CASE REPORT

Silico-Asbestosis that Responded to Steroid Therapy

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Abstract

A 73-year-old man with silico-asbestosis responded to steroid therapy. Chest CT scans showed diffuse micronodular opacities and ground glass opacities bilaterally throughout the entire lung fields, as well as progressive massive fibrosis in the bilateral upper lung fields. Diagnostic thoracoscopic biopsy revealed mixed dust pneumoconiosis with silicotic nodules, as well as fibrosis similar to that of Usual Interstitial Pneumonia (UIP) with many fibroblastic foci and alveolitis. Many asbestos bodies were also detected by iron staining.

Key words: mixed dust pneumoconiosis, dust exposure, subacute exacerbation, silico-asbestosis, steroid therapy

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Introduction

Asbestosis causes diffuse interstitial pneumonia in persons with workplace exposure to asbestos inhalation. On the other hand, silicate and silicotic (silicate) pneumoconiosis causes chronic fibro-nodular changes in the lung fields of workers with inorganic dust exposure. Silicosis is a subtype of pneumoconiosis caused by dust rich in crystalline silica (SiO₂), which features massive nodular fibrosis. The present case report concerns subacute progression of pneumoconiosis in a craftsman who had used firebrick. Thoracoscopic biopsy revealed silico-asbestosis with a usual interstitial pneumonia (UIP)-like pattern with many fibroblastic foci and mild alveolitis. Steroid therapy was partly effective. This case is of interest because subacute onset of silico-asbestosis with UIP-like features showed some response to steroid therapy. We report this case here and present a review of the literature.

Case Report

The patient was a 73-year-old man with dyspnea on exertion. He had smoked 10 cigarettes per day for 50 years and had worked as a craftsman using firebrick for 32 years since 1961. He had also used mortar containing asbestos, talc, and mica at work. He developed subacute onset of low-grade fever and dyspnea on exertion in July 2004 and received symptomatic therapy at a local hospital. His fever improved, but there was no improvement in exertional dyspnea or chest X-ray findings, so he was referred to our department and admitted.

On admission, there were no fine crackles in the lung fields and no finger clubbing. Chest X-ray films showed micronodular opacities and ground glass opacities (GGOs) entirely throughout both lungs, as well as progressive massive fibrosis in the bilateral upper lung fields (Fig. 1). CT scans of the chest revealed nodular and reticular changes in the upper lung fields, probably due to silicosis, as well as micronodular shadows and GGOs that were mainly in the middle to lower lung fields. A few areas of honeycomb change were also present in the subpleural zone of the lower lung fields (Fig. 2). Small nodular shadows in the middle and lower lung fields and a few areas with a honeycomb pattern in the subpleural zone of the lower lung fields suggested asbestosis. However, GGOs that were mainly detected in the middle and lower lung fields suggested acute or subacute deterioration of his lung disease and could not be fully explained by either silicosis or asbestosis.

Laboratory tests showed a slight increase of CRP (1.1 mg/dl), ESR (66 mm/h), LDH (672 IU/l), and KL-6 (653 U/ml). Blood gas analysis revealed only slightly abnormal val-

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duced carbon monoxide diffusion capacity (DLCO 9.45 cc/1.0% of 90.5%, suggesting restrictive impairment with reduced carbon monoxide diffusion capacity (DLCO).

Lung function tests showed a %VC of 77.4% and an FEV1.0% of 90.5%, suggesting restrictive impairment with reduced carbon monoxide diffusion capacity (DLCO).

Since we could not determine whether the UIP-like changes were related to asbestosis or silicosis, we diagnosed silicosis and asbestosis with UIP-like features including numerous fibroblastic foci and alveolitis.

The cause of the new GGOs, which corresponded to fibroblastic foci and alveolitis was unclear. Steroid pulse therapy was started on October 9, 2004, with methylprednisolone at 250 mg/day for 3 days, followed by maintenance therapy with prednisolone from 30 mg/day (tapered by 5 mg every 1 week). He has been maintained on 5 mg of prednisolone every other day since April 4, 2005, and has no fatigue or exertional dyspnea. As a result of steroid therapy, his GGOs improved on chest X-ray films and CT scans (Figs. 5, 6). Other laboratory data, such as a Six-Minute Walk Test (306 to 370 m), and KL-6 (653 to 415 U/ml), also improved.

Discussion

The patient had worked for 32 years as a craftsman using firebrick, so he had inhaled dust containing asbestos, talc, and mica over that time. The chest X-ray and CT features of pneumoconiosis including silicosis are micro-nodular shadows that are predominantly in the upper lung fields at an earlier stage, while progressive massive fibrosis occurs at an advanced stage, often accompanied with calcification of large opacities and pleural thickening. On the other hand, the chest X-ray and CT features of asbestosis are peripheral bands, lines, thickened interlobular septa, and honeycomb changes that are predominantly seen in the lower lung fields (1). In this case, nodular shadows with progressive massive fibrosis were found in the bilateral upper lung fields, while there were GGOs and micro-nodular changes in the middle to lower lung fields and a few areas of honeycombing in the subpleural zone of the lower lung. These chest X-ray and CT features suggested silico-asbestosis, but the GGOs suggested other complications. We speculated that the GGOs were relevant to the subacute onset of exertional dyspnea in this patient. There are several reports of complications of silicosis and asbestosis (2, 3). Free silica, such as silicon dioxide, is toxic, causing rapid organ damage and chest X-ray features consist of rounded or irregular opacities in the upper or middle zones. As disease advances, opacities may become massively fibrotic. On the other hand silicates are less toxic and cause slow organ damage and chest X-ray features consist of micro nodular shadows and linear shadows (4). That is why mixed dust of free silica and silicates causes atypical silicosis. Those chest X-ray features are similar to those of the present case.

To make a diagnosis, BAL and TBLB were performed. BAL did not yield specific information (5), and TBLB showed only nonspecific changes, therefore VATS was performed. The final diagnosis was silico-asbestosis with chronic interstitial pneumonia of a UIP-like pattern. Lung biopsy specimens are known to show a UIP-like pattern in patients with asbestosis (6, 7). There are also numerous case reports of pneumoconiosis associated with a UIP-like pattern. For example, Honma et al reported that 1.3% of pneu-
Chest computed tomography on admission shows nodular and reticular changes in the upper lung fields, and GGOs plus small nodules in the middle and lower fields. A few areas of honeycombing pattern are also present in the subpleural zone of the lower lung fields.

Figure 3. Histological features of S2 and S4. A) Subpleural zonal fibrosis and centrilobular nodular lesions are noted. B) One of the nodular lesions shows bronchiocentric mixed dust pneumoconiosis (MDP). HE stain, ×40. C) A tiny silicotic lesion is noted in the MDP. HE stain, ×100. D) An asbestos body detected by iron staining, ×400.

The presence of many fibroblastic foci and the rather wide area of alveolitis is unusual for the ordinary UIP pattern (14). The histological features in this case resembled a UIP pattern plus a cellular nonspecific interstitial pneumonia (NSIP) pattern like discordant UIP (15), seen in the same slide. We have previously reported that the prognosis of subacute interstitial pneumonia (SIP) with focal UIP-like changes is poorer than SIP (16), since 57% of the former patients showed worsening like the present case whereas 18% of the latter patients showed worsening. We also reported a case of subacute deterioration of interstitial pneumonia accompanied by focal UIP-like lesions (17). These reports suggest that patients with a UIP-like pattern may possibly show subacute deterioration, so it may be possible to consider interstitial pneumonia with luminal organization (including fibroblastic foci) in this case as the reason for
Figure 4. Histological features of S9. A, B) Rather diffuse involvement with structural remodeling is noted in A and B. C) In addition to pneumoconiosis, a UIP-like pattern with many fibroblastic foci was the main finding in S9. HE stain, ×200. D) There was interstitial inflammatory thickening with luminal organization, apart from the UIP pattern. HE stain, ×200.

Figure 5. A chest X-ray film obtained after steroid therapy shows amelioration of GGOs and reticulonodular shadows.

subacute exacerbation of symptoms. Asbestosis shows diffuse alveolar damage (18) which is thought to be similar to acute exacerbation of idiopathic pulmonary fibrosis (IPF). In addition to acute exacerbation of IPF and asbestosis, subacute exacerbation seems to occur in both diseases. Since there is the possibility of acute exacerbation in this case, we have to follow it up carefully.

It is difficult to determine on indications for steroid therapy, due to the lack of evidence. In this case, blood gas analysis showed almost normal findings, clinical symptoms, chest X-ray findings of one year before admission showing progressive massive fibrosis in bilateral upper lung fields and small nodular shadows in the middle and the lower lung fields, but no GGOs (data not shown) and CT films on admission showed subacute exacerbation. The pathological features of alveolitis suggested a rather good response to steroid therapy in such cases, therefore we performed steroid therapy. We gradually reduced the dose of steroid to prevent recurrence (19).

Infection, tapering of steroid therapy, and surgical stress are thought to cause acute deterioration of UIP (20), but this case had no such findings. The reason why pneumoconiosis can be complicated by a UIP pattern is unknown, but macrophages release various lysosomal enzymes and superoxides, while immune complexes that bind to the alveoli attract neutrophils, and these release toxic enzymes. At the same time, macrophages produce fibroblast growth factor and interleukin-1, which cause fibrosis (21). Silica itself is also thought to alter the antigenicity of proteins or to have an adjuvant effect (22, 23), but there is no consensus so far. This case should contribute to understanding the physiology and etiology of UIP.

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Figure 6. Chest computed tomography after steroid therapy shows amelioration of GGOs and reticulonodular shadows.

References


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