Archives of Pulmonology and Respiratory Medicine

ISSN: 2639-362X

Volume 2, Issue 2, 2019, PP: 14-19

Epidemiology, Transmission and Management of Pleuropulmonary Amoebiasis: A Review

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Abstract

With increasing travel and migration, rates of parasitic lung and pleural diseases are increasing in the immune-competent population in developed countries as well as among immune compromised patients. Amoebic pleuropulmonary disease is the most common complication of amoebic liver abscess, occurring in patients with amoebic liver disease and those with amoebic dysentery. The main source of transmission is the chronically infected human. Stools infected with the cyst form of the parasite may contaminate fresh food or water. The most common symptoms amoebic pleuropulmonary diseases include pain, cough, hemoptysis, and dyspnea. The pain may be pleuritic or localized to the right upperquadrant.The diagnosis of pleuropulmonary amoebiasis may be supported by the clinical manifestation. The diagnosis of pleuropulmonary amoebiasis may be supported by the clinical manifestation of the disease. The paper reviews the epidemiology, transmission and management of pleuropulmonary amoebiasis.

Keyword: *Amoebiasis, Entamoeba histolytica, epidemiology, pleuropulmonary.*

Introduction

Amoebiasis is defined by the World Health Organization (WHO) as a parasitic infection with the protozoan *Entamoebahistolytica* regardless of symptomatology [1]. The disease is still considered a major public health problem in developing countries of the world [2]. Amoebiasis is a cosmopolitan disease; it prevails especially in hot countries of the Third World. This is the third cause of parasite- linked death in the world after malaria and schistosomiasis [3]. It is currently estimated that 12% of the world population is affected by this germ among which only 10% are symptomatic. This is due to the fact that there are two strains of pathogenic and non-pathogenic amoebae and the majority of subjects are infested with non-pathogenic form [4]. It is considered as one of the most common parasitic infections worldwide with around 500 million infections per year and a leading cause of parasite related mortality with over 100,000 deaths annually [5].

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 [6]. 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 dyspnea [7]. Pleuropulmonary amoebiasis may also occur following haematogenous spread of organisms to the lungs or lymphatic spread from the liver to the diaphragm. Intra-thoracic complications include rupture of an amoebic liver abscess into the pleural cavity with empyema formation which carries a mortality of 15-35% [7]. Infiltration of the lung parenchyma may result in pneumonia or lung abscess formation. Further more, a hepatobronchial fistula leading to expectoration of 'anchovy sauce-like' purulent sputum can develop. Rarely, a broncho-biliary 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.

Biology of E. Histolytica

Humans are the primary known reservoir for *E. histolytica* [8]. The main source of transmission is the chronically infected human. Stools infected with the cyst form of the parasite may contaminate fresh food or water. The other common source of transmission is oral-anals exual contact [9]. Experimental infections with *E. histolytica* have been produced in some animals such as dogs, cats, rats, monkeys, and other laboratory animals. These animals may also acquire human strains as a result of close contact with humans. Natural *E. histolytica* infections with strains morphologically similar to *E. histolytica* have been found in monkeys [10]. In one study, *E. histolytica* was found microscopically in stained fecal smears from six species of locally available Kenyan non-human primates [11]. There may be some animal reservoirs of *E. histolytica* (dogs, monkeys, and probablypigs), but they represent a very small source of human infection compared with humans themselves [12]. The importance of wildlife (primates) in zoonotic infections was studied by Jackson et al., who used zymodeme analysis to investigate whether *E. histolytica* occurs as a true zoonosis [13]. However, there are no reports of sporadic zoonotic transmission of cases between infected animals and humans, although *E. histolytica* is most commonly associated with animals (cats, dogs, non-human primates, etc.).

Infective cysts may be spread by arthropods such as cockroaches and flies, suggesting that these insects are able to play a rare but important role in transmission [14]. The life cycle of *E. histolytica* is simple. It consists of an infective cyst stage and a multiplying trophozoite stage (figure 1). Humans are infected by ingesting these infective cysts, which travel through the gut lumen to the small intestine (figure 2), where each excysts to form eight daughter trophozoites. The trophozoites are motile forms, which adhere to and invade intestinal epithelial cells which line the gastrointestinal tract. Trophozoites move by extending creeping projections of cytoplasm, called pseudopodia, which pull them along. They also use these projections to surround and engulf food particles. The cytoplasm frequently contains many red blood cells (RBCs) that have been ingested. The trophozoites of *E. histolytica* always have a single nucleus. Trophozoites are easily destroyed in the outside environment, degenerating within minutes. The trophozoite of *E. histolytica* can convert to a precyst form with a nucleus, and this form matures into a tetranucleated cyst as it migrates down and out of the colon. The precyst contains aggregates of ribosomes, called chromatoid bodies, as well as food vacuoles that are extruded as the cell shrinks to become a mature cyst. It is the mature cyst that, when consumed in contaminated food or water, is infectious. In the process of becoming tetranucleated, the nucleus of the cyst divides twice. Chromatoid bodies and glycogen vacuoles cannot be seen at this stage [15,16]. Cyst scanre main alive outside the host for weeks or months, especially under damp conditions [17], but are rapidly destroyed at temperatures under 5°C and over 40°C [14]. Cysts are not invasive, but trophozoites can penetrate the gastrointestinal mucosa [15]. From there, the trophozoites are able to migrate to other organs, causing extra-intestinal infections.

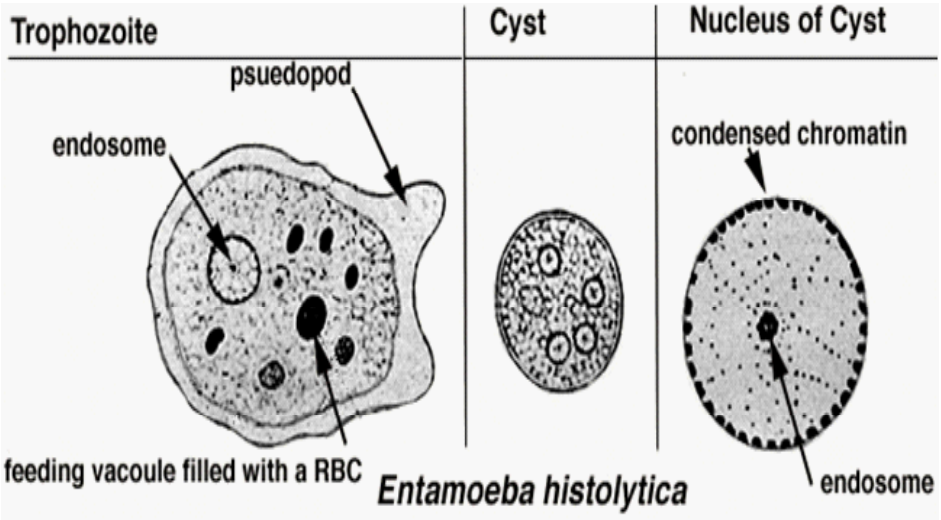

Fig 1. *Tropozoite and cyst of E. histolytica[28]*

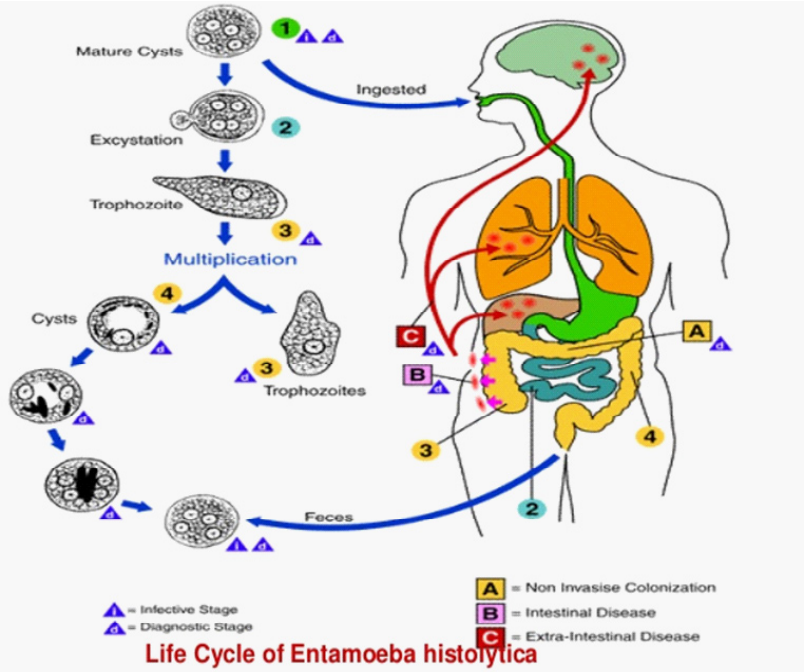

Fig 2. *life cycle of E.histolytica [28]*

Epidemiology

E histolytica is endemic in regions with poor sanitation and poor socioeconomic conditions. The motile trophozoites 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 [18]. This protozoan infection has an especially high prevalence in sub-tropical and tropical countries where poor socioeconomic and sanitary condition spredominate, while in resource-rich nations infections may be seen in travelers to and emigrants from end emicare as [19]. The majority of *Entamoeba* infections are asymptomatic. Factors that influence whether infection leads to asymptomatic or invasive disease include the *E. histolytica*strain and host factors such as genetic susceptibility, age, and immune status, where youngage, pregnancy, corticosteroid treatment, malignancy, malnutrition, and alcohol is mare considered risk factors for severe disease [18]. The 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 [6]. While amoebiasis is a rare occurrence in developed countries of the world, only found in travelers, immigrants, institutionalized persons and homosexuals, the disease is a common occurrence in the less developed and developing countries of the world. These areas include the tropical and sub-tropical countries of South and West Africa, Central and South America, India and Mexico [20].

Pathogenesis

Transmission The life cycle of *E. histolytica* is simple, with only 2 stages, existing as either an infectious cyst or invasive trophozoite. Transmission occurs after the ingestion of the infectious cyst. This most commonly arises from feacally contaminated hands, food, or water, but there is a new appreciation that exposure to fecal matter may occur during sexual contact [21]. Following ingestion, excystation to trophozoites occurs, and the released trophozoites migrate to the large intestine, multiplying by binary fission to produce more cysts. The trophozoite may invade the intestinal epithelium and even pass to extra-intestinal sites such as the liver via hepatic portal circulation or disseminate further to distant sites such as the brain and lungs hematogenously. Symptoms may occur within weeks after ingestion but may also develop years after infection. Cysts and trophozoites are passed in stools. Several properties of the cyst help it to remain hardy in the environment for weeks, whereas trophozoites do not survive. For example, cysts are resistant to gastric acidity and are

also relatively resistant to chlorine. The low infectious dose and environmental stability can predispose to the development of outbreaks [22,23].

Signs and Symptoms

Clinical manifestation of amoebiasis generally occurs in the form of intestinal involvement as acute or sub-acutecolitis, with symptoms range from mild diarrhea to severe dysentery producing abdominal pain, diarrhea, and bloody stools, to fulminant amoebic colitis. It can also present as extra-intestinal disease in the form of amoebic live rabscess and even morer are as pulmonary, cardiac, and brain involvement. Pleuropulmonary complications (i.e., pleural effusion, lung abscess, and, rarely, pleural empyema) are the second most frequent extra-intestinal complication; they occur in 7– 20% of patients with amebic liver abscesses and in 2-3% of those with invasive disease [24]. The presentation of pleuropulmonary amoebiasis is variable and depends on the type of pulmonary involvement whether it is primary simulating broncho pneumonia or tuberculosis or secondary to rupture giving the characteristic suppurative syndrome. The most common symptoms include pain, cough, hemoptysis, and dyspnea. The pain may be pleuritic or localized to the right upper quadrant. Cough can be non-productive but more often is associated with expectoration of material ranging from small amounts of sputum to large amounts of amoebicpus. If a hepato-bronchial fistula develops, the patient may expectorate necrotic material that can include liver abscess contents; such material may have are ddishbrownor "anchovysauce" appearance [25].

Transmission

The theoretical mechanisms of thoracic amoebiasis are as follows; first, the infection usually spreads to the lung by direct rupture of an amoebic liver abscess through the diaphragm. Second, the infection may disseminate to the thorax directly from the primary intestinal lesion through hematogenous orlymphatic spread. And finally, in halation of dust containing cysts of *E. histolytica* is also a hypothetical route [25, 26]. Pleuropulmonary amoebiasisis easily confused with other illnesses which makes the differential diagnosis rather a complexone, involving and not limited to pulmonary Tuberculosis, bacterial lung abscess, carcinoma of the lung, and in endemic are asmalaria and schistosomias is considered common causes of parasitic deaths that can present with unremitting fevers and hepaticor lung disease [25,27].

Diagnosis

The diagnosis of pleuropulmonary amoebiasis may be supported by the clinical manifestation and radiographic imaging such as homogenous opacity or cavitating lesion most commonly involving the right lower and middle lobes, elevated right hemidiaphragm, basilar pulmonary infiltrates with are as of focal at electasis, and pleural effusions. Light microscopic examination can often identify characteristic trophozoites and cysts through direct, concentrated, and/or permanently stained smears. Keeping in mind that the organisms may appear intermittently, specimens from patients with disseminated disease may not contain cysts and trophozoites despite repeated examinations [28]. Immunological tests such as indirect hemagglutination assay (IHA) and enzyme-linked immunosorbent assay (ELISA) for *E. histolytica* antibodies are characterized by high sensitivity. The primary disadvantage of serologic tests is that they cannot distinguish between past and current infection unless IgM is detected; IgM antibodies to E. histolytica are short- lived and rarely detected. In contrast, IgGanti bodies are long-lived but highly prevalent in endemic settings. Newserologic tests based on recombinant *E. histolytica* antigens have been developed; such as says may be especially useful in endemic areas [18,28].

Treatment

In general, amoebic pleural effusions should be aspirated. Drained pleural effusions resolve rapidly with drainage and antimicrobial therapy, which consists of metronidazole (750mg orally three times daily for 7 to 10 days) or alternatively tinidazole (2g once daily for five days) [29]. Most patients respond to a single course of treatment with resolution of symptoms before the end of therapy. In rare cases, a second course is needed because of failure to achieve complete resolution after the initial regimen. Treatment with a luminal agent such as paromomyc in (25–30mg/kg/dayorally in three divided doses for seven days), diiodohydroxyquin (650mg orally three times daily for 20 days), or diloxanidefuroate (500mg orally three times daily for 10 days) to eliminate intraluminal cysts is also warranted. The mortality rate of amoebic pleural empyemaisas high as 16%, which can increase to 42% due to theruptureofahepatic abscess into the pleural space. Empyemarequires chesttube thoracostomy and decortication to prevent recurrence and chronic infection [24, 30, 31]. Surgical

treatment of pulmonary amoebiasis may be required when there is direct pulmonary involvement or spread to pleura.

Conclusion

Inconclusion, pleura pulmonary amoebiasisis the second most frequent extra-in testinal complication that can be easily treated with drainage and antimicrobial therapy. Inhalation of dust containing cysts of *E. histolytica* is a possible route of primary infection. In patients from endemic areas all physicians should keep a high index of suspicion of amoebiasis as a cause of pulmonary disease.

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Citation: **Farouk S. Nas, Muhammad Ali.** *Epidemiology, Transmission and Management of Pleuropulmonary Amoebiasis: A Review. Archives of Pulmonology and Respiratory Medicine. 2019; 2(2): 14-19.*