



Case report

Apical loculated pleural effusion : an interesting case

Swapnil Kulkarni^a, Kapil Iyer^b, Mahesh Jansari^b^aDepartment of Pulmonary Medicine, ESIS hospital, LBS Road, Mulund(W), Mumbai – 400080, Maharashtra, India^b501, Spring Leaf, Military Road, Marol, Andheri East, Mumbai- 400059, Maharashtra, India.^b501, Spring Leaf, Military Road, Marol, Andheri East, Mumbai- 400059, Maharashtra, India.

ARTICLE INFO

Keywords:

Pleural effusion,
 loculated,
 Computed tomography,
 adenosine deaminase (ADA)

ABSTRACT

Pleural effusion is defined as the presence of fluid in the pleural cavity. It has various causes ranging from tuberculosis, bacterial empyema to congestive cardiac failure. The workup of such patients as a rule always begins with a good history, clinical examination combined with aspiration of the fluid and doing a detailed cytochemical analysis. Rarely some patients might present with loculated forms of pleural effusion which adds on to their diagnostic dilemma. We present a case of an apical loculated pleural effusion having a very interesting etiology.

© Copyright 2010 BioMedSciDirect Publications IJBMR - ISSN: 0976:6685. All rights reserved.

1. Introduction

A pleural effusion is an abnormal collection of fluid in the pleural space resulting from excess fluid production or decreased absorption or both(1). It is the most common manifestation of pleural disease, with etiologies ranging from cardiopulmonary disorders to symptomatic inflammatory or malignant diseases requiring urgent evaluation and treatment. The normal pleural space contains approximately 1 mL of fluid, representing the balance between hydrostatic and oncotic forces in the visceral and parietal pleural vessels and extensive lymphatic drainage. Pleural effusions result from disruption of this balance. Pleural effusions are generally classified as transudates or exudates, based on the mechanism of fluid formation and pleural fluid chemistry. Transudates result from an imbalance in oncotic and hydrostatic pressures, whereas exudates are the result of inflammation of the pleura or decreased lymphatic drainage. In a country like India, which has an increased prevalence of tuberculosis, one of the most common causes of pleural effusion is tuberculosis.

Rarely some patients might present with loculated pleural effusion, examples being fissural, mediastinal, apical and lamellar. We present a case of apical loculated pleural effusion, which during the course of evaluation had an interesting etiology.

Case report:

A 50 year old woman was referred to our institution for evaluation of her respiratory symptoms. She had a 4 month history of right sided pleuritic chest pain, progressive breathlessness on exertion and dry cough. Her symptoms were preceded by a few episodes of gastroenteritis. She was diagnosed as a case of right sided tuberculous pleural effusion by her family physician and was subsequently started on weight adjusted anti tuberculous

medications 3 months back. She gave no history of thoracentesis being performed. However, as she did not respond to the treatment, she was referred to our centre for further evaluation.

She was conscious and well oriented in time, place and person. Her vitals were stable. Respiratory system examination revealed signs of volume gain on the right side with decreased breath sounds. A chest x ray PA view showed right sided apical loculated pleural effusion. A computed tomography of thorax confirmed the presence of right sided apical pleural effusion. However the computed tomography also revealed 2 well defined liver abscesses with maximum dimensions of 3.5 * 2.5 cms. An ultrasonography guided thoracentesis showed thick brown colored pleural fluid, which on cytochemical analysis was an exudate with lymphocytic predominance and raised ADA levels. Her serum antibodies against Entamoeba Histolytica were positive. She was subsequently started on a course of Metronidazole followed by 10 days of Diloxanide Furoate, to which she responded clinically.

Figure 1 : chest x ray PA view showing right sided apical loculated pleural effusion.



* Corresponding Author : Dr.Kapillyer

501, Spring Leaf, Military Road, Marol, Andheri East, Mumbai- 400059, Maharashtra, India.
iyerkapil@gmail.com

©Copyright 2010 BioMedSciDirect Publications. All rights reserved.

Figure 2 : Computed Tomography of thorax confirming right apical pleural effusion.

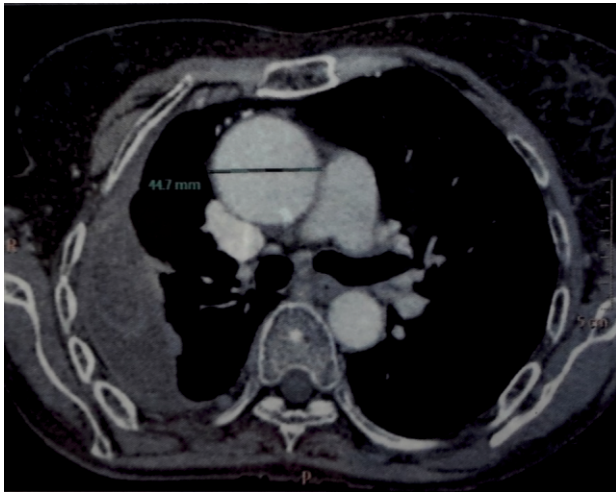
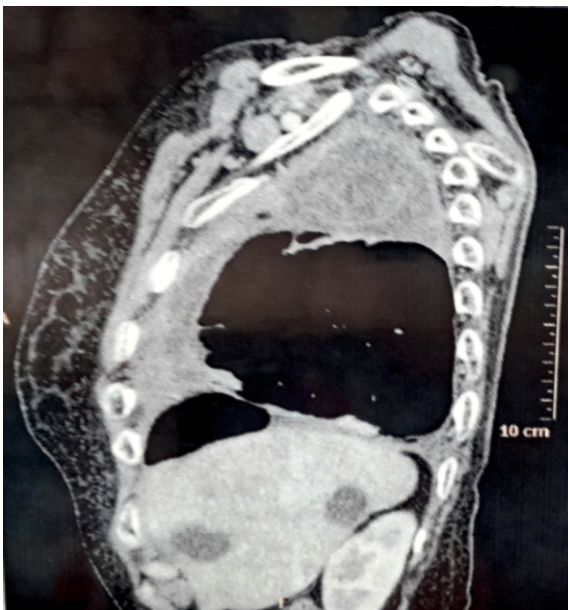


Figure 3 : Sagittal view of computed tomography of thorax showing right sided apical effusion and incidental finding of liver abscesses.



Discussion :

Amoebiasis is a parasitic infection caused by *Entamoebahistolytica*. It is the third most common cause of mortality due to parasitic infections in the world after malaria and schistosomiasis(2). It is endemic in tropical countries and in areas with deficient sanitation and poor socioeconomic conditions.

Entamoebahistolytica, an amoebic protozoan parasite, is the most invasive of the *Entamoeba* group. The life cycle of the protozoan includes an infective cyst and an invasive trophozoite form, and infection occurs due to fecal-oral mechanism through water or food contaminated with feces(3). Clinically, the disease presentation in amoebiasis ranges from asymptomatic colonization to colitis and/or liver abscess. Amoebic colitis and liver abscess are the most common intestinal and extra intestinal manifestations of the amoebiasis respectively. Infection spreads to liver via portal veins.

Pleuro pulmonary amoebiasis is the third most common manifestation of the amoebiasis in the human body and is the second most common extra intestinal manifestation followed by liver abscess. Pulmonary manifestations are invariably secondary to an amoebic liver abscess. It occurs in 2-3% of patients with invasive amoebiasis(2,4). Lung disease without liver involvement is rare and it is believed to occur as a result of haematogenous spread from a primary site, usually colon(4). Pulmonary manifestations occur as a direct spread of liver abscess through the diaphragm into the pleural cavity or into the lung parenchyma or both. As right lobe of the liver is the most common site for the amoebic liver abscess, pleuro pulmonary manifestations most commonly occur on the right side. Rarely a left lobe liver abscess may extend through the diaphragm into the pericardium and left pleural space. Rupture of the abscess into the pericardium is associated with a high mortality rate(5). Rarely trophozoites enter into the systemic circulation through rectal venous plexus leading to haematogenous spread to the lung and other organs like brain. Sympathetic plural effusion is also common in which pleural fluid remains sterile.

For diagnosis of amoebiasis, microscopy is a very useful method. Other more advanced methods such as antigen detection, polymerase chain reaction or serology are also available. Detection of antibodies is a simple and reliable test. It can be helpful in the case of ALA where patients do not have detectable parasites in feces. The sensitivity for detection of antibodies to *E. histolytica* in serum in patients with ALA is reported to be about 100%(6).

Our patient also presented with pleuro pulmonary complication of amoebiasis. Her history of a few episodes of gastroenteritis preceding her pulmonary symptoms was probably not given due importance. The present clinical case report aims to present a case of pleural effusion secondary to amoebic liver abscess which was treated wrongly with empirical anti tuberculous treatment.

Conclusion :

This case report highlights the importance of an exhaustive history taking while evaluating a case of pleural effusion. Pleuro pulmonary complications of amoebiasis should always be kept as a differential diagnosis in right sided pleural effusions, especially in countries having high prevalence of amoebiasis.

References

1. Diaz-Guzman E, Dweik RA. Diagnosis and management of pleural effusions: a practical approach. *Compr Ther*. 2007 Winter; 33(4):237-46.
2. Neghina R, Neghina AM, Merkle C, Marincu I, Iacobiciu I. A case report of pulmonary amoebiasis with *Entamoebahistolytica* diagnosed in western Romania. *J Infect Developing Countries* 2008; 2:400-2.
3. Singh U, Petri Jr WA. Amebas. In: Gillespie SH, Pearson RD, editors. *Principles and practice of Clinical Parasitology*. Chichester: John Wiley & Sons, Ltd. 2001. 197-218.
4. Gupta KB, Manchanda M, Chaudhary U, Verma M. Superior Vena cava syndrome caused by pulmonary amoebic abscess. *Indian J Chest Dis Allied Sci* 2006; 48:275-7.
5. Ibarra-Perez C: Thoracic complications of amoebic abscess of the liver: Report of 501 cases. *Chest* 1981; 79:672-677
6. Zengzhu, G., R. Bracha, Y. Nuchamowitz, W. Cheng, and D. Mirelman. 1999. Analysis by enzyme-linked immunosorbent assay and PCR of human liver abscess aspirates from patients in China for *Entamoebahistolytica*. *J. Clin. Microbiol.* 37:3034-3036.