Hemoptysis in a 49-Year-Old Man*  
An Unusual Presentation of a Sporadic Disease  
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Although amebiasis is prevalent in tropical and subtropical areas, it occurs sporadically in the United States. Pleuropulmonary involvement is seen in about 20 percent of the patients with amebic liver abscesses. We describe a patient with pleuropulmonary amebiasis who complained of hemoptysis but had no gastrointestinal symptoms. This rather unusual presentation caused a considerable delay in securing the diagnosis.  

Herein we report the case of a patient who had pleuropulmonary amebiasis. The 49-year-old man whose case is presented complained of hemoptysis without gastrointestinal symptoms.

Case Report

A 49-year-old man came to LAC-USC Medical Center with a six-week history of blood-streaked sputum. During the preceding three weeks he had lost 22 pounds in weight. He complained of general fatigue, occasional occurrences of fever, chills and night sweats. He had no cough, shortness of breath, abdominal pain or cramping. Three weeks before presentation he experienced diarrhea, which lasted for five days and subsided without any treatment. On this visit to the hospital he had a dull aching in the right lower posterior thorax.

He had no previous operations or hospital admissions. He did not know if any of his friends or family members had had tuberculosis. A gardener by profession, the patient has resided in the United States for eight years with occasional travel to Mexico. He smoked one half pack of cigarettes per day for 20 years. He was an alcoholic but gave up the habit about 15 years prior to this visit to LAC-USC Medical Center. He did not visit prostitutes and denied IVDA and homosexual contact.

On physical examination he was alert, cooperative and did not appear cachetic. His temperature was 38.8°C. The breath sounds were diminished over the lower base of the right lung and a few crackles were present. Results of the rest of the examination were normal.

Blood tests that yielded abnormal results included the following: alkaline phosphatase level, 259 units/L (normal:<110 units/L); a calcium value, 8.0 mg % (normal:8.5-10.5 mg/100 ml); total protein level, 6.2 g % (normal:6.5-9 g/100 ml); albumin value, 2.5 g % (normal:3.5-5.0). The white blood cell count was mildly elevated (15.9/cu mm./µ/100 ml), with a differential cell count of 63 percent segmented neutrophils, 15 percent band cells, 15 percent lymphocytes and 7 percent monocytes. The hematocrit value was 24.7 percent with a MCV of 85.7.

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A bronchoscopy performed six days after admission showed compression and distortion of the bronchus intermedius with blood and copious thick cottage cheese-like material coming from the right middle and lower lobes. Because the patient developed hypoxemia, bronchial biopsy specimens were not obtained. Bronchial lavage and brushing specimens showed no malignant cells, acid-fast bacilli or fungi.

Eleven days after admission a CT of the chest showed multiple abscesses in the right lower lung fields and a right pleural effusion. The diaphragm was not well visualized (Fig 2). The patient still had a fever and hemoptysis. Sixteen days after admission, an ultrasound test was done on the right upper quadrant, which showed a 6.5-cm heterogeneous lesion in the posterior right lobe with extension through the right diaphragm to the right pleural space (Fig 3 and 4). Serum amebic titers were 1:8,192 (normal: less than 1:8). He was given metronidazole, 750 mg by mouth three times a day for ten days, and 1idoquinol, 650 mg daily for 20 days. After two weeks, the patient remained asymptomatic.

Amebiasis, caused by Entamoeba histolytica, is endemic in many areas of the world including south and southeastern Asia, South Africa, Mexico and even some areas of the United States. Occasionally amebiasis is diagnosed in patients in nonendemic areas because of their recent travels. The cyst form, mainly transmitted by contaminated water and food, develops into the trophozoite stage in the intestine. The host may remain asymptomatic but may pass cysts or the disease may invade the bowel causing a dysentery syndrome. Spread of the disease to the liver leads to abscess formation. Although the liver is primarily involved, the lung is the second most commonly involved organ; the brain and spleen are rarely involved.1

Pleuropulmonary complications occur in 7 to 20 percent of patients with amebic liver abscesses and in 2 to 3 percent of those with invasive disease.1,4 Presentation ranges from minimal clinical abnormality to an overwhelming illness with respiratory failure, sepsis and shock.5 The patients often have a chronic illness with weight loss and fatigue. At times there is a history of the presence of symptoms years before the diagnosis is made.6,8 Diarrhea rarely brings the patients with pleuropulmonary involvement to the hospital, although a history of diarrhea may be elicited. Cough is the most common respiratory symptom, but dyspnea and hemoptysis are frequent. Signs of consolidation or effusion may occur along with lower thoracic and right upper quadrant pain. An enlarged, tender liver and respiratory symptoms in a patient from an area where F. histolytica is endemic would be highly suggestive of pleuropulmonary amebiasis. Unfortunately, the abdominal examination often is unhelpful in such patients. Laboratory abnormalities are
nonspecific and include leukocytosis, anemia, an elevated sedimentation rate and abnormal alkaline phosphatase and transaminase values. However, all or many of these tests may be normal in pulmonary amebiasis.

Chest roentgenograms commonly show an elevated and poorly mobile right diaphragm, right lower lobe consolidation, infiltrates or pleural effusion. Cardiomegaly, if present, may indicate pericardial effusion. The Mexican hat sign, an exotic radiographic anomaly, may be present, but is not pathognomonic.9 Lung abscesses usually involve the right lower lobe, but occasionally one or both lungs may be affected.

Ultrasound, the imaging method of choice for an amebic liver abscess, may reveal findings highly suggestive of the diagnosis, particularly in endemic areas.10 These abnormalities are a round- or oval-shaped lesion, poorly echogenic areas in the normally echoic liver parenchyma, continuity with the liver capsule and distal sonic enhancement. Usually there is just a single lesion in the right lobe. A hypoechoic liver lesion that has disrupted the diaphragm is a helpful sign of pleuropulmonary involvement.

Serologic findings significantly contribute to the diagnosis of pleuropulmonary amebiasis. Indirect hemagglutination, because of its better sensitivity and easier performance, is the recommended test.11 Complement fixation and gel diffusion also are sensitive tests. Although, all these tests are nearly always positive in patients with an amebic liver abscess, they are also positive in 60 percent of asymptomatic subjects who pass cysts.11 Thus, a positive amebic serology test alone would not prove liver involvement.

The differential diagnosis of pleuropulmonary amebiasis includes tuberculosis, empyema, cancer and pyogenic lung abscess. Chronic cough, hemoptysis and fever can occur in all of these conditions. The diagnostic process further is confounded by the similar geographic distribution of amebiasis and tuberculosis. The patients with pleuropulmonary amebiasis often are younger than lung cancer patients.

Nearly all patients with pleuropulmonary amebiasis have liver involvement. The abscess, usually in the upper aspect of the liver, can cause a reactive pleural change manifested by an exudative pleural effusion. It occurs in 9 to 34 percent12,13 of patients with pleuropulmonary involvement. The liver abscess also can rupture through the diaphragm and create an empyema, lung abscess, hepatobronchial fistula or any combination of these. Diaphragmatic rupture is common when pleuropulmonary disease is present. Sixteen of 20 patients with pleuropulmonary disease studied by ultrasound testing had diaphragmatic rupture.10 Incidences of diaphragmatic disruption may vary from 21 to 96 percent.2,7,14 Rupture is associated with a mortality varying from 5 to 36 percent.2,7,14

One of the manifestations of diaphragmatic rupture is amebic empyema, with incidence rates of 9 to 62 percent.1,4,13,15 Rarely are cysts identified in the pleural fluid, sputum or stool.8,9,12,16 The empyemas often respond well to conservative treatment which would include a chest tube,4,13,15 but in some series8,12 20 to 40 percent of the patients required invasive procedures such as rib resection and decortication. All pleural effusions should be evaluated by a thoracentesis to differentiate a reactive pleural effusion, which would require only chemotheraphy, from an empyema.

Lung abscesses occur in about one third of all the patients1,2,5 with pleuropulmonary amebiasis, whereas as many as half of the patients may have hepatobronchial fistulas.1,3,12 A patient with hepatobronchial fistulas may empty the liver abscess by expectorating a large amount of dark-colored sputum.

Hematogenous spread of amebiasis to the lung occurs only rarely.6,17 It occurred only twice in 733 patients from four published series.1,7,13,15

Emetine, previously regarded as treatment of choice for pleuropulmonary amebiasis, was extremely effective1 but toxic, and now metronidazole is regarded as such since it is as efficacious but less toxic.13 However, in rare cases of metronidazole failure, emetine is the drug of choice. Emetine causes pain at the injection site, nausea, muscular weakness and myocarditis. The patient receiving emetine should be on absolute bed rest.

**Conclusion**

Our patient is interesting for many reasons. Even though the LAC-USC Medical Center has many cases of amebiasis, in this patient the diagnosis was delayed for over two weeks. The patient was initially treated for a community-acquired bacterial pneumonia and tuberculosis. When he did not respond to adequate antibiotic therapy, bronchoscopy was performed to exclude lung cancer. The lack of abdominal findings and the history of hemoptysis and cigarette use contributed to the misdiagnosis. Hemoptysis is not uncommon in pleuropulmonary amebiasis,9,12,15,16 and in fact, massive hemoptysis has been reported.6,10 This sign also is associated with pulmonary tuberculosis particularly in many patients from areas where tuberculosis is endemic. Elevated alkaline phosphatase levels, a nonspecific marker for amebic liver abscess, are also present in tuberculosis involving the liver.

Other clues present in this case might have led an alert mind to the right diagnosis. During bronchoscopy, a cottage cheese-like material was observed. The patient was noted to have one to two cups of sputum for the first two to three days which resolved spontaneously. In retrospect, we can ascribe this to the spontaneous drainage of the abscess through the hepatobiliary fistula. The inability of the CT scan
clearly to define the liver abscess was unfortunate but not unusual. In a series of 23 patients with amebic liver abscesses who had CT scans nine had pleural effusions, and in none of these patients could the integrity of the diaphragm be established, which was true in our case.

Although a tissue diagnosis of amebiasis was not made, this patient's constellation of findings establishes the diagnosis. His travel to an endemic area, documentation of a single liver lesion with extension through the diaphragm, a clinical course compatible with a hepatobronchial fistula, high amebic titers and a response to metronidazole incontrovertibly point to pleuropulmonary amebiasis.

REFERENCES