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PLEUROPULMONARY AMEBIASIS – A CASE REPORT

Respiratory Medicine

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ABSTRACT

Amebiasis, caused by Entamoeba histolytica, remains a major health issue in developing regions, commonly affecting the intestines and liver(1,2). Rarely, it can present as a pleuropulmonary infection without prior intestinal or hepatic involvement, which can mimic conditions like tuberculosis or bacterial pneumonia(5,7). This report details the case of a 54-year-old male presenting with right-sided chest pain, fever, and difficulty breathing. Imaging and thoracentesis revealed pleural effusion containing reddish-brown fluid resembling "anchovy sauce." Microscopy confirmed the presence of E. histolytica trophozoites in the pleural fluid-an uncommon but documented manifestation of pulmonary amebiasis (3,4,6)

KEYWORDS

Entamoeba histolytica, trophozoites, pulmonary amebiasis, bacterial pneumonia

INTRODUCTION

Amebiasis, caused by the protozoan parasite Entamoeba histolytica, is an intestinal parasitic infection with a wide geographic distribution, especially prevalent in regions with inadequate sanitation and limited healthcare infrastructure. Globally, it accounts for approximately 100,000 deaths annually, ranking among the leading causes of parasite-related mortality worldwide[1].

While many infections remain asymptomatic, E. histolytica can cause invasive disease, including intestinal dysentery and extraintestinal manifestations. The most frequent extraintestinal form is the amebic liver abscess (ALA). Pulmonary complications typically arise when an ALA ruptures into the thoracic cavity, leading to pleuropulmonary involvement. However, primary pleuropulmonary amebiasis without liver or intestinal disease is extremely rare and may present with non-specific respiratory symptoms that mimic other pulmonary conditions such as tuberculosis, pneumonia, or malignancy[2–4].

Case Study

A 54-year-old male, working as a farmer, presented with a three-day history of right-sided chest pain, fever, and dyspnea. The chest pain was localized to the right lower thoracic region, with a gradual onset and progressive intensity. It worsened with movement, deep inspiration, and lying supine. The pain radiated to the back and right shoulder tip.

He was a chronic bidi smoker and consumed alcohol occasionally. The patient denied any gastrointestinal complaints such as jaundice, abdominal pain, vomiting, diarrhea, or nausea.

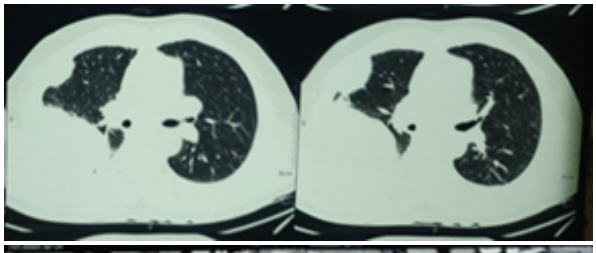
On examination, his vital signs were: pulse 108/min, blood pressure 130/80 mmHg, respiratory rate 26/min, and oxygen saturation 93% on room air. Respiratory examination showed tenderness in the right lower chest, reduced air entry, decreased tactile vocal fremitus, and crepitations on the right side. Abdominal examination revealed mild hepatomegaly with tenderness in the right upper quadrant; however, there were no signs of peritonitis.

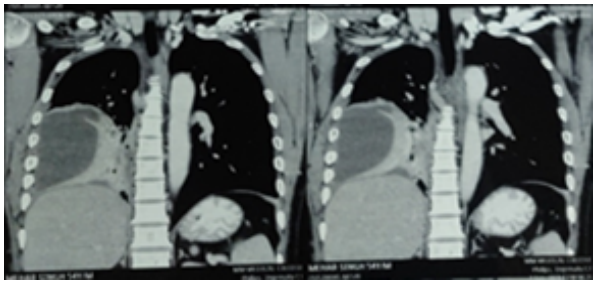
The patient was administered supplemental oxygen. Chest X-ray revealed obliteration of the right costophrenic angle, suggestive of pleural effusion. Abdominal ultrasonography showed mild hepatomegaly, gallstones with possible cholecystitis, and a mild right-sided pleural effusion. Contrast-enhanced CT (CECT) of the chest and abdomen confirmed moderate right-sided pleural effusion with loculations and associated pleural thickening. There was almost complete passive collapse of the right lower lobe and partial collapse of the middle lobe. No liver abscess was detected.

A right-sided intercostal chest drain was inserted, yielding approximately 300 mL of pleural fluid. The initial fluid appeared serous yellow, later transitioning to a reddish-brown "anchovy sauce" consistency. Microscopy of pleural fluid revealed E. histolytica trophozoites. Acid-fast bacilli staining was negative. Stool examinations (performed twice) were negative for E. histolytica cysts or trophozoites. Blood cultures showed no growth. Serological testing was not available.

Laboratory investigations showed elevated total leukocyte count, eosinophilia, increased erythrocyte sedimentation rate (ESR), and normal liver and renal function tests.

The patient was initiated on metronidazole 800 mg three times daily and the luminal amebicide diloxanide furoate 500 mg three times daily. His condition improved markedly within three days. Drain output significantly reduced, and the intercostal tube was subsequently removed.





DISCUSSION

Pleuropulmonary amebiasis usually arises due to direct extension or rupture of a hepatic abscess through the diaphragm into the pleural space [3,5]. The classical "anchovy sauce" appearance of the pleural fluid, resulting from liquefied liver tissue mixed with inflammatory exudate, is a hallmark of this complication. However, primary pulmonary involvement without liver disease, as in this case, is highly uncommon [4,6].

Diagnosis requires a high degree of clinical suspicion, particularly in endemic areas. The detection of E. histolytica trophozoites in pleural fluid is confirmatory. Imaging is crucial in identifying the extent of pulmonary involvement and ruling out hepatic abscess. Stool studies may be negative, as observed in this patient, especially in cases where

intestinal colonization has resolved or is minimal [7].

Treatment with nitroimidazole derivatives like metronidazole is the mainstay of therapy. Luminal agents such as diloxanide are used to eradicate residual intestinal cysts. In cases with significant pleural effusion or empyema, drainage via intercostal tube is essential for clinical resolution [2,5].

CONCLUSIONS

Pleuropulmonary amebiasis should be considered in patients from endemic regions presenting with unexplained pleural effusion, even in the absence of liver or intestinal disease. Prompt diagnosis through imaging and pleural fluid analysis, followed by effective anti-amebic therapy and drainage, can lead to full recovery.

REFERENCES:

[1] Stanley SL Jr. Amoebiasis. Lancet. 2003;361(9362):1025–34. doi:10.1016/S0140-6736 (03)12830-9
[2] Haque R, Huston CD, Hughes M, Houpt E, Petri WA Jr. Amebiasis. N Engl J Med. 2003;348(16):1565–73. doi:10.1056/NEJMra022710
[3] Eggleston FC, Bishop MB, Vickery WH. Primary amebic abscess of the lung without liver involvement. Am Rev Respir Dis. 1969;100(1):99–104. doi:10.1164/ arrd.1969. 100.1.99
[4] Kimura K, Kagei N, Kudo K, Shimada K. Pleuropulmonary amebiasis diagnosed by demonstration of Entamoeba histolytica in pleural fluid. J Infect Chemother. 2001;7(2):99–101. doi:10.1007/s101560100019
[5] Sharma MP, Ahuja V. Extraintestinal amebiasis. In: Singal AK, ed. Tropical Gastroenterology. Mumbai: ICMR; 1995. p. 111–8.
[6] Kundu S, Bhattacharya A, Chakrabarti S, et al. Primary amebic lung abscess: a rare entity. Lung India. 2010;27(4):213–5. doi:10.4103/0970-2113.71961
[7] Reed SL. Diagnosis and management of amoebiasis. Clin Infect Dis. 1992;14(5): 1107–16. doi:10.1093/clinids/14.5.1107