

Exudative Pleural Effusion in a Young Male: Think Beyond Tuberculosis Also

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Abstract: Tuberculosis is a common diagnosis in patients presenting with fever and exudative pleural effusion. In our case the patient presented with right sided exudative pleural effusion without any systemic manifestation. He was started on antituberculosis treatment. He was not responding to treatment and later on diagnosed to have Systemic Lupus Erythematosus on further workup.

Keywords: Exudative Pleural Effusion, Tuberculosis, Systemic Lupus Erythematosus

INTRODUCTION

Systemic lupus erythematosus, often abbreviated as SLE or lupus, is a systemic autoimmune disease that can affect any part of the body [1]. Lung and pleura inflammation can cause pleural effusion, lupus pneumonitis, chronic diffuse interstitial lung disease, pulmonary hypertension, pulmonary emboli, pulmonary hemorrhage, and shrinking lung syndrome. Pleural effusion occurs in only 2.5-3% of patients. Rarely pleural effusion can be the first manifestation of SLE, seen in 1-2% of patients [2]. The case we present illustrates the importance of careful workup of exudative pleural effusion, as other investigations can provide important clue to the diagnosis.

CASE REPORT

A 24 years old male was admitted in our hospital with chief complaint of progressive dyspnoea, for last 7 days, dry cough and low grade fever since 15 days. There was no history of chest pain, palpitation, PND, dependent edema, oliguria or joint pain. On examination his vitals were stable and air entry was decreased on right lung base. Chest X-ray showed right sided pleural effusion. His blood investigations showed haemoglobin 10.8 gm/dl, WBC 7800/mm³, ESR 18mm. Pleural fluid analysis showed: appearance slightly turbid, colour pale yellow, glucose 96.6 mg/dl, protein 5.14 gm/dl, total cells 910/mm³, lymphocytes 85%, polymorphonuclear cells 15%, ADA 35 U/L (normal limit; upto 40 U/L). He was treated with four antitubercular treatment (ATT) (INH-300mg, Rifampicin 450mg, Ethambutol 800mg, Pyrazinamide 1.5gm, his body weight was 56Kg) with oral steroid (60mg/day). As he was improved within 1 week he was

discharged with four drugs ATT with oral steroid. He continued the ATT and steroid for 3 weeks after discharge, and thereafter continued ATT only. After 6 months patient again presented with similar episode of right sided pleural effusion. On the day of admission her routine blood examination was as follows: Hemoglobin: 10 gm%, RBC 4.9 million/cumm, WBC 7500 (N 60% L 26% E2% B 0% M 22%), platelet count lakhs/cumm, ESR: 40 mm/Hr, blood sugar 98mg%, Urea-34mg%, creatinine 0.6mg%, CRP: 9mg/dl (normal upto 5 mg/dl), serum lipid profile, T3, T4, TSH, liver function test, serum LDH were within normal limits. Urine: protein was trace, no RBC, no cast was detected. Sputum for AFB: negative, sputum culture was negative. Pleural fluid ADA 23U/L. Chest X-ray showed: bilateral massive pleural effusion. Ultrasonography of abdomen showed no abnormality. Routine ECG was normal.

Echocardiography showed, a mild pericardial effusion, no chamber enlargement, no evidence of pulmonary hypertension, valves were normal, no vegetation or clot was detected. Pleural fluid Gram stain and culture was negative for microorganism. Mantoux test was negative. ELISA for HIV 1 and 2 was negative. Serum antinuclear antibody (ANA) was positive with the titer value 2.643 (done by ELISA CUT OFF 0.615). In fluorescent microscopy the pattern of ANA was homogeneous. Anti ds DNA was strongly positive in a titer of 60 ng/dl (Normal: upto 4ng/dl).

After getting all the reports, he was put on oral prednisolone (1mg/Kg/day) 60 mg per day. Within 14 days he improved clinically and her chest X-ray

showed significant radiological improvement. Four weeks after institution of steroid his chest X-ray showed near complete resolution of pleural fluid. Now he is on oral prednisolone, discharged from the hospital, and under our follow-up.



Fig. 1: Image showing X-Ray chest with right sided pleural effusion

DISCUSSION

SLE is a chronic inflammatory autoimmune disorder that can affect any organ system. The predominant manifestations include arthralgia, photosensitivity, rash, pleuritis, photosensitivity, pleuritis, renal and central nervous system involvement. Pleuritis does occur in systemic lupus erythematosus, may be a significant cause of morbidity. In addition to primary pleuritis, secondary pleural complications, especially infections, may occur [7].

SLE is typically diagnosed by a combination of physical findings and clinical laboratory testing. decades ago, the diagnosis of lupus included the lupus erythematosus (LE) cell assay. Pleural effusion is common but very rarely is the initial manifestation of disease. There are very few reports of SLE diagnosed in a cytopathology laboratory [8].

Dubois and Tuffanelli in an analysis of 520 patients pleuritis occurred in 45% of patients, and a pleural effusion occurred in 30%. Pleurisy and pleural effusion were the initial manifestation in 3% and 1% of patients, respectively [3]. In one study, it was the presenting manifestation in 27% of patients with late-onset SLE [4].

Pleural effusion due to lupus pleuritis is typically an exudate and may be unilateral or bilateral. In most cases, the glucose is >60 mg/dL and the complement levels are frequently low [5,6].

CONCLUSION

This case highlights the importance of thorough work-up of exudative pleural effusion in young patient. Pleural effusion can be the sole presenting manifestation in about 5 percent of cases with SLE

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