Case Report

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Internal Medicine Section

Bilateral Pleural Effusion in Advanced Acute Silicosis Presenting with Acute Hypercapnic Respiratory Failure: A Rare Clinical Presentation

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ABSTRACT

Silicosis is a preventable occupational lung disease caused by inhalation of crystalline silica and remains a significant cause of morbidity among young working populations in developing countries. It is characterised by progressive pulmonary fibrosis and irreversible respiratory impairment. While parenchymal lung involvement is well recognised, pleural manifestations are distinctly uncommon, and pleural effusion is rarely reported. The current case describes a 30-year-old male with a history of sandblasting exposure who presented with progressive dyspnoea and acute hypoxaemic respiratory failure. High-resolution computed tomography of the thorax demonstrated reticulonodular opacities with progressive massive fibrosis and bilateral pleural effusion, consistent with advanced silicosis. Diagnostic thoracentesis revealed a haemorrhagic exudative pleural effusion with low adenosine deaminase levels, normal glucose, and absence of malignant cells or granulomas. Microbiological studies were negative, and cardiac, hepatic, renal, and systemic causes of pleural effusion were excluded. Owing to severe respiratory failure, invasive procedures such as thoracoscopy or bronchoscopy could not be performed. The patient was treated with oxygen therapy, broad-spectrum antibiotics, systemic corticosteroids, and bronchodilators. Despite clinical stabilisation, persistent hypoxaemia necessitated Long-Term Oxygen Therapy (LTOT) and consideration for lung transplantation. After exclusion of common aetiologies, the pleural effusion was attributed to silica-induced pleural inflammation. This case highlights an unusual and under-recognised pleural manifestation of silicosis. In patients with relevant occupational exposure and unexplained exudative pleural effusion, silica-related pleural disease should be considered after excluding tuberculosis and malignancy. Early recognition is essential for appropriate management and prognostic assessment.

Keywords: Occupational lung disease, Pleural effusion, Progressive massive fibrosis, Silicosis

CASE REPORT

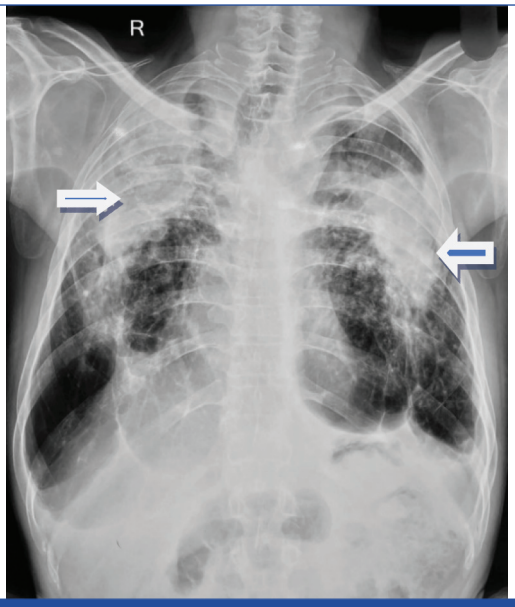
A 30-year-old male was admitted to the respiratory medicine department with chief complaints of shortness of breath, which was progressive from Modified Medical Research Council (mMRC) grade 2 to mMRC grade 4 over the last one month and dry cough over the last 20 days. He had no history of fever or chest pain. He was not a known case of hypertension, diabetes or any other chronic illness. One year back, he had constitutional symptoms like weight loss (dropped from 48 kg to 41 kg, nearly 7 kg), evening rise of temperature for one month, along with occupational exposure to silica dust. However, due to strong clinicoradiological suspicion but absence of any microbiological evidence, he had received antitubercular therapy (2HRZE+4HRE) for six months. The patients clinically improved after initiation of Anti-Tubercular Therapy (ATT). He was a non-smoker and had no other significant addiction history. He did not have any contact history of pulmonary tuberculosis nor any family history suggestive of malignancy. He was previously working in a sandblasting factory as a labourer for two years and left the occupation since last one year.

On examination, he was afebrile but had tachypnoea with a respiratory rate of 38/min and oxygen saturation of 77% in room air. There was no pallor, icterus, clubbing, cyanosis, lymphadenopathy, and Jugular Venous Pressure (JVP) was not elevated. On auscultation, late inspiratory fine crepitations were heard over the bilateral infraclavicular, mammary, upper and lower inter-scapular area along with diminished intensity over the bilateral infrascapular area. On investigation, routine complete blood count showed neutrophilic leucocytosis (Total leucocyte count- 18600/µL, neutrophil-92%). His renal function test, liver function test and serum electrolytes were within normal limits. Arterial Blood Gas analysis (with 6 litres oxygen) shows respiratory acidosis[Table/Fig-1]. Sputum induction was performed using hypertonic saline nebulisation. The sample was sent for Acid-Fast Bacilli (AFB) examination, which was negative, and CBNAAT testing did not detect Mycobacterium tuberculosis. Sputum culture showed no growth of any pathogenic organisms. Chest X-ray shows bilateral progressive massive fibrosis [Table/Fig-2]. Ultrasonography (USG) abdomen and pelvis suggestive of normal study. Cardiological evaluation and 2D-echo findings were within normal limits.

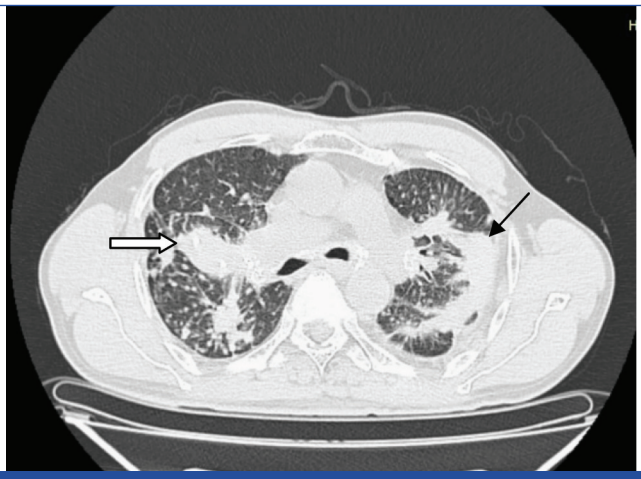
High-Resolution Computed Tomography (HRCT) Thorax demonstrated the presence of bilateral pleural effusion (left >right) with left-sided pleural thickening, bilateral septal thickening, and bilateral reticulonodular opacities and progressive massive fibrosis in bilateral upper lobes [Table/Fig-3,4]. USG guided pleural fluid aspiration and pleural analysis was done. Cytological examination shows few lymphocytes over a haemorrhagic background. Biochemical analysis values are shown in [Table/Fig-5]. Pleural fluid CBNAAT for Mycobacterium tuberculosis was negative and no growth was seen on culture. Pleural fluid cell block was negative for

Parameters	Observed value	Reference range
pH (–)	7.245	7.35-7.45
pCO_2 (mmHg)	106.3	35-45
pO_2 (mmHg)	126.2	75-100
HCO_3- (mmol/L)	46.1	22-28
SO_2 (%)	97.7	94-100

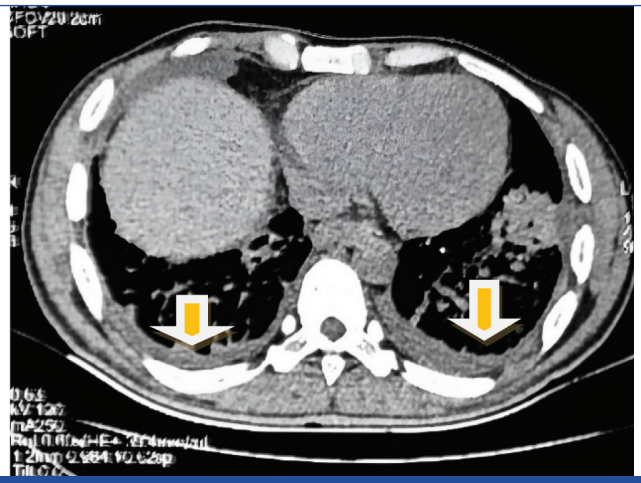
[Table/Fig-1]: ABG of patient showing respiratory acidosis.



[Table/Fig-2]: Chest radiograph in PA view reveals bilateral progressive massive fibrosis.



[Table/Fig-3]: HRCT thorax-lung window (axial section) showing reticulonodular opacities with bilateral progressive massive fibrosis.



[Table/Fig-4]: HRCT thorax- mediastinal window showing bilateral pleural effusion.

malignant cells or granuloma. No growth of pathogenic organisms was found on blood and urine culture.

Patient was managed conservatively with oxygen therapy, antibiotics (inj. meropenem 1 g i.v. TDS for 7 days and inj. teicoplanin 400 mg i.v. OD for 7 days), nebulised bronchodilators (levosalbutamol + ipratropium bromide 8 hourly) and steroids (inj. methylprednisolone 40 mg i.v. BD for five days). However, on serial ABG analysis, his PaO2 was less than 55 mmHg, but SpO_2 was maintained at 92% with 0.5 L/minute oxygen with a nasal cannula. He was prescribed metred dose inhaler (Formoterol (6 µg) + budesonide (200 µg), 2 puffs BD for 1 month, and tiotropium (9 µg), 2 puffs OD for 1 month with a spacer), advised strict exposure prevention, vaccination and pulmonary rehabilitation, lung transplantation was recommended, and he was discharged with LTOT. He was followed after first 15 days of discharge, evaluation was done and LTOT was continued. Then he was regularly followed at every two-month interval.

Parameters	Values	Reference range
Glucose (mg/dL)	93	>60
Protein (g/dL)	2.7	1.0 to 2
LDH (U/L)	2000	140-200
ADA (U/L)	18	0 to 24
Albumin (g/dL)	1.8	1.0 to 4.5

[Table/Fig-5]: Biochemical analysis of pleural fluid.
LDH: Lactate dehydrogenase; ADA: Adenosine deaminase

DISCUSSION

Silicosis is one of the most common occupational fibrotic lung disorders that affect mostly the young working class population of society. Environmental settings posing the greatest occupational risk include the following: mining and processing of stone; mining of gold and precious stones; well drilling, sandblasting; ceramic and glass production; and iron smelting [1,2]. Pleural effusion associated with silicosis is an extremely unusual presentation. Given the rarity of the manifestation, there is limited data on the chemical nature of the fluid [3]. As in this case, the patient had an occupational history of sandblasting for two years. HRCT Thorax was done which showed reticulonodular opacities, progressive massive fibrosis along with bilateral pleural effusion.

On cytological examination of pleural fluid, few lymphocytes over a haemorrhagic background were seen. On biochemical analysis, the fluid was exudative with low ADA and normal sugar levels. Pleural fluid cell block was negative for any malignancy or granuloma. USG abdomen and pelvis ruled out hepatopathy, nephropathy or other abnormalities. Cardiological evaluations were also within normal limits. Other potential causes of pleural effusions were ruled out. The patient presented with acute respiratory failure, therefore invasive procedures like medical thoracoscopy or bronchoscopy could not be performed. It is hypothesised that the pleural effusion was a result of possible inflammatory response of the pleura to crystalline silica particles.

Comparable findings have been reported by Salih M et al., and Zeren EH et al., in whom patients with long-term silica exposure developed pleural effusions and pleural thickening in association with pulmonary nodules or fibrotic lung disease after exclusion of infectious and malignant causes [3,4]. Similar pleural manifestations have also been described by Pairon JC et al., who documented diffuse pleural thickening and pleural effusions related to mineral dust exposure, occasionally occurring in the absence of advanced parenchymal involvement [5]. Zhu X et al., who reported pleural effusion as the primary manifestation of silicosis in a long-term quarry worker, with diagnosis established only after systematic exclusion of tuberculosis and malignancy [6]. Notably, Zeren EH et al., demonstrated silica particles directly within pleural biopsy specimens even without significant lung parenchymal disease, supporting the concept of primary pleural involvement by silica [4]. These observations highlight the diagnostic difficulty in distinguishing pleural silicosis from malignancy or tuberculous pleuritis, particularly in tuberculosis-endemic regions.

Taken together, the clinico-radiological and pleural fluid characteristics observed in our patient closely mirror those described by Salih M et al., and Zeren EH et al., reinforcing the hypothesis that pleural effusion in silicosis results from pleural inflammation, lymphatic obstruction, or immune-mediated injury induced by crystalline silica, particularly in advanced or progressive disease [3,4]. These

integrated findings emphasise that silica-related pleural disease, although rare, should be actively considered in exposed individuals presenting with unexplained exudative pleural effusion.

CONCLUSION(S)

This case highlights that silicosis can rarely present with clinically significant pleural effusion, even in young individuals with relatively short but intense occupational exposure to silica. The presence of bilateral exudative, haemorrhagic pleural effusion with low ADA and absence of malignancy or infection emphasises that pleural involvement may represent a direct manifestation of silica-induced pleural inflammation rather than a secondary pathology. The case further demonstrates that advanced fibrotic disease can progress rapidly and lead to refractory hypoxaemia requiring LTOT and consideration for lung transplantation. Meticulous occupational history-taking and early recognition of silica exposure are therefore essential for timely diagnosis, prevention of further exposure, and appropriate prognostic assessment.

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