

# Interstitial Pulmonary Fibrosis and Progressive Massive Fibrosis Related to Smoking Methamphetamine With Talc as Filler

Peter A Baylor MD, Juan R Sobenes MD, and Val Vallyathan PhD

**We present a case of interstitial pulmonary fibrosis accompanied by radiographic evidence of progressive massive fibrosis in a patient who had a 15–20 year history of almost daily recreational inhalation of methamphetamine. Mineralogical analysis confirmed the presence of talc on biopsy of the area of progressive massive fibrosis. The coexistence of interstitial pulmonary fibrosis and progressive massive fibrosis suggests that prolonged recreational inhalation of methamphetamine that has been “cut” with talc can result in sufficient amount of talc being inhaled to result in interstitial pulmonary fibrosis and progressive massive fibrosis in the absence of other causes. Key words: methamphetamine inhalation; pulmonary fibrosis; talc pneumoconiosis. [Respir Care 2013;58(5):e53–e55. © 2013 Daedalus Enterprises]**

## Introduction

Progressive massive fibrosis (PMF) is a radiological term that refers to lesions larger than 1 cm, that are often bilateral and asymmetric, have well defined margins, and are usually located in the posterior aspect of the upper lobes. They represent a pulmonary complication of occupational inhalation of (but not limited to) coal, silica, or talc. PMF has been caused by occupational inhalation of

talc,<sup>1,2</sup> as well as intravenous administration of talc-adulterated narcotics.<sup>3</sup> Akira et al<sup>2</sup> reported 14 cases of occupational inhalation talc pneumoconiosis, 9 of whom had PMF. The average duration of occupational talc exposure in that group was 19 years. We now report recreational inhalation (as opposed to intravenous administration) of methamphetamine adulterated with talc, associated with interstitial pulmonary fibrosis and PMF.

## Case Report

A 49-year-old African American man was evaluated at the Department of Veterans Affairs Central California Health Care system for shortness of breath in December 2002. He had a 29 year occupational history as a plant manager in a citrus fruit packing company in the San Joaquin Valley of California. He was not directly involved in picking fruit from trees or from the ground, and did not have a substantial exposure to clay/earth. He gave a 15–20 year history of almost daily inhalation of methamphetamine before coming to the clinic. He denied intravenous drug abuse, and there was no cutaneous evidence of intravenous drug abuse. He stopped using amphetamine in late 2002 and went through a clinical detoxification program.

Clinical examination showed systemic hypertension. There was no clubbing or rales. His pulmonary function tests revealed a moderate restrictive ventilatory defect consistent with pulmonary fibrosis. The diffusing capacity of the lung for carbon monoxide ( $D_{LCO}$ ) was 50% of pre-

---

Drs Baylor and Sobenes are affiliated with the Veterans Administration Central California Health Care System, Fresno, California. Dr Vallyathan (deceased) was affiliated with the National Institute for Occupational Safety and Health, Morgantown, West Virginia.

This article is dedicated to the memory of Val Vallyathan PhD who died in July 2010.

The authors have disclosed no conflicts of interest.

The findings and conclusions in this report are those of the authors and do not necessarily represent the views of the Veterans Administration or the National Institute for Occupational Safety and Health.

Dr Baylor presented a version of this paper at the International Conference of the American Thoracic Society, held May 14–19, 2010, in New Orleans, Louisiana.

Correspondence: Peter A Baylor MD, Veterans Administration Central California Health Care System, 2615 East Clinton Avenue, Fresno CA 93703. E-mail: Peter.Baylor@va.gov.

DOI: 10.4187/respcare.01595

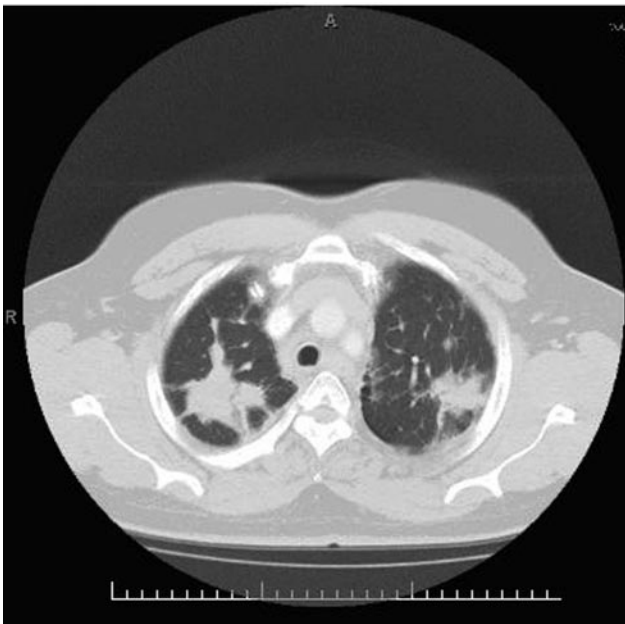


Fig. 1. Computed tomogram shows conglomerate masses in the upper lobes, consistent with progressive massive fibrosis.

dicted. There was no family history of fibrotic lung disease. Chest roentgenography revealed bilateral hilar lymphadenopathy with diffuse bilateral reticulonodular changes, the radiographic changes were more pronounced in the upper lobes. Sarcoidosis was suspected.

Bronchoscopic biopsy of the right lower lobe showed interstitial fibrosis, but no granulomas or other changes suggestive of sarcoidosis. A subsequent computed tomography revealed evidence of ill-defined reticular nodular densities consistent with interstitial pulmonary fibrosis, and infiltrative mass-like lesions in the posterior aspect of the upper lobes bilaterally, up to 2.5 cm (Fig. 1).

A fine needle biopsy of the right upper lung lobe mass lesion showed multinucleated giant cells with non-necrotizing granulomas. Stains for acid-fast bacilli and fungi were negative. Polarized light microscopy of the upper lobe biopsy material revealed highly birefringent inorganic particles consistent with talc and other silicates. Because the etiology of the pulmonary mass like lesions was obscure, examination of the tissue sections by scanning electron microscopy combined with back-scattered electron imaging (Fig. 2) and energy dispersive x-ray spectrometric analysis (Fig. 3) was carried out to identify the elemental composition of the particles in the lung lesion. These analyses showed that numerous particles in the right upper lobe mass were composed of magnesium silicate (talc) with iron, aluminum silicate (clay minerals) with potassium and iron, and aluminum silicates, which were more predominant than the magnesium silicate with iron.

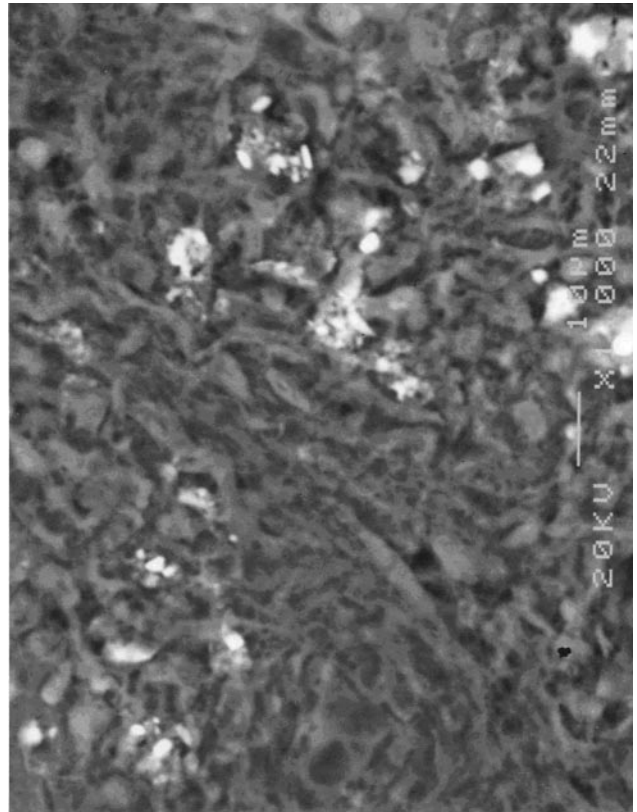


Fig. 2. Back-scattered scanning electron microscopy shows numerous particles.

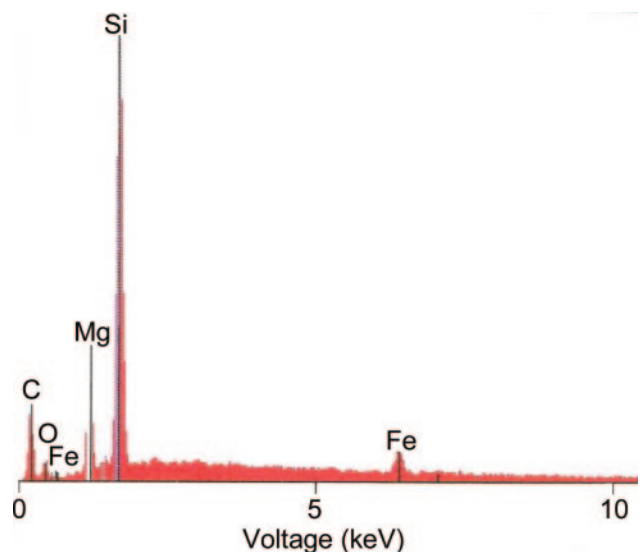


Fig. 3. X-ray spectrometry reveals that the lung particles were composed of a mix of particles, including magnesium silicate (talc) with iron, and aluminum silicate (mica) with potassium and iron (not shown).

### Discussion

Talc is hydrous magnesium silicate ( $\text{Mg}_3\text{Si}_4\text{O}_{10}[\text{OH}]_2$ ), in which ionic substitutions (such as aluminum for silicon

or iron, or aluminum for magnesium) may occur. The diagnosis of talc pneumoconiosis requires a history of talc exposure, typical radiographic changes, histological features consistent with a non-necrotizing foreign body granuloma, the presence of birefringent material seen under polarized light, as well as mineralogical confirmation. The most plausible cause of the interstitial pulmonary fibrosis associated with the PMF reported here is chronic recreational inhalation of methamphetamine.

Methamphetamine is commonly mixed or “cut” with agents known as “fillers”; these include talc, cellulose, and corn starch. Non-asbestiform talc has been shown to result in varying degrees of pulmonary fibrosis.<sup>4</sup> A previous report emphasized the necessity of obtaining an accurate occupational and exposure history in patients with interstitial lung disease. Sometimes the history of talc exposure occurred as long as 40 years prior to the diagnosis of talc pneumoconiosis.<sup>5</sup> We believe that chronic recreational inhalation of methamphetamine when “cut” with talc can result in sufficient amounts of talc being inhaled to result in interstitial pulmonary fibrosis and PMF. To our knowledge, this complication of methamphetamine inhalation

has not previously been described. Interestingly, a prior study showed that aluminum silicate is the main mineral in respirable dust samples for agricultural workers in the San Joaquin Valley of California.<sup>6</sup>

#### REFERENCES

1. Hunt AC. Massive pulmonary fibrosis from the inhalation of talc. *Thorax* 1956;11(4):287-294.
2. Akira M, Kozuka T, Yamamoto S, Sakatani M, Morinaga K. Inhalational talc pneumoconiosis: radiographic and CT findings in 14 patients. *AJR Am J Roentgenol* 2007;188(2):326-333.
3. Crouch E, Churg A. Progressive massive fibrosis of the lung secondary to intravenous injection of talc. A pathologic and mineralogic analysis. *Am J Clin Pathol* 1983;80(4):520-526.
4. Vallyathan NV, Craighead JE. Pulmonary pathology in workers exposed to nonasbestiform talc. *Hum Path* 1981;12(1):28-35.
5. Gysbrechts C, Michiels E, Verbeken E, Verschakelen J, Dinsdale D, Nemery B, Demedts M. Interstitial lung disease more than 40 years after 5 year occupational exposure to talc. *Eur Respir J* 1998;11(6):1412-1415.
6. Lee K, Lawson RJ, Olenchock SA, Vallyathan V, Southard RJ, Thorne PS, et al. Personal exposures to inorganic and organic dust in manual harvest of California citrus and table grapes. *J Occup Environ Hyg* 2004;1(8):505-514.