

# A Rare Cause of Acute Respiratory Distress Syndrome

## Abstract

A 25-year-old lady presented with acute respiratory distress syndrome following dilatation and curettage. Due to non-responding fever she was investigated further. High resolution computed tomography showed 'random nodules' with consolidation suggestive of miliary tuberculosis with acute respiratory distress syndrome, which was confirmed later on trans bronchial lung biopsy. She responded to anti tuberculosis therapy and was discharged a febrile with normal vital parameters.

**Keywords:** Arterial blood gas; Intensive care unit; Utilization

## Case Report

Volume 6 Issue 2 - 2016

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**Received:** October 29, 2015 | **Published:** November 10, 2016

**Abbreviations:** ARDS: Acute Respiratory Distress Syndrome; HRCT: High Resolution Computed Tomography; TB : Tuberculosis; D & C: Dilatation and Curettage

## Introduction

Miliary tuberculosis is a potentially fatal form of tuberculosis, occurs due to haematogenous spread of *Mycobacterium tuberculosis*. Diagnosis is often delayed because of non-specific clinical manifestations. Cryptic miliary tuberculosis is even more difficult to diagnose because of normal chest radiograph. Rarely miliary TB can lead to acute respiratory distress syndrome. We report a case of cryptic miliary tuberculosis, which presented with acute respiratory distress syndrome following dilatation and curettage.

## Case Presentation

A 25-year-old woman, housewife, was referred from postoperative intensive care unit for respiratory distress following the D & C for threatened abortion. She gave history of intermittent low-grade fever of 5 weeks duration. Beside this there were no significant past history. Her vitals before shifting to intensive care unit from procedure room were: pulse rate-140/min, respiratory rate- 32/min, and blood pressure of 108/70 mm Hg and oxygen saturation of 75% at room air. Bilateral fine basal crackles were auscultated on respiratory system examination. Other system examination was normal.

Patient was investigated. Her arterial blood gas analysis at  $\text{FiO}_2$  of 21% (room air) revealed pH-7.459,  $\text{PCO}_2$ - 34.3 mm of Hg,  $\text{PO}_2$ - 33.5mm of Hg,  $\text{SpO}_2$ -67.8 and  $\text{HCO}_3^-$ -24.6 mEq/L, with  $\text{PO}_2/\text{FiO}_2$  ratio of 159.5mm of Hg suggestive of acute respiratory distress syndrome (ARDS). She was started empirically on injectable antibiotics (imipenem, gentamicin and levofloxacin) for suspected genitourinary infection and septicemia, steroids for ARDS, and non-invasive ventilation for acute respiratory failure with a working diagnosis of ARDS secondary to septicemia. There was no evidence of giving intravenous fluid during the procedure. Initial biochemical reports were normal. Her haemogram showed haemoglobin-9.4gm/dl, total leucocytes count of 6300/mm<sup>3</sup> with normal differential cell percentage. Enzyme link immuno

sorbent assay for human immunodeficiency virus was negative. Transvaginal sonography was normal. Widal test for enteric fever, peripheral smear for malaria parasite, dengue serology, blood culture and urine culture were negative. The histopathology of specimen obtained after D & C showed only deciduas without any evidence of infection. Echocardiography revealed trivial mitral regurgitation with normal ejection fraction. The patient was transferred to ward after 5 days with partial improvement. Fever during the ICU stay had reduced but on omitting levofloxacin, gentamicin and imipenem the fever spikes increased. HRCT thorax was advised for non-responding fever, showed bilateral ground glass opacity with patchy consolidation in both upper lobes and dense consolidation in both lower lobes. It also showed presence of 'random nodules' in both lung fields with bilateral pleural effusion. The patient was started on anti-tuberculosis treatment based on HRCT finding. Transbronchial lung biopsy was performed after partial clinical improvement, which showed epitheloid cell granuloma with necrosis on histopathology. Fever responded and she was discharged with normal vital parameters. One month after the discharge chest radiograph and HRCT were repeated.

## Discussion

Acute respiratory distress syndrome describes a condition characterized by the acute onset of bilateral infiltrates on chest radiograph, hypoxaemia (defined as a  $\text{PaO}_2/\text{FiO}_2$  ratio of <200 mmHg) and no evidence of left atrial hypertension [1,2]. As per the new definition ARDS is categorized into mild, moderate and severe ARDS. Our case had findings consistent with moderate ARDS i.e.  $\text{PaO}_2/\text{FiO}_2$  between 100-200 mmHg [3]. Miliary tuberculosis is a rare cause of ARDS [3]. However, the mortality is very high ranges from 33% to as high as 100% [4], which is far higher than for ARDS from other causes. Due to low causal association, the diagnosis of miliary tuberculosis as the cause of ARDS is delayed or often missed.

Early diagnosis and timely treatment due to characteristic HRCT abnormality saved our patient. The HRCT showed areas of consolidation with “random nodules”. “Random nodules” are small nodules seen on HRCT distributed haphazardly throughout the lungs along the pleura and fissures, at the ends of small arteries, and also in a centrilobular location [5]. These nodules on HRCT are seen in miliary tuberculosis, fungal infection, metastasis and Langerhans cell histiocytosis. Possibility of fungal infection was low because she was immuno competent. Metastasis was also unlikely because of young age and absence of primary malignancy. Langerhans cell histiocytosis was also unlikely because of fever and relatively acute onset of disease. Hence, on clinico-radiological correlation the most plausible diagnosis was miliary tuberculosis presenting with ARDS. The diagnosis was further proved on lung biopsy.

The possible mechanism of ARDS is that tuberculosis causes accumulation of inflammatory cells in the alveolar spaces leading to release of granular enzymes and oxidants resulting in damage to the alveolar basement membrane. Increase in cellular permeability due to alveolar basement damage aggravates oxygen dysfunction and consequently causes ARDS [6].

The predisposing factors for development of ARDS in a case of miliary TB are prolonged illness, absolute lymphocytopenia, elevated alanine transferase (ALT), aspartate transaminase (AST), diabetes mellitus, D-dimer, low haemoglobin, and albumin [7,8]. None of the studies have shown surgery as a risk factor for development of ARDS. However surgery is known to disseminate unrecognized tuberculosis [9]. Thus, D & C possibly led to the development of ARDS in our case with existing ‘cryptic miliary TB’, which was missed during preoperative evaluation. Hence, D & C can be added as a risk factor for development of ARDS due to miliary TB.

Initial mortality due to ARDS in our patient was averted possibly because of anti micro bacterial action of gentamicin [10], levofloxacin and imipenem [11]. Partial response of ARDS and miliary TB can only be explained by anti micro bacterial action of antibiotics, as she had no evidence of pyogenic infection. Steroid also had possibly helped our patient because promising results regarding the efficacy of steroids as a treatment modality for ARDS caused by miliary tuberculosis has been reported [12].

## Conclusion

Finally preoperative evaluation is indicated prior to any

surgery. Presence of ‘random nodule’ is an important clue to the diagnosis of miliary TB in a case of ARDS. Early diagnosis and prompt treatment of ARDS due to miliary TB averts mortality.

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