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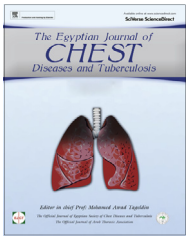

CASE REPORT

Pulmonary tuberculosis presenting with acute respiratory distress syndrome (ARDS): A case report and review of literature

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Abstract Tuberculosis is a very highly prevalent disease particularly in the developing world. In India one person dies of tuberculosis every minute. It can be a differential diagnosis of any disease ranging from infections to malignancies. But tuberculosis as a primary cause of respiratory failure requiring mechanical ventilation is an uncommon occurrence. Among patients with pulmonary tuberculosis, those with miliary or disseminated disease or having comorbidities like acquired immunodeficiency syndrome (AIDS) are especially prone to develop acute respiratory distress syndrome (ARDS). We present a case of a young female with no comorbidities or immuno suppression who presented with ARDS to us. We initially managed with mechanical ventilation and broad spectrum antibiotics, but there was no improvement. Only after anti tubercular therapy (ATT) and corticosteroids the patient recovered.

Introduction

In most countries, as ours, tuberculosis remains a major public health problem. One third of the world's population is estimated to be infected with *Mycobacterium tuberculosis*. A significant proportion of tuberculosis patients still has to be hospitalized, and in-hospital mortality remains high, with estimates ranging from 2% to 12% [1]. Some decades ago, respiratory failure resulting from tuberculosis was reported mainly in cases of miliary tuberculosis. In 1977, the first case series of

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respiratory failure in 16 patients with tuberculosis and fibrocavitary disease was described [2]. The reported frequency of acute respiratory failure in patients with active tuberculosis ranged from 1.5% to 5.0% [3,4], although pulmonary tuberculosis is rarely the primary cause of this complication. We report a case of a patient presenting with ARDS who subsequently turned out to be a case of pulmonary tuberculosis.

Case summary

Our case was a 32 year old female, normotensive, non-diabetic, presenting with a four week history of severe breathlessness, cough with expectoration with blood tinged sputum. Initially she had followed some private practitioner outside but there was no relief and symptoms got worsened.

On examination the patient was conscious, oriented, febrile to touch, cyanosed, tachypneic with respiratory rate of 34/min, pulse of 94b/min and temperature of 100F, BP was 140/82 mmHg. Oxygen saturation was 70% only. Systemic examination revealed bilateral course crepitations in the lungs. Rest of the examination was normal. Initial investigations reveal Hb of 10 g/dl, TLC of 12,000/uL, DLC of 84% neutrophils and 20% lymphocytes. Platelet count of 120,000. KFT of 40 IU urea and 1.2 mg/dl of creatinine. ABG/electrolytes showed pH of 7.34, po2 = 40 mmHg, pco2 = 45 mmHg, Hco3 = 22-meq/L, Na = 143, k = 3.5 depicting Type 1 respiratory failure. ECG showed sinus tachycardia. Po2/FiO2 = 198. Chest X-ray (Figs. 1 and 2) showed bilateral dense reticulo-nodular pattern interspersed with fine infiltrates. The patient was immediately managed with invasive mechanical ventilation and broad spectrum antibiotics but she did not improve. Influenza retroviral and other virological serologies were negative. Blood and urine culture came sterile. There was no evidence of fungal infection. Bedside echo was done which was normal. Tracheal aspiration showed strong positivity for *M. tuberculosis* but Monteux test came negative. Subsequently computed tomography (CT) scan (Fig. 3) showed typical miliary pattern of the lungs though there was no evidence of disseminated tuberculosis. So on clinical and radiological suspicion she was put on anti-tubercular therapy. Fortunately after 7 days of treatment the patient showed signs of improvement and she was extubated on 12th day of admission. In the meantime culture came positive for *M. tuberculosis* after 2 weeks of culture in Bactec. Hence she was diagnosed as a case of pulmonary tuberculosis presenting as acute respiratory distress syndrome (ARDS). She was discharged on 15th day. She is on our follow up and is doing well. Sputum for acid fast bacilli (AFB) is now negative and is symptomatically much better.

Discussion

Tuberculosis (TB) has been the scourge of civilization before recorded history, afflicting humans and domestic animals alike in all parts of the world. The multitude of names including "the white plague," "consumption," and "phthisis" that has been applied to TB attests to its protean manifestations. While the earliest classical descriptions of TB can be found in the writings of Hippocrates, it was the experiments of P.F.H. Klencke in 1843 and Jean Antoine Villemin in 1865 that elucidated the contagious nature of the disease. The identification of the tubercle bacillus by Kochin in 1882 allowed for the understanding of the pathogenesis of TB [2].

Tuberculosis is being increasingly recognized as a cause of acute respiratory distress syndrome (ARDS) [3–5]. Although exact figures as to what percentage of cases of ARDS are tubercular in aetiology are not available, in the Southeast Asia tuberculosis accounts for 3–16% of cases of community-acquired pneumonia. Malhotra et al. studied 185 cases of RICU admissions. Out of a total of 984, 18.8% admissions had ARDS over an 8-year period of which tuberculosis accounted for seven (3.8%) [6]. The pathogenesis of ARDS in patients with pulmonary tuberculosis has not been clearly elucidated. Postulated mechanisms include massive release of mycobacteria into the pulmonary circulation resulting in inflammation, obliterative endarteritis and damage of the alveolocapillary membrane [7]. Platelet aggregation in pulmonary capillaries causing endothelial injury and leucocyte activation resulting in increased vascular permeability are other hypotheses. In addition, lipoarabinomannan, a component of the mycobacterial cell wall, is thought to act in a manner similar to lipopolysaccharide in bacterial sepsis to activate macrophages to release tumour necrosis factor-α [TNF-α] and interleukin-1β [IL-1β] [8]. The activation of macrophages is thought to be a key step in the causation of lung injury [8]. It is yet to be determined, whether it is the individual host immunologic responses

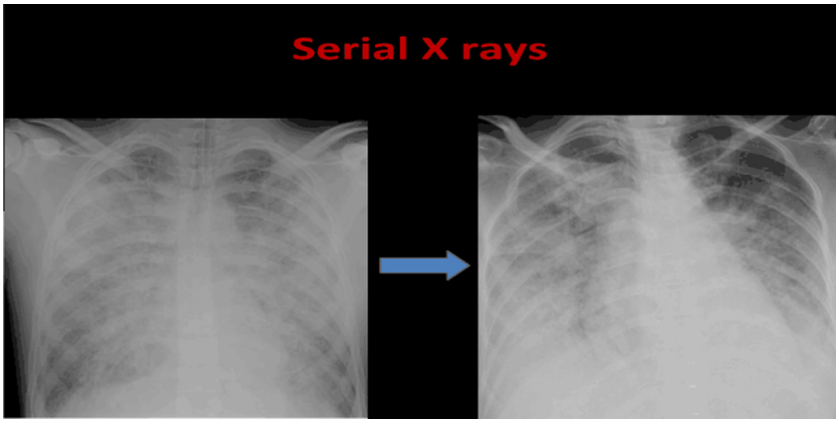

Figure 1 Chest X-ray showing bilateral infiltrates (ARDS picture).