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BMJ Case Reports

Rare disease

# Pulmonary talcosis 10 years after brief teenage exposure to cosmetic talcum powder

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## Summary

Pulmonary talcosis is a rare but debilitating variant of pneumoconiosis often presenting with isolated non-specific symptoms of progressive exertional dyspnoea or cough. Occupational exposure to talc dust and intravenous drug abuse are well-recognised aetiological factors with only a few cases related to cosmetic talc exposure being reported to date. The authors report a case of a young woman in whom a mere 4 month ritual of inhaling cosmetic talcum powder led to full-blown pulmonary talcosis being diagnosed 10 years later. The importance of a taking a pertinent history relating to environmental exposures in all patients presenting with respiratory symptoms is re-established here.

## BACKGROUND

Pure talc is a phyllosilicate ($Mg_3Si_4O_{10}$ $(OH)_2$) used in the cosmetic and pharmaceutical industries. Occupational exposure to pure talc during its production or industrial use remains to be a well-known aetiological factor in causing pulmonary talcosis, symptoms of which may occur after several years following exposure.[1 2] Other forms of pulmonary pneumoconiosis involve exposure to impure varieties of talc containing asbestiform fibres and silica affecting mainly miners and intravenous drug misusers. Isolated deliberate cosmetic talcum powder inhalation however, is a very rare entity precipitating pulmonary talcosis. Cosmetic powders usually contain high-purity talc which can produce restrictive pulmonary disease by parenchymal inflammation, resulting in the radiographic reticular pattern of diffuse interstitial thickening with or without hilar adenopathy. Patients typically present with non-specific symptoms of chronic cough and progressive dyspnoea. Radiologically, the lung fields may show findings similar to those in asbestosis, however sparing of costophrenic sulci and lung apices is typical of talcosis. Nodule confluence results in large opacities that resemble those in progressive massive fibrosis.[3] The natural history of this disease renders it to be gradually progressive in nature even when exposure to talc has been abandoned since long.[1 2] Thus, many such cases are misdiagnosed simply because history of environmental cosmetic talcum powder exposure is overlooked. This case report illustrates that obtaining a pertinent history from the patient and use of appropriate investigative modalities lead to identification of a disease process that began more than a decade ago.

## CASE PRESENTATION

A 24-year-old woman, having a 5 pack/year history of smoking in addition to recreational alcohol intake, employed as an executive at a local oil firm, presented to the pulmonology clinic with complaints of progressive dyspnoea and non-productive cough for more than a year. She did not give any history of fever, sputum production, weight loss or night sweats. She had been evaluated, a year ago, to exclude pulmonary tuberculosis at a healthcare facility outside of the country. The chest radiograph at that time demonstrated miliary shadowing which seemed to be highly suggestive of pulmonary tuberculosis. A CT scan of the thorax revealed two small cavitary lesions in the apical region of the right lung. She had no known exposure to pulmonary tuberculosis. The rest of the history was unremarkable.

Bronchoscopy did not reveal any endo-bronchial abnormalities. Broncho-alveolar lavage was negative for mycobacteria and other infectious agents, and no neoplastic cells were found.

A screening laboratory examination, which included complete blood count, liver and renal function tests, was normal. A chest radiograph showed nodular lesions in both lungs. Spirometry and diffusion capacity of the lungs for carbon monoxide was normal. The sputum smear and culture were negative for mycobacteria.

On physical examination, she did not appear to be anxious or in respiratory distress. Her room-air oxygen saturation was 98%. The pulse was 68/min, blood pressure was 110/80mm Hg and respiratory rate was 20/min. She did not have a raised jugular venous pressure. Her cardiac examination was unremarkable. On auscultation of the chest, the only significant finding was diffuse inspiratory crackles heard bilaterally. The rest of the systemic examination was unremarkable.

A repeat chest radiograph CT chest a year later demonstrated bilateral nodular opacities throughout both lungs. There was no hilar adenopathy or pleural effusion (figures 1 and 2). A tuberculin test resulted in a 10 mm induration. The patient was counselled regarding the need for antituberculous therapy and empiric treatment was initiated.

## INVESTIGATIONS

A fibre optic bronchoscopy under fluoroscopy was performed to evaluate the trachea-bronchial tree. The bronchoscopy revealed clear airways with no narrowing of the

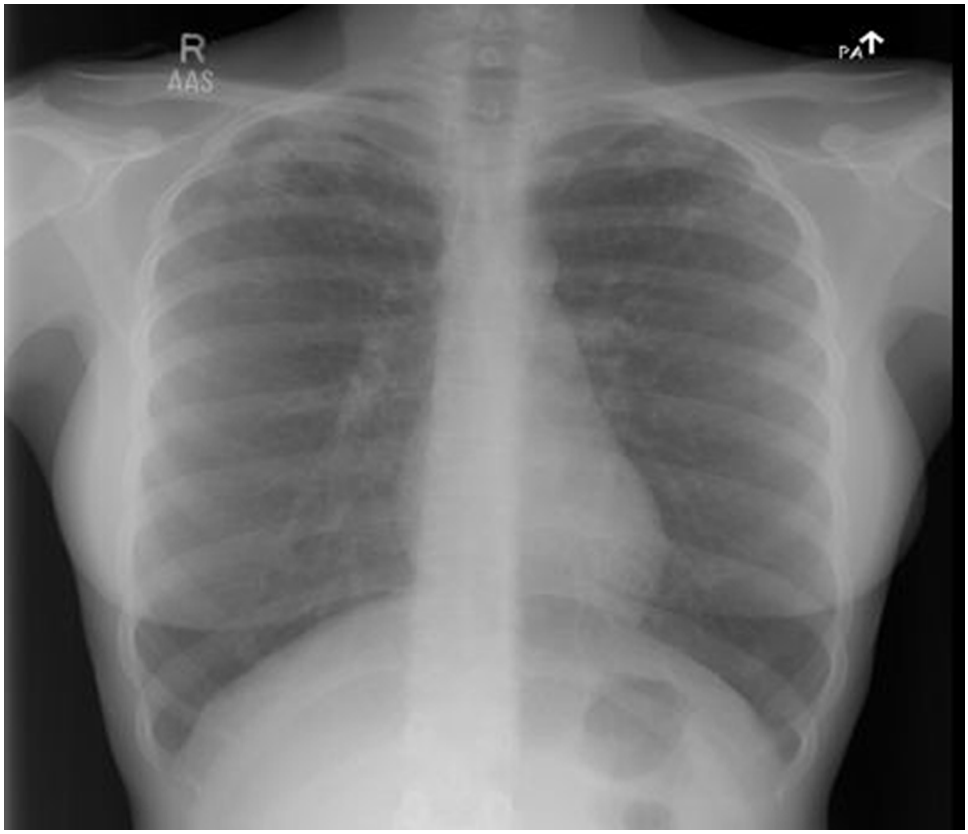


**Figure 1** Bilateral infiltrates with nodular opacities throughout both lungs.

tracheal lumen. Trans-bronchial biopsies were obtained from the lingula and lower lobe of the left lung. Broncho-alveolar lavage from both lung apices was negative for mycobacterial, fungal or other infectious agents and neoplastic cells.

Microscopic examination of the tissue samples obtained from the trans-bronchial biopsy revealed patchy lymphocytic infiltrates along with histiocytes and multinucleated giant cells in the alveoli which appeared to be engulfing a polarisable foreign material (figures 3 and 4). No epitheloid granulomas, asbestos bodies or neoplastic cells were observed.

## DIFFERENTIAL DIAGNOSIS

The differential diagnosis of a smoker presenting with progressive dyspnoea, non-productive cough and nodular opacities on chest x-ray include tuberculosis, sarcoidosis, hypersensitivity pneumonitis, Caplan syndrome, alveolar haemorrhagic syndromes (Good pasture's syndrome, systemic lupus erythematosus, Wegener granulomatosis, systemic necrotising vasculitis) certain pneumoconiosis as asbestosis and silicosis and drug-induced lung disease

## OUTCOME AND FOLLOW-UP

After a second fibre optic bronchoscopy, an in-depth, patient history was carried out at a subsequent visit to the clinic. The patient admitted to sniffing cosmetic talcum powder when she was 14 years old but had stopped after 4 months of doing so. She also admitted to smoking marijuana and 'shisha' (Persian water-pipe). She denied any intravenous drug abuse.

Based on the patient's history and the clinical, radiological and histological findings, the diagnosis of talc induced interstitial lung disease (talcosis) was made as a result of purposeful inhalation of cosmetic talcum powder. The serial chest radiographs displayed no significant improvement and the nodular lesions in both lungs remained unchanged. It was agreed by the team of treating physicians that due to the stable nature of the disease no treatment would be required. The patient was lost to follow-up.

## DISCUSSION

Talc (a hydrous magnesium silicate) is a mineral widely used in the ceramic, paper, plastics, rubber, paint and cosmetic industries.[4] Thorel proposed in 1896 that talc inhalation could lead to nodular, interstitial and fibrotic pulmonary disease.[5] Depending on the composition of the mineral dust inhaled and the duration of exposure, various intriguing patterns of restrictive respiratory pathologies emerge.

Talc induced pneumoconiosis can be divided into four variants. The first two affect mainly miners or industrial workers exposed to a high-load of impure talc dust containing either silica or asbestiform fibres. Known as talc-silicosis and talc-asbestosis, these variants have pathological and radiological findings almost identical to silicosis and asbestosis.[6] The third variant is common among intravenous drug misusers indulging in crushing oral tablets and using them parenterally resulting in a pulmonary foreign body granulomatosis being precipitated via haematogenous spread. Abusers of intravenous drugs who inject talc or similar substances may experience a rapid decline


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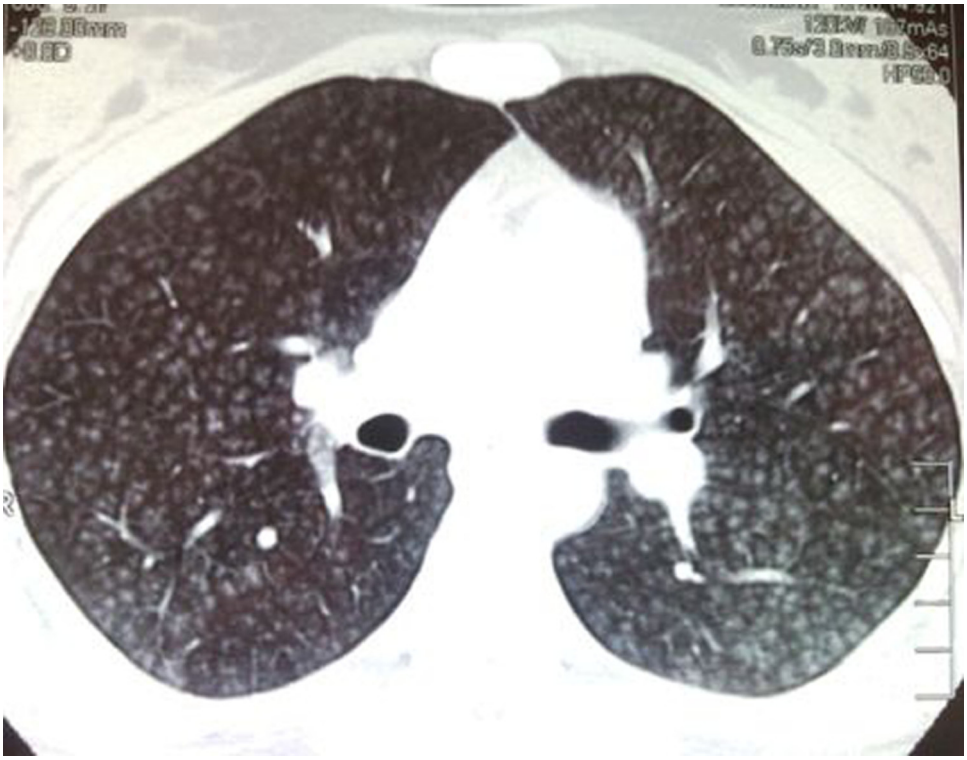

**Figure 2** Diffuse micronodular pattern with well-defined centrilobular nodules and diffuse ground-glass opacities.

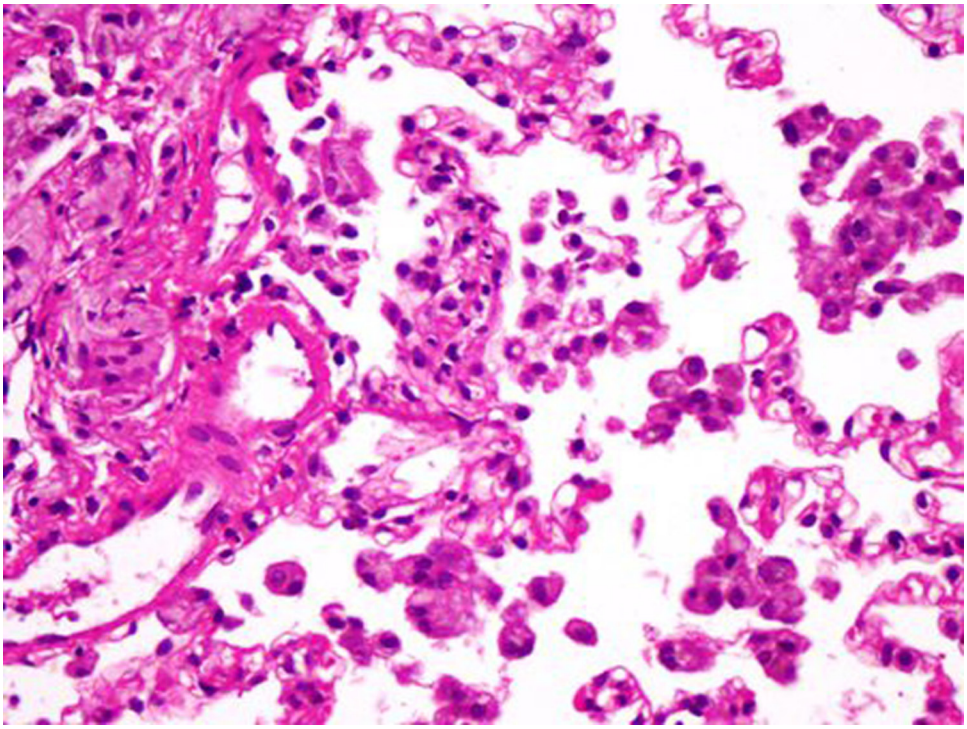

**Figure 3** Perivascular and peribronchial fibroses with non-caseating foreign body granulomas are present within the fibrotic areas and in the alveolar septa.

in pulmonary function when compared to abusers of non-adulterated agents such as heroin.

The fourth variant is pure talc induced pneumoconiosis or simply talcosis. This form is precipitated in individuals exposed to high-grade pure talc utilised in cosmetic and pharmaceutical industries with the victims ranging from industrial workers to the unaware cosmetic consumer. Interestingly, so far there is no conclusive evidence that cosmetic talc if used as intended can pose a health hazard.[7] Nevertheless, a number of cases have been identified

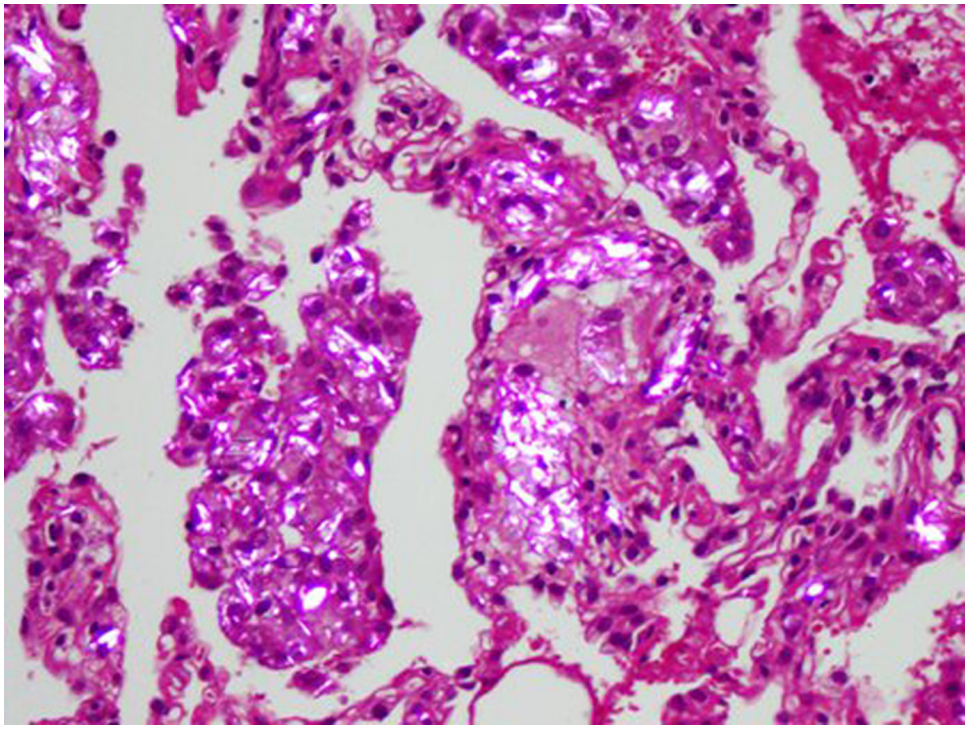

**Figure 4** Numerous bi-fringent needle-shaped crystals inside and outside macrophages being visualised under polarised light.

where isolated exposure to pure talc was identified as the only possible precipitant of pulmonary disease. For instance, massive inhalation of talc can cause pulmonary disease even if it occurs once and this has been reported in a 10-year-old child who had a history of massive exposure to talc at 2 years of age.[8] Talcosis has also been reported secondary to cocaine sniffing in the absence of any intravenous drug use.[6] Other case reports have revealed interesting exposure histories. To name some, a brief 5 year occupational exposure to pure talc resulted in symptomatic disease four decades later[2] or cases of patients presenting with progressive restrictive respiratory symptoms who on probing reported their chronic daily routine of cosmetic talcum powder dusting of their bodies since many years.[3 9] In this regard, the case we have reported shows a fascinating exposure history. A mere 4 month daily ritual of sniffing her favourite brand perfumed talc in teenage rendered our young patient to fall prey to full-blown interstitial lung disease which only became symptomatic 10 years after exposure.

The importance of taking a pertinent history relating to all possible environmental, occupational or recreational exposures in every patient presenting with restrictive respiratory symptoms is irrefutable. The clinical manifestations vary from mild cough and exertional dyspnoea to severe debilitating disease with respiratory failure in addition to night sweats, weight loss and progressive dyspnoea. Physical examination may be completely unremarkable or may reveal varying distribution of crackles and/or wheezes.

Radiologically, the lung fields may show findings similar to those in asbestosis, however sparing of costophrenic sulci and lung apices is typical of talcosis. Nodule confluence results in large opacities that resemble those in progressive massive fibrosis.[4] CT findings consist of a diffuse micronodular pattern with well-defined centrilobular nodules or diffuse ground-glass opacities. These lesions are similar to those seen in progressive massive fibrosis caused by silicosis.[10–12] The size and location of the nodules in our patient strongly supports the diagnosis of talcosis.

Pulmonary talcosis can either present as a fibrosis reaction diffusely involving the interstitium or showing an irregular nodular pattern or as a non-caseating granulomatous reaction observed on histology. The definitive diagnosis of the latter is made by careful light microscopic examination of lung tissue specimens. Tissue can be obtained via transbronchial or open lung biopsy. Perivascular and peribronchial fibroses with non-caseating foreign body granulomas are present within the fibrotic areas and in the alveolar septa. Findings consist of an interstitial inflammatory reaction composed of macrophages, foreign body multinuclear giant cells and numerous bi-fringent needle-shaped crystals inside or outside macrophages being visualised under polarised light.[13 14] The size of these crystals in most cases is not more than 5 picometre. In exposures unrelated to inhalation such as intravenous drug abuse, the particles are larger and characteristic intravascular and perivascular granulomas are seen depicting the haematogenous spread of talc. The histopathological reporting in our case, the size and location of the particles and the apparent lack of vascular lesions indicate that the mode of entry of the talc particles in our patient was via inhalation.

This case report highlights the importance of a thorough but relevant medical history.


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## Learning points

- A detailed medical history with special focus on environmental exposures together with relevant radiological and histological investigations can facilitate the diagnosis of talc pneumoconiosis-a relatively rare disease.
- The onset of talc induced interstitial lung disease may be insidious and the time span between exposure and onset of clinically apparent disease may be quite prolonged, as reported in our patient.
- The patient can be spared of the tedious hurdles of reaching a diagnosis in cases like these if a thorough history is taken at the first clinical visit.

**Competing interests** None.

**Patient consent** Obtained.

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