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Case report

Microscopic polyangiitis associated with pleuropericarditis, pulmonary embolism and pulmonary hemorrhage as a complication of silicosis

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ABSTRACT

Silica (silicon dioxide) occupational exposure has been linked to both pulmonary and extra-pulmonary toxicity. Silicosis is the major pulmonary toxicity, which has also been associated with the development of collagen-vascular disease and with anti-neutrophil cytoplasmic antibody (ANCA)-positive vasculitis, especially perinuclear anti-neutrophil cytoplasmic antibodies (P-ANCA). The most common pulmonary manifestations of microscopic polyangitis (MPA) are interstitial fibrosis and alveolar hemorrhage. We describe a patient who had unusual presentation of microscopic polyangitis, characterized by lung hemorrhage, rapidly progressive glomerulonephritis, pleuropericarditis and pulmonary embolism that was associated with a history of silica exposure and radiologic evidence for silicosis.

1. Introduction

Occupational exposures to silica dust has been linked to both pulmonary and extra-pulmonary toxicity [1–6]. The major pulmonary toxicity is silicosis. Silicosis has also been associated with the development of collagen-vascular disease and with anti-neutrophil cytoplasmic antibody (ANCA)-positive vasculitis, especially anti-myeloperoxidase (MPO) antibody [1,7]. The most common pulmonary manifestations of microscopic polyangiitis (MPA) are interstitial fibrosis and alveolar hemorrhage [8]. We describe a patient who had microscopic polyangiitis, characterized by lung hemorrhage, pulmonary embolism and pleuropericarditis that was associated with a history of silica exposure and radiologic evidence for silicosis.

2. Case report

A 68-year-old man reported a 2-week history of dry cough with a central chest discomfort radiating to the epigastrium. There was no associated hemoptysis, breathlessness or palpitations. He was seen in another institution 11 days prior to admission where he was found to have atrial fibrillation. A CT scan of the chest with contrast revealed small filling defects at the sub segmental level in both lower lobes (Fig. 1) diagnostic of pulmonary embolism. No alveolar filling was seen. The patient was treated with IV heparin and amiodarone.He was discharged on oral anticoagulation (Rivaroxaban). However, cough persisted and he developed daily scanty hemoptysis with progressive breathlessness, for which he sought attention at our institution.

On initial evaluation there was no fever or hemodynamic instability. There was no clinical evidence of congestive heart failure. Oxygen requirements were high (90% saturation; FiO2 1.0) and he was admitted to the intensive care unit for further evaluation and monitoring.

Review of his occupational history revealed a 10-year history of sandblasting without respiratory protection while working in a automobile body shop between 1994 and 2004.

Laboratory tests showed a microcytic hypochromic anemia (Hgb 9 g/l, MCV 78.5 fl), INR 1.82, PTT 42.8, creatinine 150 μmol/L (Normal: 55–110 μmol/L) and positive anti-myeloperoxidase (MPO) antibodies (>200 RU/ml). Urine analysis showed: 60-80 RBC/high-powered field, several granular casts, with a few RBC and fatty casts. Multiple alveolar opacities were noted in both lungs on chest radiography (Fig. 2.1).

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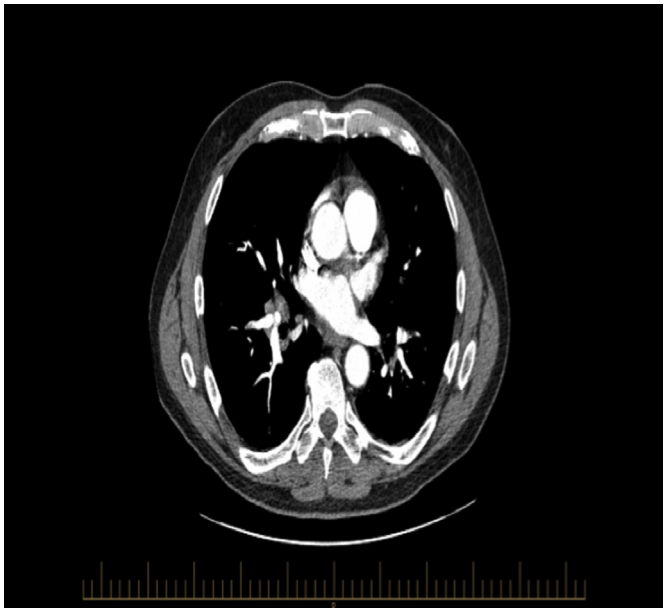

Fig. 1. CT chest with contrast showing small filling defects at sub segmental left lower lobe and right lower lobe.

A CT scan of the chest showed diffuse heterogeneous opacification of the lungs centrally with sparing of the lung periphery consistent with a diagnosis of diffuse alveolar hemorrhage. There were numerous small sub pleural nodules as well as mediastinal and hilar adenopathy, consistent with a diagnosis of silicosis (Fig. 3).

There were small bilateral pleural effusions and a moderate pericardial effusion with contrast enhancement (Fig. 4).

Kidney ultrasound was normal. Echocardiography revealed a moderate pericardial effusion with normal global and regional LV systolic function. A renal biopsy revealed changes compatible with active pauci-immue glomerulonephritis with focal and segmental fibrinoid necrosis associated with small cellular crescents (Fig. 5).

Treatment was initiated with methyl prednisone 1 g IV daily for two days that was followed by oral prednisone 80 mg daily.

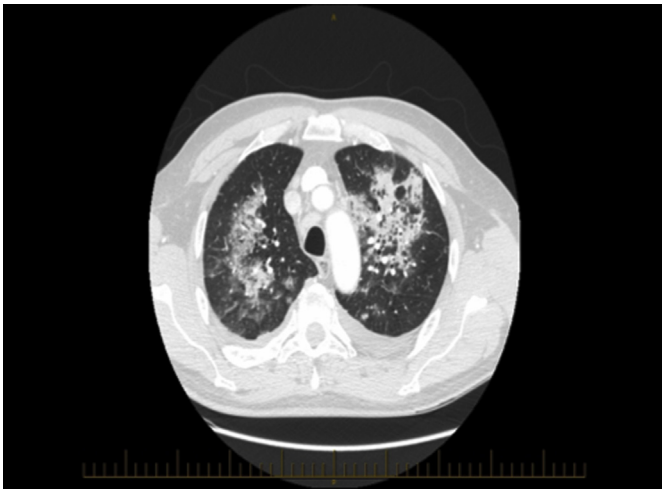

Fig. 3. Heterogeneous opacification of the lungs centrally with sparing the lung periphery diagnostic of the lung hemorrhage, with small sub pleural nodular opacities in the upper lobes diagnostic of silicosis.

Cyclophosphamide 1540 mg intravenously was given as a single dose in addition to Mesna.

There was dramatic and rapid improvement with reduction in oxygen requirements and significant radiographic clearing (Fig. 2.2) over the next 4 days. The patient was transfused packed red blood cells. The acute kidney injury persisted with elevated creatinine. One month following discharge there was resolution of the airspace lung opacities.

3. Discussion

Silica (silicon dioxide) exposure is the cause of silicosis. Occupations at risk include miners, foundry workers, sandblasters, as well as workers in the ceramic and glass manufacturing industries [1,4]. There are a number of recognized complications of silica exposure including silicosis, progressive massive fibrosis, chronic obstructive pulmonary disease, and increased risk of TB infection and lung cancer [4,9]. Systemic complications that have been linked to silica exposure with silicosis include: rheumatoid arthritis,

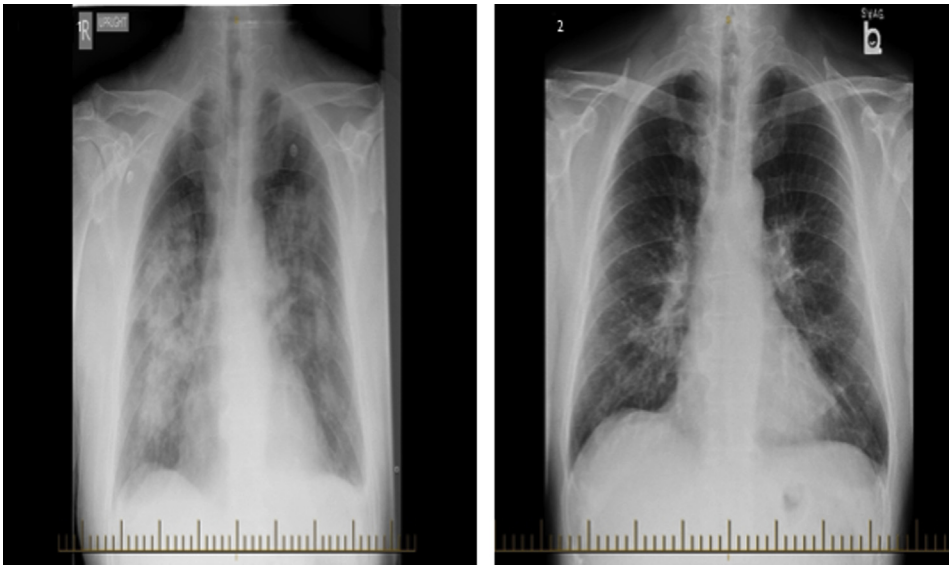

Fig. 2. 1 and 2. CXR before and after treatment.