Thoracic Amebic Empyema after Complete Resolution of Liver Abscess

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ABSTRACT

A 67-year-old diabetic male was hospitalized through pulmonary OPD with history of exertional dyspnea, right sided chest pain and pyrexia. Before hospitalization, he was diagnosed to have a solitary liver abscess that was treated in the community with various antibiotics and at the time of admission, there was no residual abscess in the liver. He was found to have a huge opacity involving the upper and middle portions of right hemithorax which was consistent with a large pleural effusion on cross sectional chest CT imaging. There was sparing of lower hemithorax, this was quite unusual for a pleural collection which in majority accumulates in lower dependent zones. Under ultrasound guidance, a small bore (10 Fr) pleural catheter was placed in the upper zone of right pleural cavity that lead to therapeutic drainage of 800 ml of pus having anchovy sauce like (chocolate colored) appearance suggestive of amebic empyema. Besides drainage, he required intrapleural streptokinase instillations for adhesiolysis and was medically managed with metronidazole. He responded well to the treatment with complete clinical and radiological resolution of amebic empyema & lung expansion.

Key words: Amebic, empyema thoracis, intrapleural streptokinase, small bore pleural catheter, tube thoracostomy.

INTRODUCTION

Thoracic amebiasis is an uncommon complication due to rupture of amebic liver abscess caused by Entamoeba histolytica. E. histolytica is estimated to infect 50 million people worldwide every year and accounts for an estimated 100,000 annual deaths. Infection ranges from asymptomatic colonization of the large bowel to severe invasive intestinal and extra-intestinal disease. E. histolytica is transmitted primarily through the fecal-oral route. The parasite life cycle is relatively simple, humans orally ingest the cysts from contaminated sources, excystation to the trophozoite form occurs in the small bowel, then the trophozoite either colonizes or invades the large bowel. When the trophozoite encysts, the life cycle is complete and the organism can be transmitted. Although most cases of amebiasis are asymptomatic, dysentery and invasive extra intestinal disease can occur. Amebic liver abscess is the most common manifestation of invasive amebiasis, but other organs can also be involved, including pleuropulmonary, cardiac, cerebral, renal, genitourinary, peritoneal, and cutaneous sites especially in immunocompromised hosts.

CASE HISTORY

A 67-year-old male was hospitalized through OPD with history of exertional dyspnea, right hypochondrial pain, right sided chest pain and fever of 100°F for 2 months. Before hospitalization, he was diagnosed to have a solitary liver abscess that was managed in the community with various intravenous (ceftriaxone, ceftazidime) and oral (co-amoxiclav, moxifloxacin, ciprofloxacin) antibiotics and at the time of admission there was no residual abscess in the liver on hepatobiliary ultrasound examination. He was suffering from diabetes mellitus which was controlled on oral hypoglycemic agents. In the past, he was never hospitalized for
any medical or surgical ailment. He kept no pet animal or birds at home and denied any aerosol or chemical exposure. He was married, had four healthy children and belonged to lower middle socioeconomic class. On general physical examination, he was alert and cooperative, febrile with temperature of 101°F, regular pulse 90/min, BP 130/80 mmHg & respirations were 22/m with 97% saturation on pulse oximetry. Respiratory system examination revealed dull percussion note with reduced intensity of breath sounds on the right side. He had pain in right hypochondrium on deep palpation. Remaining general and systemic examinations were normal. Laboratory evaluation showed hemoglobin 11.2 g/dl, TLC 23000/cmm and platelets 273000/cmm. Serum biochemical analysis showed BUN 18 mg/dl, creatinine 0.8 mg/dl and albumin 2.5 mg/dl, LFTs and electrolytes were normal. Serology for E. histolytica was surprisingly negative. Chest radiographic findings included mass like homogenous opacity with curved inferior margin involving right hemithorax with slight sparing of base and raised right hemidiaphragm (Fig. 1). Contrast enhanced CT scan of chest (Fig. 2) showed pleural based opacity involving the right hemithorax sparing the lower portion, with positive split pleura sign suggestive of empyema. Ultrasonography chest showed multiloculated collection at the antero-lateral aspect of the upper part of the pleura.

Diagnostic aspiration showed thick brownish pus with anchovy sauce like appearance (Fig. 3) highly suggestive of amebic empyema, a consequence of ruptured liver abscess into pleura. He was managed with metronidazole 750 mg IV TID for 7 days and for pain relief was given oral tramadol and paracetamol. Pleural ultrasound was utilized to localize the pleural empyema found at the antero-lateral aspect of the upper part of the chest. To decompress the pleural space, a small bore (10 Fr) pleural catheter was placed under local anesthesia at-the-bed-side in localized collection of the right upper part of the chest. Almost 500 ml of amebic pus was drained after pleural catheter placement but the lung failed to expand due to presence of multiple pleural loculations (Fig. 4a). Adhesiolysis was successful with the use of intrapleural streptokinase instillations (total 1.5 million units in six divided doses over 3 days) in combination with external suction of -20 cmH₂O. Further 300 ml brownish pus was drained over next 3 days leading to partial expansion of right lung.
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(Fig. 4b). At this stage, patient was clinically improved, CRP and TLC counts were normal and pleural drainage was nil, therefore pleural drain was removed and patient was discharged. Follow up chest radiograph (after 2 weeks) was unremarkable except insignificant scarring and pleural thickening at the right apical region (Fig. 5).

Fig. 3: Chocolate colored (anchovy sauce appearance) aspirated pleural fluid.

DISCUSSION

Amebiasis is caused by *E. histolytica*, a protozoan that is found worldwide. The highest prevalence of amebiasis is in developing countries where barriers between human feces and food and water supplies are inadequate. Risk factors for development of pulmonary amebiasis include malnutrition, chronic alcoholism and atrial septal defect with left to right shunt. Pleural manifestations may include development of a sympathetic serous effusion. Liver abscess rupturing into the pleural space results in an amebic empyema; rupture into the lung can lead to consolidation, abscess formation, or a hepatobronchial fistula. Other mechanisms of pleural involvement include lymphatic spread from the liver through the diaphragm, or hematogenous embolic spread from the liver or colon. Hematogenous spread is an unusual disorder and should be suspected when there is pulmonary

Fig. 4 (A) CXR-PA after small bore pleural catheter placement showing entrapped right lung with air fluid level due to loculated nature of pleural collection (B) partial expansion of lung after intrapleural fibrinolytic treatment in combination with external suction.

Fig. 5: Follow-up radiograph showing area of small residual scarring and pleural thickening at the right apical region.
amebiasis in the absence of hepatic disease, or noncontiguous pulmonary and hepatic disease. Approximately 10% of patients with amebic liver abscess develop pleuro-pulmonary amebiasis, which presents with cough, pleuritic pain, and dyspnea. It is 7-10 times more common among adult men, despite equal gender distribution of colonic amebic disease and is observed most frequently in the fourth and fifth decades of life. Patient described in this case had similar demographic characteristics and also presented with pleuritic chest pain, cough, sputum and dyspnea. A hepatobronchial fistula is an unusual problem characterized by the expectoration of sputum resembling anchovy paste. The trophozoites of *E. histolytica* may be found in the sputum sample. Primary amebic pneumonia as a result of hematogenous spread has been reported, though rarely. Our patient had loculated pleural effusion which on gross appearance resembled anchovy sauce, grew no microorganism on culture and in this clinical scenario was highly consistent with amebic empyema. We successfully drained the pus with a small bore (10 Fr) pleural catheter placement in accordance with British Thoracic Society (BTS) guidelines about pleural infection. Recommendations for fibrinolytics in pleural infections include combination of DNase and tissue plasminogen activator (TPA). We utilized intrapleural streptokinase (only available fibrinolytic in Pakistan with anecdotal evidence of successful use) that in combination with external tube suction probably assisted in adhesiolysis and expansion of entrapped lung. A study carried out by Taylor RF & colleagues found that intrapleural streptokinase was an effective adjunct in the management of complicated empyema and may reduce the need for surgery. Several agents are active against anaerobic bacteria and protozoa. Metronidazole is the drug of choice for symptomatic, invasive amebic disease and paromomycin is the preferred drug for noninvasive disease. Patient described above also received intravenous metronidazole 750 mg three times daily and experienced remarkable improvement.

CONCLUSION

Amebic empyema should be considered in patients presenting with loculated or non loculated right sided pleural effusions/empyemas in countries endemic for amebic diseases.

REFERENCES


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