AN UNUSUAL CASE OF A PLEURAL EFFUSION WITH AN ABDOMINAL MASS

Preetam R Acharya¹, Rameshchandra Sahoo² and Poornima Baliga³

(Received on 10.12.2007. Accepted after revision on 22.4.2008)

Summary: A 38-year old man presented to us with a left sided pleural effusion. Pleural fluid was aspirated and analysis revealed it to be an exudate with predominant lymphocytes and an elevated ADA level. He was discharged on anti-tuberculous treatment. Patient returned with re-accumulation of pleural fluid. Computed tomography done in our institute picked up not only parenchymal disease in the lung which was not evident on chest radiographs but also picked up an abdominal mass in the left renal fossa. Pathological examination of excised mass revealed its tuberculous nature. The repeated recollection of pleural fluid was attributed to a “paradoxical response”; the patient was reassured and his anti-tuberculous treatment continued. Recognition of the fact that evidence of tuberculosis at distant sites may occasionally be needed to substantiate the diagnosis of tuberculous pleural effusion in a difficult and bacteriologically “negative” case prompted us to report this case. [Indian J Tuberc 2008; 55: 153-156]

Key words: Tuberculous Pleural Effusion, Paradoxical Response, Incidentaloma

INTRODUCTION

Establishing Tuberculosis as the cause of pleural effusion can be difficult at times. The diagnosis of Tuberculous Pleuritis is commonly made from observation of granulomas in pleural biopsy specimens or a culture positive for Mycobacterium Tuberculosis from pleural tissue or pleural fluid. However, the diagnosis can be uncertain or missed in “bacteriologically negative” cases. Proof of co-existing pulmonary tuberculosis or getting evidence of the presence of mycobacterium at other extra-pulmonary sites may indicate dissemination and establish sufficient grounds for institution of anti-tuberculous treatment in an otherwise microbiologically ½ negative½ case.

CASE REPORT

A-38-year old man presented to us with left sided pleuritic chest pain and exertional breathlessness (Medical Research Council Grade I) for last seven days. He also had cough with minimal expectoration for a month. He denied having had haemoptysis, fever, chills or night sweats. On review of systems, the patient reported that he had lost some weight since last one month. He had no past history of respiratory disease and was a non-smoker. Family history for pulmonary tuberculosis was positive in an elder sibling who had completed a course of anti-tuberculous treatment 15 years back.

Respiratory system examination was suggestive of a left sided pleural effusion. Chest radiograph revealed a moderate left sided pleural effusion with contra lateral tracheal shift and no evidence of any parenchymal abnormalities (Fig.1). Routine blood investigations like haemogram, ESR, fasting blood glucose, total bilirubin, transaminases and creatinine were all within normal limits. Human immuno-deficiency virus (HIV) ELISA was negative. Three samples of sputum examined for AFB by Ziehl-Neelsen method were negative. Thoracentesis was done and serosanguinous fluid aspirated. Pleural fluid was an exudate (Protein: 7.1 gm/dl) with a leukocyte count of 4,800 cells/cumm and a lymphocyte differential of 95 %. Pleural fluid examination by Gram stain and ZN stain were negative. AFB culture of the pleural fluid sample did not show any
mycobacterial colonies at the end of eight weeks of incubation. Cytological examination of pleural fluid was negative for malignant cells. Pleural fluid adenosine deaminase (ADA) levels were elevated (patient value: 122.61 iu/L, normal: up to 36.0 iu/L). A provisional diagnosis of tuberculous pleural effusion was made and the patient discharged on a four drug antituberculous regimen consisting of Isoniazid, Rifampicin, Ethambutol and Pyrazinamide (HERZ).

Over a period of next four weeks, our patient did not show any improvement. A chest radiograph taken at follow-up at this point showed re-accumulation of pleural fluid. A closed pleural biopsy was inconclusive. Pleural fluid carcinoembryonic antigen (CEA) was normal (1.7). Pleural fluid was re-aspirated and a contrast enhanced computed tomography of chest was advised. This revealed calcified nodular opacities in apices of both the lungs. Additionally, a hypo dense

![Fig. 1: Chest radiograph showing left sided effusion](image1)

![Fig. 2: Follow-up chest radiograph at 12 weeks](image2)

![Fig. 3: Chest radiograph at treatment end.](image3)

![Fig. 4: Image showing granulomas consisting of epitheloid cells and Langhan’s giant cells (H & E X10)](image4)
enhancing mass lesion with a few areas of hyper intensity was seen to arise from the upper pole of the left kidney. CT guided biopsy and FNAC was done from the mass which showed only necrotic material.

Because of the rapid re-accumulation of pleural fluid despite regular anti-tuberculous treatment, a decision was taken to surgically explore the abdomen and excise the mass to rule out a malignant etiology. Surgical exploration revealed a pale brown mass measuring approximately 8×7×4 cms not involving the kidney. The mass was excised in its entirety. Histopathological examination revealed caseating granulomas consisting of epitheloid cells, Langhan’s giant cells and mature lymphocytes suggesting tuberculosis (Fig.4).

The presence of caseating granulomas in the excised mass in addition to the pleural fluid characteristics helped us to substantiate our initial diagnosis of tuberculous pleural effusion. Patient was asked to continue his anti-tuberculous drugs. Follow-up at regular intervals and serial chest radiographs (Fig.2, Fig.3) have not revealed any further re-accumulation of pleural fluid.

DISCUSSION

Pleural effusions can occur in any form of pulmonary tuberculosis. It is a well-known fact that neither the clinical features nor any of the imaging modalities are of much help in the diagnosis of a tuberculous pleural effusion. Co-existing parenchymal disease detected radiographically in about one third of the patients with an effusion serves as a marker of active pulmonary tuberculosis. Computed tomography of chest may show lymphadenopathy, pulmonary infiltrates or cavitation not seen on chest radiographs; which although non-specific, may help to distinguish tuberculous pleurisy from other causes like malignancy.

The onus of proving tuberculosis as a cause of pleural effusion rests on microbiological (smear/culture), and histological analysis of aspirated pleural fluid and biopsied pleural tissue. Mycobacteria are seen in pleural fluid only in 10 - 20 % of cases whereas a culture though positive in 25 - 50 % of the cases takes a minimum of 6-8 weeks by conventional methods to be of any clinical utility. Pleural biopsy will show granulomas in the parietal pleura in 50 – 80 % of patients and if cultured will grow mycobacterium in 55 – 80% of the cases. High levels of pleural fluid ADA (The reported cut-off value for PADA varies from 47 to 60 U/l) may be helpful in distinguishing tuberculous effusions from malignant effusions. Several reports have suggested that an elevated pleural fluid ADA level predicts tuberculous pleuritis with a sensitivity of 90 to 100% and a specificity of 89 to 100%.

However, controversy surrounds the use of elevated pleural fluid ADA as a diagnostic aid for tuberculosis effusion. Treatment with anti-tuberculous drugs is the mainstay of management of extra pulmonary tuberculosis. The term “paradoxical response” refers to enlargement of old lesions or unexpected appearance of new lesions during anti-tuberculous therapy. Re-crudescence of fever, enlarging adenopathies, worsening of pulmonary infiltrates, pleural effusion, ascites and appearance of intracranial tuberculomas have all been described. An incidence of 16% of paradoxical worsening of tuberculous effusion following the start of anti-tuberculous treatment has been observed. Such a paradoxical worsening can occur in both HIV uninfected and infected starting TB therapy. The syndrome poses a diagnostic challenge since the apparent clinical deterioration may raise the suspicion of drug resistant tuberculosis, non-compliance to prescribed regimen or presence of a concomitant disorder unrelated to TB. These patients generally need no alteration in their drug regimen.

An incidentaloma is a tumor (-oma) found by coincidence (- incidental). The widespread use of ultrasound, CT and MRI has resulted in the incidental discovery of asymptomatic adrenal masses. Adrenal incidentaloma is not a single pathological entity and the differential diagnosis includes adenoma, adrenocortical carcinoma, phaeochromocytoma, metastases, Cushing’s...
syndrome, primary aldosteronism etc. The incidental adrenal masses may also be infiltrative disease, fungal and tuberculous granulomas, hemorrhage and lesions that masquerade as adrenal but arise from adjacent organs (e.g. kidneys, pancreas, gall bladder, spleen, lymph nodes). The adrenal gland is also a common site for metastases from the breast, lung, renal, melanoma, lymphoma. Tuberculosis may affect many of the endocrine glands the commonest being the adrenal gland; acute tuberculosis adrenalitis producing mass-like enlargement of the gland. Ultrasonography and computed tomography do not always differentiate between adrenal and extra-adrenal masses and between malignancy and non-malignancy; surgical excision therefore seems to be desirable in such cases.

In our patient, CT thorax was requested for purpose of evaluating lung parenchymal abnormalities. The upper abdominal cuts in the CT helped us to pick up the incidental adrenal mass. Unfortunately, transcutaneous needle biopsy of adrenal mass proved to be inconclusive prompting us to subject the patient to exploratory laparatomy with excision of the mass in its entirety. Histological examination of the excised mass showed features suggesting tuberculosis. Considering the pleural fluid characteristics i.e. exudate with lymphocytic predominance and a high pleural fluid ADA levels, the diagnosis of tuberculous pleural effusion was substantiated. In retrospect, the increase in effusion size on anti-tuberculous treatment may be attributed to Immune reconstitution syndrome – a syndrome of paradoxical worsening known to occur during treatment with ATT. Our assumption is strengthened by the fact that the patient has completed treatment and has had no recurrence of the effusion on follow-up.

REFERENCES