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Polymicrobial Community Acquired Pneumonia with Occult Lower Lung Field Tuberculosis A Case Report & Review of the Subject

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SUMMARY

A middle aged diabetic male was admitted with a respiratory infection having right middle & lower zones pneumonic infiltrates on chest radiograph. He had no response to the broad spectrum antibiotics (non responding pneumonia) and therefore underwent bronchoscopy for bronchoalveolar lavage (BAL) which showed unusual polymicrobial community acquired pneumonia (*Staphylococcus aureus*, *Pseudomonas aeruginosa*, and *Aspergillus fumigatus*) along with smear positivity for Mycobacterium tuberculosis. The lower lung field involvement is unusual for mycobacterium tuberculosis which typically involves the upper lung zones. His pneumonic infection was treated with culture specific antibiotics and lower lung field TB was managed with conventional antituberculous regimen resulting in excellent response.

INTRODUCTION

Pulmonary tuberculosis can involve any lung region but most patients with reactivation or secondary tuberculosis have abnormalities on chest radiograph with typical involvement of the apical-posterior segments of the upper lobes (80 to 90 % of patients), followed in frequency by the superior segment of the lower lobes and the anterior segment of the upper lobes.[1] Lower lung field tuberculosis (LLFTB) refers to disease involvement below the hila (including the perihilar regions) on a frontal chest radiograph.[2] LLFTB is frequently misdiagnosed initially as viral or bacterial pneumonia, bronchiectasis, or carcinoma. The diagnosis and management requires the same principles as other forms of pulmonary tuberculosis.

CASE REPORT

A 57 years old gentleman was admitted via emergency department due to high grade continuous fever with rigors, loose watery stools, crampy abdominal pain and dry cough of five days duration. There was no history of chest pain, wheeze or haemoptysis and the systemic review was negative other than generalized weakness and gastrointestinal complaints. He was also suffering from diabetes mellitus with history of moderate glycemic control while using insulin. One year ago, he was hospitalized with pamphigus vulgaris and was treated with intravenous immunoglobulins, prednisolone and azathioprin, with a history of recent successful tapering of these immunosuppressive agents. He was a never smoker and an office worker in education department, married with four healthy children and had insignificant surgical, family and social history. He kept no pet animals or birds at home. On examination, he was cooperative, and oriented with a regular pulse of 110/min, blood pressure of 130/80 mmHg, temperature 101^{o}F, respirations of 25/min and oxygen saturation of 94%. Chest examination was consistent with bilateral equal air intensity and crackles on the right middle and lower part of the chest with increased vocal resonance. There were normal heart sounds, mild tenderness in the epigastric region, and normal musculoskeletal and central nervous system examinations. His investigations included CBC; Hb 11.1 g/dl, TLC 22.82/cmm, platelets 185/cmm and DLC showed 90% neutrophills and 10 % lymphocytes and INR of

1.0. Serum electrolytes, LFTs and RFTs were within normal limits. Viral serologies for hepatitis B, C and HIV were all negative. Chest radiograph (Fig. 1a,b) showed an inhomogeneous opacity in the right middle and lower zones, on the basis of which he was diagnosed as suffering from community acquired pneumonia (CAP) and was, started with intravenous antibiotics including ceftriaxone 1000 mg bd & levofloxacin 750 mg od. Besides treatment

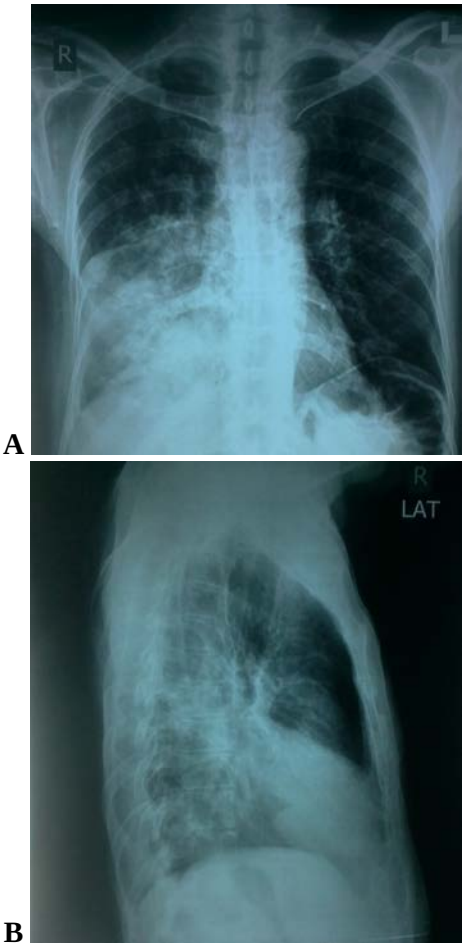

Fig. 1: CXR-PA (a) & lateral (b) projections showing right middle and lower zones patchy air space (pneumonic) shadows causing obliteration of right heart border & right hemidiaphragm.

with broad spectrum antibiotics, his fever and cough did not respond and the chest radiograph remained unchanged. Patient was not able to expectorate so bronchoscopy was done to obtain lower respiratory specimen for analysis and to rule out any obstructive lesion that could be responsible for post obstructive pneumonia. Airways were clear on either side of bronchial tree and bronchoalveolar lavage (BAL) was taken from right middle and lower lobe segments to evaluate the microbiological cause of non responding CAP. Gram staining of BAL fluid showed rare epithelial and numerous pus cells with numerous Gram negative bacilli, and BAL culture showed growth of both Staphylococcus Aureus and Pseudomonas Aeruginosa, fungal smear showed few budding yeast cells with pseudohyphae (aspergillus) and ZN stain was also positive (4 AFB per 100 fields). His antibiotics were changed (ceftazidime and linezolid) and continued for 2 weeks according to antibiogram sensitivity patterns and he was also put on fixed dose antituberculous regimen (rifampicin, isoniazid, ethambutol and pyrazinamide) continued for three months followed by six months of rifampicin, isoniazid and ethambutol. He responded well to treatment and the follow up chest radiographs showed remarkable improvement (Figs 2, 3).

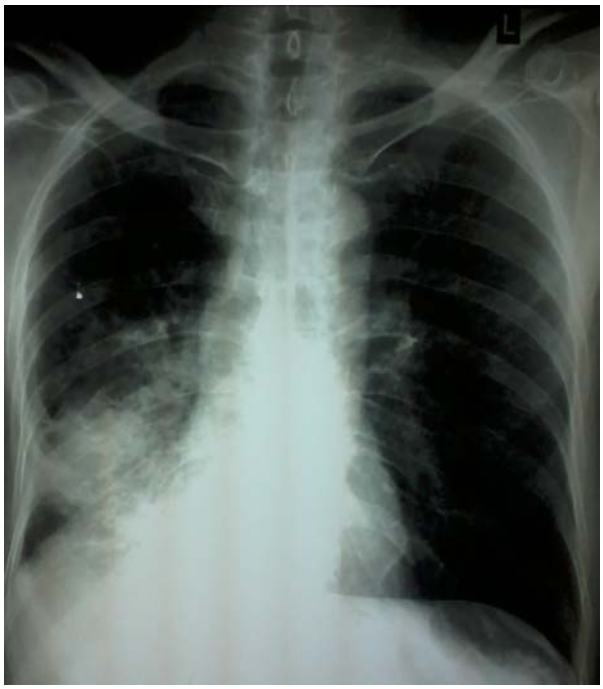

Fig. 2: Partial resolution of pneumonic infiltrates with loss of volume on right side of hemithorax, and incomplete visibility of right heart and diaphragmatic contours after 4 months of antituberculous treatment.

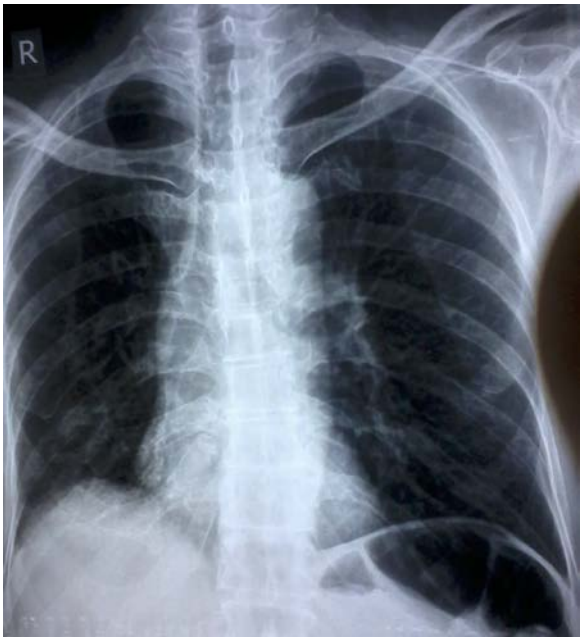

Fig. 3: CXR-PA at 9 months of treatment completion showing complete resolution of air space shadows and full visibility of right heart and diaphragmatic contours but with moderate volume loss on right side.

DISCUSSION

LLFTB is infrequent in the adult population (typical appearance of primary TB in children) and is generally associated with immunodeficiency.[3] The most likely explanation for the development of lower lung field tuberculosis is transbronchial perforation of a hilar lymph node, with spread to the adjacent lung.[4] There are some conditions which have been reported to occur more frequently in patients with lower lung field disease than in the general tuberculous population including advanced age, diabetes mellitus, pregnancy, silicosis, and kyphoscoliosis.[5,6,7] LLFTB can be present without associated comorbidities and must be suspected in pneumonias that have a torpid evolution regardless of pulmonary localization.[3] The radiographic findings in lower lung field tuberculosis differ significantly from those found in upper lobe disease and often resemble bacterial or viral pneumonia more than tuberculosis.[4] Right lung involvement is more common and consolidation in lower lung field disease tends to be more confluent and extensive than that found in upper lobe disease, and acinar shadows are less prominent.[4] Tuberculosis should be considered a diagnostic possibility in patients with "lower lung field pneumonia" who do not appear acutely ill or have had symptoms for more than several days and particularly several weeks before seeking medical aid.[4,5,8] The diagnosis of LLFTB is based on demonstration of mycobacterium tuberculosis on respiratory specimen (sputum or bronchial washings or lavage) staining or culture and the results of treatment with anti-tuberculosis drugs are similar to those in upper lobe tuberculosis.[3]

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