Chapter 106
Pulmonary Manifestations of Tropical Parasitic Diseases

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INTRODUCTION

With globalization and frequent travelling, the tropical lung disease is now being reported from many parts of the world. With the emergence of human immune deficiency virus/acquired immunodeficiency syndrome (HIV/AIDS), the frequent use of immunosuppressive drug and increasing number of organ transplants have resulted in renewal of interest in tropical lung diseases (Table 1).

In tropical countries parasitic lung diseases are due to protozoa and helminthic parasites. (Indian J Chest Dis allied Sci. 2008;49-66).

Protozoal parasites that cause pulmonary disease are Entamoeba histolytica, Leishmania donovani, malarial parasite, Toxoplasma gondii, Babesia microti and Babesia divergens.

PULMONARY AMEBIASIS

The causative organism is E. histolytica and pulmonary amebiasis occurs mainly by the extension from amebic liver abscess.1,2

The clinical features are fever, chest pain, cough, hemoptysis of "anchovy sauce like" pus indicates pleuropulmonary amebiasis. Diagnosis is confirmed by elevated right hemidiaphragm, pleural effusion, basal pulmonary infiltrates and tender hepatomegaly. Active trophozoites of E. histolytica can be demonstrated in sputum or pleural pus. Stool microscopy may reveal cyst or trophozoite. A combination of serological tests like polymerase chain reaction, enzyme-linked immuno sorbent assay (ELISA), indirect hemagglutination test (IHA) with detection of parasite by antigen detection is the best approach for diagnosis.

Treatment

Metronidazole is the treatment of choice. Luminal amebicidal drug, diloxanide furoate can eliminate intestinal entameba cyst.

PULMONARY LEISHMANIASIS

It is caused by L. donovani and infection is transmitted by various species of phlebotomus, the sand fly.4 Pneumonitis, pleural effusion and mediastinal adenopathy are reported in patient coinfected with HIV and in lung transplant patients. L. donovani amastigotes can be found in the alveoli and bronchoalveolar lavage (BAL) fluid.5,6

The drugs for the treatment of leishmaniasis include pentavalent antimonials, amphotericin B especially the liposome formulation and pentamidine. Oral drug miltefosine is now available.7

PULMONARY MALARIA

The four types of malarial parasites (Plasmodium vivax, P. falciparum, P. malariae, and P. ovale) infect man. It is an intraerythrocytic protozoa transmitted by the bite of female anopheles mosquito. P. falciparum is the most deadly. Sequestration of erythrocytes containing mature form of P. falciparum in the microvasculature of the organs is the main feature.

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<td>Pulmonary trichinellosis</td>
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Pulmonary manifestation ranges from cough to severe and rapidly progressive fatal noncardiogenic pulmonary edema and acute respiratory distress syndrome (ARDS).

Acute respiratory distress syndrome can also occur in vivax malaria. Superadded viral and bacterial infection cause pneumonitis.

**Diagnosis**
The gold standard task is identification of malarial parasite by thick and thin stained blood smears. Radiological findings may include lobar consolidation, diffuse interstitial edema, pulmonary edema and pleural effusion.

**Treatment**
Infusion of intravenous quinine 600 mg/hourly, injection Artemisinin derivatives are the drugs of choice. Parenteral chloroquine is used in susceptible *P. falciparum* infection. Artemisinin-based combination therapies (artemether + lumefantrine, artesunate + amodiaquine, artesunate + mefloquine or artesunate + sulfadoxine-pyrimethamine) are the best antimalarial drugs. The patient with ARDS requires ventilatory support. The best prophylaxis is prevention from mosquito bites by the use of insecticide-treated bed-nets.

**PULMONARY TOXOPLASMOSIS**
Toxoplasmosis is caused by one unicellular protozoan parasite: *Toxoplasma gondii*. Cats are the primary carriers of the organism. Humans get infection by eating parasitic cyst-contaminated raw or undercooked meat, vegetables or milk products. The symptoms of toxoplasmosis are flu-like syndrome, enlarged lymph nodes or myalgia. Infection in early pregnancy can cause fetal death, and chorioretinitis and neurologic symptoms in the new born. Chronic toxoplasmosis can cause chorioretinitis, jaundice, encephalitis and convulsions. Pulmonary toxoplasmosis has been reported in HIV patient. It can manifest as interstitial pneumonia/diffuse alveolar damage or necrotizing pneumonia. Diagnosis is based on detection of protozoa in body tissues. Polymerase chain reaction-based assay in BAL fluid has been reported in HIV positive patient. Toxoplasmosis can be treated with a combination of pyrimethamine and sulfadiazine.

**PULMONARY BABESIOSIS**
Babesiosis is caused by hemoprotozoan parasites—*Babesia microti* and *Babesia divergens*. Humans get infected by the bite of an infected tick *Ixodes scapularis*. This tick borne illness can have co-infection with ehrlichiosis and Lyme disease. The parasites attack the red blood cells like *P. falciparum*. Clinical features are fever, drenching sweat, tiredness, loss of appetite, myalgia and headache. Acute respiratory distress occurring a few days after initiation of the medical therapy is the important pulmonary manifestation. Diagnosis is made by Giemsa stained thin blood smear and PCR or detection of specific antibody. Treatment is with combination of clindamycin (600 mg every 6 hrs) and quinine (600 mg every 8 hourly) or atovaquone (750 mg every 12 hrs) and azithromycin 500 mg od for 7-10 days.

**HELMINTHIC PARASITES**
All three classes (cestoidea, trematoda and nematoda) cause pulmonary disease in humans.

**Cestodes**

**Pulmonary Hydatid Disease**
It is caused by *Echinococcus granulosus* and *E. multilocularis*. Hydatid cysts are found mainly in the liver and lungs. Pulmonary alveolar echinococcosis (AE) is due to hematogenous dissemination from hepatic lesion. The definitive host is the dog. The eggs excreted by the dogs in the feces are ingested by the intermediate hosts including man.

**Trematodes**

**Pulmonary schistosomiasis** and paragonimiasis are caused by trematodes.

**Pulmonary Schistosomiasis**
The schistosomiasis that cause human disease are Schistosomiasis hematobium, *S. mekongi* and *S. japonicum*. The eggs are passed in urine or stools of infected humans and from water they are ingested by snails (intermediate host) in which the eggs hatch and develop into cercariae. The infective cercariae penetrate human skin and gut. Pulmonary schistosomiasis can manifest clinically as an acute form and a chronic form. The acute form, also known as Katayama syndrome presents with fever, chills, weight loss, diarrhea, abdominal pain, myalgia and urticaria and is seen in nonimmune patients. Pulmonary manifestations include shortness of breath, wheezing and dry cough. Small pulmonary nodules in CT have been described in acute schistosomiasis. Massive hemoptysis and lobar consolidation and collapse have been reported in schistosomiasis.

Patients with chronic schistosomiasis present with cor-pulmonale and pulmonary hypertension.

**Diagnosis:** Peripheral blood eosinophilia with mild leukocytosis, abnormal liver function test and elevated lgE levels are reported during this phase.

**Treatment:** Acute schistosomiasis at presentation can be treated with corticosteroids alone followed by praziquantel (20–30 mg/kg orally in two doses within 12 hours). Praziquantel can be repeated several weeks later to eradicate the adult flukes.

**Pulmonary Paragonimiasis**
Paragonimiasis is caused by infection with paragonimus species and manifest as subacute or chronic inflammation of the lung. The main species that cause paragonimiasis in man is *paragonimus westermani*. Adult worms live in the lungs and the eggs are voided in the sputum or feces. The eggs hatch in the fresh water to release miracidiae which are ingested by the first intermediate host, fresh water snails. The man gets infection, when raw or undercooked crabs or crayfishes infected with infective metacercariae are ingested. Pulmonary paragonimiasis manifests as fever, chest pain, chronic cough and hemoptysis chest radiographs may show infiltrative, nodular and cavitating shadows. Pleural effusion or pneumothorax is an important finding in paragonimiasis.
Pulmonology

- The chronically-infected people may be development when environment conditions become favorable.
- The upper part of small intestine.
- trachea, larynx and pharynx and are ultimately swallowed to reach pulmonary circulation through the lymphatics and venules.
- The second-stage larvae moult twice more in the wall of the intestine and travel via capillaries and lymphatics to the hepatic circulation and to the right side of the heart and then reach the lungs. The second-stage larvae moult twice more in the alveoli to produce third and fourth-stage larvae. The migrating larvae can induce granuloma formation with eosinophils, neutrophils and macrophages.
- Clinical features: Pulmonary migration of larvae is usually asymptomatic. Respiratory symptoms are due to larval pulmonary migration, airway hyper-reactivity and bronchospasm. Symptomatic pulmonary disease may range from mild cough to a Loeffler’s syndrome. Loeffler’s syndrome is a self-limiting inflammation of the lung and is associated with blood and lung eosinophilia. The respiratory symptoms include chest pain, cough with mucoid sputum, hemoptysis, shortness of breath and wheezing.
- Diagnosis: Pulmonary ascariasis is diagnosed with chest radiographs demonstrating unilateral or bilateral, transient, migratory, fleeting nonsegmental opacities of various sizes. These opacities are often peripherally situated and appear to be pleural based. Sputum may show Charcot-Leyden crystals and the measurement of ascariss-specific IgG4 by ELISA may be useful in the serodiagnosis of ascariasis.
- Treatment: It is self-limiting disease. The specific anthelmintic treatment is given. Mebendazole and albendazole have been found to be equally effective in the treatment of ascariasis. Albendazole is given as a single dose of 400 mgm or single dose of pyrantel pamoate (11 mg/kg, maximum dose 1 gram) is useful.
- Pulmonary Ancylostomiasis
- Hookworm disease in humans results from infections with two species, Ancylostoma duodenale and Necator americanus. Hookworm is a habitual blood sucker. The eggs containing segmented ova with four blastomers are passed out in the feces. A rhabditiform larvae develops from each egg in the soil and this larva moult twice and develops into a filariform larva, which is infective to man. The filariform larva penetrates through the intact skin. These larvae reach pulmonary circulation through the lymphatics and venules. The larvae then pierce the alveolar walls and ascend the bronchi, trachea, larynx and pharynx and are ultimately swallowed to reach the upper part of small intestine. Ancylostoma duodenale larvae can developmentally get arrested in the gut or muscle and restart development when environment conditions become favorable.

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- Bronchitis and bronchopneumonia can occur when the larvae break through the pulmonary capillaries to enter the alveolar spaces. Chronic blood loss can result in iron deficiency anemia.
- Clinical features: Ancylostoma dermatitis which manifests as intense pruritus, erythema and rash occurs at the site of skin penetration during pulmonary larval migration, patient may present with fever, cough, wheezing and transient pulmonary infiltrates in chest radiographs. Patient may present with fatigue, exertional dyspnea, poor concentration and cardiac murmurs. Prominent gastrointestinal symptoms in hookworm disease are abdominal pain, nausea, anorexia and diarrhea.
- Diagnosis: Eosinophilia in the peripheral blood is a prominent finding. A polymerase chain reaction (PCR) to differentiate between A. duodenale and A. americanus has been developed.44
- Treatment: Both mebendazole and albendazole are useful in the treatment of hookworm. Recent studies have demonstrated that ivermectin can also be used in the treatment of hookworm infection.

Pulmonary Strongyloidiasis
- The parasitic females live in the wall (mucous membrane) of the small intestine of man. The parasitic males remain in the lumen of the gut and they have no capacity to penetrate the mucus membrane. The rhabditiform larvae emanating from the eggs pierce the mucous membrane and reach the lumen of the intestine. These larvae are then passed with the feces and eggs.
- Clinical features: The chronically-infected people may be asymptomatic. Pulmonary sign and symptoms include cough, shortness of breath, wheezing and hemoptysis. In addition, acute anemia, acute renal failure and systematic inflammatory response syndrome are also reported in hyperinfection.
- Diagnosis: The examination of a single stool specimen using conventional techniques usually fails to detect larvae in up to 70% of cases.
- Treatment: Ivermectin, thiabendazole or albendazole can be used for the treatment of strongyliodisis in immunocompetent individuals. 49,50

Tropical Pulmonary Eosinophilia
- Tropical pulmonary eosinophilia (TPE) is characterized by cough, dyspnea and nocturnal wheezing, diffused reticulomnodular infiltrates in chest radiographs and marked peripheral blood eosinophilia. The syndrome results from immunologic hyper responsiveness to human filarial parasites, Wuchereria bancrofti and Brugia malayi. Tropical pulmonary eosinophilia is one of the main causes of pulmonary eosinophilia in the tropical countries. These patients had extensive bilateral military motting in chest radiographs and can be wrongly diagnosed as military tuberculosis. However, they were in good physical condition and did not have a high mortality as observed in military tuberculosis. They described this entity as “a pseudo-tuberculosis condition associated with eosinophilia.” The name “tropical eosinophilia” syndrome was coined by Weingarten in 1943 who described 81 patients with severe spasmodic bronchitis, leukocytosis and very high eosinophilia and disseminated motting of both the lungs.
- Tropical pulmonary eosinophilia, an occult form of filariasis, is endemic in the Indian subcontinent, Southeast Asia and South Pacific islands. It has been estimated that at least 120 million persons are infected with mosquito-borne lymphatic filariasis worldwide. However, TPE is seen in only less than 1% of filarial infections. Tropical pulmonary eosinophilia is being increasingly reported from countries which are not endemic to filarial infection. Therefore,
Tropical pulmonary eosinophilia (TPE) should be considered in the differential diagnosis, if a patient traveling from a filarial endemic region presents with “asthma-like” symptoms. Various studies have shown that filarial infection is the cause of TPE. Elevated concentrations of filarial-specific IgG and IgE have also been reported in TPE. Ultrasound examination of the scrotal area of the patient with TPE had demonstrated living adult *Wuchereria bancrofti* in the lymphatic vessels of the spermatic cord. Tropical pulmonary eosinophilia resulted from immunologic hyper-responsiveness to human filarial parasites.

Histopathological examinations have demonstrated microfilaria in the lungs, liver and lymph nodes of patients with TPE. The microfilaria was sheathed and had the anatomical features of *W. bancrofti*. The histopathological study in TPE had shown widely scattered nodules of varying sizes (1-5 mm) over lung surface. Three types of histopathological reactions can be seen in TPE:

1. Interstitial, peribronchial and perivascular exudates consisting of histiocytes in patient with a short duration of symptoms (less than 3 weeks)

2. Acute eosinophilic infiltration of interstitial, peribronchial and perivascular tissues leading to the formation of eosinophilic abscesses and eosinophilic bronchopneumonia

3. A mixed cell type of infiltration consists of histiocytes, eosinophils and lymphocytes with well-marked interstitial fibrosis after 6 months. A predominant histiocytic response developed 2 years after the onset of the disease and ultimately progressed to fibrosis with marked scarring. In the end stage, a combing might develop in some cases, if untreated. Lung biopsies after 1 month's treatment with diethylcarbamazine (DEC) demonstrate incomplete histological regression, although symptoms subside within 7 days of therapy and peripheral eosinophilia return to normal.

The current concept of the pathogenesis of TPE suggests that it begins with a lung parenchymal inflammation in individuals highly sensitized immunologically to filarial parasites. The microfilariae released from adult worms living in lymphatics are cleared in the pulmonary circulation, degenerate and release their antigenic constituents which trigger a local inflammatory process. Though the lung bears the major brunt of the disease as a result of trapping of microfilariae in the pulmonary circulation, the antigenic material released from the microfilariae can reach the systemic circulation and cause extrapulmonary manifestations.

Bronchoalveolar larvae (BAL) studies have demonstrated that TPE characterized by intense eosinophilic inflammatory process in lower respiratory tract is many folds greater than that in the blood, suggesting that eosinophils accumulate selectively in the lung parenchyma. The bronchospasm in TPE may result from leukotrienes released by the eosinophils. A major IgE inducing antigen (Bm23-25) of the filarial parasite, *B. malayi* has been identified from patient with TPE. Patients with TPE show striking elevations of total IgE, IgG (hypergamma globulinemia) and filarial-specific IgG, IgM and IgE antibodies in peripheral blood and lung epithelial lining fluid (ELF). A major IgE-induced antigen (Bm23-25) of the filarial parasite, *Brugia malayi* has been identified from patients with TPE. Bronchoalveolar lavage fluid of patients with TPE contains IgE antibodies that recognize *B. malayi* antigen, Bm23-25. A marked reduction in lung ELF filarial-specific IgG and IgE levels within 6-14 days of therapy with DEC has been observed.

Clinical features: Tropical pulmonary eosinophilia is a systemic disease involving not only the lungs, but other organs such as liver, spleen, lymph nodes, brain, gastrointestinal tract, etc. may also be involved. The disease occurs predominantly in males, with male to female ratio of 4:1 and is mainly seen in children and young adults between the age 15 years and 40 years. The systemic symptoms include fever, weight loss and fatigue. Patient with severe cough can lead to fractured ribs. Sputum is usually scanty, viscous and mucoid. Sputum often shows clumps of eosinophils. Bilateral scattered rhonchi and rales may be heard on auscultation.

Leukocytosis with an absolute increase in eosinophils in the peripheral blood is the hallmark of TPE. Spontaneous fluctuations in the eosinophilia count can occur. Absolute eosinophilia counts are usually more than 3,000 cells/mm³ and may range from 5,000 to 80,000. Erythrocyte sedimentation rate is elevated in 90% of cases and returns to normal following specific treatment. Microfilariae are rarely seen in the peripheral blood. As patients with TPE especially from endemic areas can be simultaneously infected with other helminthic parasites, stool examination may reveal ova or larvae of other helminthes. This observation does not deter the physician from making a diagnosis of TPE, if other conditions for diagnosis are fulfilled. The chest radiological features of TPE include reticulonodular shadows predominantly seen in mid and lower zones and military mottling of 1-3 mm in diameter often indistinguishable from miliary tuberculosis. Radiological improvement occurs on specific therapy with DEC, but some degree of radiological abnormality persists in some patient.

CT scan studies have shown bronchiectasis, air trapping, calcification and mediastinal lymphadenopathy in patient with TPE. Lung function test reveal mainly a restrictive ventilation defect with superimposed airways obstruction. Arterial hypoxemia (PaO₂ < 80 mm Hg) was observed in 41% of untreated TPE patient. Lymphadenopathy may occur in TPE, especially in children. Cardiovascular changes especially electrocardiographic abnormalities, pericarditis, pericardial effusion and cor-pulmonale have also been reported. Tropical pulmonary eosinophilia may also present with gastrointestinal, skeletal muscle and central nervous system manifestations.

**Diagnosis:** Infestation with helminthes (cestodes, nematodes and trematodes) are the most common causes of pulmonary eosinophilia in tropical countries. Tropical pulmonary eosinophilia of filarial etiology may sometimes be clinically indistinguishable from TPE-like syndrome caused by other helminthes. In addition, elevated levels of antifilarial antibodies (IgG, IgG4 and IgE) are also observed in patients with TPE-like syndrome. Noninfecious causes of pulmonary eosinophilia include allergic bronchopulmonary aspergillosis (ABPA), bronchial asthma, acute eosinophilic pneumonia, chronic eosinophilic pneumonia, idiopathic hypereosinophilic syndrome, cryptocogenic pulmonary fibrosis, Wegener’s granulomatosis, lymphomatoid granulomatosis, eosinophilic granuloma of the lung, Churg-Strauss syndrome and drug hypersensitivity reactions. Till a diagnostic test that can differentiate filarial TPE from other TPE-like syndrome is available, the following diagnostic criteria can be used for the diagnosis of TPE:

- **Appropriate exposure history (mosquito bite) in an endemic area of filariasis**
- **History of paroxysmal nocturnal cough and breathlessness**
- **Chest radiographic evidence of pulmonary infiltrations**
- **Leukocytosis in blood**
- **Peripheral blood eosinophils more than 3,000 cells/mm³**
- **Elevated serum IgE levels**
- **Elevated serum antifilarial antibodies (IgG and/or IgE), and**
- **A clinical response to DEC.**

**Treatment:** The efficiency of DEC in TPE was proved by Ganatra and Lewis and Dhanraj. Baker et al. had shown in a controlled trial that DEC in a dose of 5 mg/kg/day for 7 days was sufficient in the treatment of TPE. The standard treatment recommended by the World Health Organization for treatment of TPE is oral DEC (6 mg/kg per day) for 3 weeks. One month after the start of the treatment most patient show marked symptomatic and radiographic improvement and significant improvement in almost all aspects of
Pulmonary Trichinellosis

Human trichinellosis is an important food-borne zoonosis. Five species of trichinellosis (T. spiralis, T. nativa, T. nelsoni, T. britovi and T. pseudospiralis) can infect man.65 The most important species that infect man is trichinellosis spiralis. The infective larvae in the muscle are surround by a host capsule which is modified striated muscle known as “nurse” cell. Symptomatic treatment of trichinellosis includes analgesics and corticosteroids. Specific treatment is with mebendazole 200 to 400 mg three times a day for 10 days. Albendazole 200 to 400 mg three times a day for 10 days. Albendazole is a pro-drug that is metabolized to mebendazole. Ivermectin is an effective treatment for trichinellosis in the acute phase. Ivermectin is administered to patients as a single dose of 200 μg/kg. The parasites are usually seen in the pulmonary artery where they produce an embolism ultimately leading to the formation of a pulmonary nodule or “coin lesion”. Clinical symptoms are chest pain, cough, fever, hemoptysis and dyspnea. CT scan may show a well-defined nodule with smooth margin connected to an arterial branch.82

Pulmonary Dirofilariasis

Pulmonary dirofilariasis is a zoonotic infection caused by filarial nematodes, parasites has been named as “dog heart worm”. Humans are accidental hosts of this parasite which is transmitted to man by the mosquito. The parasites are usually seen in the pulmonary artery where they produce an embolism ultimately leading to the formation of a pulmonary nodule or “coin lesion”. Clinical symptoms are chest pain, cough, fever, hemoptysis and dyspnea. CT scan may show a well-defined nodule with smooth margin connected to an arterial branch.82

REFERENCES