Silica-Induced Pleural Disease*

An Unusual Case Mimicking Malignant Mesothelioma

E. Handan Zeren, MD; Thomas V. Colby, MD, FCCP; and Victor L. Roggli, MD, FCCP

A 57-year-old man with a history of exposure to silica for 32 years presented with pleural thickening of the lower lobe of the left lung and a chronic right-sided pleural effusion without any radiographic evidence of parenchymal nodules in either lung. Light microscopic examination of a left visceral pleural biopsy specimen revealed markedly thickened pleura with fibrosis and macrophages containing birefringent silica and silicates. Occasional rounded intrapleural silicotic nodules were present. The underlying lung tissue did not show fibrosis or silicotic nodules. An energy-dispersive x-ray analysis confirmed the presence of silica. In the absence of lung involvement, this case represents a very unusual pathologic reaction caused by silica and silicates and adds to the clinical differential diagnosis of chronic pleuritis and malignant mesothelioma.

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Key words: pleura; scanning electron microscopy; silicosis

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nhalation of silica and silicate-containing mineral dust causes a variety of pathologic changes in the lungs and other tissues.1 The main morphologic changes occur in the lungs: silicotic nodules, alveolar proteinosis, interstitial cellular infiltrates, and interstitial fibrosis. Unusual extrapulmonary manifestations of silica exposure via lymphohematogenou spread in humans and animals also have been reported.2,3 Silicotic nodules have been described in tonsils, spleen, liver, extrathoracic lymph nodes, and retroperitoneum, but in all the described cases, pulmonary silicosis with typical clinical and radiographic findings also has been present. This is a report of a case of silicosis in which the disease was pleural and did not involve the underlying lung parenchyma. Review of the medical literature did not reveal any similar cases.

CASE REPORT

A 57-year-old man presented with chest pain, shortness of breath, and fever. He had a 32-year history of exposure to silica and silicates in a plumbing fixture factory where he sprayed glazing compound (primarily comprised of clay) onto the fixtures prior to their being fired. The patient was required to wear a mask. He described airborne dust resulting from the spraying process itself and from the sweeping up of the dried glazing compound that had fallen on the floor. The chest radiographs and CT scans showed thickening of the left pleura consistent with a chronic process and a small right pleural effusion without any evidence of parenchymal disease typical of silicosis in either lung (Fig 1). A biopsy specimen taken from the visceral area of the pleura of the left lung, which included underlying lung parenchyma, was performed to rule out infection and malignant mesothelioma. A follow-up was done for 3 years. The patient (who was personally interviewed by one of the authors [T.V.C.]) had changed his work environment and is currently working with plastic injection molds and continues to have occasional mild chest pain, which has not worsened. Chest radiographs and CT scans show persistent thickening of the left pleura; the right pleural effusion and fever initially present resolved within a month of original presentation. No other explanation for his presenting symptoms was identified. No parenchymal nodules have developed radiologically. He has had one or two episodes of pneumonia presumed to be infectious.

Histologically, the pleura was thickened with spindle-shaped macrophages containing birefringent silica and silicate particles

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responsible tonsils, nodes, cannot were examined well-described. In an phosphate (3%). Lymphatic logic changes with silicosis with necrosis, and cytokeratin positivity and recognition of the spindle cells as histiocytes containing silica and silicates led to the diagnosis of a silica-induced reactive lesion.

Mineralogic analysis was performed on a 5-µm thick paraffin section using scanning electron microscopy viewed by backscattered electron detector and an energy dispersive x-ray spectrometry. This showed many (nonfibrous) particles ranging from 1 to 5 µm in diameter (Fig 3). One hundred consecutive particles were examined by energy dispersive x-ray spectrometry and most consisted of silica or silicates. Specifically, 20% of the particles displayed peaks for silicon only or silicon plus phosphorus with an additional 70% consisting of potassium aluminum silicates, aluminum silicates, calcium aluminum silicates, calcium silicates, and magnesium aluminum silicates. The remaining 10% of the particles in this case were titanium (4%), tin (3%), and calcium phosphate (3%).

COMMENTARY

Involvement of the pleura in pulmonary silicosis is well-described. In advanced cases of silicosis, the pleural surfaces of the lungs are fibrotic with typical silicotic nodules and rarely diffuse plaques. Typically, there are multiple white spherical silicotic nodules protruding from the surface of the pleura. Al-Kassimi2 reported a case of lung silicosis with pleural effusion. In his case, no pathological changes in the pleura other than mesothelial cell hyperplasia were noted.

Extrapulmonary silicotic nodules have been described involving the spleen, liver, extrathoracic lymph nodes, tonsils, peritoneum, and retroperitoneum. Lymphatic and venous spread of the dust is thought to be responsible for such events since silica particles cannot be destroyed with phagocytosis and enzymatic digestion. It is presumed that the immune system plays an important role in the formation of the silica-induced fibrosis. Such a relationship is supported by cases of silica exposure-associated scleroderma. Slavin et al3 have stated that formation of typical silicotic nodules depends on lysosomal injury and fibrogenesis as two separate processes working synchronously. In another study, it was shown that there was no significant difference between the silicon content of the lungs and pleura between exposed individuals with and without silicosis. This finding also indicates that factors other than silica exposure alone may play a role in silica-induced fibrosis. In our case, pleural preference for the silica and silicate remains unexplained. We have considered the possibility that the accumulation of silica and silicate particles occurred in the pleura as a consequence of prior pleural inflammation and fibrosis and disturbance of the lymphatic drainage.

Silica and silicates commonly occur together naturally in rocks and pure exposures are relatively uncommon. The occurrence of silica and silicates together in our case is not unexpected. Although silicates were more numerous than silica (70% vs 20%), the presence of silicotic nodules in the pleura indicates the fibrogenic effect of silica was present and led us to conclude that this patient’s pleural nude would be responsible for such events since silica particles cannot be destroyed with phagocytosis and enzymatic digestion. It is presumed that the immune system plays an important role in the formation of the silica-induced fibrosis. Such a relationship is supported by cases of silica exposure-associated scleroderma. Slavin et al3 have stated that formation of typical silicotic nodules depends on lysosomal injury and fibrogenesis as two separate processes working synchronously. In another study, it was shown that there was no significant difference between the silicon content of the lungs and pleura between exposed individuals with and without silicosis. This finding also indicates that factors other than silica exposure alone may play a role in silica-induced fibrosis. In our case, pleural preference for the silica and silicate remains unexplained. We have considered the possibility that the accumulation of silica and silicate particles occurred in the pleura as a consequence of prior pleural inflammation and fibrosis and disturbance of the lymphatic drainage.

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disease was at least in part related to silica although silicates may also have contributed.

Although light microscopic examination allows detection of silica and silicate particles under polarized light, their precise identification requires additional techniques. Scanning electron microscopy is very useful in the detection of smaller particles, including those less than 1 μm.10 Ferrer et al9 have focused attention on the importance of the analysis of pleural tissue in the detection of pneumoconiosis and have successfully demonstrated high contents of silicon and calcium in the pleura and lung of occupationally exposed individuals using scanning electron microscopy and energy-dispersive x-ray analysis. In another study, the examination of hematoxylin-eosin-stained sections under polarized light was used as a guide to select the areas for scanning electron microscopy with energy dispersive analysis.10 In that study, polarizing light microscopy was shown to be very successful in the detection of silica particles, although some very small ones may be missed.10 Analytical scanning electron microscopy was then used to confirm the presence of silica and silicates consistent with the patient’s occupational history.

The case reported here demonstrates an unusual pattern of silica-related injury that apparently has not been previously reported. Silica-associated pleuritis must be kept in mind in the differential diagnosis of other reactive conditions as well as of neoplasms (including malignant mesothelioma) involving the pleura.

REFERENCES


Superior Vena Cava Obstruction Secondary to Mediastinal Lymphadenopathy in a Patient With Cystic Fibrosis*

Benjamin J.W. Chow, MD; Douglas A. McKim, MD, FCCP; Hani Shennib, MD, FCCP; and Robert E. Dales, MD

Superior vena cava (SVC) obstruction most often is a complication of malignant tumors such as lung cancer or lymphoma. The common use of long-term indwelling central venous catheters also has added to the prevalence of SVC obstruction. This report describes the first case of SVC obstruction in a patient with cystic fibrosis due to extrinsic compression from benign reactive mediastinal lymphadenopathy. Although in these circumstances intravascular thrombosis should be ruled out, extrinsic compression from mediastinal lymphadenopathy should be considered.

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Key words: cystic fibrosis; indwelling catheters; lymph nodes; superior vena cava syndrome

Abbreviations: CF=cystic fibrosis; SVC=superior vena cava

Mediastinal lymphadenopathy is a common sequela found in cystic fibrosis (CF) patients.1,2 This is the first reported case of superior vena cava (SVC) syndrome

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