Appearances can be deceiving: What is the diagnosis for this community-acquired pneumonia?

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A 65-year-old male with a past history of cigarette smoking and asbestos exposure (in the 1970s) presented to the emergency department with a one-month history of progressive dyspnea, right-sided pleuritic chest pain, cough productive of white-coloured sputum and malaise. His health problems had commenced four months before presentation while he was vacationing at a northern Ontario resort. At that time, he had felt unwell and had developed a fever with right-sided pleuritic chest pain that radiated to his right shoulder. The diagnosis was an upper respiratory tract infection, made by the local physician; the patient was treated with a 10-day course of cephalexin. Although his condition had initially improved after the antibiotic therapy, during the month before presentation he had experienced increasing fatigue, cough with clear sputum production and a loss of appetite. He also developed worsening right-sided pleuritic chest pain that radiated to the right shoulder, dyspnea and orthopnea. He had no nausea, vomiting, diarrhea or hemoptysis. However, he had lost 4 kg and had drenching night sweats over the previous three and a half months. Further history revealed that he had drunk well water during his vacation in northern Ontario and that several families who were with him at that time also became ill, although he was not aware of the nature of their symptoms.

On examination, he was a thin male in some respiratory distress and was afebrile. His blood pressure was 140/90 mmHg, and he had a heart rate of 80 beats/min and a respiratory rate of 30 breaths/min. Chest examination revealed percussion dullness, decreased breath sounds, crackles, and bronchial breathing in the right base and mid-lung fields. No clubbing was observed. There was no jugular venous distention, and heart sounds were normal without any murmurs. His abdomen was flat, but he had mild right upper quadrant tenderness on deep palpation. No abdominal masses were palpable, no organomegaly was appreciated and there were no stigmata of chronic liver disease. Bowel sounds were present.

The patient's oxygen saturation was 89% on room air. The white blood cell count revealed mild leukocytosis of $12.2 \times 10^9/L$ without a left shift, but hemoglobin and platelet counts were normal. The erythrocyte sedimentation rate was 38 mm/h. The level of serum aspartate aminotransferase was 28 U/L, while the alanine aminotransferase level was mildly elevated at 47 U/L; the alkaline phosphatase and gamma-glutamyl transpeptidase levels were moderately elevated at 380 U/L and 243 U/L, respectively. The patient's total bilirubin concentration was normal, but the albumin concentration was depressed at 32 g/L. All other serum chemistry values were within normal limits.

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Figure 1) *Chest radiograph demonstrating right middle and lower lobe consolidations with effusion*

A chest x-ray (Figure 1) showed a large right pleural effusion with associated consolidation or subsegmental atelectasis in the right middle and lower lung zones. An underlying parenchymal mass could not be excluded. Computed tomography of the thorax with contrast enhancement (Figure 2) revealed no masses in the lung parenchyma or the mediastinum. However, it demonstrated a 10 cm fluid collection occupying most of the right lobe of the liver, with peripheral enhancement consistent with a massive liver abscess.

Figure 2) *Computed tomography scan of the abdomen showing massive liver abscess in the right lobe of the liver*

Percutaneous computed tomography-guided drainage of the liver abscess ensued, and 200 mL of bloody purulent fluid was aspirated. In addition, a chest tube was inserted, and 900 mL of fluid of similar consistency with numerous leukocytes was removed from the thorax. Microscopic examination of the fluids did not reveal any microorganisms, and cultures of the fluids and blood were negative for bacteria. Thereafter, therapy consisting of intravenous clindamycin 600 mg every 8 h and oral ciprofloxacin 750 mg bid was initiated.

What is your diagnosis?

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DIAGNOSIS

Entamoeba histolytica serology revealed a titre of 1:12,800 by immunoglobulin G-specific ELISA. However, examination of the stool did not reveal any eggs or parasites. On receipt of the serology report, the patient's treatment was changed to oral metronidazole 750 mg tid for 10 days. A convalescent titre for *E histolytica* was performed one month after the initial test and showed a fourfold rise in titre. Follow-up ultrasonography of the liver demonstrated gradual resolution of the abscess over four months.

DISCUSSION

Our patient presented with symptoms consistent with community-acquired pneumonia with concomitant pleural involvement. His clinical picture was actually precipitated by an amoebic liver abscess with extension to the right pleural cavity and lung. This case illustrates how pneumonia can be caused not only by microaspiration of organisms, inhalation of airborne organisms or seeding of the lung by blood-borne pathogens, but also by contiguous spread of a pathological process across the diaphragm.

Amebiasis caused by *E histolytica* is endemic in tropical and subtropical countries. Cases of amebiasis seen in North America are typically caused by fecal contamination of the water supply by foreign-born individuals arriving from endemic areas. It is presumed that the patient acquired his disease in this manner via the well water in northern Ontario; this was not proven, however, because it is not known whether any subsequent tests were performed on this water source. Recently, it has been determined by genetic analysis that there are distinct species of *Entamoeba* that are morphologically identical (1). *Entamoeba dispar*, a more prevalent species, is associated with an asymptomatic carrier state. In contrast, *E histolytica*, the pathogenic species, is associated with symptomatic infection. The cyst form of *E histolytica* exists in contaminated water and food, and once indigested, it results in excystation in the small bowel and trophozoite infection of the colon. This produces symptomatic dysentery, the most common manifestation of amebiasis.

Liver involvement accounts for about 10% to 40% of symptomatic patients (2-4). Infection ascends from the colon, through the portal system and to the liver. Typically, patients with amoebic liver abscesses have an acute course with symptoms for fewer than 10 days. However, at times, a subacute course, with symptoms lasting many months, may prevail (4). Fewer than 30% of patients have active diarrhea at some time before presentation (1). The most common presenting symptom is pain, which may emanate from the right upper quadrant of the abdomen, the epigastrium, the right lower chest or the right shoulder (4). The pain may be dull, stabbing or pleuritic in nature. Fewer than 50% of the patients have an enlarged, painful liver. Long standing cases show weight loss, anemia, cachexia and low grade fevers.

Amoebic liver abscesses may involve the pleura, lung, pericardium or peritoneum. Pleuropulmonary involvement, particularly of the right lung, occurs in about 50% of cases (5). Pulmonary complaints include pleuritic chest pain, cough and dyspnea. The most common findings on radiographic examination of the chest are right-sided pleural effusions and elevations in the right hemidiaphragm.

The diagnosis of intestinal amebiasis is based on examination of the stool or a biopsy of mucosal tissue. Invasive disease is confirmed by finding hematophagous trophozoites of *E histolytica* in the stool. Microscopic examination of a wet mount from a hepatic aspirate will reveal the diagnosis in only 30% of cases. Serology is the best way to confirm the diagnosis of invasive amebiasis. An immunoglobulin G titre of 1:100 to *E histolytica*-specific antigen by ELISA is considered positive. A titre greater than 1:800 has a sensitivity and specificity of greater than 95%, especially in nonendemic areas (6).

The differential diagnosis of amoebic liver abscess includes pyogenic abscess, echinococcal cyst and hepatoma. If the diagnosis is uncertain, a fine needle aspiration under ultrasound or computed tomography guidance can be performed to rule out pyogenic disease.

The treatment of amoebic liver abscesses involves a five- to 10-day course of oral metronidazole 750 mg bid. The cure rate is greater than 95% (6), and the mortality rate associated with uncomplicated amoebic liver abscess is less than 1%. Although aspiration of the abscess is rarely needed because there is no adequate evidence that evacuation of large lesions leads to more rapid healing, it should be entertained in the following situations: to rule out a pyogenic abscess, particularly with multiple lesions; as an adjunct to medical therapy if it appears that the patient is a therapeutic failure within three to five days; and if rupture of the abscess is believed to be imminent (7,8).

Our patient was an interesting case of 'community-acquired pneumonia' as a result of pleuropulmonary involvement from an amoebic liver abscess. A high index of suspicion is necessary to make this diagnosis in nonendemic areas in combination with epidemiological risk factors, radiological findings, microbiology and serology.

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