Parasitic infections of the lung: a guide for the respiratory physician

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ABSTRACT
Parasitic infections of the lung occur worldwide among both immunocompetent and immunocompromised patients and may affect the respiratory system in a variety of ways. This review provides an update on the presenting symptoms, signs, investigation and management of diseases affecting the lung caused by protozoa, nematodes and trematodes. The clinical presentations and radiographic findings of several of these diseases may mimic tuberculosis and malignancy. It is important to consider parasitic infections in the differential diagnosis of such lung diseases. If identified early, most parasitic diseases that affect the lung are curable with medical or surgical treatments.

INTRODUCTION
With increasing travel and migration, rates of parasitic lung and pleural diseases are increasing in the immunocompetent population in developed countries as well as among immunocompromised patients. Respiratory physicians should consider parasitic diseases in the differential diagnosis of lung conditions such as tuberculosis and malignancy, with which parasitic lung diseases may be confused.

This review describes the presentation, investigation and management of common parasitic infections affecting the lung caused by protozoa, nematodes and trematodes. The diseases have been grouped according to their manner of presentation: (1) those presenting with focal lesions and (2) those which characteristically present with diffuse lung disease. Focal lung lesions have been divided into cystic lung lesions, coin lesions and consolidation/pleural effusion. Diffuse lung disease has been divided into transient pulmonary infiltrates and alveolar/interstitial lung changes. Diseases that may present in a variety of ways are fully described only the first time they are mentioned.

CONDITIONS PRESENTING WITH FOCAL LUNG LESIONS

cystic lung lesions

hydatidosis

Distribution and life cycle
Hydatid disease is caused by larvae of Echinococcus tapeworm species, the definite hosts of which are members of the Canidae family (dogs and foxes). Most cases are caused by Echinococcus granulosus which has a worldwide distribution including South America, countries surrounding the Mediterranean, the Middle East, some sub-Saharan African countries, Russia and China. Although most cysts form in the liver, 20–50% form in the lung.

Dog are the definitive host for E. granulosus and harbour the adult worms in their gut. The eggs shed in dog faeces remain viable for many weeks and are able to contaminate food sources of intermediate hosts such as sheep, cattle and horses. When humans become accidental intermediate hosts after eating food contaminated with eggs, the ingested eggs hatch, releasing larvae which migrate from the gastrointestinal tract to the circulation. The eggs travel to the liver or lungs and slowly develop into hydatid cysts over a period of several months or years. Occasionally, lung cysts form after transdiaphragmatic spread of parasites following the rupture of liver cysts.

Presentation
Primary infection is asymptomatic and patients may remain asymptomatic for years, during which time lung lesions may be discovered incidentally on a chest x-ray. In Europe the average patient is in their 50s at diagnosis.3 Cysts may cause symptoms by compression of adjacent structures, and lung cysts may present with chest pain, cough, haemoptysis or pneumothorax. Symptoms may also occur if antigenic material is released from the cyst, causing a hypersensitivity reaction with fever, wheeze and urticaria and, rarely, anaphylaxis. Cysts may become secondarily infected causing empyema or lung abscess formation.

Imaging features
Cysts can be seen as single or multiple well-defined homogenous lesions surrounded by otherwise normal lung parenchyma on a plain chest x-ray. The lower lobes, posterior lung segments and the right lung are affected most frequently.2 The cyst wall often calcifies over time. A CT scan of the chest may reveal further diagnostic features including collapse of the laminated membrane from the surrounding host tissue, the presence of daughter cysts and the presence of cyst rupture.3 Air between the host tissue and laminated membrane of ruptured cysts may produce crescent-shaped lucencies. A pleural effusion or a hydro-pneumothorax may develop following the rupture of a pulmonary cyst into the pleural space. Collapsed laminated membrane may float in the cyst cavity, producing the ‘water lily’ sign (figures 1 and 2).3 4

Laboratory diagnosis
Peripheral blood eosinophilia may be found in no more than 50% of cases. A substantial rise in eosinophil count is often associated with a leakage of antigenic material from the cyst. Serological tests to support the initial diagnosis are available at reference laboratories but are less sensitive for the diagnosis of lung disease than for hepatic disease.1
Postoperatively, serological tests may be used to monitor the immunological response to treatment.5

While percutaneous aspiration of liver cysts under ultrasound guidance can support the diagnosis by demonstrating the presence of protoscolices or hooklets, this is not generally recommended because of the risk of leakage resulting in an anaphylactic reaction.

Management
In a small number of cases hydatid cysts resolve spontaneously.6 In the majority of cases the treatment of choice is surgical excision of cysts with a view to preserve as much lung parenchyma as possible.7 Albendazole should be avoided preoperatively as it may soften the cyst wall and increase the chance of rupture. It may be given as adjunctive therapy once the cyst has resolved.9 Expert advice should be sought regarding chemotherapeutic regimens before and after surgery and for cases where surgery is not to be carried out. While aspiration followed by injection of a scolicidal agent and re-aspiration (PAIR) has been used as a therapeutic tool when surgery is contraindicated,10 the World Health Organization states that PAIR is contraindicated for lung cysts11 and the authors endorse that view.

Coin lesions
Dirofilariasis
Distribution and life cycle
Pulmonary dirofilariasis is caused by the dog heartworm Dipetalonema immitis and has been reported from Japan, Australia, the USA and Italy (D repens). Adult worms live in the right ventricle of the definitive canine hosts and produce circulating microfilaria which can be transmitted by a variety of mosquito species to humans. In humans the worms pass through the right ventricle but fail to mature and are swept away to peripheral pulmonary arteries.

Presentation
Most patients are asymptomatic, although symptoms including chest pain, cough, haemoptysis, wheezing, fever, chills and malaise have been reported.12

Imaging features
The most common presentation is with a coin lesion on chest radiography. These are usually 1–3 cm in diameter and sharply defined. In established lesions a central necrotic area is surrounded by a granulomatous reaction and fibrous wall.13 Dead worms may calcify. Positron emission tomographic scanning may show an increased uptake of fluorodeoxyglucose around necrotic nodules, a feature also seen in other infectious lung nodules.14

Laboratory diagnosis
Eosinophilia may be present in a small number of patients. There are no reliable serological tests available commercially. Most cases are diagnosed by identifying the worm in biopsy specimens after thoracotomy for a pulmonary nodule thought to be a malignancy.15

Management
Chemotherapy is ineffective. The only treatment is surgical excision if indicated.

Consolidation/pleural effusion
Paragonimiasis
Distribution and life cycle
Pulmonary paragonimiasis is most frequently seen in south east Asia but also occurs in central Africa and South America. It is caused by lung flukes of the genus Paragonimus. Paragonimus westermani is responsible for most cases. The adult worms produce eggs which are secreted in sputum or faeces. Humans are usually infected by eating the second intermediate host, fresh water crab or crayfish, if raw or lightly cooked. After ingestion the larvae penetrate the intestinal wall and migrate to the lung parenchyma. They mature in a fibrous host-derived capsule, usually in the upper zones of the lung.

Presentation
Presenting symptoms include pleuritic chest pain, haemoptysis, chronic cough and fever. If pulmonary cysts erode into adjacent bronchi, patients may suffer life-threatening haemoptysis.16

Imaging features
Radiographic features of paragonimiasis include patchy air space consolidation, cystic changes and ring shadows. Pleural effusions and pneumothoraces may occur. On the CT scan round low-attenuation cystic lesions filled with fluid or gas may be seen within an area of consolidation. Peripheral linear opacities seen on chest x-rays are suggestive of worm migration tracks on corresponding CT scans. Intracystic worms may be detected on a CT scan of the thorax.17 18 Patients with pulmonary...
paragonimiasis who present with parenchymal lesions on a CT scan of the thorax may have bronchial stenosis on bronchoscopy. Paragonimiasis is often misdiagnosed as tuberculosis or malignant disease and should be considered in the differential diagnosis of these conditions in those who have lived in endemic countries.

**Laboratory diagnosis**
Peripheral blood eosinophilia is mainly seen during larval migration through the lung and total serum IgE may be raised. The eosinophil counts are usually elevated in pleural and bronchoalveolar lavage fluids. The identification of the characteristic operculated eggs in faeces, sputum or bronchoalveolar lavage fluid confirms the diagnosis. Specific serological testing is not performed in the UK but samples may be referred to overseas laboratories for specific IgG and IgM antibody testing when paragonimiasis is suspected.

**Management**
Medical treatment is usually with praziquantel. Some patients may require surgery for persistent pneumothorax, pleural effusion or empyema.

**Amoebiasis**

*Distribution and life cycle*:
Amoebiasis is caused by *Entamoeba histolytica*, a protozoan found worldwide. *E histolytica* is the causative agent of invasive amoebiasis but is morphologically indistinguishable from a related but non-pathogenic species, *E dispar*. *E histolytica* is endemic in regions with poor sanitation and poor socioeconomic conditions. The motile trophozoite forms of the parasite live in the lumen of the large intestine where they multiply and differentiate into the cyst forms. Cysts are passed in the faeces and are highly resistant to environmental conditions, facilitating faecal-oral transmission. Ingested cysts are transformed into trophozoites.

*Presentation*:
In most infections the organism does not invade the gut mucosa. In a subset of cases the *E histolytica* trophozoites bind to intestinal epithelial cells via a lectin expressed on the parasite surface. This leads to lysis of the epithelial cells and invasive amoebiasis resulting in colitis. Blood-borne spread of the parasite may lead to the formation of an amoebic liver abscess which may present several months after the initial infection when diarrhoea may be absent.

Amoebic liver abscess may cause irritation of the diaphragm and the patient may present with cough. A serous pleural effusion may occur in conjunction with an amoebic liver abscess and does not indicate extension of the disease. Amoebic liver abscess may also present with chest pain due to hepatic enlargement and inflammation. Elevation of the hemidiaphragm can cause segmental atelectasis of the lung and lead to secondary bacterial pneumonia.

Amoebic pleuropulmonary disease is the most common complication of amoebic liver abscess, occurring in 15% of patients with amoebic liver disease and in 1% of patients with amoebic dysentery. It most commonly occurs by direct extension from a superior right lobe hepatic abscess through the diaphragm into the right lower lobe of the lung, presenting with cough, pleuritic pain and dyspnoea. Pleuropulmonary amoebiasis may also occur following haematogenous spread of organisms to the lungs or lymphatic spread from the liver to the diaphragm. Intrathoracic complications include rupture of an amoebic liver abscess into the pleural cavity with empyema formation which carries a mortality of 15–35%. Infiltration of the lung parenchyma may result in pneumonia or lung abscess formation. Furthermore, a hepatobronchial fistula leading to expectoration of ‘anchovy-sauce-like’ purulent sputum can develop. Rarely, a bronchobiliary fistula may occur causing bile expectoration.

*Radiographic features*:
Common radiological features include right pleural effusion and basal lung disease. Right lower lobe consolidation may develop, which may progress to abscess formation (figure 3).

*Laboratory investigations*:
Eosinophilia is not a feature of amoebic infections. If symptoms of dysentery are present, freshly passed stools should be examined for amoebic trophozoites. Trophozoites are seldom found in sputum, pleural fluid or abscess aspirates. Antibodies directed against the parasite may be detected by a variety of methods. Antibody tests start to become positive after approximately 1 week and have a sensitivity of 99% for the diagnosis of amoebic liver abscess but are only positive in approximately 75% of cases of intestinal infection. The *E histolytica* adhesin may be detected in faecal specimens by ELISA. This test is also helpful in differentiating between *E histolytica* and *E dispar* in asymptomatic patients who pass amoebic cysts.

*Management*:
Medical treatment consists of a course of tinidazole or metronidazole to kill trophozoites in tissue, followed by diloxanide furoate or paromomycin to eliminate cysts from the intestinal lumen. Surgical treatment of pulmonary amoebiasis may be required when there is direct pulmonary involvement or spread to pleura.

**CONDITIONS PRESENTING WITH DIFFUSE LUNG DISEASE**

**Transient pulmonary infiltrates**

*Ascariasis*

*Distribution and life cycle*:
Ascariasis is caused by *Ascaris lumbricoides*, the round worm. It is common worldwide in areas with poor sanitation where there is faecal contamination of soil or food. Transmission of the disease is faecal-oral. After eggs are ingested they hatch and larvae migrate via the portal circulation to the liver then via the heart to reach the lungs 2 weeks after ingestion. Larvae then ascend to the trachea, are swallowed and eventually develop into adults in the small intestine, producing eggs 10–12 weeks after ingestion.

![Figure 3](http://thorax.bmj.com/content/10.1136/thx.2009.132217.figure3)

*Figure 3* Chest x-ray showing right basal consolidation and pleural effusion secondary to amoebic liver abscess.
Presentation
During the second week of infection, as the larvae invade the lung tissue, a small proportion of patients become symptomatic. Loeffler’s syndrome is caused by larval migration into the alveoli which triggers an allergic response leading to respiratory symptoms including cough, wheeze, dyspnoea, chest pain, fever and haemoptysis. The illness usually resolves spontaneously after several weeks.

Imaging features
During pulmonary migration, transient nodular or diffuse pulmonary infiltrates on the chest x-ray support the diagnosis of Loeffler’s syndrome. Chronic eosinophilic pneumonia has been associated with ascariasis of the lung.30 Spontaneous pneumothoraces have been described infrequently.21

Laboratory diagnosis
During larval migration a leucocytosis and peripheral blood eosinophilia are often present and total serum IgE may be elevated. Eosinophils and larvae may be found in sputum. The diagnosis is confirmed by finding characteristic Ascaris eggs in the stool on microscopy, although these may not be passed until after resolution of the pulmonary symptoms. Serological tests are not routinely available.

Management
A single dose of albendazole is the treatment of choice. Mebendazole, piperazone and pyrantel pamoate are alternatives.28

Hookworm infections
Distribution and life cycle
Ancylostomiasis is common in tropical and subtropical countries, caused by two species of hookworm (Ancylostoma duodenale and Necator americanus). A duodenale is also found in the Middle East and southern Europe; N americanus is found sporadically in the southeastern USA. Hookworm eggs are passed in faeces and develop in soil into filariform larvae able to penetrate the skin of the human host. Poor sanitation and the absence of footwear favour transmission. The larvae penetrate blood vessels and undergo heart–lung migration before breaking out into the alveoli and ascending to the pharynx from where they are swallowed. The hookworms subsequently mature into adults in the small intestine. Eggs first appear in the stool 5 weeks after skin invasion.

Presentation
Pulmonary manifestations are usually mild. During larval migration in the lung the hookworm may cause symptoms and signs consistent with Loeffler’s syndrome including dry cough, wheeze, dyspnoea and fever.

Imaging features
The radiographic changes are consistent with Loeffler’s syndrome and an eosinophilic pneumonia with transient non-segmental areas of consolidation.15

Laboratory diagnosis
Peripheral blood eosinophilia is found during larval migration and commonly persists during gastrointestinal infection. Although their appearance may be delayed, finding eggs in the stool supports the diagnosis. Serological tests are not useful in view of many cross-reactions with other helminthic infections.22

Management
Recommended treatment consists of a single dose of albendazole. Mebendazole and pyrantel pamoate are alternatives.28

Toxocariasis
Distribution and life cycle
Human toxocariasis is usually caused by the dog round worm Toxocara canis which commonly infects juvenile dogs and has a worldwide distribution. Humans, who are incidental hosts, are usually infected by the ingestion of eggs. The disease primarily affects children, who acquire the infection through contact with soil contaminated with embryonated eggs. Fresh animal faeces are not infectious as the eggs require 2–3 weeks to embryonate in the soil. Ingested eggs hatch in the stomach. Larvae penetrate the mucosa of the gut and enter the mesenteric vessels from where they migrate to the lungs and other organs.

Presentation
Most infections occur in children and most are asymptomatic. Patients may present with immunologically-mediated symptoms including cough, dyspnoea and wheeze which may present as asthma or bronchitis. Presenting signs may include hepatomegaly, splenomegaly and ocular lesions.

Radiographic features
Radiological features may include transient localised changes or widespread patchy areas of consolidation.15

Laboratory diagnosis
Toxocara larvae do not develop into adults in the human host, so eggs are not passed in human faeces. Visceral larva migrans is characterised by persistent peripheral eosinophilia and leucocytosis. A high degree of eosinophilia in the differential cell count of bronchoalveolar lavage fluid may support the diagnosis.12 Serological tests are available at reference laboratories. Although positive Toxocara serology supports a diagnosis of visceral larva migrans, titres may remain elevated for years and other causes of illness should be considered.54 Children suspected of ingesting infective Toxocara eggs may be investigated with full blood count and Toxocara serology tests at baseline and at 3 months. In practice this is seldom required as, in many cases where dog faeces have been ingested, it is possible to establish that the sample is relatively fresh and thus not likely to contain embryonated eggs. Tissue biopsy is not required for the diagnosis of visceral larva migrans.

Management
Ocular disease is treated under the supervision of an ophthalmologist with local or systemic steroids. Anthelminthics are not usually given. Visceral larva migrans is treated with albendazole. Corticosteroids may be required in more severe cases.28

Alveolar/interstitial changes
Schistosomiasis
Distribution and life cycle
Schistosomiasis is endemic throughout the tropics and it is estimated that more than 200 million people worldwide are infected.55 There are three main species of schistosomes: Schistosoma mansoni, Schistosoma haematobium and Schistosoma japonicum. The adult trematode worms are found in the vesical plexus (S haematobium) or in the mesenteric veins (S mansoni and S japonicum). Their eggs are excreted in urine or faeces respectively. Humans are infected by cercariae during contact with fresh water. The organisms enter the circulation and pass through the heart, lungs and then the liver to reach the target venous plexus. There the worms mature and mate, releasing eggs 4–6 weeks after skin penetration. The initial release of eggs may give rise to acute schistosomiasis which occurs predominantly in non-immune hosts. Eggs that are not passed into the
bladder or intestinal lumen are the main cause of chronic disease, causing granulomatous reactions and fibrosis. In severe long-
standing \textit{S mansoni} and \textit{S japonicum} infections, the development of hepatosplenomegaly and portal hypertension may lead to
diversion of eggs to the lung vasculature. This results in oblitrative arteritis which may cause pulmonary hypertension.

**Presentation**

In non-endemic countries, returned non-immune travellers may present with acute schistosomiasis while migrants from endemic
countries may present with chronic disease. Acute schistosomiasis (Katayama fever) may present with fever, cough, dyspnoea, rash and arthralgias. The precise pathogenesis of Katayama fever is unknown, but it is thought to be due to an immune complex phenomenon initiated by eggs laid by maturing schistosomes. Typically, symptoms occur 4–6 weeks after infection. In acute schistosomiasis, serological tests are often negative and eggs are not
detectable, requiring the diagnosis to be made on clinical and epidemiological grounds. In heavy infections, the migratory phase
through the lungs may produce a pneumonitis resembling that
produced during hookworm migration. A Loeffler-like syndrome
can occur during the treatment of heavy infections. Acute symp-
toms are usually self-limiting. In the chronic stage the commonest
symptoms are dyspnoea, reduced exercise tolerance and chest pain.
Clinical signs of severe hypoxaemia such as digital clubbing may be
seen. Since schistosomiasis is an important cause of pulmonary hypertension, it should always be considered as a differential
diagnosis in patients from endemic countries. The chronic form of
schistosomiasis may also cause pulmonary arteriovenous fistulas.

**Radiological features**

Nodular ill-defined lesions or reticulonodular changes may be
seen on the chest x-ray in acute pulmonary schistosomiasis (figure 4). Although radiological findings are usually transitory in acute infection, chest x-rays may show features resembling
granulomatous disease or tuberculosis. In chronic lung
disease, interstitial or granulomatous changes are caused by the
widespread deposition of eggs in the pulmonary vasculature. A CT scan of the thorax may show nodular changes and ground
glass opacification (figure 5).

**Laboratory investigation**

Peripheral blood eosinophilia is present in more than 65\% of
patients. Eggs of the \textit{Schistosoma} species may be found in
faeces, urine and sputum or bronchoalveolar lavage 6 weeks after
infection. Eggs of \textit{S haematobium} are most likely to be found in
late morning urine samples. Terminal urine specimens max-
imise sensitivity. IgG antibodies to \textit{Schistosoma} egg antigen are
detectable 6–12 weeks following infection and can be measured by
ELISA. Such assays have sensitivities of >90\% for the diag-
nosis of schistosomiasis. These tests may remain positive for
several years and are not helpful for monitoring response to
treatment. Serological testing in asymptomatic travellers should be
delayed for a period of 3 months following exposure to fresh
water to allow time for seroconversion. Schistosomal serology
may be negative, especially in Katayama fever, but if positive it
provides helpful diagnostic information.

**Management**

The treatment of choice for schistosomiasis is praziquantel.\textsuperscript{28 48} In acute schistosomiasis (Katayama fever) praziquantel is
administered under steroid cover. A second course of prazi-
quantel may need to be given 3–6 months later to eradicate any
schistosomes that may have survived the first course of treatment.\textsuperscript{47} Treatment is less beneficial in advanced chronic disease.

**Strongyloidiasis**

**Distribution and life cycle**

Strongyloidiasis is caused by the nematode \textit{Strongyloides stercoralis} and is prevalent in tropical and subtropical countries.
Filariform larvae penetrate the skin, enter blood vessels and
undergo heart and lung migration. They migrate into alveoli
and subsequently ascend to the trachea. Larvae are swallowed
and develop in the small intestine into adult worms which
produce eggs. These are deposited in the intestinal mucosa
where they hatch into rhabditiform larvae which are excreted in
faeces. Occasionally, rhabditiform larvae mature to the filariform
stage while still in the bowel lumen. These filariform larvae may
penetrate the bowel wall or the perianal skin to initiate a new
individual may harbour \textit{Strongyloides} for several decades.

**Presentation**

Clinically the infection is often asymptomatic, but mild symp-
toms may occur in the initial stages of lung migration. In the
immunocompromised patient (including those on steroid
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<td>Hydatidosis</td>
<td>Symptoms due to mass effect</td>
<td>Single or multiple lung cysts</td>
<td>Wide distribution: Mediterranean borders,</td>
<td>Months to decades after exposure</td>
<td>Hydatid serology positive in 50-60%</td>
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<td>Chest pain, cough,</td>
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<td>East and Central Asia, South America</td>
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<td>Blood eosinophilia uncommon</td>
<td>Albendazole with or without praziquantel</td>
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<td>haemoptysis</td>
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<td>Coin lesions</td>
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<td>Coin lesion with or without calcification</td>
<td>Pulmonary dicrofilariosis reported from USA, Japan,</td>
<td>May be years after exposure</td>
<td>Blood eosinophilia uncommon</td>
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<td>Eggs in s.tol or sputum</td>
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<td>Paragonimiasis</td>
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<td>Does not cause eosinophilia; usually does cause</td>
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<td>fever, haemoptysis</td>
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<td>Amebiasis</td>
<td>Right upper quadrant or shoulder tip pain</td>
<td>Pleural effusion, atelectasis, Empyema</td>
<td>Widely distributed</td>
<td>Weeks to years after exposure</td>
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<td>Tindazole (or metronidazole) followed by diloxanide</td>
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<td>Cough and bile expectoration</td>
<td>Amebic lung abscess</td>
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<td>Does not cause eosinophilia; usually does cause</td>
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<td>Ascariasis</td>
<td>Cough, wheeze, dyspnoea,</td>
<td>Transient pulmonary infiltrates</td>
<td>Worldwide in areas where sanitation is poor</td>
<td>1-2 weeks from infection to onset of pulmonary symptoms</td>
<td>Larvae in pulmonary, gastric secretions.</td>
<td>Albendazole, mebendazole, piperazine, or pyrantel pamoate</td>
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<td>Pneumothorax</td>
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<td>Hookworm infection</td>
<td>Cough, wheeze, dyspnoea,</td>
<td>Transient pulmonary infiltrates</td>
<td>Widely distributed: (infection usually by contact of bare feet with faecally contaminated soil)</td>
<td>Pulmonary manifestations start within 10 days of exposure, can continue for more than one month</td>
<td>Blood eosinophilia during larval migration</td>
<td>Albendazole, mebendazole or pyrantel pamoate</td>
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<td>Eggs may be absent in larval migratory phase</td>
<td></td>
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<tr>
<td>Toxocariasis</td>
<td>Cough, dyspnoea, wheezing, asthma or bronchitis, splenomegaly, ocular lesions</td>
<td>Pulmonary infiltrates</td>
<td>Worldwide distribution (adult worms live in gut of cats and dogs)</td>
<td>Several weeks</td>
<td>Peripheral blood eosinophilia common</td>
<td>Albendazole for visceral disease</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Secondary bacterial pneumonia</td>
<td></td>
<td></td>
<td>Eosinophilia in bronchoalveolar lavege</td>
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<td></td>
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<td></td>
<td></td>
<td></td>
<td>Toxocara serology</td>
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<tr>
<td>Alveolar' interstitial changes</td>
<td></td>
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<td></td>
<td></td>
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<tr>
<td>Condition</td>
<td>Presentation</td>
<td>Imaging features</td>
<td>Geographical distribution</td>
<td>Incubation period</td>
<td>Investigations</td>
<td>Treatment</td>
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<tr>
<td>Schistosomiasis</td>
<td>Acute disease: Katayama fever with cough, dyspnoea, rash, and arthralgias</td>
<td>Acute disease: transient reticulonodular changes</td>
<td>Africa, South America, south east Asia, China</td>
<td>Acute disease: 5–7 weeks after exposure. Chronic disease: years</td>
<td>Acute disease: blood eosinophilia common</td>
<td>Praziquantel</td>
</tr>
<tr>
<td></td>
<td>Loeffler’s syndrome, pneumonia</td>
<td>Chronic disease: granulomatous lung disease, pulmonary hypertension, pulmonary AV fistula</td>
<td></td>
<td>Chronic disease: eggs in sputum, or bronchovascular lavage after 6 weeks</td>
<td>Chronic disease: eggs in stool and/or urine (S. haematobium) after about 6 weeks</td>
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<tr>
<td></td>
<td>Chronic disease: dyspnoea, pulmonary hypertension</td>
<td></td>
<td></td>
<td></td>
<td>Schistosomal serology positive after 6-12 weeks</td>
<td></td>
</tr>
<tr>
<td>Strongyloidiasis</td>
<td>In hyperinfection syndrome: asthma, ARBS, intra-alveolar haemorrhage</td>
<td>In hyperinfection syndrome: pulmonary infiltrates, mililiary nodules, airspace opacities ARDS in severe disease, rarely granulomatous changes</td>
<td>Worldwide distribution where sanitation poor</td>
<td>Pulmonary symptoms may occur days after acute infection; hyperinfection may occur up to decades after infection</td>
<td>Blood eosinophilia common</td>
<td>Ivermectin, albendazole less effective</td>
</tr>
<tr>
<td></td>
<td>Bilateral reticulonodular shadowing</td>
<td></td>
<td>Asia, sub-Saharan Africa, South America</td>
<td>Up to years after leaving endemic area</td>
<td>Blood eosinophilia</td>
<td>Diethylcarbamazine with or without albendazole</td>
</tr>
</tbody>
</table>

**Tropical pulmonary eosinophilia (filariasis)**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Presentation</th>
<th>Imaging features</th>
<th>Geographical distribution</th>
<th>Incubation period</th>
<th>Investigations</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cough, dyspnoea, wheezing, fever, malaise, weight loss</td>
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<tr>
<td></td>
<td>Bilateral reticulonodular shadowing</td>
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<tr>
<td></td>
<td>Mediastinal lymphadenopathy</td>
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<td></td>
<td>Lake Victoria, East Africa, Central Africa</td>
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<tr>
<td></td>
<td>Bilateral reticulonodular shadowing</td>
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</tr>
<tr>
<td></td>
<td>Fever, malaise, weight loss</td>
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</tbody>
</table>

**Laboratory investigations**

- Blood eosinophilia is seen in more than 75% of patients with chronic infection. Antibody assays have sensitivities of approximately 90%.
- Culture techniques are more likely to yield positive results.
- Loeffer’s syndrome is caused by larval obstruction of lymphatic vessels. The syndrome of TPE is thought to be an immunological response to microfilarial antigens. Antigenic stimulation of lymphatic vessels is common in areas of south east Asia, India, China and Africa. The most common manifestation of south east Asia, India, China and Africa.
- Bilateral reticulonodular shadowing is seen on chest radiography. Radiographic changes may include miliary nodules, reticular opacities and airspace consolidation. Systemic features including fever, malaise and weight loss may be present.
- Rare manifestations of pulmonary strongyloidiasis include granulomatous changes leading to pulmonary fibrosis.
- Pulmonary hypertension may cause right ventricular failure.
- Aperistalsis of the small intestine may lead to bowel obstruction. The syndrome of TPE is caused by larval obstruction of lymphatic vessels.
- Other features include fever, malaise and weight loss.
- Tuberculosis may include miliary nodules, reticular opacities and airspace consolidation. Systemic features including fever, malaise and weight loss may be present.
- Rare manifestations of pulmonary strongyloidiasis include granulomatous changes leading to pulmonary fibrosis.
- Pulmonary hypertension may cause right ventricular failure.
to detect reticulonodular pulmonary opacities and mediastinal lymphadenopathy.60

Laboratory investigations

High titres of antifilarial antibodies and peripheral blood eosinophilia are present. The extensive immune response leads to rapid clearance of microfilariae from the blood and, in order to diagnose TPE, it is essential that no microfilariae can be found in the peripheral blood. Total serum IgE is raised, as are filarial-specific IgE and IgG antibody levels. Bronchoalveolar lavage may show evidence of an eosinophilic alveolitis with a high concentration of eosinophils.61 Specific antifilarial IgE and IgG antibodies have also been detected in bronchoalveolar lavage samples.62

Management

Pulmonary abnormalities usually progressively worsen without treatment. Concomitant infection with other filarial species must be investigated prior to treatment. Treatment is usually with diethylcarbamazine under expert guidance; however, it is sometimes given in combination with albendazole to improve efficacy.63 64 Even after treatment, pulmonary symptoms may progress and interstitial lung disease might develop leading to chronic respiratory failure.65

Non-infectious causes of pulmonary eosinophilia

Other causes of pulmonary eosinophilia need to be considered in the differential diagnosis of parasitic lung infections. Idiopathic lung diseases such as acute eosinophilic pneumonia, chronic eosinophilic pneumonia and idiopathic hypereosinophilic syndrome and lung diseases of known cause such as allergic bronchopulmonary aspergillosis, bronchocentric granulomatosis, drug reactions and eosinophilic vasculitis (Churg–Strauss syndrome) can cause pulmonary and peripheral blood eosinophilia.66 67

Conclusions

This review outlines the symptoms, signs, investigation and treatment of parasitic lung diseases which are summarised in table 1. The clinical presentation and radiology of many of these diseases may share common features with tuberculosis and malignancy. When symptoms of lung disease are associated with gastrointestinal, hepatobiliary or cutaneous symptoms or a peripheral eosinophilia, there should be a high index of suspicion for parasitic diseases. With increasing travel and migration, it is important to consider parasitic infections in the differential diagnosis of lung diseases. Parasitic lung infections have been described in patients following chemotherapy or transplantation and need to be considered in the differential diagnosis of lung infections in immunocompromised individuals.86–70 Direct identification of the causative organisms may be achieved definitively through microscopic examination of stool or respiratory tract samples, or indirectly through serological testing. If identified early, most parasitic lung diseases are curable with medical treatment. Further information on the distribution of parasitic lung diseases and treatment may be obtained from the World Health Organization.71

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References