Pleural plaques and effusion in a patient exposed to asbestos

Kenneth Iwuji RN, BSN, Jirapat Teerakanok MD

Figure 1 A and B show calcified pleural plaques (arrows). There is no residual pleural effusion.

CASE REPORT

A 72-year-old white man with a past medical history of pulmonary hypertension, sleep apnea, rheumatoid arthritis, hypertension, gastroesophageal reflux disease, diabetes mellitus type II, coronary artery disease, peripheral vascular disease, anemia, and asbestos exposure presented to the clinic with a four month history of progressive dyspnea. He had been hospitalized two weeks prior to this presentation with worsening dyspnea and a low serum sodium. He denied cough, sputum production, hemoptysis, persistent fever, a history of recurrent lung infection, and a prior history of lung disease or trauma to the chest. He reported occupational exposure to asbestos many years ago but could not provide useful details about that job. The following studies had been completed during his hospitalization:

1) An echocardiogram revealed elevated right ventricular systolic pressure,

2) Pulmonary function tests showed a severe restrictive defect with a normal corrected diffusion capacity,

3) CT scan of the chest showed bilateral calcified pleural plaques and thickening which were more pronounced in the posterior areas adjacent to the ribs (Figures) and scarring in the right middle lobe,

4) Thoracentesis revealed a hemorrhagic pleural fluid consistent with asbestosis-related pleural effusion.

Corresponding author: Kenneth Iwuji
Contact Information: Kenneth.Iwuji@ttuhsc.edu
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**DISCUSSION**

Despite increasing awareness and campaigns for the reduction or elimination of occupational asbestos exposure, asbestosis still remains a significant clinical problem. Asbestos exposure from direct occupational exposure or para-occupational exposure can lead to the following pulmonary problems: asbestosis, pleural plaques, diffuse pleural thickening, benign asbestos pleural effusion, lung cancer, and mesothelioma.

Benign asbestos pleural effusion was first described by Eisenstadt in 1964. Pleural effusion is a common manifestation and can be the earliest abnormality seen within 10 years of exposure to asbestos. The overall occurrence of asbestos effusions in exposed workers from one study was 3.1%, and most asbestos effusions were small. Clinical presentations vary from asymptomatic with an incidental radiologic finding to pleuritic chest pain with fever. Diagnosis requires: 1) a history of exposure, 2) the absence of other possible causes, and 3) no malignancy detected within 3 years after the effusion. Asbestos pleural effusion is usually exudative and hemorrhagic; bloody effusion in a patient with asbestos exposure does not necessarily imply mesothelioma. Increased eosinophils in pleural effusion is found in one third of patients; asbestos bodies (iron coated asbestos fibers) are rare or never found. The differential diagnosis of these effusions includes malignancy, tuberculosis, and pulmonary embolism. The presence of pleural effusion with pleural plaques suggests the diagnosis of asbestos pleural effusion. Pleural effusions can be transient and resolve in 3-4 months in many patients. These effusions recur in 30-40% of patients and persist and progress to diffuse pleural thickening in 50% of patients. In one cohort study, approximately 38.2% of the asbestos workers had parietal pleural plaques, and most were incidental findings. Robinson et al followed 22 patients with benign asbestos pleural effusion for 17 years and no mesothelioma developed. Management of patients with asbestos effusion is observed as in the case of our patient, and drainage if symptomatic. In summary, this case reminds clinicians that asbestos exposure is associated with several thoracic diseases including hemorrhagic pleural effusion and calcified pleural plaques.

**Author affiliations**: Kenneth Iwuji is a medical student at Texas Tech University Health Sciences Center in Lubbock, TX; Jirapat Teerakanok is a resident in Internal Medicine at TTUHSC in Lubbock.

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