndrome	Demonstration larvae in bronchial secretions, pleural fluid, stool and duodenal aspirate, serology	Thiabendazole, ivermectin
Ascariasis					
Ascaris lumbricoides	Africa, Asia, Central and South America	Oral (ingestion of contaminated food or fluids)	Loeffler-like syndrome	Demonstration of eggs in stool	Mebendazole, albendazole
Hookworm disease					
Ancylostoma duodenale	Tropical and subtropical areas of Africa, Asia and America	Skin penetration (in soil) and oral contamination	Loeffler-like syndrome	Demonstration of eggs in stool	Mebendazole, albendazole
Necator americanus		Skin penetration (in soil)			
Dirofilariasis					
Dirofilaria immitis	Tropical and subtropical areas	Mosquito bite	Coin lesion on chest x-ray	Identification of worm in lung tissue	None
Tropical pulmonary eosinophilia					
Wuchereria bancrofti *Brugia malayi*	Tropical and subtropical areas (particularly India, South-east Asia)	Mosquito bite	Eosinophilic pneumonia, paroxysmal coughing, dyspnoea and wheezing	Serology	Diethylcarbamazine
Visceral larva migrans					
Toxocara canis *Toxocara catis*	Worldwide	Oral (ingestion of soil contained the eggs)	Eosinophilic pneumonia, episodes wheezing or asthma	Serology	Thiabendazole, albendazole

South America. These parasites cause polycystic echinococcosis and are of minor importance [1,4•,10].

Hydatid disease primarily affects the liver, and one of its potential local complications is transdiaphragmatic thoracic involvement. The lung can also become involved via haematogenous or lymphatic dissemination [3,12]. The clinical presentation of hydatidosis of the lung depends on whether the cysts are intact or ruptured. Most intact hydatid cysts in pulmonary tissue are either noted as incidental findings or cause manifestations such as cough, dyspnoea or chest pain. The signs and symptoms of these cysts almost always result from pressure caused by the lesion. Hydatid cysts in lung tissue may rupture into the pleural space or into a bronchus. Perforation into a bronchus can lead to expectoration of vomit-like cystic fluid and remnants of parasitic membrane, as well as recurrent haemoptysis. Patients with ruptured cysts in the lung may also present with persistent pneumonia [13] or an infected cyst lesion [14••,15•]. Sometimes ruptured hydatid cysts cause severe complications, such as massive haemoptysis [16•], hypersensitivity reaction [17,18], asthma-like symptoms [19] or sepsis [14••]. Perforation of cyst into the pleural space may cause pneumothorax, tension pneumothorax, pleural effusion, empyema, or allergic or anaphylactic reactions [14••,20, 21••,22,23].

Cystic echinococcosis is initially diagnosed on the basis of identification of cysts using different imaging techniques.

Figure 1 Chest radiograph showing well-defined rounded opacity surrounded by normal lung tissue in the left lung of a patient with unruptured hydatid cyst

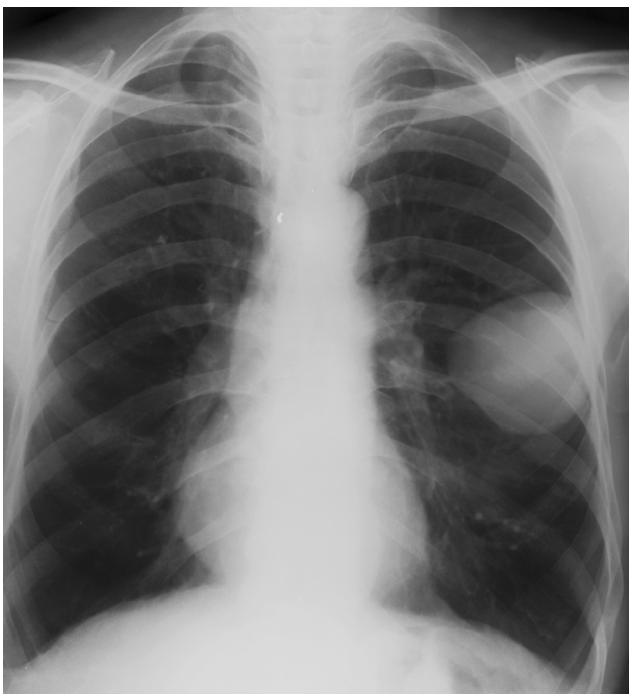
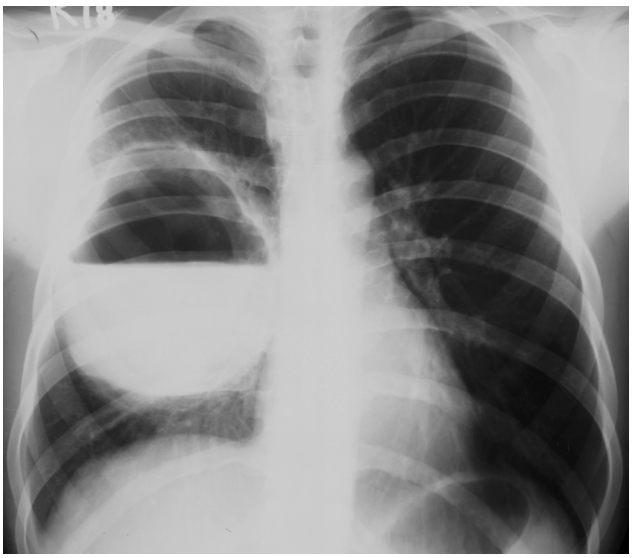


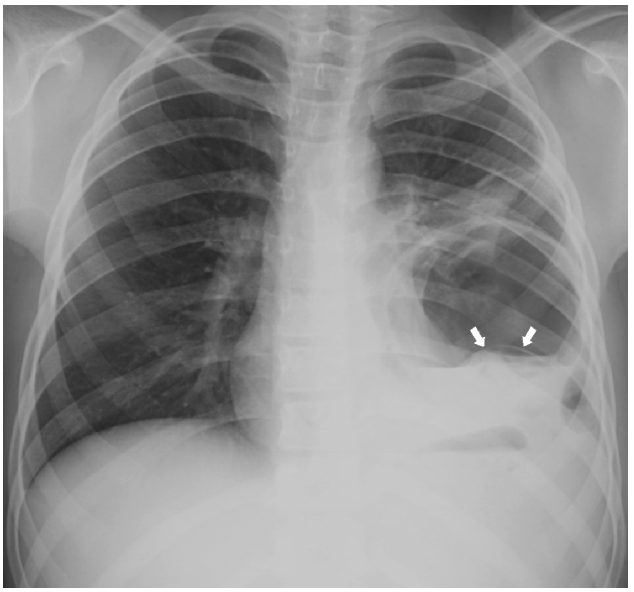
Figure 2 Chest radiograph revealing a ruptured hydatid cyst with an air–fluid level



The disease may be confirmed when specific serum antibodies are detected on immunodiagnostic testing [24•]. Plain chest radiography reveals solitary lesions in approximately 60% of cases, and multiple, unilateral or bilateral lesions in 20–50% of cases [25••]. On x-ray films, the cysts appear as homogeneous, dense, round or oval lesions that have well-defined borders and are surrounded by normal lung tissue (Fig. 1). If a cyst has ruptured, there may be consolidation adjacent to the lesion and the inflammatory reaction may mask the ruptured lesion. If the ruptured cyst communicates with the tracheobronchial tree, air enters the space between the pericyst and laminated membrane causing an air–fluid level (Fig. 2). The meniscus sign (or crescent sign), Cumbo's sign (or onion peel sign), water lily sign (Fig. 3), and mass-within-a-cavity sign are well-known identifiers on chest radiography and computed tomography (CT) [1,25••,26]. Several newer signs of hydatid disease on CT and magnetic resonance imaging have also been described. These include the rim sign, serpent sign (Fig. 4), spin or whirl sign, cyst wall sign, ring enhancement sign, halo sign [26], and inverse crescent sign [27]. If the ruptured cystic membrane leads to an occlusion in the bronchial system, diagnosis can be made with the use of bronchoscopy [13,21••,28,29].

Surgery is the main treatment for pulmonary hydatidosis, as the parasite must be eliminated to achieve complete cure. In patients with pulmonary cysts, the principle of surgery is to preserve as much lung tissue as possible. In countries where hydatidosis is sporadic, the rates of resection (i.e. lobectomy or pneumonectomy) are very high for hydatid disease. Figures as high as 74% have been reported in some series [30].

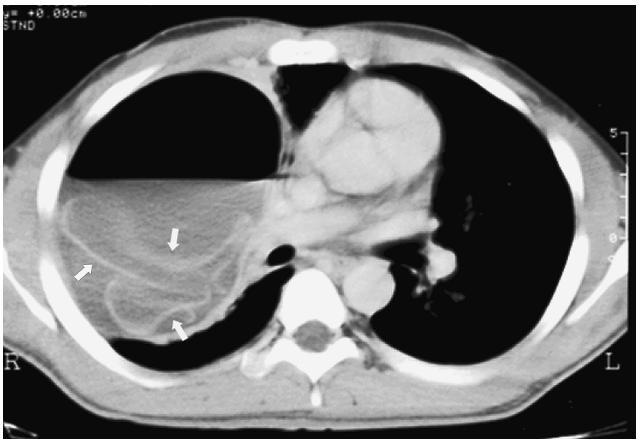
Figure 3 Chest radiograph showing a ruptured hydatid cyst with a free-floating membrane inside the cystic cavity as water lily sign



The free-floating membrane is indicated by arrows.

Our team at Turgut Ozal Medical Centre believes that radical resection is too aggressive for managing pulmonary hydatidosis, even if a patient presents with giant cysts, multiple cysts or lung abscess. Removal of all parasitic material is usually adequate treatment. The parenchyma around a hydatid cyst is often affected by the lesion, and may show chronic congestion, haemorrhage, bronchopneumonia or interstitial pneumonia [31•]. These inflammatory changes in the lung tissue often resolve after surgery. Parenchymal resection is only indicated rarely,

Figure 4 Chest computed tomography image demonstrating detached and collapsed membranes seen floating within the cystic fluid



Detached and collapsed membranes are indicated by arrows and produce the serpent sign.

when the tissue adjacent to a cyst is seriously and irreversibly destroyed. It is considered that even after an initial surgical cure, there is always a possibility of reinfestation if the patient lives in an area where *E. granulosus* is endemic.

Most surgeons prefer the lung conserving approaches of enucleation, pericystectomy, simple cystotomy, or cystotomy with capitonnage when treating pulmonary hydatid cysts. Regardless of the surgical procedure performed, spillage of cyst contents must be avoided to prevent intraoperative dissemination of daughter cysts and eventual recurrence. Recently, a case of secondary echinococcosis after surgical therapy for pulmonary hydatid cyst was reported [32•]. Once the surgeon is certain that all cyst membrane is completely removed, the cavity is irrigated with saline solution, large bronchial openings are closed, and the cavity is obliterated with separate purse-string sutures. These are placed from the deepest level to the surface of the cavity (capitonnage). Some authors suggest that capitonnage offers no benefit with respect to outcome [33], but this technique is the safest way to avoid prolonged air leakage and protect the cavity from infection and abscess formation [34].

Bilateral pulmonary hydatid disease can be managed with one or two-stage surgery involving bilateral thoracotomy, or median sternotomy [14,35–37]. When bilateral thoracotomy is performed in patients with bilateral 'uncomplicated' pulmonary hydatid disease (no ruptured cysts), it is best to first treat the side with the larger cysts or greater number of cysts [35]. If there is a ruptured cyst on one side and an intact cyst on other side, the intact cyst is treated first unless the ruptured lesion is causing urgent serious symptoms [35]. In select cases, lung and liver cysts may be treated during the same operation via thoraco-phrenotomy [35,37,38••,39]. Recent reports have described video-assisted thoracic surgery in the treatment of pulmonary hydatid cysts [40], but information about long-term outcomes is needed before this procedure can become widely accepted.

In contrast to surgical therapy, some authors contend that hydatid disease may be treated with regimens of anthelmintics, such as oral mebendazole or albendazole [41••,42]. Research has shown that 68–70% of patients with pulmonary hydatidosis show some degree of response to medical management [41••,43••]. The reported cure rates, however, are only 25–34% [41••,44]. Anthelmintics weaken the cyst wall, thus increasing the likelihood of rupture. The incidence of cyst rupture with these drugs was found to be approximately 77% in a study [44]. If a cyst does rupture but the cyst membrane and contents are completely expectorated, then the patient may be cured. Even if the parasite dies due to the drug, however, the cyst membrane will usually remain in the cavity, and this often

leads to secondary bacterial infection and other complications [41••,43••]. Considering the high risk of complications, hydatidosis patients who are treated with anthelmintics should be followed closely. Unfortunately, this is usually not possible because affected individuals tend to live in rural areas where medical care is far away or inadequate. In addition to the potential problems mentioned above, anthelmintic therapy is a long and tedious process, and the drugs can cause adverse effects such as neutropenia, alopecia and liver dysfunction. These are other good reasons for thorough follow-up care. When considering how to proceed with a case, all the potential problems with medical treatment should be weighed carefully.

It seems that hydatid cysts in the lungs cause more problems than those in the liver. The lung lesions grow faster, perhaps because the elastic nature of lung tissue offers minimal resistance to cyst expansion [45]. Our previous research has revealed that complicated pulmonary cysts are associated with increased preoperative and postoperative morbidity, need for more extensive surgery, and longer hospital stays [14••]. It is generally agreed that, regardless of whether symptoms are present, all pulmonary hydatid cysts should be surgically treated as soon as they are diagnosed in order to avoid complications. Currently, most experts believe that medical therapy should only be used to prevent recurrence or in patients who cannot tolerate surgery.

Paragonimiasis

Paragonimiasis, or 'lung fluke disease', is caused by *Paragonimus westermani* or other *Paragonimus* species. Humans become infested by ingesting raw or undercooked crabs or crayfish that contain the metacercariae (infective larvae) of *Paragonimus* spp. *P. westermani* is endemic to southeast Asia, some parts of Latin America, and Africa [46,47,48••]. After a human ingests the larvae, they penetrate the intestinal wall, enter the peritoneum, and then migrate directly through the diaphragm and pleura into the lung where they mature to adult flukes [46,47]. Typically paragonimiasis is characterized by fever, chest pain and chronic cough with haemoptysis [46,48••,49••,50].

Chest radiographs of patients with paragonimiasis may show pleural lesions (pleural effusion, pneumothorax, empyema and pleural thickening), parenchymal lesions (patchy infiltration, nodular opacities and fluid-filled cysts), or combinations of pleural and parenchymal lesions [48••,49••,50].

On CT, paragonimiasis usually manifests as single or multiple nodules in the pleura or lung parenchyma [49••]. One report [51••] describes that the findings of worm cyst, migration tract, peripheral density, bronchial wall thickening and centrilobular nodules in the lung demonstrated by high-resolution CT are suggestive of a diagnosis of *P. westermani* infestation. Prolonged bronchial inflammation may contribute to the development of bronchiectasis [48••].

The clinical and radiological manifestations of paragonimiasis can resemble those of lung cancer [48••], tuberculosis [48••,52], mesothelioma [53] or metastatic malignancy [54]. As well, this disease can mimic lung cancer on fluorodeoxyglucose positron emission tomography [49••,55]. Definitive diagnosis is based on demonstration of eggs in sputum samples [48••,52,55], bronchoalveolar lavage fluid [48••,54], transthoracic lung biopsy [48••,56], or open biopsies of lung tissue [48••,53]. Immunodiagnostic testing can be useful for diagnosis [57•]. Praziquantel is the drug of choice for treating paragonimiasis, and the next best option is bithionol [46,47].

Schistosomiasis

Schistosomiasis is caused by blood flukes of the genus *Schistosoma*. Three species, *S. mansoni*, *S. japonicum* and *S. haematobium*, are responsible for the most frequent and clinically significant forms of this condition in humans [58]. Infestations occur by skin contact with freshwater containing *Schistosoma* cercaria (infective larval forms that are excreted by snails). Once the cercaria have penetrated the skin, they pass into the bloodstream, migrate to the lung and liver, and eventually reach their target site, the portal (in the case of *S. mansoni* and *S. Japonicum*) and vesical venous system (in the case of *S. haematobium*) [58,59]. The most prevalent areas for *S. mansoni* and *S. haematobium* are sub-Saharan Africa and South America and for *S. Japonicum*, far-east Asia.

Schistosomiasis causes acute illness (Katayama fever) and chronic manifestations. In the acute form of disease, patients present with shortness of breath, wheezing and dry cough associated with fever, myalgia, headache, hepatosplenomegaly and marked eosinophilia [60]. In chronic schistosomiasis, embolization of eggs to the portal tracts leads to periportal fibrosis, portal hypertension and portosystemic anastomoses. Pulmonary involvement can occur in this phase, with ectopic migration of ova from the portal system to the pulmonary vascular bed. In the pulmonary vasculature, the eggs trigger a granulomatous response that results in fibrosis, pulmonary hypertension, and subsequent development of cor pulmonale [58••]. Salama *et al.* [61] suggested that apoptosis of endothelial cells in the pulmonary vasculature also plays an important role in the pathogenesis of shistosomal cor pulmonale.

In cases of pulmonary schistosomiasis, radiography and CT may show small nodular lesions with ill-defined

borders or, less commonly, a reticulonodular pattern or areas of diffuse, ground-glass increased opacity bilaterally [25••]. Such findings can mimic tuberculosis, sarcoidosis or metastatic disease [62]. Vawda *et al.* [63] described a patient with pulmonary schistosomiasis who presented with bilateral pneumothorax and had honeycombing in the lung parenchyma in the CT.

Pulmonary schistosomiasis can be diagnosed based on detection of eggs in stool or urine [58,59]. Serologic testing may be helpful in the clinical setting [64•]. The drug of choice for schistosomiasis is praziquantel [58,65]. This agent has no effect, however, on the juvenile stages of these parasites [66,67••]. Recent research indicates that some derivatives of artemisinin offer promise as anti-schistosomal drugs [66,67••]. These derivatives are active against immature forms of *Schistosoma* spp., so it seems possible that combinations of praziquantel and artemisinins may useful for treating the acute stage of schistosomiasis [58].

Strongyloidiasis

Strongyloides stercoralis is a roundworm that is endemic throughout the tropics and subtropics. Humans become infested when larvae in the soil penetrate the skin [68••]. The larvae migrate through the soft tissue, enter the bloodstream, and travel to the lungs. Upon reaching the large airways, they are coughed up and swallowed, and eventually settle in the small intestine. The larvae are then either excreted from the host via faeces, or re-enter the circulatory system to return to the lungs and cause autoinfection [68••]. The *Strongyloides* life cycle is unique among helminths because it is completed entirely within one host. In the acute and chronic stages of infestation, the host may develop a variety of signs and symptoms, including fever, cough, dyspnoea, wheezing and haemoptysis [69,70].

In patients with deficient cellular immunity, *S. stercoralis* infestation can lead to development of overwhelming hyperinfection associated with exacerbation of gastrointestinal and pulmonary symptoms, and may even result in death. Numerous recent reports have noted development of hyperinfection syndrome in patients with latent strongyloidiasis who are receiving systemic corticosteroids [71–76]. Other known risk factors for this syndrome include infection with HIV [77,78], infection with human T-lymphotrophic virus type 1 [79], haematologic malignancies [80], chronic lung disease [70], chronic alcoholism [79], malnutrition [70] and use of H_2 blockers and antacids [70]. According to some reports, mortality rates for patients with hyperinfection syndrome is 26–50% [70,79]. Indeed, infestation with *S. stercoralis* is a potentially lethal helminthic infection, so it is important to screen patients who may be at risk before initiating immunosuppressive therapy.

Strongyloidiasis is diagnosed by demonstrating larvae in stools, duodenal aspirate, sputum, bronchoalveolar lavage fluid, lung biopsies or pleural fluid [81]. Affected individuals often show only mild eosinophilia. Enzyme-linked immunosorbent assay (ELISA) using filariform larval antigen may be employed for the diagnosis of strongyloidiasis [82•]. Chest radiographs sometimes demonstrate patchy alveolar infiltrates in acute infection. In severe cases, chest radiography may reveal diffuse interstitial infiltrates, segmental or diffuse alveolar infiltrates, or pleural effusion [25••,26,69,70]. Mayayo *et al.* [83] described one patient with strongyloidiasis whose chest x-ray film showed pulmonary condensation that resembled a neoplastic lesion. Thiabendazole [68••,69,70] or ivermectin [68••,84] are the drugs of choice for treating strongyloidiasis.

Ascariasis and hookworm infections

The roundworm *Ascaris lumbricoides* and the hookworms *Ancylostoma duodenale* and *Necator americanus* are common causes of disease throughout the world. *A. lumbricoides* is transmitted via ingestion of food or fluids that are contaminated with faeces that contain its eggs [85]. Hookworm larvae enter via skin penetration (*A. duodenale* larvae are also orally infective) [86].

Ascarid and hookworm infestations both involve larval migration through the lungs, and this causes a hypersensitivity response that presents as transient eosinophilic pneumonia (Löffler's syndrome) [85–87]. The symptoms of this pneumonia are usually limited to mild and self-limited cough, wheezing, haemoptysis and dyspnoea [88,89]. Patients who ingest a large number of *A. duodenale* larvae can develop a condition known as Wakana disease, which is characterized by nausea, vomiting, pharyngeal irritation, cough, dyspnoea and hoarseness [86].

It can be difficult to diagnose ascariasis or hookworm infection during the pulmonary phase of the life cycle. At this stage, infestation may be suspected based on clinical findings, and can be rarely confirmed by identifying larvae in the sputum. Stool examination is negative for eggs in this phase because the parasites are in larval form [85]. In cases of ascarid or hookworm infection, chest radiography and CT show transient migratory, patchy alveolar infiltrates. The agents most often used to treat ascarids and hookworms are mebendazole and albendazole [85,86].

Dirofilariasis

The filarial nematode *Dirofilaria immitis*, and less commonly *D. repens*, are transmitted from dogs to humans by mosquitoes. This form of parasitic disease occurs throughout Southern Europe, Asia, Australia, and North and South America [90].

Occasionally, the injected larva passes into a peripheral vein and travels to the right ventricle where it develops into a sexually immature worm [91]. When an immature adult worm washes out into the pulmonary arteries, it induces a vasculitis in the pulmonary arterial tree. Ultimately it dies as a result of the inflammatory responses and resultant granuloma formation [92••].

Most patients with dirofilariasis are asymptomatic [91,93], but symptomatic patients exhibit cough, chest pain, fever, dyspnoea or haemoptysis [91]. Eosinophilia is uncommon. The typical clinical manifestation of this disease is a solitary pulmonary nodule that measures 3 cm or less in diameter [25••,94••]. The lesions are usually located at the periphery of the lung and are attached to the pleura [25••,94••]. Foroulis *et al.* [95] described one patient with pulmonary dirofilariasis who had a peripheral pulmonary mass invading the chest wall and anterior mediastinum. Pulmonary dirofilariasis can cause an increased metabolic activity on fluorodeoxyglucose positron emission tomography scan [96].

In most human cases of dirofilariasis, the diagnosis can only be made by directly identifying a worm in an excisional biopsy of the lung [90,91,93,94••,95,96,97•]. In rare cases, this condition is diagnosed based on demonstration of parasite fragments in a needle biopsy [91].

Dirofilariasis should be included in the differential diagnosis for any small, solitary or multiple pulmonary nodules in patients who live in areas where these organisms are endemic. In humans, this disease does not require any specific treatment [91,97•], and it is important to keep a high index of suspicion in order to avoid unnecessary aggressive surgery. Wand *et al.* [98] reported a case in which patients with dirofilariasis were mistakenly operated on for lung cancer.

Tropical pulmonary eosinophilia

Tropical pulmonary eosinophilia (TPE) is a type of parasitic infection caused by microfilaria of the lymphatic-dwelling organisms *Wuchereria bancrofti* and *Brugia malayi*. This condition usually affects people who live in the tropics and the subtropics (particularly India and Southeast Asia) [99••,100]. Humans contract TPE when they are bitten by mosquitoes that carry infective larvae [100]. The larvae develop into mature worms that reside in the lymphatic vessels, and the adults release microfilaria into the circulation. The microfilaria are rapidly opsonized with anti-filarial antibodies and become trapped in the lung on their first pass through the circulatory system [99••,100,101].

The pathological features of TPE mainly reflect immunologic processes that are involved in the clearance of microfilaria from the bloodstream [99••,100]. Patients typically present with an asthma-like syndrome that includes paroxysmal coughing, shortness of breath and wheezing. Most receive therapy for asthma but show minimal or no improvement [99••]. In addition to the respiratory symptoms noted above, some affected individuals exhibit non-specific signs, such as fever, weight loss, or fatigue [99••,100].

Most patients with TPE exhibit leukocytosis, an elevated erythrocyte sedimentation rate, marked eosinophilia, elevated serum total immunoglobulin IgE and high titres of antifilarial antibodies [87,89,99••,100]. It was noted that other helminthiases may be ruled out via immunodiagnostic tests using recombinant filarial antigens [102,103•]. Although anti-filarial antibodies can be detected in blood, microfilaria are almost never found [89,99••]. The chest radiograph findings in TPE include reticulonodular opacities predominantly in the middle and lower zones of the lungs, miliary mottling, and predominant hila with increased vascular markings over both lung bases [89,100]. Pulmonary function tests may indicate a predominantly obstructive defect in the early stages of TPE, and a combination of obstructive and restrictive ventilatory impairment in the chronic phase [89,100].

It is important to recognize and treat TPE early, because chronic untreated TPE may lead to progressive and irreversible pulmonary fibrosis [99••,100,101]. The treatment for TPE is diethylcarbamazine [99••,100,101]. Some patients, however, develop chronic fibrosis and permanent, irreversible impairment of pulmonary function despite this therapy. It has been suggested that priority should be given to developing combined antifilarial and anti-inflammatory treatment strategies in order to prevent the serious fibrotic sequelae that occur in the acute eosinophil-mediated syndrome of TPE [101].

Toxocariasis

Toxocariasis is caused by larvae of roundworms that affect the dog and cat, *Toxocara canis* and *T. cati*, respectively. This condition occurs worldwide and is transmitted to humans via ingestion of soil that contains eggs passed in the faeces of these animals [88,104]. The larvae migrate through the host's somatic tissues, particularly the liver, central nervous system, eyes and lungs [104].

The disease is categorized as three clinical forms: visceral larva migrans, ocular larva migrans, and covert (subclinical) toxocariasis [105•]. The classical form of toxocariasis (VLM) is characterized by eosinophilia, pulmonary abnormalities and hepatomegaly [105•]. The respiratory signs and symptoms include wheezing, coughing and dyspnoea (mimicking asthma) and pulmonary infiltrates at chest x-ray. Bachmeyer *et al.* [106] reported an adult

patient with toxocariasis who exhibited hilar and mediastinal lymphadenopathy and bilateral pleural effusion, all signs that mimicked lymphoma.

Figueiredo *et al.* [105•] emphasized that toxocariasis should be considered in any patient from an endemic region who presents with asthma (particularly children older than 3 years), hepatomegaly, eosinophilia and increased IgE levels [105•]. The diagnosis of toxocariasis is established by an ELISA with the larval antigens [87,104]. Thiabendazole or albendazole is the treatment of choice for toxocariasis [87,104].

Conclusion

Although most parasites that affect the lung are endemic to tropical and subtropical regions, immigration and travel practices have made parasitic diseases part of the scope of medicine throughout the world. Changing demographics and the concomitant changes to the environment, climate, technology, land use and in human behaviour converge to favour the emergence and spread of parasitic zoonoses [6]. Most pulmonary parasitic infestations are either asymptomatic or cause non-specific clinical and radiological signs and can make diagnosis challenging. It is important for physicians to be aware of the epidemiologic characteristics, clinical presentations, and treatments of choice for these conditions in order to make the differential diagnosis and administer appropriate therapy.

References and recommended reading

Papers of particular interest, published within the annual period of review, have been highlighted as:

- • of special interest
- •• of outstanding interest

Additional references related to this topic can also be found in the Current World Literature section in this issue (p. 248).

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