

Ischaemic Cavitation in Conglomerate Silicosis

Rajendra Prasad, Nitin Goel, Pawan Gupta, Viswesvaran B. and Abhijeet Singh

Department of Pulmonary Medicine, Vallabhbhai Patel Chest Institute, University of Delhi, Delhi, India

[Indian J Chest Dis Allied Sci 2015;57:233-234]

Clinical Summary

The most common cause of cavitation in progressive massive fibrosis (PMF) in patients with silicosis is infection with *Mycobacterium tuberculosis*. However, cavitation of PMF as a result of ischaemic necrosis has also been identified as a rare entity in silicosis.

A 40-year-old male, non-smoker, working as a stone-cutter for 30 years presented with a five years history of exertional breathlessness and eight months of dry cough. He had no history of fever, haemoptysis, chest pain, weight loss or loss of appetite. Physical examination was unremarkable.

Investigations

Routine haematological and biochemical investigations were found to be normal. Chest radiograph (postero-anterior view; Figure 1) showed well-defined, multiple nodular opacities in both lung fields along with massive fibrosis with cavitation in upper zones.

Computed tomography (CT) of the chest (Figure 2A,B,C) revealed bilateral multiple randomly distributed nodules of uniform size and attenuation

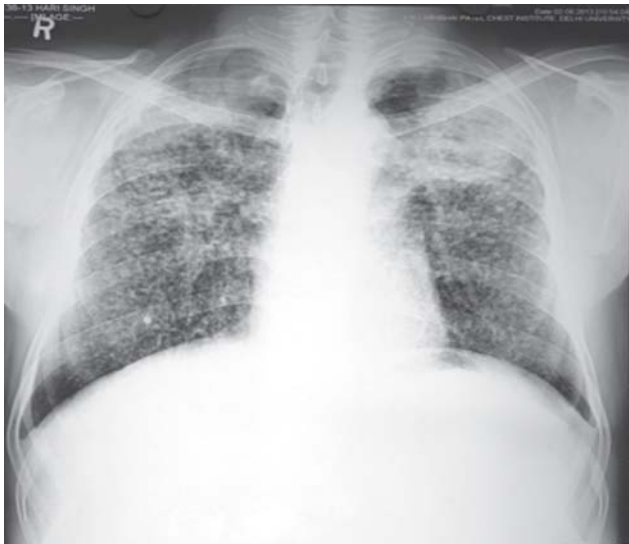


Figure 1. Chest radiograph (postero-anterior view) showing multiple nodular opacities along with progressive massive fibrosis with cavitation in both upper zones.

involving all lobes with upper lobe predominance with massive fibrosis with cavitation in both upper lobes (left more than right). Two samples of sputum for acid-

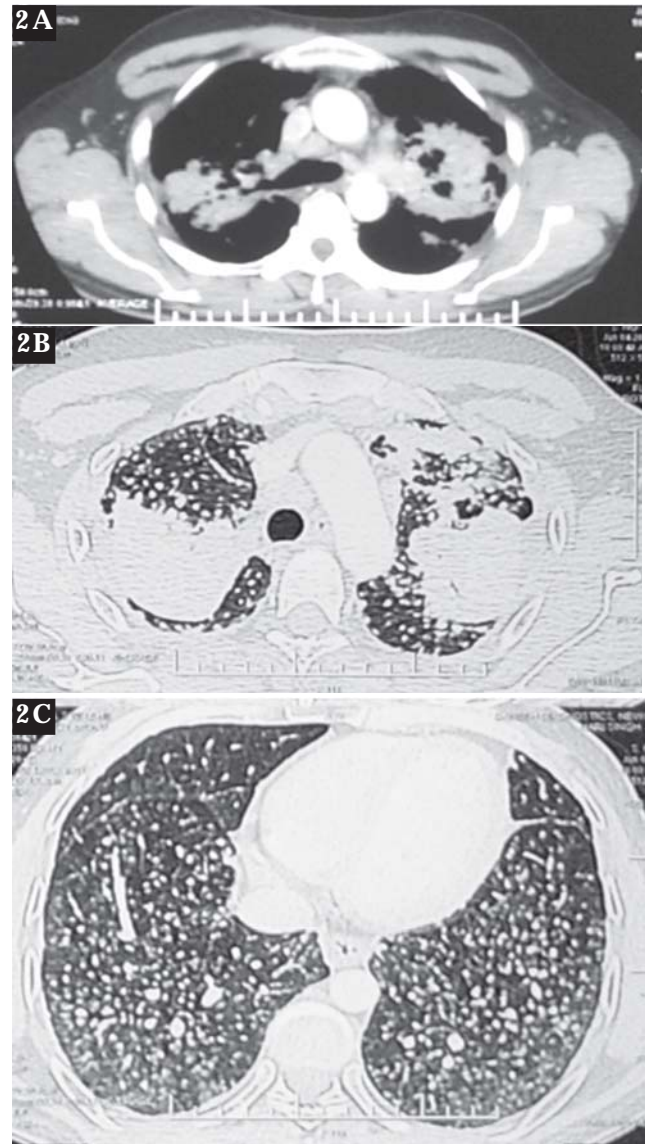


Figure 2A,B,C. Computed tomography of the chest showing bilateral multiple randomly distributed nodules in upper, middle and lower lobes with progressive massive fibrosis with cavitation in both upper lobes (Left > right).

[Received: July 30, 2014; accepted after revision: April 30, 2015]

Correspondence and reprint requests: Professor Rajendra Prasad, Former Director, Vallabhbhai Patel Chest Institute, University of Delhi, Delhi-110 007, India; E-mail: rprasadkgmc@gmail.com

fast bacilli (AFB) were negative. Mantoux test was negative. Pulmonary function test showed mild impairment of lung functions.

Fibreoptic bronchoscopy was done and transbronchial lung biopsy revealed ill-defined granulomatous inflammation surrounding anthracotic pigment. Bronchoalveolar lavage and endobronchial brush were negative for atypical cells, Gram's stain, fungal stain and AFB. A diagnosis of silicosis with progressive massive fibrosis was made on the basis of occupational history and radiological imaging characteristic of silicosis.

Diagnosis

Silicosis with progressive massive fibrosis

Discussion

Silicosis also known as "potters rot" is pneumoconiosis attributable to inhalation of respirable particles of crystalline silica. Crystalline silica has been classified as a group 1 substance by the International Agency for Research on Cancer.¹ A number of clinical and pathologic varieties of silicosis have been identified including simple, or nodular, silicosis, silico-proteinosis (acute silicosis), complicated silicosis (progressive massive fibrosis), and interstitial fibrosis.² In the case presented, the patient had significant exposure to silica dust during stone crushing and radiologically had complicated silicosis. Conglomerate silicosis or complicated silicosis results from the coalescence and agglomeration of several smaller nodules. In addition to the enlargement of nodules, profusion of nodular lesions results in PMF. Histopathologically the condition, is said to develop when the above described pulmonary lesions coalesce forming pulmonary masses 2cm or larger. However, according to the Silicosis and Silicate Disease Committee, a PMF lesion is defined as a lesion greater than 2cm in diameter in contrast to the 1cm or larger radiographic size established by the International Labour Office (ILO).³ Progressive massive fibrosis is often characterised by large fibrotic masses in conjunction with a distortion of the lung architecture often involving an upward displacement of the mediastinal and hilar structures attributable to volume loss. In addition, lower areas of the lung may appear hyperinflated and emphysematous, often in conjunction with multiple bullae.² Progressive massive

fibrosis lesions have a predilection to occur in the upper lobes of the right lung. However, in advanced cases, lesions are bilateral⁴ and the appearance being described sometimes as an "Angel's Wing" appearance.⁵ The conglomerate lesions appear against a background of smaller nodules and may obliterate bronchi and vessels and cause marked distortion of lung structure and function. Tuberculosis (TB) should be suspected when chest radiograph reveal the rapid emergence of new opacities, together with pleural effusion and cavitation.⁶ Nevertheless, cavitation is a relatively uncommon finding in silicosis. When it does occur, one should consider TB as the likely cause.⁷ In our case, we excluded possibility of pulmonary TB. The PMF lesions in accelerated silicosis may cause cavitation due to the resultant ischaemia and necrosis. In a series of 44 patients with silicosis and conglomerate masses who underwent chest high resolution CT, cavitations was noted in eight patients, and of these, six had concomitant TB.⁸ Occasionally, cavitation due to ischaemic necrosis may occur in a conglomerate mass.^{9,10}

References

1. IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, Silica, Some Silicates, Coal Dust and Para-Aramid Fibrils. In: *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans*. Vol. 68; France: Lyon; 1997.
2. Greenberg MI, Waksman J, Curtis J. Silicosis: a review. *Dis Mon* 2007;53:394-416.
3. Craighead JE, Kleinerman J, Abraham JL, Gibbs AR, Green FHY, Harley RA, *et al*. Diseases associated with exposure to silica and non-fibrous silicate minerals. *Arch Pathol Lab Med* 1998;112:673-720.
4. Castranova V, Vallyathan V. Silicosis and coal workers' pneumoconiosis. *Environ Health Perspect* 2000;108:675-84.
5. Yang JC, Liu KL. Coal workers' pneumoconiosis with progressive massive fibrosis. *CMAJ* 2012;184:E878-E878.
6. Bailey WC, Brown M, Buechner HA, Weill H, Ichinose H, Ziskind M. Silico-mycobacterial disease in sandblasters. *Am Rev Respir Dis* 1974;110:115-25.
7. Souza Filho AJ, Alice SH. *Fibrose maciça pulmonar progressiva*. *J Pneumol* 1991;17:147-53.
8. Ferreira AS, Moreira VB, Ricardo HMV, Coutinho R, Gabetto JM, Marchiori E. Progressive massive fibrosis in silica-exposed workers: high-resolution computed tomography findings. *J Brasileiro de Pneumologia* 2006;32:523-8.
9. Chong S, Lee KS, Chung MJ, Han J, Kwon OJ, Kim TS. Pneumoconiosis: comparison of imaging and pathologic findings. *Radiographics* 2006;26:59-77.
10. Kim KI, Kim CW, Lee MK. Imaging of occupational lung disease. *Radiographics* 2001;21:1371-91.