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TUBERCULAR ARDS: A RARE PRESENTATION OF A COMMON DISEASE

Pulmonary Medicine

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ABSTRACT

ARDS is a common cause of morbidity and mortality in intensive care settings. Tuberculosis is the leading infectious killer and commonly causes respiratory failure in patients with extensive involvement of the lung parenchyma, however it is a rare cause of ARDS. But only a few studies have reported the association between ARDS and pulmonary tuberculosis in the past. Patients with tuberculosis developing ARDS have a significantly higher mortality when compared with ARDS of other infectious etiology. Thus clinicians should keep a high suspicion of TB in the setting of ARDS since the use of empiric anti tubercular treatment could potentially reduce mortality in these patients. We hereby report a case of Pulmonary tuberculosis diagnosed on microbiological basis presenting with pneumothorax, complicated by a rare manifestation ARDS. It should prompt the clinicians to have a high degree of suspicion to rule out tuberculosis in a patient with sepsis and to not merely limit themselves to a single diagnosis for optimal patient care.

KEYWORDS

PULMONARY TUBERCULOSIS, ARDS, GASTRIC ASPIRATE

INTRODUCTION:

ARDS is an important cause of morbidity and mortality in intensive care units. Pulmonary tuberculosis commonly causes respiratory failure in patients with extensive lung parenchymal involvement, but it is a rare cause of ARDS. [2]

Tuberculosis is one of the most common cause of death due to a single infectious agent worldwide. Mortality from ARDS secondary to TB is around 50%.[1]

Among patients with tuberculosis, those with miliary or disseminated disease or having comorbidities like acquired immunodeficiency syndrome (AIDS) are especially prone to develop ARDS. The reported frequency of acute respiratory failure in patients with active tuberculosis ranged from 1.5% to 5%, although pulmonary tuberculosis is rarely the primary cause of this complication.[3]

Case Report

A 55 yr old female with no known comorbidities presented to the emergency room in an unconscious state. BP was non recordable, carotid was non palpable. Hence, CPR was started immediately according to ACLS protocols. Return of spontaneous circulation was achieved after 3 cycles of CPR and patient was put on invasive mechanical ventilation. Patient's attendants gave history of shortness of breath since 3 months aggravated since 5 days, not associated with orthopnea. There is history of chest pain since 2 days along with history of fever since 3 days. There was history of TB contact with daughter 4 years ago. General physical examination revealed Tachycardia (PR-110BPM), hypotension (BP-NR) and no clubbing or lymphadenopathy. Ionotropic support and IV fluids were started.

Auscultation revealed diminished air entry on the right side along with expiratory wheeze and crepitations in left infra-axillary and infrascapular region. Chest X ray revealed right sided pneumothorax along with bilateral diffuse infiltrates. Intercostal drainage tube was inserted in the right 5th ICS.

Hematological investigations were suggestive of Anemia (Hb-8.2), leukocytosis (TLC- 41.2K N-93, L-3). LFT was within normal limits and RFT was suggestive of Urea-57 Creat- 1.48.

HRCT was done which was suggestive of Right sided pneumothorax with ICD insitu along with multiple patchy consolidation with bronchiectasis changes in bilateral lower lobes. 2D echo was done which was suggestive of no RWMA, EF-60%), Mild TR and Mild MR. Gastric aspirate was sent for AFB and CBNAAT and ET secretions were sent for gram stain and culture sensitivity. Patient was empirically started on IV antibiotics and nebulization. Serial Chest x ray showed increased infiltrates and PaO2/FiO2 ratio showed deterioration (230160125).

TLC trend- 41.2k29k21.2k24k

ET secretions for gram stain and culture along with blood and urine culture was sterile.. Gastric aspirate for CBNAAT was suggestive of Mtb detected, RR not detected. Patient was started on ATT under NTEP HRZE according to weight band. Sedation vacation was given to check for neurological parameters, however there was no response or motor movements. NCCT head was done and neurology call was done for NCCT head findings which revealed Global hypoxia, cerebral and cerebellar edema along with pseudo arachnoid hemorrhage.

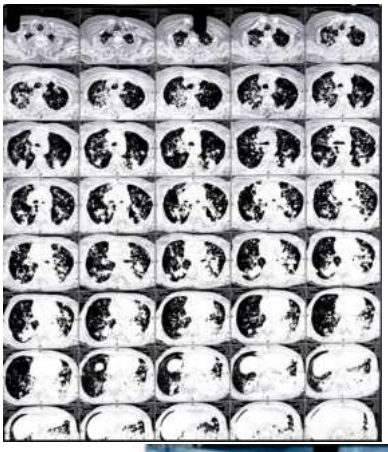
Patients repeat chest x ray showed slight improvement however patient's condition deteriorated and ionotropic support was later increased. Later patient succumbed to the condition.

DISCUSSION

Pulmonary tuberculosis causing ARDS in a patient is a rare manifestation which ranges from 2-5%, studies suggest that around 4% of admissions of ARDS is due to pulmonary tuberculosis. In view of the complete blood count findings which revealed leukocytosis with neutrophilia and lack of comorbidities, provisional diagnosis in our patient consisted of Bacterial pneumonia superimposed on viral pneumonia with ARDS and right sided pneumothorax. In view of acute clinical history, chest X ray findings and PaO2/FiO2 ratio on ABG (165), diagnosis of ARDS was made. She was started on empirical antibiotics. However, ET cultures in the patient came out to be negative. ET secretion for AFB was negative. Gastric aspirate was sent for AFB and CBNAAT in view of suspicion of TB due to history of TB contact with the daughter. Gastric aspirate for CBNAAT revealed MTB detected, Rifampicin resistance not detected. Patient was immediately started on Antitubercular drugs. The likely pathogenesis of ARDS in pulmonary tuberculosis patients is thought to be massive release of mycobacteria into pulmonary circulation resulting in inflammation, obliterative endarteritis and damage of the alveolo-capillary membrane as well as platelet aggregation in pulmonary capillaries causing endothelial injury and leucocyte activation resulting in increased vascular permeability is another hypothesis. In addition, lipoarabinomannan, a component of mycobacterial cell wall is thought to act in manner similar to lipopolysaccharide in bacterial

sepsis to activate macrophages to release tumor necrosis factor alpha and interleukin 1 beta. The activation of macrophages is thought to be the key step in causing lung injury.[3] The sensitivity of gastric aspirates for the diagnosis of pulmonary tuberculosis is 21.2% and 91.9% specificity for smear positivity and 55.8% sensitivity and 99.6% specificity for nucleic acid amplification test positivity. [4] Thus gastric aspirate is valuable diagnostic tool in pulmonary tuberculosis, especially for individuals who cannot produce sputum or have smear negative results. It can be used in conjunction with NAAT and microscopy to enhance the accuracy and speed of diagnosis.

As in our patient with sepsis presenting with leukocytosis and neutrophilia, one might miss the underlying tuberculosis. But in our case we further confirmed the microbiological diagnosis of tuberculosis. This emphasizes the importance of ruling out alternate diagnosis as a part of holistic approach.



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