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Research Article

METICULOUSLY, KEEP AN EYE ON WORMY LUNG INFECTIONS

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ABSTRACT

Parasitic infection is a severe issue that impacts millions of people around the world. The normal distribution of parasitic illnesses far distant from endemic areas has been altered by immigration and global warming. A wide range of helminthic and protozoan parasitic illnesses can impact the respiratory system. The identification of parasitic infections of the lungs may be delayed due to the wide range of clinical and radiographic manifestations that make diagnosis difficult. Parasitic lung infections affect mainly immunocompetent and immunocompromised persons globally, and they can impact the lung tissue in a variety of ways. Some of these diseases have clinical and radiographic features that are similar to tuberculosis and cancer. In the differential diagnosis of these kind of lung illnesses, parasite infections should be considered. Most parasite disorders of the lungs can be treated medically or surgically if caught early enough. Entamoeba histolytica, Plasmodium falciparum, *Trypanosoma gambiense, Ascaris lumbricoides, Strongyloides stercoralis, Dirofilariasis, Echinococcus granulosus, Schistosoma species, Paragonimus westermani,* commonly affect the lungs.

INTRODUCTION

Pleuropulmonary amoebiasis is easily confused with pulmonary TB, lung cancer, bacterial lung abscess.(1,2) Lung disorder in patients with HIV vary alter with opportunistic infections. (3) *Strongyloides stercoralis,* occurred when larvae from contaminated feces penetrate the skin.(4,5)

Un-ruptured hydatids on computed tomography appeared as well demarcated spherical or oval cystic lesions with magnify the walls in the lung.(6) Calcification of hydatid cyst is rare in lungs.(7) Cyst Rupture of the cyst can lead to lung abscess formation. (8) Alveolar echino coccosis (AE) is also rare. (9,10)

The fatal complication of chronic schistosomiasis is *Schistosoma*-associated pulmonary arterial hypertension (SchPAH). *Paragonimus westermani,* primarily infect the lung, but extrapulmonary infections are also encountered. (11-13) Respiratory symptoms at admission have been related to worse prognosis in children, possibly due to existing pulmonary infection. (14)

In USA, Toxoplasma gondii pneumonia is observed most frequently in acquired-immunodeficiency-syndrome (AIDS) patients. However, pulmonary involvement is poorly described (15) Amoebiasis is caused by the enteric protozoan Entamoeba histolytica, a leading basis of deaths accounted to parasites, succeeding malaria and schistosomiasis. (16) Pleuro-pulmonary involvement, seen as the second most common extra-intestinal infection, is frequently associated with amebic liver abscess.(17)

The overall prevalence of schistosomiasis infection, disease forms, is expected to be between 200 and 300 million individuals worldwide. (18,19)

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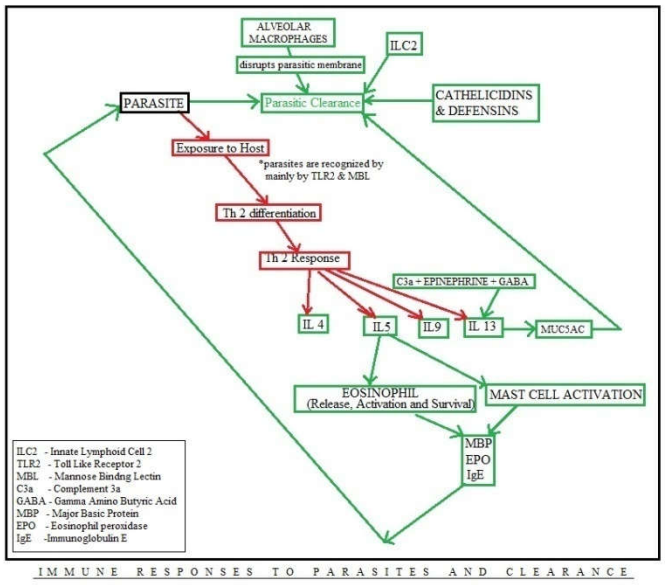
Even mild cases of Ascaris infection should be treated to prevent complications from parasite migration, and however, during active migration through the lungs, medical therapy is not indicated, secondary to the increased risk of pneumonitis.(20)

Parasitic immunity

Immunity is a rule. Entamoeba histolytica, Plasmodium falcparum, *Trypanosoma gambiense, Ascaris lumbricoides, Strongyloides stercoralis, dirofilariasis, Echinococcus granulosus, schistosoma species, paragonimus westermani,* and other parasitic infections commonly affect the lungs.
It is highly common in intestinal roundworms.

In these conditions there is ample opportunity for prolonged absorption of antigenic materials from the parasites, which can give rise to hypersensitivity reactions at the site where they are released. The best evidence that this can be seen in hydatid disease. The cysts contain excretory products. These cysts are surrounded by inflammatory cells, with many eosinophilia and plasma cells. If the hydatid fluid is absorbed into circulation, the patient is liable to undergo an acute anaphylactic attack. In some patients delayed type of skin reactions may occur. (21)

Diagram



IMMUNE RESPONSES TO PARASITES AND CLEARANCE

Pulmonary Amoebiasis

Entamoeba histolytica is a protozoan infection of humans responsible for up to 100,000 deaths annually. It is endemic in most temperate and tropical areas of the world. Serological evidence of prior or current infection with Entamoeba histolytica is present in 5 to 50 percent of individuals. Entamoeba histolytica is infectious in its cyst form. Transmission usually occurs as a result of contamination of food or water, but may also occur by means of oral-anal contact. Infection is common in developing countries. There does not seem to be an increased risk of invasive disease in persons with HIV infection.

Entamoeba histolytica has a simple life cycle involving an infectious cyst and an amoeboid trophozoite phase. Cysts may survive in the external environment for several weeks to months, especially in damp conditions and temperatures between −5◦C and 40◦C.

Clinical Manifestations

The incubation period for intestinal amebiasis is usually 1 to 4weeks, but ranges from a few days to months. Infection with *E .histolytica* is asymptomatic in up to 90 percent of cases, but may cause a range of gastrointestinal symptoms from mild diarrhoea to severe colitis with bloody diarrhoea. Extraintestinal disease most commonly occurs in the liver, and manifests as a liver abscess. In this instance, trophozoites migrate to the liver via the portal veins, and infection results in inflammation, necrosis, and ultimately an abscess. Eighty percent of amoebic hepatic abscesses are solitary, and 80 percent occur in the right lobe of the liver.

Thoracic manifestations of *E. histolytica* infection are rare; they have been reported in 2 to 3 percent of cases of invasive amebiasis. They can be considered chiefly as either pleuro-pulmonary or pericardial. Amoebic pulmonary disease may occur by a number of mechanisms. It most often occurs as the result of a concomitant liver abscess. The abscess may result in a sympathetic pleural effusion that does not require specific therapy.

Pleuritic chest pain along with respiratory symptoms such as cough, dyspnea and physical examination consistent with pleural effusion further suggest involvement of the pleuro-pulmonary system. Sputum production may range from scant to copious, and sputum may contain purulent material, particularly if a hepatobronchial fistula has developed. Classically, the purulent material of amoebic abscess is reddish “anchovy paste.” Jaundice is uncommon. Leukocytosis may be present, but eosinophilia is not a feature of the disease.

Laboratory Diagnosis

Stool microscopy may reveal the presence of *E. histolytica* trophozoites or cysts, but microscopy is neither a sensitivenor a specific method for diagnosis of amebiasis. Microscopyis unable to distinguish *E. histolytica* from non-pathogenic *E. dispar* and extraintestinal disease is associated with presenceof the pathogen in stool in only a minority of cases. Stoolmay be concentrated and stained with iodine to evaluate forthe presence of cysts. Trophozoites are best seen on a freshsmear with iron haematoxylin and trichome stain. Microscopyof abscess fluid occasionally may reveal presence of trophozoites. Diagnosis of extra intestinal disease is best made by a combination of microscopic examination of samples, antigen detection, and serology tests. Antigen detection methods are increasingly commercially available to detect *E. histolytica.*
Serological analysis may be negative in early infection. The presence of IgG antibodies does not always indicate active disease; IgG may persist for many years after active infection.

Treatment

Metronidazole or imidazole is effective in the treatment of individuals with intestinal and extra intestinal amebiasis. To prevent a relapse, a luminal agent such as iodoquinol or paromomycin also should be administered.

Pulmonary Toxoplasmosis

Toxoplasma gondi causes toxoplasmosis, which is frequently reported in immunosuppressed conditions like HIV AIDS and post bone marrow / solid organ transplantation. Pulmonary involvement is commonly seen. This can range from bilateral pulmonary interstitial infiltrated, discrete pulmonary opacities to ARDS. Toxoplasma pneumonia is associated with 40%

mortality. Most common symptoms include cough and breathing difficulty.

Diagnosis

Sputum PCR can identify pathogen but sensitivity is still not known. Confirmation of pulmonary involvement may need biopsy/ BAL.

Treatment

Pyrimethamine and a sulphonamide are the treatment of choice for toxoplasmosis. In case of sulphonamide intolerance, clindamycin or atovaquone can be given. Primary suppressive therapy is given to treat the condition. Secondary suppressive therapy is given throughout immunosuppression to prevent the relapse. Leucovorin therapy is combined to prevent bone marrow suppression.

Pulmonary Babesiosis

Usually tick borne infection but may also occur due to infected blood transfusion. *Babesia microti* is distributed in United states where as *Babesia divergens* is seen in European countries. Usually Babesia invades RBC, multiplies and lyses RBCs (haemolysis).Disease is asymptomatic or mild in immunocompetent hosts. Symptoms can be flu like characterised by fever and GI symptoms. Severe disease is most common in asplenia, immunosuppressed and elders. Persistent disease is seen in HIV AIDS. Pulmonary involvement includes ARDS / NON CARDIOGENIC PULMONARY EDEMA / Ground glass opacities. Appropriate exposure history , fever and sepsis syndrome needs evaluation for babesia.

Diagnosis is confirmed by microscopic examination of peripheral blood. Treatment is combination therapy of anti babesia drugs - atovaquone, azithromycin, quinine and clindamycin.

Pulmonary Leishmaniasis

Leishmaniasis is caused by an intracellular protozoa parasite in the *Leishmania donovani.* The Leishmania parasites are widely distributed and are usually transmitted through the bite of an infective sand fly (*Phlebatomus argentipus).* Infection of humans may be asymptomatic, or may involve the skin (cutaneous leishmaniasis), the mouth or nose (mucocutaneous leishmaniasis), or generalized (visceral leishmaniasis). It is most common in South and Central Asia, Middle East, Mediterranean, Balkans, North Africa and South America. Specific organisms are often responsible for specific clinical syndromes, although overlap occurs. The parasite exists as an intracellular organism (amastigote) in host macrophages and as an extracellular promastigotes in the sand fly gut—being inoculated into the host's skin during the bite of the fly.

Clinical Features

Cutaneous syndromes may range from localized disease, usually on exposed areas of skin, to widespread cutaneous involvement. In localized disease, painless ulcers develop and most often resolve spontaneously over months Severe destruction and disfiguration of the face with resultant aspiration pneumonitis may occur.

Visceral leishmaniasis is usually caused by *L. donovani, L. infantum or L. chagasi.* Individuals with visceral leishmaniasis usually present with fever, hepato-splenomegaly, weight loss, pancytopenia and hypergammaglobulinemia. Lymphadenopathy is commonly present. Gastrointestinal symptoms are common in advanced disease, and individuals with untreated visceral leishmaniasis develop a wasting syndrome. Pneumonia and tuberculosis may be the cause of death.

Pulmonary involvement from leishmaniasis in immune competent hosts is rare, but interstitial pneumonitis has been reported. However, immunosuppressed patients, particularly those with HIV infection, are at increased risk of atypical disease ,an increase in the incidence of HIV infection has caused an increase in the number of advanced cases of visceral leishmaniasis. Patients with leishmaniasis and HIV infection may present with cough, dyspnea, hemoptysis, granulomatous mediastinal lymphadenopathy, solitary pulmonary nodules, or pleural effusions. Treatment of leishmaniasis depends on type of disease (cutaneous vs. mucocutaneous vs. visceral), infecting species, parasite resistance patterns, severity of disease, and status of the host. Therapeutic options include amphotericin B, liposomal preparation of amphotericin, pentavalent antimony (sodium antimonylgluconate or N-methylglucamine antimonate), pentamidine and miltefosine

Pulmonary Malaria

The majority of malaria-related fatalities are a result of infection with *P. falciparum*. Malaria is acquired by the bite of an infective female *Anopheles* sp. mosquito.

Clinical Features

Malaria related morbidity and mortality occurs predominantly in children and pregnant women. Most individuals who live in endemic areas develop partial immunity to symptomatic disease as they age. This immunity does not prevent infection, but reduces the frequency of symptoms and severity of disease despite ongoing parasitemia. Infection with any of the four human-specific species of malaria causes haemolysis resulting in anaemia that may sometimes be severe. Patients with malaria typically present with fever and a constellation of other nonspecific symptoms such as headache, chills, and myalgias. Vomiting, nausea, diarrhoea and cough also may be present. Anaemia, thrombocytopenia, and hepatosplenomegaly may be present. Sequestration of infected erythrocytes in the brain may cause cerebral malaria. Individuals with cerebral malaria may present with altered mental status, seizures, focal neurologic findings, or coma. Even with treatment in modern intensive care units, mortality during cerebral malaria is high. Other organ-specific complications of infection are also largely related to microvascular sequestration of parasitized red blood cells in specific tissues, and may manifest as placental, renal, or pulmonary dysfunction.

Pulmonary Features

Pulmonary symptoms such as cough and increased respiratory rate are common during malaria. In African children, malaria frequently presents with symptoms and signs suggestive of pneumonia. Metabolic acidosis is an important cause of respiratory distress in these children, but pneumonitis as a result of sequestered parasitized red blood cells is also responsible. In more severe disease, noncardiogenic pulmonary edema may develop. Initial acute lung injury may progress to acute respiratory distress syndrome (ARDS).

ARDS has also been reported in cases of malaria caused by *P. vivax,* and one case of pulmonary edema has been reported complicating a case of *P. ovale* malaria; however, significant

pulmonary morbidity is largely confined to infection by *P. falciparum*, and usually occurs in the context of multisystem involvement. The principal differential diagnosis for pulmonary disease in malaria infection is metabolic acidosis and bacterial pneumonia.

Diagnosis

The diagnosis of malaria should be considered in any symptomatic patient who has had exposure to the parasite in a malaria endemic area. Rare cases of transmission by blood products, organ transplantation or congenital infection also have occurred. Individuals with malaria caused by *P. falciparum* usually develop symptoms within 3 months of the mosquito bite, and usually within 1 month. Since *P. vivax* and *P. ovale* have potentially dormant liver phases, individuals infected with these parasites may not develop symptoms for many months after exposure.

Malaria is usually diagnosed through microscopic examination of blood smears. A thick smear examined by an experienced microscopist is highly sensitive for detecting infection. Review of a thin smear provides further detail to allow identification of the infecting species. Antigen detection assays identifying falciparum-specific histidine rich protein 2 (HRP-2), or parasite lactate dehydrogenase (pLDH, which is present in all four species), are also commercially available.

Treatment

Appropriate treatment of individuals with malaria depends on the infecting species, severity of disease, age, pregnancy status and the ability of the patient to take drugs by mouth.

Non-falciparum malaria is generally susceptible to chloroquine phosphate. Non-severe cases of chloroquine-resistant falciparum malaria can be treated with oral atovaquone, proguanil or mefloquine, or quinine sulfate in combination with either doxycycline or clindamycin. Severe cases of falciparum malaria should be treated parenterally with quinine dihydrochloride, quinidine gluconate, or artesunate. Therapy with artemisinin-based combinations of drugs is increasingly being recommended, particularly in endemic areas; however, artemisinin derivatives are not commercially available in many countries. Artesunate is the drug of choice in severe malaria.

Primaquine should not be administered toindividuals deficient in glucose-6-phosphate dehydrogenaseor pregnant women. Pulmonary manifestations and otherorgan-specific complications of severe infection are treatedsupportively. Mechanical ventilation may be required. Earlyinstitution of renal replacement therapy may prevent subsequentdevelopment of ARDS in severe malaria. Exchangetransfusion has been recommended for patients with severefalciparum-associated disease with parasitemia levels over5 percent, although data on efficacy are controversial.

Pulmonary Ascariasis

Clinical features

Respiratory symptoms occur as a result of airway hyper reactivity and bronchospasm.
Loffler's syndrome:
Self-limiting inflammation of lungs with hyper eosinophilia.
Cough with mucous sputum
Fever, malase, Loss of Apetite, headache,myalgia, hemoptysis, wheezing, shortness of breath.

Diagnosis

sputum: eosinophilia, Charcot laden crystals
chest x ray- unilateral or bilateral, transient,migratory opacity

Treatment

albendaole 400mg once for 2 to 3 weeks
mebendazole 00mg per day for 2 to 3 days.
Ivermectin 200ug/kg per day for 2 weeks.

Pulmonary Ancylostomiasis

Clinical features

Intense pruritus, erythema, rash,fever, cough, wheeze, transient pulmonary infiltrates.
Complete blood picture; iron deficiency anemia.

***Treatment*:**

Mebendazole 10mg two times per day for 3 days
Albendazole 400mg single dose
Pyrantel pamoate 11mg/kg single dose

***Strongyloides stercoralis*:**

Clinical features:
Cough Shortness of breath, wheee, hemoptysis.

***Treatment*:**
Ivermectin 200ug/kg for 1 or two days
Thiabendazole 25ug/kg twice a day for 2 days
Albendazole 400mg twice a day for 5 days

Pulmonary Dirofilariasis:

***Clinical features*:**

Cough chest pain, hemoptysis, fever Shortness of breath
CT Chest:; well defined nodule with margins connected to arterial branch
Wedge biopsy for HPE
Thoracoscopy biopsy for HPE.

***Treatment*:**
No specific treatment

Toxocariasis (visceral larva migrans)

Clinical features

Cough, wheezing, pulmonary infiltrates,
Chest x ray transient migratory infiltrates

Treatment

Benign self limiting disease.
Corticosteroid to decrease inflammation

Pulmonary trichinellosis

Caused by Trichenella spiralis, native, nelosin, britovi, pseudo spiralis
Pulmonary symptoms:
Shortness of breath, cough.
Chest x ray: pulmonary infiltrates.
Blood: leukocytosis, esonophilia, raised sr LDH,

Treatment

Mebendazole 200mg to 400mg 3 times a day for 3 days followed by 400mg 3 times a day for 10 days Albendazole 400mg per day for 3 days followed by 800mg per day for 15 days

Lung fluke infections are treated with praziquantel, a drug used to eliminate flukes from the body (called an anthelmintic drug). An alternative is triclabendazole. If the brain is infected, corticosteroids may also be given. They help control the inflammation that develops when the drug kills the flukes

Popular Parasitic Infection Drugs

- Flagyl. metronidazole.
- Stromectol. ivermectin.
- Tindamax. tinidazole.
- Vaniqa.
- Albenza. albendazole.
- Emverm.
- Biltricide. praziquantel.
- Ticovac.

Tropical pulmonary eosinophilia or Weingarten's' lung or Eosinophilic lung,

It is by *Brugia malayi* and *Wucheraria bancrufti* TPE is pervasive in endemic regions of the world.
Patients suffer from fever, cough and massive eosinophilia. This is described as pseudo-Tuberculosis condition. It is called wingarten syndrome.
Pulmonary dirofilariasis, Visceral larva migrans, Pulmonary richinellosis, schistosomiasis, Paragonimiasis, Hydatid, Eosinophilia and pulmonary tuberculosis, and Brucellosis. Fungus induced eosinophilic lung disease are Coccidiomycosis, Aspergillosis, Cryptococcosis and Histoplasmosis. Clinical features of TPE-.

Clinical features

Nocturnal dry cough,
Low grade fever, fatigue.
PFT Restrictive pattern
Chest radiograph: reticulonodular opacity
BAL: increased IgE, Esinophils>3000

Treatment

There is good evidence that albendazole alone; or addition of albendazole to diethylcarbamazine or ivermectin, makes minimal difference in clearing microfilaria or adult worms from blood circulation (25) Diethylcarbamazine-medicated salt is effective in controlling lymphatic filariasis while maintaining its coverage at 90% in the community for six months (26)

Parasitic infections

Dirofilaria immitis are frequently found in the lung periphery, particularly.the right lower lobe (22,23) The lung lesion usually less than 3 cm in size appear as a solitary-coin nodule or multiple nodules (24) *Trichinella spiralis* larvae penetrate into the submucosa and are carried in the circulatory and lymphatic systems to various organs, including lungs.(27)

The *Necator americanus* larvae break through the pulmonary capillaries to enter the alveoli.and cause Bronchitis and bronchopneumonia. Bronchitis and bronchopneumonia can occur when the *Ancylostoma duodenale* larvae break through the pulmonary capillaries to enter the alveolar spaces *Ascaris lumbricoides* may produce hypersensitivity in the lungs and result in peribronchial inflammation, During migration of filariform larvae through the lungs, bronchopneumonia and alveolar hemorrhages can occur (28)

The metacercariae from the human intestine passes through several organs and tissues to reach the lungs (29) Pulmonary paragonimiasis produce cough, rusty brown or blood-stained sputum or recurrent hemoptysis, chest pain, fever, chest tightness, pneumonitis.(30)

Cough, hemoptysis, fever, and chest pain were observed in the *Paragonimus* ova-positive patients, particularly cough and hemoptysis. There is no difference noted in most of the chest signs of both paragonimiasis and tuberculosis . Presence of crepitations in children with tuberculosis is a significant finding.(31) Tropical pulmonary eosinophilia (TPE) is one of the main causes of pulmonary eosinophilia in tropical countries and is prevalent in filarial endemic regions of the world particularly Southeast Asia(32)

Patients with babesiosis are frequently complicated by noncardiogenic diffuse-bilateral-interstitial pulmonary edema and adult respiratory distress syndrome (33) Early pregnancy with toxoplasmosis can cause fetal death and chorioretinitis and neurological symptoms in the newborn, whereas chronic disease can cause chorioretinitis, jaundice, convulsion, and encephalitis (34)

Chest roentgenograms in three cases of malaria with sickle cell anemia also reported that all reported patients demonstrated bilaterally pulmonary infiltrates (35) Sanklecha et al. reported three cases of childhood falciparum malaria (36) Chest roentgenograms are usually nonspecific, but they should be recognized in high endemic areas of malaria (37). Chest roentgenogram in a patient with *Plasmodium vivax* malaria demonstrated diffuse bilateral alveolar opacities which indicated acute respiratory distress syndrome (38)

A few of these eggs of *Schistosoma hematobium* remain in the host tissue and can cause granuloma formation around them (39) Acute manifestations, called "Katayama syndrome" can develop three to eight weeks after skin penetration (40)

CONCLUSION

Immigration and global climate change have altered the natural distribution of parasitic diseases .Helminth and protozoan parasitic diseases can be affected by a broad spectrum of the respiratory system. Although on one aspect parasitic infections are decreasing globally as a result of enhanced socioeconomic conditions and improved sanitary conditions, urbanization, global climate change etc, on an another aspect issues such as international travel, and an increase in the number of immunocompromised people have increased the number of people who are vulnerable to parasitic diseases.As a result, pulmonologists must understand the etiology, life cycles, clinical manifestations, laboratory diagnostics, and therapies of these "pneumatodes" in order to provide appropriate care to these patients.

The declaration of interest forms were reviewed, and no conflicts were identified.

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