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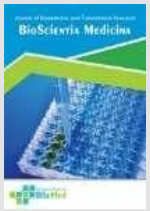

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Pleural Amebiasis Mimicking Pleural Effusion: A Case Report

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A B S T R A C T

Background: Pleural amebiasis is a rare manifestation of extraintestinal amebiasis, primarily occurring due to the rupture of an amebic liver abscess into the pleural space. We present an unusual case of pleural amebiasis in an elderly woman without any evidence of liver involvement. **Case presentation:** A 78-year-old female presented with a two-month history of progressive dyspnea, cough, and right-sided chest pain. She had a history of consuming raw vegetables. Examination revealed right-sided pleural effusion, which was confirmed by chest radiography and thoracic ultrasound. Pleural fluid analysis showed exudative effusion and microscopic examination demonstrated the presence of Entamoeba histolytica trophozoites. Despite the absence of a liver abscess on abdominal ultrasound, the patient was diagnosed with pleural amebiasis based on the pleural fluid findings. She was successfully treated with metronidazole and drainage of the pleural fluid. **Conclusion:** This case highlights the importance of considering pleural amebiasis as a differential diagnosis in patients with pleural effusion, even in the absence of liver involvement. Early diagnosis and prompt treatment with metronidazole and drainage are crucial for a favorable outcome.

1. Introduction

Amebiasis, a widespread parasitic infection primarily affecting the human gastrointestinal tract, is caused by the protozoan *Entamoeba histolytica*. This ubiquitous parasite is responsible for significant morbidity and mortality worldwide, particularly in regions with poor sanitation and limited access to clean water. The World Health Organization (WHO) estimates that approximately 50 million people are infected with *E. histolytica* globally, leading to around 100,000 deaths annually. The clinical manifestations of amebiasis range from asymptomatic colonization to severe invasive disease. The majority of infected individuals remain asymptomatic, harboring the parasite without experiencing any noticeable symptoms. However, in a subset of individuals, *E. histolytica* can invade the intestinal mucosa, leading to amebic colitis, characterized by abdominal pain, bloody diarrhea, and, in severe cases, intestinal perforation.[1-3]

More rarely, *E. histolytica* can spread beyond the confines of the intestinal tract, causing extraintestinal amebiasis. The liver is the most common site of extraintestinal involvement, resulting in amebic liver abscess, a potentially life-threatening condition. Other less frequent sites of extraintestinal amebiasis include the lungs, pleura, brain, and skin. Pleural amebiasis is a rare but serious complication of amebiasis, accounting for approximately 1-3% of all cases. It typically arises from the direct extension of an amebic liver abscess into the pleural space, leading to pleural effusion, empyema, or bronchopleural fistula. Hematogenous spread of *E. histolytica* to the pleura without liver involvement is exceedingly rare. The

diagnosis of pleural amebiasis can be challenging, as the clinical and radiological findings are often nonspecific, mimicking other causes of pleural effusion. A high index of suspicion is required, especially in patients from endemic areas or those with a history of amebiasis. The diagnosis is confirmed by the demonstration of *E. histolytica* trophozoites in the pleural fluid, either by microscopic examination or culture.[4-7]

Treatment of pleural amebiasis involves a combination of medical therapy and drainage of the pleural fluid. Metronidazole is the drug of choice, effectively eradicating the parasite. Drainage of the pleural fluid can be achieved by thoracentesis or chest tube insertion, relieving symptoms and preventing complications.[8-10] In this case report, we present an unusual case of pleural amebiasis in an elderly woman who presented with pleural effusion without any evidence of liver involvement. This case highlights the diagnostic challenges and the importance of considering this rare entity in the differential diagnosis of pleural effusion.

2. Case Presentation

A 78-year-old female presented to the pulmonary clinic of Dr. M. Djamil General Hospital with a two-month history of progressive dyspnea. The dyspnea was not accompanied by chest tightness but worsened with exertion and coughing. The patient had a non-productive cough that had persisted for two months. The cough was intermittent and characterized by difficulty expectorating sputum. Additionally, she complained of intermittent right-sided chest pain for the past month. The pain was not constant and varied in intensity. The patient had been treated at M. Natsir Hospital three days prior to her presentation at Dr. M. Djamil General Hospital. During her stay at M. Natsir Hospital, she underwent thoracentesis, which drained 800 cc of reddish-brown pleural fluid. However, her symptoms did not improve significantly after the procedure. A sputum examination conducted at M. Natsir Hospital was negative for Mycobacterium tuberculosis (MTB).

The patient reported consuming raw vegetables without washing them regularly. She denied any fever, hemoptysis, weight loss, or gastrointestinal symptoms, such as abdominal pain, changes in bowel habits, nausea, or vomiting. She was a non-smoker and lived alone in a house with adequate ventilation and lighting. She had a history of cooking with a stove for 30 years, with exposure of fewer than 6 hours per day. On physical examination, the patient appeared moderately ill but was alert and oriented. Her vital signs were as follows: blood pressure 125/70 mmHg, pulse 88 beats per minute, respiratory rate 20 breaths per minute, temperature 36.7°C, and oxygen saturation (SpO_2) 99% on room air. She had a body mass index (BMI) of 15.2 kg/m², indicating underweight status. Examination of her head and neck revealed no conjunctival pallor or scleral icterus. There was no jugular venous distension, and no enlarged lymph nodes were palpable. Respiratory examination revealed an asymmetrical chest wall, with the right side appearing more convex than the left. The movement of the right chest wall was reduced compared to the left. Tactile fremitus was decreased on the right side. Percussion of the right lung field was sonor above the sixth rib and dull below the sixth rib. Auscultation revealed bronchovesicular breath sounds over the right upper lung field, with absent breath sounds below the sixth rib. There were no rhonchi or wheezes. Examination of the left lung field was unremarkable. Abdominal examination revealed no hepatomegaly or splenomegaly. There was no tenderness or guarding. Examination of the extremities revealed no edema.

Laboratory investigations revealed mild eosinophilia (7%) and normal liver function tests. The complete blood count showed a white blood cell count of 8,920/mm³, with a differential count of neutrophils 63%, lymphocytes 19%, monocytes 10%, and eosinophils 7%. The hemoglobin level was 12.9 g/dL, and the platelet count was 345,000/mm³. The serum albumin level was 3.9 g/dL, and the globulin level was 3.9 g/dL. The total bilirubin level was 0.2 mg/dL, with a direct bilirubin of 0.1 mg/dL and an indirect

bilirubin of 0.1 mg/dL. The aspartate aminotransferase (AST) level was 32 U/L, and the alanine aminotransferase (ALT) level was 26 U/L. The blood urea nitrogen (BUN) level was 32 mg/dL, and the creatinine level was 0.7 mg/dL. The serum sodium level was 143 mmol/L, the potassium level was 4.4 mmol/L, and the chloride level was 108 mmol/L. The random blood glucose level was 93 mg/dL. A chest radiograph taken at M. Natsir Hospital showed right-sided pleural effusion (Figure 1A). A repeat chest radiograph taken at Dr. M. Djamil General Hospital revealed an increase in the pleural effusion (Figure 1B). Thoracic ultrasound confirmed the presence of pleural effusion (Figure 2A).

Thoracentesis was performed twice, draining a total of 1900 cc of pleural fluid. The pleural fluid was reddish-brown in color (Figure 2B). Pleural fluid analysis revealed an exudative effusion with a total cell count of 5,200/mm^3, with a differential count of polymorphonuclear leukocytes (PMNs) 25% and mononuclear cells (MN) 75%. The pleural fluid protein level was 7.1 g/dL, lactate dehydrogenase (LDH) 1481 U/L, glucose 20.8 g/dL, and albumin 3.1 g/dL. The Rivalta test was positive, indicating an exudative effusion. Cytology of the pleural fluid showed a lymphocytic effusion. The patient was referred to a gastroenterologist to investigate the possibility of a liver abscess. An abdominal ultrasound was performed, which was unremarkable (Figure 3). Stool examination was also negative for *E. histolytica*. Based on the pleural fluid findings, a diagnosis of pleural amebiasis was made. The presence of *E. histolytica* trophozoites in the pleural fluid, despite the absence of liver involvement, confirmed the diagnosis. The patient was treated with intravenous fluids, metronidazole 750 mg three times daily, and paracetamol 500 mg four times daily. A pigtail catheter was inserted to drain the pleural fluid. The patient's condition improved significantly after five days of treatment, and she was discharged home. Follow-up thoracic ultrasound showed minimal loculated pleural effusion. The pigtail catheter was removed on the 15th day of treatment. The patient remained asymptomatic at her follow-up visits.

Table 1. Timeline of disease.

Timeline	**Description**
2 months prior to admission	Onset of progressive dyspnea and non-productive cough.
1 month prior to admission	Intermittent right-sided chest pain develops.
3 days prior to admission	Admitted to M. Natsir Hospital for 3 days.
During hospitalization at M. Natsir Hospital	800 cc of reddish-brown pleural fluid drained via thoracentesis.
At a presentation to Dr. M. Djamil General Hospital	Dyspnea, cough, and chest pain continue. 2.5 kg weight loss reported.
During hospitalization at Dr. M. Djamil General Hospital	1900 cc of pleural fluid drained via thoracentesis.
Day 1 of hospitalization	A pigtail catheter was inserted for continuous drainage.
Day 5 of hospitalization	Clinical improvement noted. Discharged home with oral metronidazole.
Follow-up visit	Symptoms resolved. Pleural fluid drainage < 100 cc/day.
Day 15 of treatment	The pigtail catheter was removed.

Table 2. Detailing the diagnostic approach for pleural amebiasis.

Diagnostic modality	Findings	Rationale
History and Physical Examination	Progressive dyspnea, cough, right-sided chest pain. Weight loss. No fever or gastrointestinal symptoms. Decreased breath sounds, and dullness to percussion.	Suggestive of pleural effusion. Atypical presentation for pleural amebiasis (lack of fever and gastrointestinal symptoms).
Complete blood count	Mild eosinophilia (7%). Leukocyte count: 8,920/mm³ (within normal limits).	Eosinophilia can be seen in parasitic infections, but not always in pleural amebiasis. Leukocytosis may be present in some cases.
Chest radiography	Right-sided pleural effusion.	Confirms the presence of pleural effusion. Non-specific for pleural amebiasis.
Thoracic ultrasound	Right-sided pleural effusion. No evidence of liver abscess.	Confirms pleural effusion and guides thoracentesis. Helps rule out hepatic amebiasis.
Pleural fluid analysis	Exudative effusion. Lymphocytic predominance. Elevated LDH (1481 U/L). Low glucose (20.8 mg/dL). Presence of *E. histolytica* trophozoites.	Exudative effusion is typical in pleural amebiasis. Lymphocytic predominance suggests a chronic process. Elevated LDH and low glucose are consistent with an infective process. Identification of *E. histolytica* trophozoites confirms the diagnosis.
Abdominal ultrasound	Normal abdominal organs, no liver abscess.	Rules out hepatic amebiasis as the source of pleural involvement.
Stool microscopy	Negative for parasites.	Can be negative in cases of extraintestinal amebiasis.

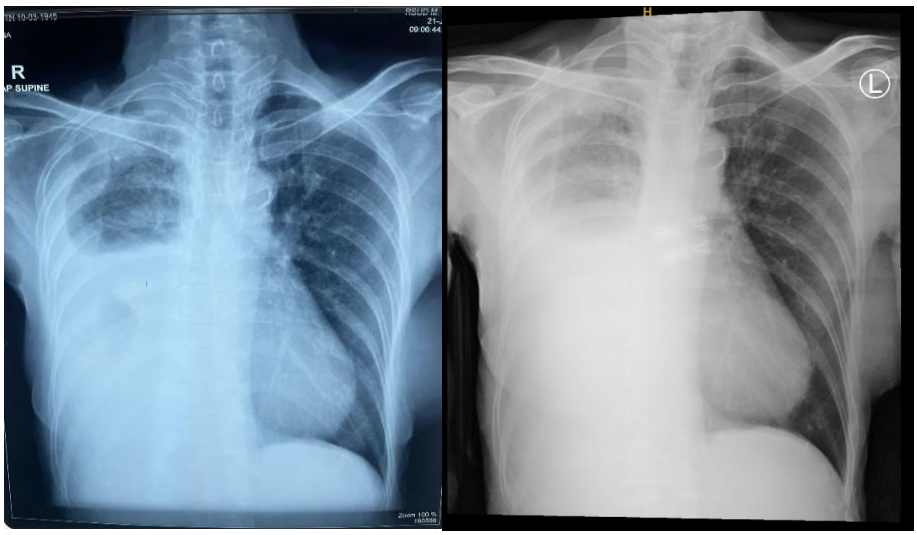

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Figure 1. A. Chest photo at M. Natsir Hospital taken July 21st, 2024. B. Chest photo at Dr. M. Djamil General Hospital taken August 1st, 2024.

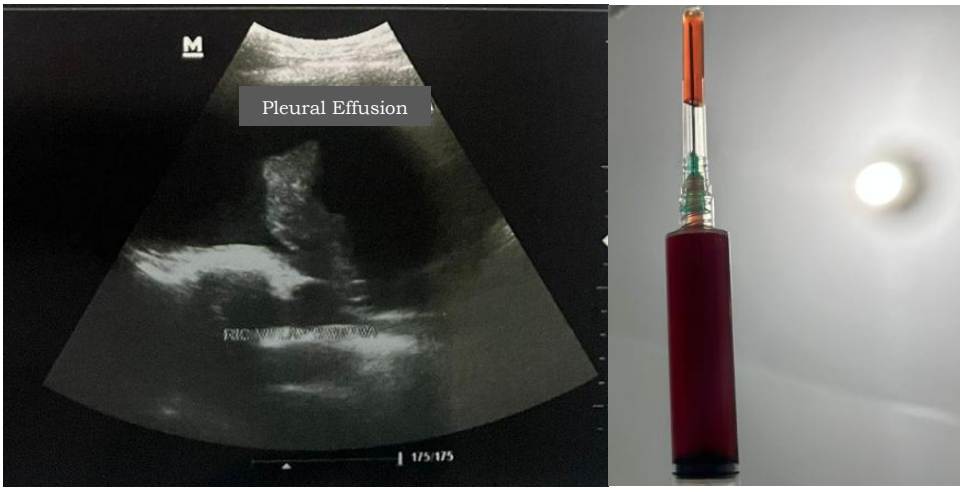

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Figure 2. A. Thoracic ultrasonography with the impression of pleural effusion. B. Proof of pleural fluid with a brownish-red color.

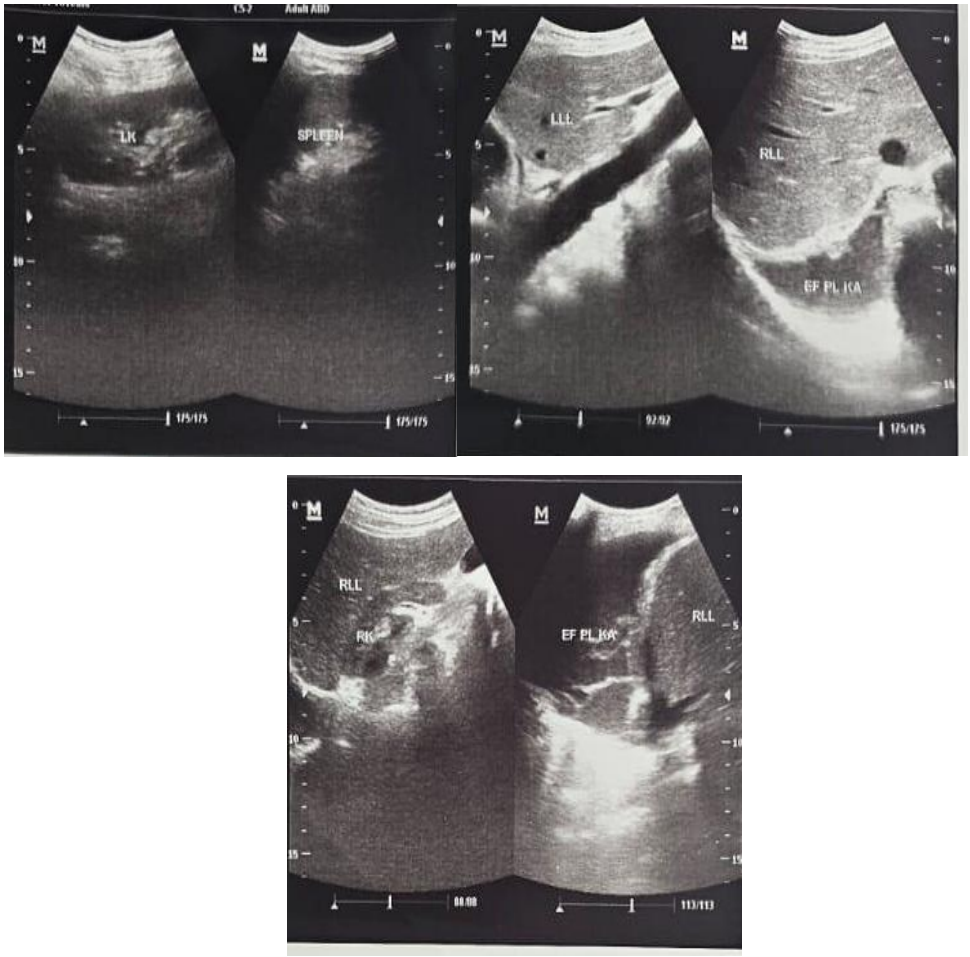

Figure 3. Abdominal ultrasound with impressions of abdominal organs within normal limits.

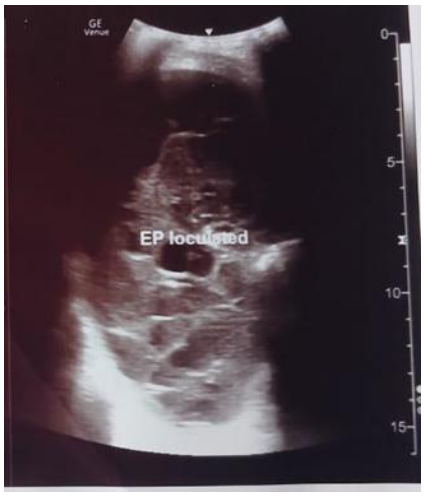

Figure 4. Thoracic ultrasound evaluation on day 15 at the outpatient clinic.

3. Discussion

E. histolytica trophozoites primarily inhabit the large intestine, where they can cause amebic colitis. In some cases, these trophozoites can breach the intestinal barrier and gain access to the bloodstream. *E. histolytica* trophozoites possess remarkable cytolytic capabilities, enabling them to disrupt the intestinal epithelium and invade deeper tissues. The parasite utilizes a combination of virulence factors. Gal/GalNAc lectin mediates adherence to host cells, facilitating colonization and invasion. Cysteine proteases enzymes degrade extracellular matrix components, allowing the parasite to penetrate tissues. Amoebapores pore-forming proteins induce cell lysis and contribute to tissue damage. Once the trophozoites breach the intestinal barrier, they can enter the mesenteric venules and subsequently gain access to the portal circulation. Once in the bloodstream, the trophozoites can disseminate to various organs, including the liver, lungs, brain, and, less commonly, the pleura. The liver is the most common site of extraintestinal involvement due to its direct connection to the portal circulation. However, in some cases, the trophozoites can bypass the liver and reach other organs through the systemic circulation. This hematogenous spread is likely the mechanism responsible for pleural amebiasis in our patient, given the absence of a detectable liver abscess. Individuals with compromised immune systems, such as those with HIV infection, malnutrition, or chronic alcoholism, are more susceptible to invasive amebiasis and hematogenous spread. Malnutrition can impair immune function and compromise the integrity of the intestinal barrier, increasing the risk of invasion and dissemination. Chronic alcohol consumption can damage the liver, impairing its ability to filter out pathogens from the bloodstream. In our patient, her advanced age and underweight status may have contributed to a degree of immunosuppression, increasing her susceptibility to hematogenous spread. Once the trophozoites reach the pleura, they can induce an inflammatory response, leading to the accumulation of pleural fluid. The pleural fluid may be serous, serosanguineous, or purulent, depending on the severity of the infection. In some cases, the trophozoites can invade the pleural tissue, causing pleural thickening and fibrosis. Hematogenous spread of *E. histolytica* can be difficult to diagnose, as it may not always be associated with a detectable liver abscess. A high index of suspicion is required, especially in patients from endemic areas or those with risk factors for amebiasis. Thorough clinical evaluation, including a detailed travel history and dietary habits, is essential. Imaging studies, such as

abdominal ultrasound and chest radiography, may help to rule out other causes of pleural effusion. Pleural fluid analysis is crucial for the diagnosis of pleural amebiasis, as it allows for the direct detection of *E. histolytica* trophozoites. Treatment of pleural amebiasis due to hematogenous spread is similar to that of amebiasis with liver involvement. Metronidazole is the drug of choice, and it should be administered for a sufficient duration to eradicate the parasite. Drainage of the pleural fluid is essential to relieve symptoms and prevent complications. Close follow-up is necessary to monitor for recurrence or complications. Amebic liver abscess (ALA) is the most common extraintestinal manifestation of amebiasis, typically occurring when *E. histolytica* trophozoites reach the liver via the portal circulation. *E. histolytica* trophozoites, after breaching the intestinal barrier and entering the portal circulation, are carried to the liver. The liver, acting as a filter for the portal blood, becomes a prime target for *E. histolytica* invasion. The trophozoites utilize their cytolytic capabilities to destroy liver cells, leading to the formation of an abscess. The abscess typically contains necrotic liver tissue, inflammatory cells, and *E. histolytica* trophozoites. In some cases, an ALA can rupture into adjacent structures, such as the pleural space, peritoneum, pericardium, or bronchi. Rupture into the pleural space is the most common complication, leading to pleural amebiasis. The rupture can occur spontaneously or be precipitated by trauma or certain medical procedures. It is conceivable that our patient had a small, transient liver abscess that ruptured into the pleural space, leading to pleural amebiasis. This hypothetical abscess may have subsequently resolved spontaneously or become undetectable by abdominal ultrasound, explaining the lack of radiological evidence. However, this scenario is less probable given the typically indolent nature of amebic liver abscesses, which tend to persist and require treatment. While rare, spontaneous resolution of ALA has been reported in some cases. The resolution may be facilitated by the host's immune response, which can contain and eliminate the infection. Factors that may favor spontaneous resolution include small abscess size, early stage of infection, and good overall health of the patient. Abdominal ultrasound is a valuable tool for diagnosing ALA, but it has limitations. Small abscesses or those located in certain areas of the liver may be difficult to detect by ultrasound. The sensitivity of ultrasound can also be affected by factors such as operator experience and patient body habitus. The possibility of a transient liver abscess should be considered in patients with pleural amebiasis, even in the absence of radiological evidence. Other diagnostic modalities, such as computed tomography (CT) or magnetic resonance imaging (MRI), may be helpful in detecting small or subtle liver abscesses. Serological tests for amebiasis can also be useful in supporting the diagnosis, especially in cases where imaging is inconclusive. If a transient liver abscess is suspected, treatment with metronidazole is recommended to eradicate any residual infection. Drainage of the pleural fluid is essential to manage pleural amebiasis and prevent complications. Close follow-up is necessary to monitor for recurrence or complications. Although less common than hepatic or pleural involvement, primary pulmonary amebiasis can occur through the inhalation of *E. histolytica* cysts. *E. histolytica* cysts are the infective form of the parasite, responsible for transmission through the fecal-oral route. These cysts are highly resistant to environmental conditions and can survive outside the host for extended periods. Inhalation of aerosolized cysts can occur in settings of poor sanitation or close contact with infected individuals. The cysts can also be inhaled through aspiration of contaminated material, such as vomitus or fecal matter. Once the cysts reach the alveoli of the lungs, they can excyst, releasing trophozoites. Excystation is triggered by the favorable conditions in the lungs, such as the warm temperature and the presence of nutrients. The trophozoites can then invade the lung parenchyma, leading to pulmonary amebiasis. The trophozoites utilize their cytolytic capabilities to destroy lung tissue, leading to the formation of abscesses and cavities. The infection can spread to the pleura,

causing pleural effusion and pleural amebiasis. In severe cases, the infection can disseminate to other organs through the bloodstream. The clinical presentation of primary pulmonary amebiasis can vary depending on the extent of the infection. Common symptoms include cough, chest pain, dyspnea, and fever. Hemoptysis may occur if the infection erodes into blood vessels. In severe cases, respiratory failure and sepsis can develop. Primary pulmonary amebiasis can be challenging to diagnose, as it may mimic other respiratory infections, such as bacterial pneumonia or tuberculosis. A high index of suspicion is required, especially in patients from endemic areas or those with a history of amebiasis. Chest radiography may reveal pulmonary infiltrates, abscesses, or pleural effusion. Computed tomography (CT) of the chest can provide more detailed imaging of the lungs and pleura. Sputum examination and bronchoscopy may be performed to obtain samples for microscopic examination and culture. Serological tests for amebiasis can also be helpful in supporting the diagnosis. Treatment of primary pulmonary amebiasis typically involves metronidazole, the drug of choice for amebiasis. Drainage of pleural fluid or abscesses may be necessary in some cases. Supportive care, such as oxygen therapy and mechanical ventilation, may be required in severe cases. Although our patient did not exhibit any respiratory symptoms suggestive of primary pulmonary amebiasis, such as productive cough, hemoptysis, or pulmonary infiltrates on chest radiography, this possibility cannot be entirely ruled out. It is conceivable that she had a mild or subclinical pulmonary infection that extended to the pleura, leading to pleural amebiasis.[11-13]

Diagnosing pleural amebiasis can be challenging due to its non-specific clinical presentation, often mimicking other causes of pleural effusion such as bacterial pneumonia, tuberculosis, or malignancy. Our patient presented with common symptoms of pleural effusion dyspnea, cough, and chest pain. The absence of fever, weight loss, or gastrointestinal symptoms further complicated the diagnostic picture. The symptoms of pleural amebiasis are often non-specific and can mimic other respiratory conditions, making it difficult to distinguish based on clinical presentation alone. Pleural amebiasis can manifest with a wide range of respiratory symptoms. Dyspnea (shortness of breath) is often the most prominent symptom and can vary in severity from mild exertion-induced breathlessness to severe resting dyspnea. The cough may be dry or productive, with sputum production ranging from scant to copious. The sputum may be mucoid, purulent, or even blood-tinged. Chest pain is often pleuritic in nature, meaning it is sharp and worsens with deep breathing or coughing. The pain may be localized to the area of pleural involvement or may radiate to the shoulder or back. Fever may or may not be present. When present, it can range from low-grade to high-spiking. Less common symptoms may include fatigue, malaise, weight loss, and anorexia. The non-specific nature of these symptoms makes it challenging to differentiate pleural amebiasis from other respiratory conditions based on clinical presentation alone. Bacterial pneumonia often presents with fever, cough, chest pain, and dyspnea. However, it may also be associated with other symptoms such as chills, rigors, and sputum production. Tuberculosis can present with a chronic cough, hemoptysis, weight loss, and night sweats. However, it may also present with non-specific symptoms such as fever, fatigue, and anorexia. Lung cancer or pleural malignancies can cause pleural effusion and present with dyspnea, cough, and chest pain. However, they may also be associated with other symptoms such as hemoptysis, weight loss, and hoarseness. Pulmonary embolism can cause sudden onset dyspnea, chest pain, and cough. However, it may also be associated with other symptoms such as tachycardia, tachypnea, and hemoptysis. Heart failure can cause dyspnea, cough, and orthopnea. However, it may also be associated with other symptoms such as peripheral edema, jugular venous distension, and hepatomegaly. Our patient presented with dyspnea, cough, and chest pain, which are common symptoms of pleural effusion regardless of the underlying cause. The absence of fever, weight loss, or gastrointestinal

symptoms further complicated the diagnostic picture, as these symptoms are often associated with more severe or systemic infections. This non-specific presentation highlights the importance of maintaining a broad differential diagnosis and considering pleural amebiasis as a possibility, even in patients without typical risk factors or symptoms. A thorough clinical evaluation, including a detailed medical history, physical examination, and review of systems, is essential in evaluating patients with suspected pleural amebiasis. Particular attention should be paid to risk factors for amebiasis, such as travel to endemic areas, consumption of potentially contaminated food or water, and history of amebic colitis. The presence of other symptoms, such as fever, weight loss, or gastrointestinal complaints, should also be carefully assessed. Chest radiography may reveal pleural effusion, but it cannot differentiate amebic effusion from other causes. Other radiological findings, such as atelectasis or consolidation, may also be present, further confounding the diagnosis. In our patient, the chest radiograph showed right-sided pleural effusion, which is a non-specific finding. Chest radiography is often the initial imaging modality used to evaluate patients with suspected pleural effusion. While it can confirm the presence of pleural fluid, it cannot distinguish between different causes of effusion. The appearance of pleural effusion on chest radiography is similar regardless of the underlying etiology, whether it is due to infection, malignancy, or other causes. In our patient, the chest radiograph showed right-sided pleural effusion, which is a non-specific finding. In addition to pleural effusion, other radiological findings may be present on chest radiography, further complicating the diagnostic picture. These findings may include atelectasis (collapse of lung tissue), consolidation (filling of air spaces with fluid or inflammatory cells), elevated hemidiaphragm, pleural thickening. These findings can be seen in various respiratory conditions, including pleural amebiasis, bacterial pneumonia, tuberculosis, and malignancy. In cases where the chest radiograph is inconclusive or suggests other potential diagnoses, additional imaging studies may be necessary. CT scan can provide more detailed images of the lungs and pleura, helping to identify subtle abnormalities or complications. Ultrasound can be used to confirm the presence of pleural fluid and guide thoracentesis. It can also help to assess the nature of the effusion and identify any loculations or masses. MRI can provide detailed images of the chest and may be helpful in evaluating complex cases or those with suspected involvement of surrounding structures. In our patient, the initial chest radiograph showed right-sided pleural effusion, which is a non-specific finding. A repeat chest radiograph showed an increase in the pleural effusion, prompting further investigation. Thoracic ultrasound confirmed the presence of pleural effusion and guided thoracentesis. Abdominal ultrasound was also performed to rule out a liver abscess, which is a common cause of pleural amebiasis. However, the abdominal ultrasound was unremarkable. Radiological findings should always be interpreted in conjunction with clinical and laboratory findings to arrive at an accurate diagnosis. In our patient, the non-specific radiological findings of pleural effusion were correlated with her clinical presentation and the results of pleural fluid analysis to diagnose pleural amebiasis. It is important to recognize that radiological findings alone are not sufficient to diagnose pleural amebiasis. Other conditions can mimic the radiological appearance of pleural amebiasis, highlighting the need for a comprehensive evaluation. Radiological imaging can also play a role in guiding the management of pleural amebiasis. For example, imaging can help to determine the optimal site for thoracentesis or chest tube insertion. It can also help to monitor the response to treatment and identify any complications. Pleural amebiasis can mimic other conditions that cause pleural effusion, such as bacterial pneumonia, tuberculosis, malignancy, pulmonary embolism, and heart failure. These conditions may present with similar symptoms and radiological findings, making it challenging to distinguish them from pleural amebiasis based on clinical and radiological grounds alone. Bacterial

pneumonia is an infection of the lung parenchyma that can lead to pleural effusion as a complication. Symptoms of bacterial pneumonia may include fever, cough, chest pain, dyspnea, and sputum production. Radiological findings may include consolidation, pleural effusion, and atelectasis. Distinguishing bacterial pneumonia from pleural amebiasis can be challenging, as both conditions can present with similar symptoms and radiological findings. However, bacterial pneumonia is often associated with a more acute onset of symptoms and a higher fever. Pleural fluid analysis can help to differentiate the two conditions, as bacterial pneumonia typically results in a neutrophilic predominance in the pleural fluid, whereas pleural amebiasis may have a lymphocytic predominance. Tuberculosis is a chronic infectious disease that can affect the lungs and pleura. Symptoms of tuberculosis may include chronic cough, hemoptysis, weight loss, night sweats, fever, fatigue, and anorexia. Radiological findings may include pulmonary infiltrates, cavities, pleural effusion, and lymphadenopathy. Distinguishing tuberculosis from pleural amebiasis can be challenging, as both conditions can present with similar symptoms and radiological findings. However, tuberculosis is often associated with a more chronic course and a history of exposure to tuberculosis. Pleural fluid analysis can help to differentiate the two conditions, as tuberculosis typically results in a lymphocytic predominance in the pleural fluid, whereas pleural amebiasis may have a neutrophilic predominance. Lung cancer or pleural malignancies can cause pleural effusion and present with dyspnea, cough, and chest pain. Other symptoms may include hemoptysis, weight loss, hoarseness, and fatigue. Radiological findings may include pleural effusion, pleural thickening, and masses. Distinguishing malignancy from pleural amebiasis can be challenging, as both conditions can present with similar symptoms and radiological findings. However, malignancy is often associated with a history of smoking or exposure to carcinogens. Pleural fluid analysis can help to differentiate the two conditions, as malignancy typically results in a bloody or serosanguineous effusion, whereas pleural amebiasis may have a purulent or "anchovy sauce" appearance. Pulmonary embolism is a condition in which a blood clot travels from the legs or pelvis to the lungs, blocking blood flow to a portion of the lung. Symptoms of pulmonary embolism may include sudden onset dyspnea, chest pain, cough, tachycardia, tachypnea, and hemoptysis. Radiological findings may include atelectasis, pleural effusion, and a wedge-shaped infiltrate. Distinguishing pulmonary embolism from pleural amebiasis can be challenging, as both conditions can present with dyspnea, chest pain, and cough. However, pulmonary embolism is often associated with a more sudden onset of symptoms and risk factors such as recent surgery, immobilization, or a history of deep vein thrombosis. CT pulmonary angiography is the gold standard for diagnosing pulmonary embolism. Heart failure is a condition in which the heart is unable to pump enough blood to meet the body's needs. Symptoms of heart failure may include dyspnea, cough, orthopnea, peripheral edema, jugular venous distension, and hepatomegaly. Radiological findings may include cardiomegaly, pulmonary edema, and pleural effusion. Distinguishing heart failure from pleural amebiasis can be challenging, as both conditions can present with dyspnea and cough. However, heart failure is often associated with a history of cardiovascular disease and other signs and symptoms of heart failure. Echocardiography is the gold standard for diagnosing heart failure. There is no single specific diagnostic test for pleural amebiasis. The diagnosis is often made based on a combination of clinical, radiological, and laboratory findings. A high index of suspicion is required, especially in patients from endemic areas or those with risk factors for amebiasis. The diagnosis of pleural amebiasis can be challenging due to the lack of a single, definitive diagnostic test. Currently, no specific blood test, imaging modality, or clinical finding can definitively confirm or exclude pleural amebiasis. This diagnostic uncertainty often leads to delays in diagnosis and treatment, potentially

resulting in worse outcomes for patients. In the absence of a specific diagnostic test, clinicians must rely on a combination of clinical, radiological, and laboratory findings to diagnose pleural amebiasis. This approach requires careful consideration of the patient's medical history, physical examination findings, imaging results, and pleural fluid analysis. A high index of suspicion is crucial, especially in patients from endemic areas or those with risk factors for amebiasis. Clinical findings alone are often non-specific and can mimic other respiratory conditions. Common symptoms include dyspnea, cough, chest pain, and fever, which can also be seen in bacterial pneumonia, tuberculosis, and malignancy. A detailed medical history, including travel history, dietary habits, and history of amebiasis, can help to raise suspicion for pleural amebiasis. Chest radiography may reveal pleural effusion, but it cannot differentiate amebic effusion from other causes. Other radiological findings, such as atelectasis or consolidation, may also be present, further confounding the diagnosis. Additional imaging studies, such as CT scan or ultrasound, may be necessary to further evaluate the pleural effusion and rule out other potential causes. Pleural fluid analysis is crucial in establishing the diagnosis of pleural amebiasis. The pleural fluid is typically exudative, with a predominance of neutrophils. However, in some cases, the fluid may be lymphocytic predominant. The presence of *E. histolytica* trophozoites in the pleural fluid, either by microscopic examination or culture, confirms the diagnosis. However, the sensitivity of microscopic examination can be variable, and culture may take several days to yield results. Serological tests for amebiasis can be helpful in supporting the diagnosis, especially in cases where the pleural fluid is negative for *E. histolytica*. However, serological tests cannot differentiate between current and past infections, as antibodies may persist for years after successful treatment. The lack of specific diagnostic tests for pleural amebiasis highlights the need for improved diagnostic tools. These tests could include molecular diagnostic techniques, such as polymerase chain reaction (PCR), or novel biomarkers for amebiasis. In the absence of a single, definitive diagnostic test, clinical judgment plays a crucial role in diagnosing pleural amebiasis. Clinicians must carefully consider all available clinical, radiological, and laboratory findings to arrive at an accurate diagnosis. A high index of suspicion is essential, especially in patients from endemic areas or those with risk factors for amebiasis. Thoracentesis and pleural fluid analysis are crucial in establishing the diagnosis of pleural amebiasis. The pleural fluid is typically exudative, with a predominance of neutrophils. However, in some cases, the fluid may be lymphocytic predominant, as in our patient. The presence of *E. histolytica* trophozoites in the pleural fluid, either by microscopic examination or culture, confirms the diagnosis. In our patient, the pleural fluid was reddish-brown in color, often described as "anchovy sauce pus," which can be a clue towards amebic etiology. However, this finding is not always present and can also be seen in other conditions like complicated parapneumonic effusions or malignant effusions. Pleural fluid analysis is an essential diagnostic procedure in evaluating patients with pleural effusion. It involves inserting a needle into the pleural space to collect a sample of pleural fluid for laboratory analysis. This analysis provides valuable information about the nature of the effusion and can help to identify the underlying cause. One of the primary uses of pleural fluid analysis is to distinguish between exudative and transudative effusions. Exudates are caused by inflammation or infection, whereas transudates are caused by imbalances in hydrostatic or oncotic pressures. This distinction is crucial as it helps to narrow down the differential diagnosis and guide further investigations. Cell count and differential helps to identify the types of cells present in the fluid, such as neutrophils, lymphocytes, or malignant cells. Elevated protein levels are typically seen in exudative effusions. Low glucose levels may indicate infection or malignancy. Elevated LDH levels may indicate inflammation or infection. Gram stain and culture tests can help to identify bacterial infections. Cytology tests can help to

identify malignant cells. In the case of pleural amebiasis, pleural fluid analysis is crucial for confirming the diagnosis. The presence of *E. histolytica* trophozoites in the pleural fluid, either by microscopic examination or culture, confirms the diagnosis. However, the sensitivity of microscopic examination can be variable, and culture may take several days to yield results. In our patient, pleural fluid analysis revealed an exudative effusion with a predominance of lymphocytes. This finding, along with the reddish-brown color of the fluid, raised suspicion for pleural amebiasis. Microscopic examination of the pleural fluid confirmed the presence of *E. histolytica* trophozoites, establishing the diagnosis. In some cases, additional tests may be performed on pleural fluid to further evaluate the effusion. These tests may include adenosine deaminase (ADA) levels, which can be elevated in tuberculosis, and tumor markers, which can be elevated in malignancy. In our patient, the ADA level was not elevated, further supporting the diagnosis of pleural amebiasis. Serological tests for amebiasis can be helpful in supporting the diagnosis, especially in cases where the pleural fluid is negative for *E. histolytica*. However, serological tests cannot differentiate between current and past infections, as antibodies may persist for years after successful treatment. Serological tests detect the presence of antibodies in the blood that are produced by the immune system in response to *E. histolytica* infection. These tests are particularly useful in diagnosing extraintestinal amebiasis, such as amebic liver abscess or pleural amebiasis, where direct detection of the parasite in the affected organ may be difficult. Several types of serological tests are available for amebiasis, including Enzyme-linked immunosorbent assay (ELISA), Indirect hemagglutination assay (IHA), Indirect fluorescent antibody test (IFA). ELISA is the most commonly used serological test for amebiasis and is widely available. Serological tests for amebiasis generally have high sensitivity and specificity, meaning they are accurate in detecting both the presence and absence of infection. However, the accuracy of these tests can vary depending on the type of test used, the stage of infection, and the patient's immune status. One of the main limitations of serological tests is their inability to differentiate between current and past infections. Antibodies to *E. histolytica* can persist for years after successful treatment, leading to false-positive results. Therefore, serological tests should be interpreted in conjunction with other clinical and laboratory findings. In pleural amebiasis, serological tests can be helpful in supporting the diagnosis, especially in cases where the pleural fluid is negative for *E. histolytica*. However, it is important to note that a negative serological test does not rule out pleural amebiasis, as the sensitivity of these tests may be lower in extraintestinal amebiasis. In our patient, serological tests were not performed as the diagnosis of pleural amebiasis was confirmed by the presence of *E. histolytica* trophozoites in the pleural fluid. However, if the pleural fluid had been negative for *E. histolytica*, serological tests would have been considered to support the diagnosis. Diagnosing pleural amebiasis often requires a multidisciplinary approach, involving pulmonologists, gastroenterologists, and infectious disease specialists. Collaboration among these specialists is essential to ensure accurate diagnosis and appropriate management. Pleural amebiasis can present with a complex clinical picture, involving both respiratory and gastrointestinal symptoms. The diagnosis may not be immediately apparent, and it often requires the expertise of multiple specialists to arrive at the correct diagnosis. Pulmonologists play a key role in the diagnosis and management of pleural amebiasis. They are often the first point of contact for patients presenting with respiratory symptoms, such as dyspnea, cough, and chest pain. Pulmonologists perform a thorough clinical evaluation, including a detailed medical history, physical examination, and review of systems. They also order and interpret imaging studies, such as chest radiography and CT scans, to evaluate the pleural effusion and rule out other potential causes. Pulmonologists perform thoracentesis and send pleural fluid for analysis, which is crucial in establishing the diagnosis of

pleural amebiasis. Gastroenterologists are involved in the evaluation and management of patients with suspected pleural amebiasis, especially if there is a suspicion of liver involvement. They perform a thorough abdominal examination and order imaging studies, such as abdominal ultrasound or CT scans, to evaluate the liver and rule out amebic liver abscess. Gastroenterologists may also perform endoscopic procedures, such as colonoscopy, to evaluate the colon for evidence of amebic colitis. Infectious disease specialists provide expertise in the diagnosis and treatment of amebiasis. They are consulted to confirm the diagnosis of pleural amebiasis, especially in cases with diagnostic uncertainty. They also recommend appropriate antimicrobial therapy and monitor the patient's response to treatment. Collaboration among these specialists is essential to ensure accurate diagnosis and appropriate management of pleural amebiasis. By sharing their expertise and perspectives, these specialists can arrive at a comprehensive understanding of the patient's condition and develop an individualized treatment plan. This multidisciplinary approach can help to improve patient outcomes and reduce the risk of complications. In our patient's case, a multidisciplinary approach was essential in establishing the diagnosis and ensuring appropriate management. The patient was initially seen by a pulmonologist for her respiratory symptoms. The pulmonologist performed thoracentesis and sent pleural fluid for analysis, which revealed the presence of *E. histolytica* trophozoites. The patient was then referred to a gastroenterologist to rule out amebic liver abscess. An infectious disease specialist was also consulted to confirm the diagnosis and recommend appropriate antimicrobial therapy.[14-17]

Thoracentesis and pleural fluid analysis were crucial in establishing the diagnosis in this case. The exudative nature of the effusion, along with the presence of *E. histolytica* trophozoites on microscopic examination, confirmed pleural amebiasis. Interestingly, our patient's pleural fluid showed a predominance of mononuclear cells, which is less common in amebic effusions. Typically, a neutrophilic predominance is observed. This variability highlights the diverse presentations of pleural amebiasis. Pleural fluid analysis is an essential diagnostic procedure in evaluating patients with pleural effusion. It involves inserting a needle into the pleural space to collect a sample of pleural fluid for laboratory analysis. This analysis provides valuable information about the nature of the effusion and can help to identify the underlying cause. In the case of pleural amebiasis, the pleural fluid may exhibit unique characteristics that can aid in diagnosis. The appearance of pleural fluid can provide valuable clues about the underlying cause of the effusion. In pleural amebiasis, the pleural fluid may have a distinctive reddish-brown color, often described as "anchovy sauce pus." This characteristic appearance is attributed to the presence of blood and necrotic debris in the fluid. However, it is important to note that this finding is not always present in pleural amebiasis and can also be seen in other conditions, such as complicated parapneumonic effusions or malignant effusions. In our patient, the pleural fluid exhibited this characteristic reddish-brown color, which raised suspicion for pleural amebiasis. Biochemical analysis of pleural fluid can help to differentiate between exudative and transudative effusions. Exudative effusions are typically characterized by high protein content, high lactate dehydrogenase (LDH) levels, and low glucose levels. In pleural amebiasis, the pleural fluid is usually exudative, reflecting the inflammatory nature of the infection. In our patient, the pleural fluid analysis revealed an exudative effusion, consistent with pleural amebiasis. Microscopic examination of pleural fluid is crucial for identifying the presence of *E. histolytica* trophozoites. This involves examining a stained smear of the pleural fluid under a microscope. The trophozoites are typically identified by their characteristic morphology, including their amoeboid movement and the presence of ingested red blood cells. In our patient, microscopic examination of the pleural fluid confirmed the presence of *E. histolytica* trophozoites, establishing the diagnosis of pleural

amebiasis. Culture of pleural fluid can also be performed to isolate and identify *E. histolytica*. However, culture may take several days to yield results and is not always successful in isolating the parasite. In our patient, culture of the pleural fluid was not performed as the diagnosis was confirmed by microscopic examination. In some cases, additional tests may be performed on pleural fluid to further evaluate the effusion. These tests may include adenosine deaminase (ADA) levels, which can be elevated in tuberculosis, and tumor markers, which can be elevated in malignancy. In our patient, the ADA level was not elevated, further supporting the diagnosis of pleural amebiasis.[18-20]

4. Conclusion

This case underscores the importance of considering pleural amebiasis in patients with pleural effusion, even in the absence of classic risk factors like liver abscess. A high index of suspicion is crucial, especially in individuals with a history of consuming raw vegetables or residing in areas where amebiasis is prevalent. Thorough clinical and radiological investigations, coupled with pleural fluid analysis, are essential for accurate diagnosis. Prompt treatment with metronidazole and drainage, as demonstrated in our patient, usually leads to a favorable outcome. This case emphasizes the need for increased awareness and a multidisciplinary approach in managing this rare but potentially serious condition.

5. References

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