Chapter 7

Tropical Lung Diseases

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1. Introduction

Infectious disease results from the disruption of the balance between the host and the pathogen. Pathogen influencing factors include virulence, immunoevasion capacities, and drug resistance ability. According to the host, disease outcome relay on many factors such as: immunocompetence, comorbidities, terrain (ageing, malnutrition).

The lung epithelium is a large surface exposed to outside aggression. The environment plays a key role in the onset and development of illness by many factors such as: climate, social and cultural habits, vegetations, degree of industrial, or domestic pollutions...

Despite this exaggerated exposition, the lung is nevertheless protected through non specific and/ or specific defense mechanisms (Agostini CV, Chilosi M, Zambello R et *al*, 1993).

1.1. Non specific defense mechanisms

According to its size, a foreign substance can reach the upper or lower respiratory tract, and be cleaned via the mucociliary escalator, during coughing or sneezing. Epithelial cells also secrete many agents such as lysozyme, toxic oxygen radicals, with antimicrobial properties. The renewal of the epithelium, as for the skin, is an additional protecting property.

Innate immune recognition is a second protective mechanism implicating immune cells and secreted mediators. Its under the control of cells carrying receptors for recognition of foreign antigens (PRRs: pathogen recognition receptors) such as macrophages, dendritic cells, neutrophils, mastocytes, epithelial cells, NK cells, and fibroblasts which can link microbial structures (PAMPS: pathogen associated molecular patterns) for further destruction (Ahnen DJ, 1985).

1.2. Specific defense mechanisms

Adaptive immunity relays on the interaction between antigen-presenting cells (macrophages, dendritic cells, neutrophils) with specific T-lymphocytes in the context of cell mediated immunity, as well as, on the antibodies production by activated B-lymphocytes (humoral immunity) (Kohlmeter JE, Woodland DL, 2006).

2. Tropical parasitic lung diseases

2.1. Overview

Protozoa and helminthes can affect the lung as a primary site, or a complication. Some parasites have a migration cycle through the lung (larva migrans), inducing blood and tissue eosinophilia. Tissue and peripheral blood eosinophilia are elicited by chimiotactic activity of released inflammatory mediators, such as cytokines (IL-3, IL-5), which play a key role in activation and differentiation of eosinophils. Eosinophils secrete various substances, some with antiparasitis properties, others favoring tissue damage in targeted organs. Elevated IgE level observed in these conditions relay on the Th2-lymphocytes, stimulating antibodies production by B-lymphocytes (Om P Sharma, 1991; VijayanVK, 2008).

Clinical manifestations of the lung involvement could be acute: asthma –like syndrome, or Loeffler's syndrome, with dyspnea, wheezing, cough (Ford RM, 1996); or chronic such as hemoptysis or right heart failure signs. Acute manifestations depend on immunological reaction (hypersensitivity), and chronic feature relay on the mechanical action of pathogen on the vessels and tissues. (vg: schistosoma eggs in the pulmonary artery and pulmonary hypertension). (Santiago M et *al*., 2005).

Löeffler's syndrome represent transient clinical, immunological and radiological manifestations due to parasites whose life cycle elicit a transit through the lung or not, and to drug reactions

Hypereosinophilia observed in this syndrome is antigen –induced, and circulating IL-5, is the key mechanism for the recruitment and differentiation of the eosinophils.

2.2. Helminthic parasites

The three classes of helminths (Cestoidea, Trematoda, and Nematoda) can affect the lung.

2.2.1. Nematodes and the lung

This group include: ascariasis, strongyloidiasis,ancylostomiasis, tropical pulmonary eosinophilia, pulmonary dirofilariasis, and pulmonary trichinellosis.

2.2.1.1. Pulmonary ascariasis

Ascariasis is a round worm infection caused by Ascaris lumbricoides. This nematode disease affects ± 25% of the world population whose 95% are in Africa (Crompton, 1999).

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Mode of contamination

The starting point is the survival of eggs able to contaminate ingesta by the new host. Poor sanitation, fecal contamination of food or water, are the main risk factors of dissemination. Embryonated eggs (2-4 weeks), when ingested are dissolved in the stomach juice and then release rabdoid larvae in the duodenum, before migration through the intestine. Larvae then enter the portal system via capillaries and lymphatics, after penetrating the wall of the intestine. The involvement of the hepatic circulation allow the right heart and lung invasion. The eggs reach the alveolar space after crossing the capillary walls and can be swallowed and then reach again the small intestine to mute in adult forms. This pilgrimage can take 14 days after ingestion (Sarinas PS, Chitkara RK, 1997).

Pathophysiology

Adult worms or migrating larvae exert a mechanical pressure on lung structures inducing inflammatory responses, leading to granuloma formation with eosinophils, neutrophils and macrophages. Activated cells release cytokines such as IL-3 and IL-5 involved in the recruitment and differenciation of eosinophils, explaining the blood and tissue eosinophilia reported. TH2 Lymphocytes are responsible for the high IgE (Yazicioglia, 1996) and IgG4 levels (Santra A, 2001) described.

Hypersensitivity reaction inducing peribronchial inflammation, mucus production, and sometimes bronchospasm is responsible for the clinical manifestations.

Diagnosis

Abdominal manifestations are currently reported: gastric pain, vomiting, diarrhea, abdominal discomfort. In some complicated cases pancreatitis or obstruction of biliary duct or small intestine can occur, caused by adult worms.

Respiratory symptoms due to larval migration in the lungs, consist in mild cough or Loeffler's syndrome (Ford RM, 1996). This syndrome associates respiratory symptoms (dry cough, wheezing, dyspnoea) with blood and lung eosinophilia, and chest radiograph with fleeting infiltrates. General symptoms such as fever, loss of appetite, myalgia can be observed.

Pneumonia is a more rare condition with ascariasis infection

Laboratory findings

Stool examination may show eggs or adult worms.

Larvae may be found in respiratory secretions

Serological approach (specific IgG4 antibodies) could be helpful (Santra A *et al*., 2001; Bhattacharya T, 2001).

Blood hypereosinophilia and high IgE level are common.

Chest radiograph may show migrating inhomogenous alveolar infiltrates.

Treatment

The treatment aims to eradicate intestinal colonization responsible for recurrent respiratory episodes.

Mebendazole (100 mg twice a day for three days, or 500mg one day) and Albendazole (400mg, single dose) are the drugs of choice. Pyrantel Pamoate, Levamisole, and Piperazine are alternative choices. Ivermectine, an antifilarial drug has shown efficacy in the treatment of ascariasis.

2.2.1.2. Toxocariasis

a roundworm of dog and cat can infect human, who is an intermediate host, and determine a Loeffler's like syndrome caused by larva migrans as with Ascaris.

Severe respiratory syndromes (ARDS) have rarely been observed (Bartelink AK et *al*., 1983), while asthma-like symptoms are currently reported among pulmonary manifestations.

Defects in neutrophil function have been reported in children with visceral larva migrans. This defect should be explained by the neutrophilic adherence to larvae illustrated elsewhere in animal models (Martin Huwer, 1989).

Exacerbations of inflammatory reactions during antihelminthic treatment emphasize the need of combination with corticosteroids.

2.2.1.3. Pulmonary strongyloidiasis

The causative agent is Strongyloides stercoralis, endemic in the tropics and subtropics. Eggs containing larvae ready to hatch, are the contaminating form after penetrating the skin; they then disseminate in all tissues via venous or lymphatic route in immunodeficient host (Cook, 1987; Longworth and Weller, 1986).

Autoinfestation is a common feature of this parasite, meaning the penetration of filariform larvae in the perianal skin of the infected subject without leaving the host. This phenomenon can determine the persistence of infection even many years after (Scowden EB, 1978).

Lung invasion result from larvae carried by the blood stream to the right heart and then to the lung. Larvae can pierce the pulmonary capillaries and reach the alveoli through the alveolo-capillary membrane, inducing non cardiogenic edema and hemoptyses. After their migration through the bronchi and superior respiratory tree, some larvae can be swallowed in the intestine. Hyperinfection syndrome is related to severity of symptoms in the lung and the intestine, which are common sites of the parasitic life cycle, while disseminated disease represent the invasion of other organs not generally involved in the growth of the parasite (Longoworth DL, and Weller PF, 1986).

Pathophysiology

The skin penetration by the larva determines an hypersensitivity reaction as the result of a strong cell mediated immunity reaction in immunocompetent host preventing the tissue invasion (Neva FA, 1986). Marked autoinfection and subsequent hyperinfection are the main determinants of tissue dissemination in immunosuppressed subjects such as AIDS patients

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(Lessman KD, 1993), individuals on long term corticosteroid therapy, or with malnutrition, lymphomas etc..(Casati A, 1996; Genta RM, 1989).

Mechanical action by the adult worms and host reaction are responsible for digestive manifestations.

Secondary gram negative bacterial infection by gram- pathogen is frequent, bacteria being carried by larvae during the crossing of the intestinal wall. The migration through the lung can determine bronchopneumonia,alveolar hemorrhages, and pulmonary abscess and haemoptyses.

Diagnosis

Lung strongyloidiasis is commonly asymptomatic in immunocompetant individuals, or present with mild symptoms.

Gastrointestinal manifestations, cough, dyspnoea, wheezing and haemoptysis are frequent during lung involvement. Hyperinfection and disseminated disease are commonly fatal, which elderly individuals and those on long term corticosteroid therapy, or having hematologic malignancies being at higher risk for the latter.

Eosinophilic pleural effusion have been reported among pulmonary manifestations of strongyloidiasis; and rare cases of acute respiratory failure due to respiratory muscle paralysis have been observed (da Silva OA, 1981).

The hyperinfection syndrome with Strongyloides stercoralis can worsen asthma or COPD exacerbations in some patients (Sen P, 1995; Ossorio MA, 1990).

Peripheral blood eosinophilia, anemia, and hypoalbuminemaia are current laboratory findings.

Larva may be observed in repeted stool specimen examination due to the low parasitic load in immunocompetent individuals.

Pulmonary secretions, duodenal juice, may be contributive for parasite identification.

Serology is also interesting for detection of specific antibodies.

Treatment

Thiabendazole (25 mg/kg, twice a day for two days), Albendazole (400 mg, twice a day/5 days), Ivermectine (200 microgr/kg for one or two days) are recommended.

2.2.1.4. Pulmonary ancylostomiasis

Ancylostoma duodenale and *Necator americanus* are the two helminths in this group.

Eggs eliminated with the feces continue the maturation in the soil, where larvae will penetrate the intact skin to infect the man, which is the only definitive host. The oral route of infection is possible for *Ancylostoma duodenale*. Migrant larvae reach the lung structures has illustrate for other helminths, inducing a Loeffler's syndrome. The larvae of *Ancylostoma du-*

odenale can reach the mammary glands and be transmitted to the child by maternal breast-feeding (Yu Sen-Hai, 1995).

Pathophysiology

The lung migration can elicit blood eosinophilia as for other larva migrans. Larvae release low molecular weight proteins with anticoagulant properties (Cappello M, 1993), favoring blood loss during the intestinal capillaries destruction. Anemia with iron deficiency is often associated with this infection.

Local prurit and erythema follow skin penetration. Clinical, immunological and radiological manifestations of Loeffler's syndrome can be observed during larval migration through the lung.

Diagnosis

Gastrointestinal symptoms associated with respiratory asthma-like symptoms in an exposed individual are suggestive of parasitic lung infections

Laboratory findings

Stool examination may demonstrate the presence of eggs, blood samples may identify eosinophilia and iron deficient anemia.

Treatment

Mebendazole and Albendazole have equivalent efficacy.

Ivermectine has been reported as an alternative.

2.2.1.5. Tropical Pulmonary Eosinophilia (TPE)

This syndrome is an immunological response of the host to filarial parasites invasion, mainly Wuchereria bancrofti and Brugia malayi, affecting only 1% of patients with filariasis (Johnson S, 1994).

The filarial etiology has been suggested by the prevalent occurrence of the syndrome in the world regions with reported high filarialsis prevalence (South-East Asia), as well as the recovery after antifilarial drug administration.

Pathophysiology

The hypersensitivity reaction to filarial antigens induce a strong eosinophilic inflammatory response in the lungs. The microfilariae in lymphatics invade the pulmonary circulation and parenchyma with further granulomatous and fibrosing pattern. The concentration of eosinophils in the lung has been shown to be more significant than in the peripheral blood suggesting the compartimentalisation, and a prominent role of these cells in the pulmonary involvement and clinical manifestations (Pinkstori P, 1987).

Diagnosis

Epidemiological data state a male predominance in TPE (sex ratio M/F; 4:1), a disease of children and young adult (15-40 yrs).

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Respiratory manifestations are frequently associated with systemic symptoms such as: fever, weight loss, and fatigue.

The diagnosis should be evoked after exclusion of other causes of pulmonary eosinophilia as other helmintic diseases, or drug use.

Laboratory findings

Leucocytosis is common in TPE, with marked peripheral blood eosinophilia; and elevated erythrocyte sedimentation rate is often reported.

Serological examinations may reveal high level of specific IgG or IgE antibodies to microfilariae.

Stool examination is very important to determine co-infection with other helminths.

The chest radiograph may show miliairy nodules mimicking miliairy tuberculosis.

Histopathological analysis of lung biopsies may illustrate microfilariae

Treatment

Diethylcarbamazine (6 mg/kg/day for 3 weeks), a current treatment of filariasis, has been successfully indicated In patients with TPE.

Steroids have shown additional improvement of symptoms in TPE patients.

2.2.1.6. Pulmonary dirofilariasis

This is a zoonosis caused by Dirofilaria immitis and repens. The nematode is a vascular parasite, with the human as accidental host. The parasite is transmitted by mosquito. The vascular location induce embolism of pulmonary artery, with subsequent pathophysiological manifestations such as: hemoptyses, chest pain, dyspnoea …

The disease is mainly asymptomatic

Thoracic imaging (TTDM) shows well delimited nodule neigbouring an arterial branch.

Histopathological analysis of pulmonary biopsies is strongly contributive for the diagnosis of this disease lacking specific treatment.

2.2.1.7. Pulmonary Trichinellosis

The food-borne disease is caused by *Trichinella spiralis* in the man.

The larvae grow in striated muscles after invading the bloodstream. Man is infected after ingesting partially or cooked or raw meat, and the larvae develop in the gut into adult worms.

Pulmonary symptoms include: dyspnoea due partially to the involvement of diaphragm, and cough.

Peripheral blood eosinophilia and elevated IgE level are depending on Th-2 cytokines released by TCD4 cells recruited by parasitic antigens. Elevated LDH enzyme suggest muscle involvement.

Larvae may be also identified in striated muscle biopsies.

Treatment

Mebendazole (for almost 2 weeks), associated to analgesics and corticosteroids is recommended.

2.2.1.8. CESTODES and the lung

Lung disease due to cestodes are caused by *Echinococcus granulosis* and *Echinococcus multilocularis* in the man. The lung and the liver are the main sites of cysts formation. Dogs are definitive hosts and eggs excreted in their faeces contaminate human when ingested with food or water.

Pulmonary symptoms are non specific and resemble asthma manifestations. Mechanical compression by hydatid cysts may influence clinical features. Rupture of Cysts in the bronchi could explain haemoptysis or excretion of cyst fluid, and may lead to anaphylaxis. Pneumothorax, pleural effusion, and emphysema are possible lung presentation.

Blood laboratory findings extend from eosinophilia to IgE production.

Serodiagnosis is helpful by detection of specific antibodies.

Chest radiograph manifestations may consist in multiple nodules mimicking lung tumors.

Treatment

Surgical resection of the lesion is the most relevant approach. Mebendazole, Albendazole and praziquantel are indicated, mainly in recurrent disease.

2.2.1.9. Trematodes and the lung

This group include pulmonary scistosomiasis and paragonimiasis.

Pulmonary Shistosomiasis

Schistosoma haematobium, mansoni, and *japonicum* are the major pathogenic species for human. *Shistosoma intercalatum* and *Shistosoma mekongi* are rarely encountered. Eggs excreted in urine or faeces of the infected patient contaminate water and infect the snail, intermediate host. The eggs then evolve in cercariae wich can penetrate the skin or be ingested by man. The adult worm stay in the bladder (*haematobium*), or in the gut (*S. mansoni, S. japonicum*).

Pathophysiology

Local inflammation occurs at the penetration site, while the onset of pulmonary manifestations may be acute or chronic.

Pulmonary inflammatory reaction may induce a cytotoxic reaction to migrating agents, and facilitate the secretions of chimiotactic mediators for eosinophils, involved in the *schistosoma* immunity (Schwartz E et *al*, 2000).

Acute manifestations result from immunologic hypersensitivity reactions, and consist of systemic complaints such as fever, myalgia, chills, diarrhea, abdominal pain, urticaria,

named Katayama syndrome. Pulmonary acute manifestations mimic Loffler's syndrome or Asthma-like syndrome (Walt F., 1954).

Chronic manifestations result from mechanical action of eggs or adult worm on the tissues. Pulmonary hypertension, haemoptysis, cor pulmonale could be observed. Pulmonary embolism is the consequence of small blood vessels obstruction by foreign bodies surrounded by various cells (eosinophils, neutrophils, lymphocytes, giant multinucleated cells (Wyler and Postlehwaite WE, 1983); Granuloma formation is the end-stage of the maintained inflammatory response to schistosomal antigens.

Optic microscopical identification of eggs in urine or stools is mandatory for the diagnosis. Rectal or bladder's mucosal biopsies could help demonstrating eggs of Schistosoma.

Extrapulmonary manifestations include hepatosplenomegaly due to portal hypertension. Schistosomiasis is an infectious cause of liver cirrhosis.

Peripheral blood eosinophilia and high IgE level are frequent.

Treatment

Cortcosteroides are indicate during acute phase and praziquantel (10-15 mg/kg, each 12 hours, one day). Artemether has shown effectiveness on juvenile forms of schistosomes.

Pulmonary Paragonimiasis

The food-borne zoonosis is more frequent in Asia, affecting± 20 million people (Schwartz E, 2002); Subacute or chronic lung manifestations are described. The agent Paragonimus westermani lives in the lung, and eggs are eliminated in the sputum or faeces. Miracidiae develop into cercariae in the snail before infecting the second intermediate host, the crabs. The man get infection after eating partially cooked or raw crabs.

Clinical manifestations are not specific, and chest radiograph may demonstrate cavitations as in tuberculosis. Pleural effusion or pneumothorax are frequently seen in paragonimiasis.

Treatment

Praziquantel is the first choice for treatment of this helminthic disease.

2.3. Protozoal lung diseases

2.3.1. Pulmonary amoebiasis

Entamoeba histolytica is the pathogenic form of infectious agent for the man.

Lung involvement is mainly linked to extension of amoebic liver abscess; hematogenous spread and aspiration have rarely been reported (Shamsuzzaman SM et *al*, 2002).

During intestinal transit and mutations, Trophozoites released after the cysts digestion by digestive secretions, may reach the muscularis mucosa and erode the lymphatics or the walls of mesenteric venules to invade the portal system of the liver. The parasitic embols

then obstruct the bloodstream and lead to abscess development with necrosis. The lung is the most frequent site of extra-intestinal invasion.

Clinical symptoms are related to the hepatic and intrathoracic implications. General symptoms including fever, right upper quadrant pain, cough, chest pain are frequent in the lung amoebiasis. Pleural effusion could develop, following hepatobronchial fistula. The parenchymal disease can present as pulmonary abscess with characteristic chocolate pus and air-space consolidation at chest radiograph. Elevation of right hemidiaphragm is an earlier radiographical feature in liver abscess.

E histolytica may be identified in sputum, in stools specimen or pleural pus.

The accuracy of serodiagnosis is established in the tissue amoebiasis, mainly in non endemic populations. PCR should also be more contributive, even not routinely performed in many institutions.

Treatment

Metronidazole is widely used, with established effectiveness. Lactoferrin and lactoferricins combined to low metronidazole doses has been proposed as an alternate therapeutic option.

2.3.2. Pulmonary malaria

Malaria is a public health problem in tropical and subtropical areas. With the increasing population travelling, mosquitos which transmit the disease can be carried out of the natural frontiers and cause illness in naïve, non exposed patients. Four species of *Plasmodium* are identified (*P falciparum, P. ovale, P. malariae, P. vivax*). *Plasmodium falciparum*, *vivax*, and *ovale* can cause acute lung injury, or acute respiratory distress syndrome (Mohan A et *al*, 2008).

Pathophysiology

The pathogen lives in the erythrocytes and could impair their functions. Impaired red cells motility, favored by exaggerated cytoadherence to the capillaries endothelium (Corbett CE et *al*, 1989), induce sequestration of the red and white blood cells in different organs, with subsequent deprivation in oxygen delivery, endothelial dysfunction, and enhancement of anaerobic metabolism. Multiple organ dysfunctions (MODS) is the condition leading to death. Red cells sequestration and destruction enhances the release of parasites and erythrocyte material in the bloodstream, inducing a vigorous inflammatory response

Pulmonary involvement extend from cough and dyspnoea, to fatal ARDS, non cardiogenic pulmonary edema, and intra-alveolar hemorrhages. Parenchymal disease due to plasmodial infections has not yet been clearly evidenced, due to numerous viral or bacterial co-infections, mainly in child under 5 years.

ARDS in malaria is more common in adults than in children, as well as in pregnant women and non immune individuals

The pathogenesis of ARDS in severe malaria is poorly understood. Sequestration of parasitized red cells in small vessels seems not to be the only underlying mechanism. Recent study

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in Indonesia has reported occurrence of ARDS in uncomplicated and severe malaria, in patients within the first 5 days after the start of the treatment, while peripheral parasitemia was decreased. The authors hypothesize that lung injury could then be related to an inflammatory response following treatment (Louis Schofield, Georges E. Grau, 2005). Their work suggest that impaired lung function is not exclusively the fact of microvascular obstruction by parasitized red cells, but include also white blood cells, contributing to impairment of gas transfer, subsequent to ventilation and perfusion mismatch (Anstey NM et *al*, 2002).

Pulmonary edema follows increased alveolar capillary permeability with extravasation of capillary content into the alveoli (Mohan Alladi et *al*, 2008)

Diagnosis

Systemic symptoms of malaria are: fever, myalgia, headache, loss of appetite, nausea, vomiting. Severe respiratory symptoms may be observed, following the onset of edema and respiratory distress syndrome.

Thick and thin stained blood smears are the routine laboratory examination to identify the plasmodium species.

Serodiagnosis and PCR of *plasmodium* in urine or saliva, may be contributive where available.

Chest radiograph demonstrates variable patterns such as lobar consolidation, pleural effusion, alveolar infiltrates suggesting pulmonary edema, or hemorrhages.

Treatment

Parenteral quinine is the drug of 1st choice for the treatment of severe malaria. Artemisinine derivatives are an alternative in case of contra-indications. Adjunctive therapy with clindamycine or doxycycline has been proposed in complicated malaria.

General resuscitation measures could be indicated in life threatening cases.

Antivectorial eradication, using insecticide treated bed-nets is widely utilized in endemic regions.

2.3.3. Pulmonary Toxoplasmosis

The disease caused by the Protozoan parasite, *Toxoplasma gondii* infects the man, after ingestion of cyst-contaminated food.

Immunocompromised individuals are at higher risk of developing toxoplasmosis with the central nervous system involvement as the most common complication.

Toxoplasma infection is asymptomatic in most immunocompetent humans. The pathogen is then destroyed by strong antibody dependent reactions or delayed type hypersensitivity mechanism. A strong Th1 cytokine profile is elicited by cells of innate immunity for efficient protection, and pathogen could be destroyed also by monocytes- derived mediators such as nitric oxide, which inhibits the parasite growth in different organs, mainly the lung and the central nervous system, as prominent targets.

Diagnosis

Encephalitic symptoms are very contributive for the diagnosis of toxoplasmosis in HIV positive patients. Pneumonitis occurring in less than 1% of AIDS cases may induce septic shock. Tissue biopsy should be very important for an early diagnosis

Cervical or occipital lymphadenopathy are the common clinical feature with flu-like symptoms. Target organs involved are: the brain, the liver, the lung, the muscle, the heart, the eyes, with related symptoms. Pulmonary symptoms may resemble tuberculosis, or severe Pneumocystis jiroveci pneumonia

Reactivation of latent infection is frequent in immunosuppressed patients (AIDS, organ transplantation).

2.3.4. Pulmonary Trypanosomiasis

Trypanosoma cruzi, the etiological agent of trypanosomiasis is frequent, as Chagas disease, in Central and South America. Man is infected through the bite of an insect, inoculating trypomastigotes which multiply within the macrophages. Macrophages then release amastigotes, the invading form of tissues through bloodstream. The heart and the gut are the most involved organs with predominant clinical manifestations (myocarditis, arythmia, achalasia, megacolon). Pulmonary manifestations (pleural effusion, edema), are linked to heart involvement. Tracheomegaly and bronchiectasis have been infrequently encountered (Lemle A. Chagas' disease, Chest 1999; 115, 906). Acute manifestations consist of flu-like syndrome and facial edema.

Serological diagnosis is helpful in chronic forms.

3. Conclusion

The internalization of neglected tropical diseases due to migrations across the world highlights the awareness of healthcare givers. The diagnosis of parasitic tropical pulmonary disease could be evoked in recent travelers or immigrants from endemic zones, presenting with respiratory symptoms with peripheral blood or tissue eosinophilia.

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