

Opaque Hemithorax. Primary Tuberculous Pneumonia Presenting with Acute Respiratory Failure A Case Report & Review of the Subject

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SUMMARY

A young previously healthy male was hospitalized in intensive care unit due to progressively worsening dyspnea (mMRC grade 4), cough with scanty sputum and fever. He exhibited signs of acute respiratory failure including tachypnea (respiratory rate 40/m), cyanosis and oximetric oxygen saturation (SpO₂) of 73 %. There was bronchial breathing on right hemithorax and coarse crackles on left side of chest auscultation. Chest radiograph was consistent with almost opaque right hemithorax with slight sparing of upper zone along with patchy air space shadowing involving the middle and lower zones of left lung. He received high flow oxygen, empirical broad spectrum antibiotics and had HRCT chest that showed large pneumonia involving almost complete right lung with air bronchograms and a small pneumonia on left side with ipsilateral pleural effusion (exudative lymphocytic). He was found to have primary tuberculous pneumonia because his sputum was positive for acid-fast bacilli (AFB ++) on Ziehl-Neelsen (ZN) staining and there was no bacterial growth on culture. His respiratory failure responded well to anti-tuberculosis treatment (ATT), broad spectrum antibiotics and corticosteroids and he was off oxygen after 10 days of intensive treatment.

Key words: Acute respiratory failure, air space consolidation, opaque hemithorax, sputum analysis, tuberculosis.

INTRODUCTION

Pulmonary tuberculosis (TB) is one of the treatable diseases rarely causing acute hypoxemic respiratory failure which is often fatal.¹ TB as a primary cause of acute respiratory failure (ARF) has an incidence of 1.5 % in patients hospitalized with pulmonary TB.² Patients with miliary or disseminated disease are especially prone to develop acute respiratory failure.^{3,4} Acute TB pneumonia presents as parenchymal consolidation with or without endobronchial spread mimicking bacterial pneumonia.² It probably represents an exudative hypersensitivity reaction to tuberculo-protein, rather than actual inflammation caused by the mycobacterial tuberculosis organism per se.¹ These infiltrates can appear within a matter of days and can clinically simulate acute bacterial

pneumonia.^{1, 2} ATT has been considered to be an important factor affecting clinical outcome.³ This case report describes a patient with tuberculosis who developed ARF and was successfully treated with early appropriate anti-tuberculosis therapy. The experience with this case serves to emphasize the importance of quality routine sputum examination for AFB in patients at risk of TB (particularly in endemic areas) and presenting with respiratory failure and pneumonic infiltrates, since specific and effective therapy for TB is available in contrast to most other conditions associated with respiratory failure.

CASE REPORT

A 35-year-old male, never smoker, running a pesticide shop with no previous co-morbid diseases

was hospitalized having cough with muco-purulent sputum, exertional dyspnea, right sided chest pain and low grade pyrexia with evening rise and night sweats of 3 weeks duration. There was no history of hemoptysis, wheezing or upper respiratory symptoms. His systemic review showed slight weight loss, occasional nausea and vomiting but no other complaints. He was given antibiotics (cefixime and co-amoxiclav for 7 days) by his family physician but there was no relief in his symptoms. In the past, he was never hospitalized for any medical or surgical ailment. He kept no pet animal or birds at home, denied any aerosol or chemical exposures other than his pesticides business that according to him had no aerosol production. His family history revealed that his wife was in good state of health, he had one 3 year old healthy son, his father was suffering from coronary heart disease and mother had chronic hepatitis C. There was no history of TB in the family or other close contacts.

On general physical examination, he was obviously dyspnoeic but was alert and cooperative, febrile (temperature 102°F), pulse rate of 124/m, BP 120/70 mmHg, respiratory rate 32/min with SpO₂ 73%. Chest examination was consistent with bronchial breathing, increased vocal resonance and impaired percussion note over right middle and lower part of the chest posteriorly and inspiratory crackles over left lower part of chest. Remaining general and systemic examination was unremarkable.

Laboratory evaluation showed hemoglobin of 10.5 g/dl, WBC count 11500/cmm and platelets 630000/cmm. His serum biochemical analysis included blood urea of 36 mg/dl, creatinine 0.9 mg/dl, sodium 132 mmol/L, potassium 4.2 mmol/L and chloride 104 mmol/L. Chest radiographic findings included almost opaque right hemithorax with patchy areas of opacification involving left middle and lower zones (Fig. 1). Arterial blood gas analysis revealed Ph 7.49, PO₂ 58 mmHg, PCO₂ 39 mmHg and HCO₃ 29 mmol/L. Anti-HIV IgM by ELISA was negative. Urine analysis showed microscopic hematuria. HRCT chest (Fig. 2) revealed a large air space consolidation with air bronchograms involving almost entire right hemithorax along with patchy air space shadowing

involving left upper and lower lobes with ipsilateral small pleurisy and small volume mediastinal lymphadenomegaly. Sputum analysis for ZN staining turned out to be positive for acid-fast bacilli (AFB++) while Gram stain and bacterial culture revealed no pathogen. He was given empirical broad spectrum antibiotics (piperacillin-tazobactam and moxifloxacin) on initial suspicion of bacterial pneumonia which were stopped after availability of sputum culture.

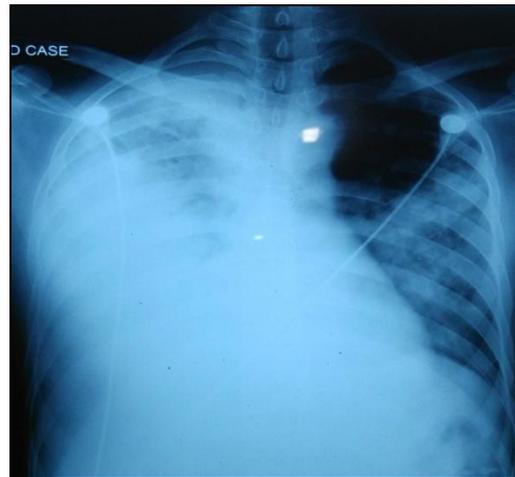


Fig. 1. CXR-AP view: Opaque right hemithorax with partial sparing of right upper zone and patchy air space shadowing involving left middle and lower zones.

In the light of his clinical presentation, radiological features of air space consolidation with air bronchograms, falling oxygen saturation and AFB positive sputum smear, he was finally diagnosed as having primary tuberculous pneumonia with acute respiratory failure. Combination ATT (rifampicin, isoniazid, pyrazinamide and ethambutol) was started according to his body weight along with high flow oxygen, and IV corticosteroids. After 10 days of his hospitalization and treatment, his oxygen therapy was stopped when he was able to maintain SpO₂ to 90% while breathing air. During course of his treatment, he developed ATT induced hepatitis with derangement of liver biochemical tests (ALT 113 U/L, AST 175 U/L, bilirubin 3 mg/dL & alkaline phosphatase 319 U/L). He was given modified, non-hepatotoxic anti tuberculous drugs including

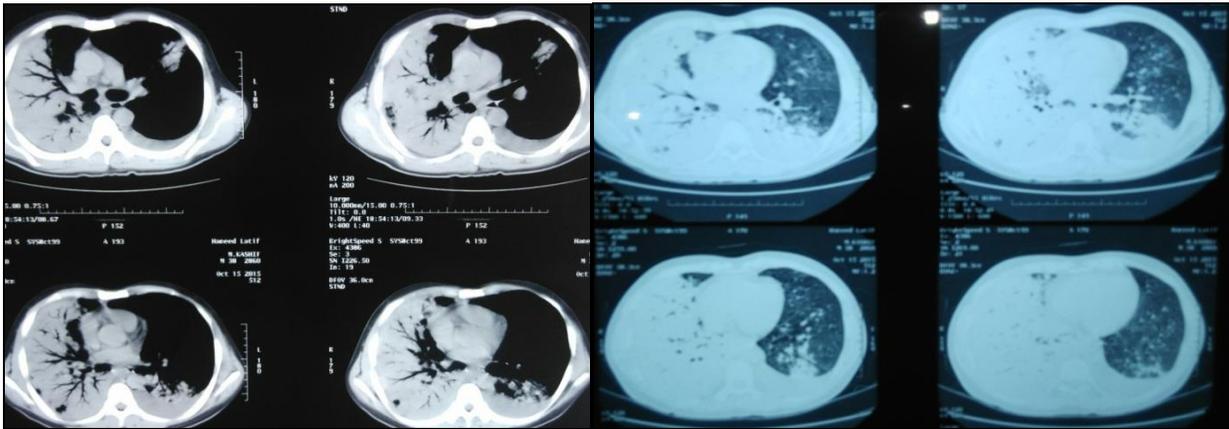


Fig. 2. HRCT chest showing large air space consolidation with air bronchograms involving almost entire right lung and patchy air space shadowing involving left upper and lower lobes. Small volume mediastinal adenopathy and small left sided pleural effusion is also seen.

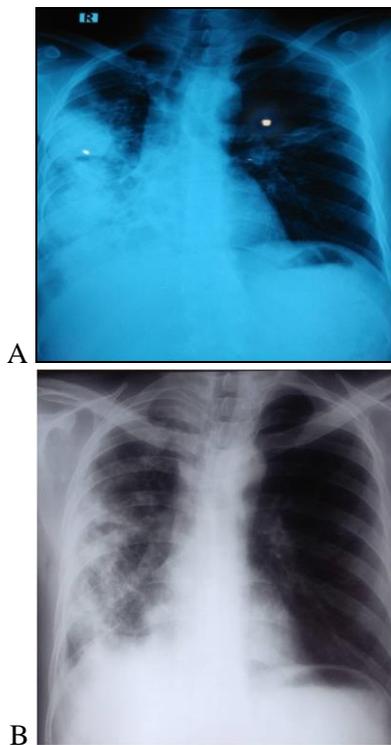


Fig. 3. Follow up chest radiograph (PA view) after 2 weeks (A) and 2 months (B) showed almost clear left lung field with residual linear infiltrate in left middle zone and considerable resolution of consolidation on right side.

enzymes, moxifloxacin & linezolid were stopped and rifampicin, INH and pyrazinamide were reintroduced one by one that were tolerated without any further rise in liver transaminases. His follow up chest radiograph (Fig. 3) after 2 weeks of discharge from hospital had considerable improvement on both sides. There was great improvement in his respiratory symptoms, he was afebrile and was mobilized and maintaining oxygen saturation around 90-94% on room air. He was advised to continue ATT, while tapering steroids over next 4 weeks and obtain regular follow up at monthly intervals.

DISCUSSION

Tuberculosis, a disease caused by the bacillus mycobacterium tuberculosis complex affects 9.4 million individuals and is responsible for the death of 1.8 million individuals across the world annually.⁵ The disease usually has a smoldering onset and progression, primarily due to the bacilli multiplying once every 18-24 hours, a duration much longer than that taken by most pathogenic bacteria.⁴ Patients usually present with the symptoms of cough, weight loss, anorexia, night sweats and malaise that is usually present for a few weeks before presentation.^{4, 5} However, in relatively rare circumstances the disease can present as an acute pneumonia with respiratory failure, masquerading as

ethambutol, streptomycin, moxifloxacin and linezolid. Later on with the improvement in liver

a community acquired pneumonia (CAP).^{5, 6} The incidence of TB being diagnosed among patients presenting with clinical and radiological signs of CAP has varied across series and can be as high as 35 percent of microbiologically confirmed pneumonias, the incidence being higher in the HIV-positive subgroup of patients.^{6, 7} The incidence of acute respiratory failure secondary to such an acute pneumonia has been reported to be close to 1.5 % among patients hospitalized with pulmonary TB.⁸ The acute presentation of TB can result from the primary infection, progressive primary disease, reactivation of latent TB, or atelectasis caused by the effects of compression or rupture of enlarged tuberculous lymph nodes.^{6, 7, 8}

TB is seldom thought of when the patient presents as an acute pneumonia with respiratory failure, and the results of this can have two implications. Firstly, the patient gets treated with antibiotics, a variety of which are known to have anti tuberculous effects, resulting in transient improvement of the clinical picture.^{8, 9} The most potent of the anti tuberculous antibiotics are fluoroquinolones, recommended in the guidelines as one of the first-line drug options in the management of hospitalized patients with community acquired pneumonia.⁹ The empiric use of antibiotics in the absence of an index of suspicion for TB results in delays in diagnosis and institution of anti tuberculous therapy, and such delays are known to cause increased morbidity and mortality, and increased risk of forward transmission.¹⁰ The second, and equally worrisome result of the use of fluoroquinolones in the management of tuberculosis misdiagnosed as community acquired pneumonia, is the acquisition of resistance to fluoroquinolones during the period that the bacillary load is exposed to mono therapy with the drug.¹⁰ A course of fluoroquinolones mono therapy for as short duration as 13 days has been found to select resistant mutants.¹¹ Our patient also received moxifloxacin in the start as empirical treatment of CAP but was stopped after diagnosis of TB was confirmed and ATT was started.

It is especially important to strongly suspect TB in the differential diagnosis of CAP in countries that are endemic for TB and among immigrants from such countries.¹² The treatment of pulmonary

tuberculosis does not change based on the severity of the initial presentation, and even the presence of respiratory failure does not warrant a change in the standard treatment of TB that comprises an intensive phase, consisting of two-months of rifampicin (10 mg/kg/day), isoniazid (5 mg/kg/day), ethambutol (15-20 mg/kg/day) and pyrazinamide (25-30 mg/kg/day) followed by a four-month course of rifampicin, isoniazid and ethambutol.¹³ The beneficial effect of corticosteroids in the presence of respiratory failure are not proven but can be considered to reduce the severe inflammation causing impairment in pulmonary gas exchange.^{12, 13} This case highlights the fact that pulmonary tuberculosis, though commonly a disease with a subacute vague presentation, can occasionally masquerade as an acute CAP with respiratory failure, and (particularly in endemic regions) should be entertained as a possible differential diagnosis in any patient with CAP. We treated this case empirically with antibiotics as CAP but in the start kept a high clinical suspicion for TB and therefore sent AFB smears (confirmed later with conventional TB culture consistent with pan drug sensitive TB) and obtained an earlier accurate diagnosis and therefore treated him precisely with ATT in combination with steroids.

CONCLUSION

Tuberculosis should be considered in the differential diagnosis of acute pneumonic infiltrate with respiratory failure as timely diagnosis and thus treatment besides carrying the potential to reduce morbidity and mortality can also prevent the misuse, acquired resistance and toxicity of unnecessary antibiotics.

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