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CASE REPORT – OPEN ACCESS

Rare Complications of Silica Dust Exposure

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ABSTRACT

Crystalline silica inhalation causes silicosis, one of the ancient occupational lung diseases. It leads to an irreversible fibrotic response in the lung parenchyma and, consequently, causes diffuse interstitial lung disease. Asymptomatic to chronic irreversible forms are various presentations of silicosis, which has a high-risk predisposition to various comorbidities. We documented two cases of rare presentations of silica dust exposure alveolar silicoproteinosis and silicotuberculosis.

Keywords: Silicosis, alveolar silicoproteinosis, silicotuberculosis, silica dust

INTRODUCTION

Silicosis is a diffuse irreversible interstitial lung disease caused by continuous inhalation of crystalline silica (SiO_2) leading to lung fibrosis (1). Because of the absence of a definitive silicosis treatment, prevention of the disease is the main concern. To prevent the disease, regular close monitoring of the workplaces and routine screening of the workers are required, in addition to the establishment of effective workplace regulations (2). In this paper, we report two cases of rare complications of silica dust exposure.

CASE REPORTS

Case 1

A 34-year-old male, who used to work in glass grinding, complained of exertional dyspnea and dry cough for 1 year. Three months before, dyspnea worsened to occur with mild exertion, and the patient developed cyanosis. On examination, he was cyanotic with oxygen saturation 84% on room air. A high-resolution computed tomography (HRCT) chest scan showed diffuse ground-glass opacification, septal thickening, and areas of a "crazy-paving" pattern (Fig. 1a). Bronchoalveolar lavage (BAL) from the right lower lobe was milky white (Fig. 1b), and transbronchial lung biopsies (TBLB) form the right lower lobe revealed intra-alveolar amorphous proteinaceous material associated with hyperplastic alveolar macrophages and interstitial inflammation (Fig. 1c). The intra-alveolar material was periodic acid Schiff (PAS) stain-positive (Fig. 1d), confirming the diagnosis of alveolar proteinosis. The whole hole-lung lavage was performed for both lungs sequentially.

The more severely affected lung, as detected by a CT scan, was lavaged first, and the other after 24–48 hr. Accordingly, the patient was intubated with a double-lumen endotracheal tube, and after 15 min of ventilation with 100% O_2, one lung was lavaged with sterile isotonic saline at 37°C. The volume used for each filling was 1000 ml, and then the lung is left to drain by gravity. The filling and drainage was repeated till the effluent is clear, and 10 L of saline was required for each lung.

Thereafter, the patient's symptoms and oxygen saturation had improved, and his oxygen saturation was 93%.

Case 2

A 31-year-old male, who used to work in sandblasting, ex-smoker, complained of exertional dyspnea for 1 year. One month before, dyspnea worsened to occur with mild exertion, and the patient developed night fever and night sweats with loss of weight and appetite, and productive yellowish sputum. On examination, he was cachectic and run a fever 38°C, with bilateral infraclavicular crepitations. A chest CT scan showed diffuse ground-glass opacification, bilateral apical reticulations, and perilymphatic nodules (Fig. 2a). His laboratory findings were normal, apart from a high ESR (1st hour=100), and his tuberculin skin test was 12 mm. BAL from the right upper lobe revealed a positive Ziehl–Nelseen stain confirming tuberculosis (TB), and the TBLB from the posterior segment of the right upper lobe and the posterior basal segment of the right lower lobe for histopathological examination revealed silica crystals due to silica dust exposure (Fig. 2b). Anti-TB treatment with isoniazid, rifampicin, pyrazi-

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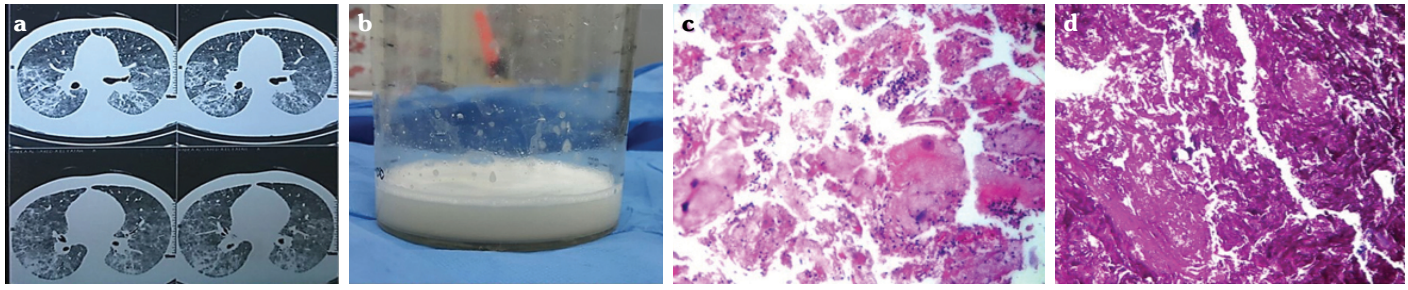

Figure 1. a–d. (a) A high-resolution computed tomography chest scan shows diffuse ground-glass opacities, septal thickening, and the areas of "crazy paving." (b) Milky white bronchoalveolar lavage from the right lower lobe. (c) Transbronchial lung biopsy from the right lower lobe reveals intra-alveolar amorphous proteinaceous material deposition associated with hyperplastic alveolar macrophages and interstitial inflammation (hematoxylin and eosin stain). (d) Positive periodic acid-Schiff stain

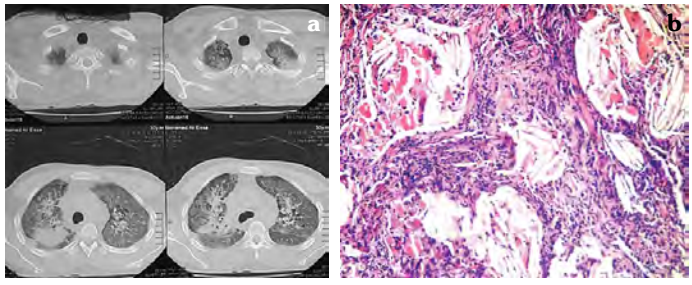

Figure 2. a, b. (a) A chest computed tomography scan shows diffuse ground-glass opacification and bilateral apical reticulations and perilymphatic nodulation. (b) Transbronchial lung biopsy from the posterior segment of the right upper lobe and posterior basal segment of the right lower lobe reveals silica crystals

namide, and ethambutol was initiated, and the patient continued therapy for 6 months.

DISCUSSION

Silicosis is an incurable occupational interstitial lung disease caused by free crystalline silica inhalation and its accumulation in the lung interstitium. Variable forms of the disease can be identified, but mainly three can be identified: chronic/classic, accelerated, and acute. Alveolar silicoproteinosis is one of the acute complications of silica dust exposure with subsequent alveolar filling with proteinaceous material (3). Massive inhalation of silica dust leads to shortness of breath, productive cough, chest pain and loss of weight, fever, and fatigue (4).

The standard treatment for alveolar silicoproteinosis is whole lung lavage, which removes large amounts of silica dust and inflammatory cells. Thereafter, it relieves pulmonary symptoms and improves oxygenation (5).

Exposure to silica, even without initiation of silicosis disease is associated with a high-risk predisposition of TB, which has been reported as 1.9% per year. A reduction in silica dust exposure, continuous medical surveillance, and TB screening in high-risk occupations and securing anti-TB treatment are strategies to minimize the rate of silicotuberculosis among workers or employees (6).

CONCLUSION

In the current cases, we have confirmed a rare diagnosis of acute silicoproteinosis and silicotuberculosis. Given the patients' occupational history of glass grinding and sandblasting and known high-risk exposure to respirable silica dust in their professions, we have considered a possible occupational etiology. Finally, this was confirmed by clinical, cytological, and histopathological findings.

Informed Consent: Written informed consent was obtained from patients who participated in this study.

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Conflict of Interest: The authors have no conflict of interest to declare.

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