

Pleural plaques and effusion in a patient exposed to asbestos

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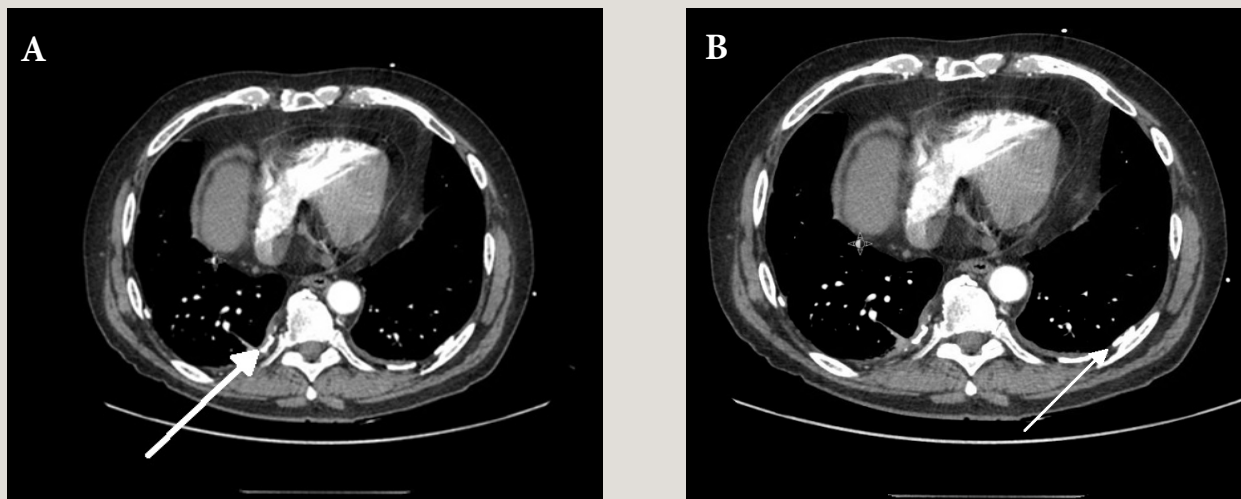


Figure 1 A and B show calcified pleural plaques (arrows). There is no residual pleural effusion.

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.

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DOI: 10.12746/swrccc 2016.0413.170

DISCUSSION

Despite increasing awareness and campaigns for the reduction or elimination of occupational asbestos exposure, asbestosis still remains a significant clinical problem.¹ 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.²

Benign asbestos pleural effusion was first described by Eisenstadt in 1964.³ Pleural effusion is a common manifestation and can be the earliest abnormality seen within 10 years of exposure to asbestos.⁴ The overall occurrence of asbestos effusions in exposed workers from one study was 3.1%, and most asbestos effusions were small.⁵ Clinical presentations vary from asymptomatic with an incidental radiologic finding to pleuritic chest pain with fever.⁶ 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.⁴ Asbestos pleural effusion is usually exudative and hemorrhagic; bloody effusion in a patient with asbestos exposure does not necessarily imply mesothelioma.⁷ Increased eosinophils in pleural effusion is found in one third of patients; asbestos bodies (iron coated asbestos fibers) are rare or never found.⁸ 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.⁴ 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.² In one cohort study, approximately 38.2% of the asbestos workers had parietal pleural plaques, and most were incidental findings.¹² Robinson et al followed 22 patients with benign asbestos pleural effusion for 17 years and no mesothelioma developed.⁸ Management of patients with asbestos effusion is observation 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.

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Submitted: 10/28/2015

Accepted: 12/22/2015

Reviewers: Eman Attaya MD, Ebtesam Islam MD

Conflict of Interest: None

Published electronically: 1/15/2016

References

1. Cugell DW, Kamp DW. Asbestos and the pleura: A review. *Chest* 2004; 125(3):1103-1117.
2. Eisenstadt HB. Asbestos pleurisy. *Dis Chest* 1964; 46:78-81.
3. Epler GR, McLoud TC, Gaensler EA. Prevalence and incidence of benign asbestos pleural effusion in a working population. *JAMA* 1982; 247(5):617-622.
4. Larson TC, Meyer CA, Kapil V, et al. Workers with libby amphibole exposure: Retrospective identification and progression of radiographic changes. *Radiology* 2010; 255(3):924-933.
5. Mossman BT, Bignon J, Corn M, Seaton A, Gee JB. Asbestos: Scientific developments and implications for public policy. *Science* 1990; 247(4940):294-301.
6. Myers R. Asbestos-related pleural disease. *Curr Opin Pulm Med* 2012; 18(4):377-381.
7. Paireon JC, Laurent F, Rinaldo M, et al. Pleural plaques and the risk of pleural mesothelioma. *J Natl Cancer Inst* 2013; 105(4):293-301.
8. Peacock C, Copley SJ, Hansell DM. Asbestos-related benign pleural disease. *Clin Radiol* 2000; 55(6):422-432.
9. Prazakova S, Thomas PS, Sandrini A, Yates DH. Asbestos and the lung in the 21st century: An update. *Clin Respir J* 2014; 8(1):1-10.
10. Robinson BW, Musk AW. Benign asbestos pleural effusion: Diagnosis and course. *Thorax* 1981; 36(12):896-900.

11. Rockoff SD, Kagan E, Schwartz A, Kriebel D, Hix W, Rohatgi P. Visceral pleural thickening in asbestos exposure: The occurrence and implications of thickened interlobar fissures. *J Thorac Imaging* 1987; 2(4):58-66.
12. Rudd RM. New developments in asbestos-related pleural disease. *Thorax* 1996; 51(2):210-216.