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Pulmonary talcosis in the setting of cosmetic talcum powder use

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ABSTRACT

Pulmonary talcosis is a rare pneumoconiosis that is difficult to diagnose and may progress to debilitating lung disease. Four types of talcosis are described in literature: talc-silicosis and talc-asbestosis secondary to inhalation in industry workers and talc-emboli in intravenous drug users that self-inject talc-containing oral tablets. Although found in common household products, talc is overlooked as a cause of pneumoconiosis. Talcosis caused by cosmetic face powder is even rarer. Here we discuss a woman in her 50s who developed talcosis from inhalation of cutaneous cosmetics two years prior, and how comprehensive history may be crucial in diagnosing this rare disease.

1. Introduction

Talc is a magnesium silicate mineral widely used in ceramics, paper, oral medications, and even household cosmetics such as baby powder [1]. It is also the causative agent of a rare type of pneumoconiosis that is difficult to diagnose.

Four types of talc-induced lung injury are described in the literature. The first two are found in industry workers with recurrent occupational exposure to talc that leads to talc-silicosis and talc-asbestosis [1]. Talc may also spread hematogenously to involve the lungs in intravenous drug users that self-inject talc-containing oral tablets [2,3]. Patients may present with nonspecific symptoms, including chronic cough and progressive dyspnea. If left unrecognized, the disease may progress to pulmonary hypertension and fibrosis. Here we discuss a patient who developed pulmonary talcosis from inhalation of cutaneously applied cosmetic powder, an especially rare mode of exposure to talc.

2. Case presentation

A woman in her 50s presented to an outpatient pulmonologist with dyspnea on exertion. She had a history of gastric adenocarcinoma, which resolved after a partial gastrectomy in 2013, hypertension, hyperlipidemia, and post-traumatic stress disorder. She reported no prior history of pulmonary or cardiac disease, and denied smoking exposure or injection drug use. She lived on farmland but did not have any exposure to silos, birds, mold, or hay. The patient's medications were not associated with any risk for pulmonary toxicity. Her dyspnea was associated with nonproductive cough, fatigue, and weight loss. She denied any new rash, arthralgias, fevers, chills, or diaphoresis. The patient was functional at baseline and independent with her activities of daily living (ADLs).

No prior pulmonary function tests were available for review at the time of presentation. Computerized tomography (CT) scan of her chest on initial evaluation showed diffuse centrilobular ground-glass nodules bilaterally. Bloodwork was notable for a positive perinuclear anti-neutrophil antibodies (*p*-ANCA). Her outpatient pulmonologist performed a transbronchial biopsy, however pathology was non-diagnostic. Bronchial fluid sample cell count was 294/μl; 98% was polymorphonuclear neutrophils and 2% mononuclear cells. The patient was presumptively diagnosed with Granulomatosis with Polyangiitis (GPA) and prednisone and cyclophosphamide were initiated.

In the following year, the patient had multiple admissions to the hospital due to continued abdominal pain and pneumonias. Cyclophosphamide was discontinued due to worsening liver function tests. In the months preceding our admission, the patient was found to have bilateral pleural effusion and underwent thoracentesis. Fluid analysis demonstrated a transudative effusion with negative cultures and cytology. Ultimately, she was treated for presumed pneumonia with intravenous antibiotics with some symptomatic improvement.

Approximately one year after initial presentation to her outpatient pulmonologist, the patient was then readmitted for progressive dyspnea, lethargy, encephalopathy, weight loss, and inability to perform ADLs.

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She also developed hepatic steatosis and unprovoked pulmonary embolism in interim. On examination, the patient was breathing comfortably. She was afebrile, normotensive, breathing at a rate of 18 breaths per minute, and oxygen saturations per pulse oximetry was 97% on ambient air. Cardiac exam showed regular rate and rhythm without murmurs, rubs, or gallops on auscultation. Breath sounds were diminished in left lower base fields, but without wheezes or crackles. She exhibited anasarca in her lower extremities.

Complete blood count was notable for a slight neutrophil-predominant leukocytosis. HIV was negative. Autoimmune labs, including antinuclear antibody, anti-ribonucleoprotein antibody, anti-smooth muscle antibody, anti-mitochondrial antibody, alpha 1 anti-trypsin, myositis panel, anti-Ro/anti-La, cytoplasmic-ANCA and perinuclear-ANCA were all negative. CT chest demonstrated bilateral centrilobular nodules, now with upper lobe predominance without hilar adenopathy along with a left sided pleural effusion (Fig. 1). Transthoracic echocardiogram showed normal ventricular function without significant valvular abnormalities.

The patient underwent bronchoscopy with bronchoalveolar lavage and transbronchial biopsies along with pigtail chest tube placement on the left side to drain the pleural effusion. Blood cultures were positive for candida and the patient was started on antifungal therapy. The bronchoalveolar lavage fluid showed cell count of 74/μl; 74% polymorphonuclear neutrophils, 3% lymphocytes, 12% monocytes, and 11% mesothelial cells. It was negative for malignant cells, fungi, and bacteria. The pleural fluid was transudative and negative for malignant cells, fungi, and bacteria as well. Biopsy samples revealed abundant macrophages with white crystals that were negatively birefringent under polarized light indicative of talc crystals (Fig. 2). These results were consistent with inhalational pulmonary talcosis. Around the talc crystals, there were also significant titanium particles deposited in the lung tissue (Fig. 3).

In summary, we present a non-smoking Caucasian female patient with chronic progressive dyspnea and cough. Her chest imaging was notable for bilateral centrilobular lung nodules with transudative pleural effusions. Differential diagnoses included atypical mycobacterial infection, bacterial pneumonia, lymphangitic carcinomatosis, hypersensitivity pneumonitis, sarcoidosis, bronchiolitis, pulmonary vascular disease, GPA, and Goodpasture's syndrome. However, they were inconsistent with the patient's overall clinical picture and lab findings.

After transbronchial biopsy results returned consistent with pulmonary talcosis and further probing, the patient admitted to excessive application of talcum face powder for two years prior to her presentation. Diagnosis of pulmonary talcosis was made. She experienced symptomatic improvement after removal of her pleural effusion and remained nonhypoxic on room air. She was discharged to her home.

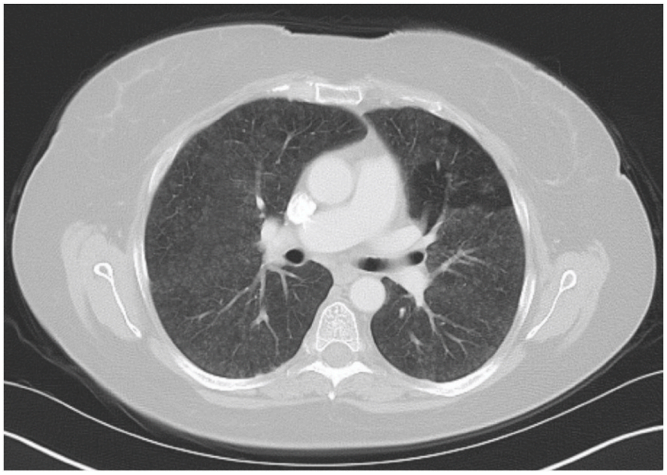

Fig. 1. Diffuse, bilateral ground-glass nodules in centrilobular pattern.

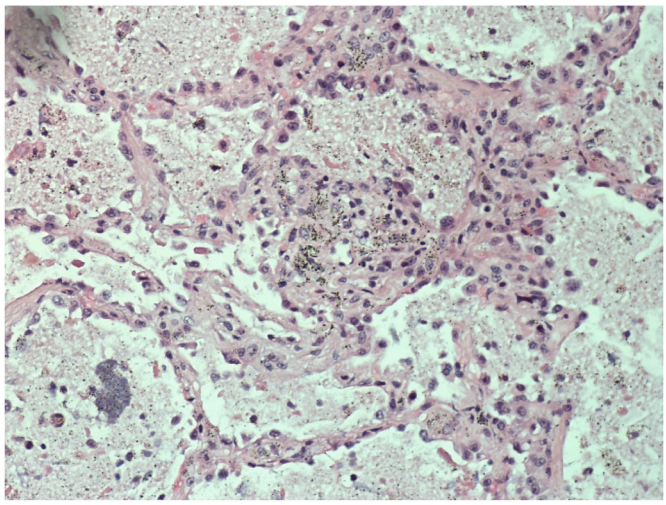

Fig. 2. Transbronchial biopsy with abundant macrophages and talc crystals. Color should be used. (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

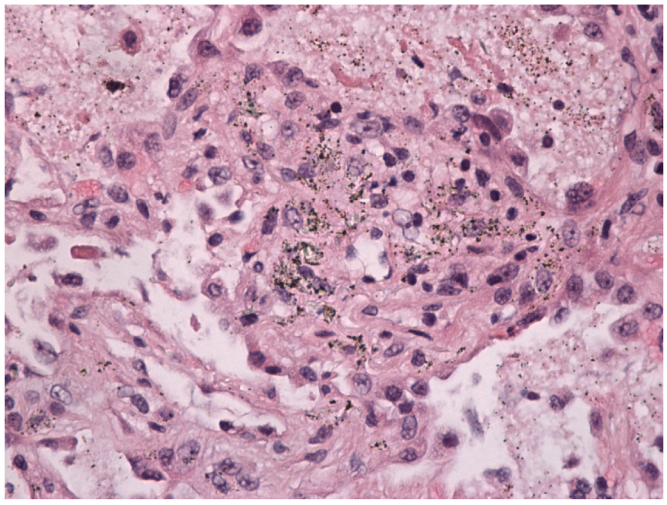

Fig. 3. Transbronchial biopsy with titanium particles in addition to talc crystals Color should be used.

3. Discussion

Pulmonary talcosis can be broadly categorized into two groups: those resulting from inhaling talc versus those from injecting talc, which is typically associated with intravenous drug users [1–3]. Talc-silicosis and talc-asbestosis from long-term inhalation exposures in miners and industrial workers are well-known and documented. However, talc exposure from household products is often overlooked as a causative agent of inhalation pneumoconiosis [4]. Pulmonary talcosis caused by cosmetic face powder is even less frequently described in the literature [4–6].

Diagnosis remains challenging, as the presenting symptoms of pulmonary talcosis are nonspecific and include dyspnea on exertion, cough, weight loss, anorexia, and diaphoresis [4–8]. The duration and amount of talc exposure necessary to cause symptoms are variable. Among documented cases, there have been patients who manifested symptoms only months after exposure, as well as those who became symptomatic up to four decades after exposure [7,8].

Chest CT in patients who inhale talc is typically characterized by middle- or upper-lobe predominant centrilobular nodules and ground

glass opacities [4–7,9]. Hilar adenopathy may or may not be present. On tissue biopsy, talc manifests as birefringent needle-shaped crystals under polarized light [4–7]. Once in the lungs, talc can cause non-necrotizing granulomas to form [1]. The resulting chronic inflammation causes fibrosis, and potentially restrictive pulmonary disease and pulmonary hypertension in later phases. There is no specific treatment. The disease may continue to progress even if all talc exposures have ceased [7,8].

The degree to which the titanium crystals in our patient contributed to this disease process is unclear [10]. Titanium oxide is another major ingredient in talc-based cosmetic products, often used to produce their characteristic white color. In this setting, titanium crystals should be a consistent finding in those who have had significant inhalation exposure to talc-containing products. However, patient cases with heavy titanium burden found on biopsy remain rare in current literature.

While imaging and pathology can confirm the diagnosis of pulmonary talcosis, a sufficient suspicion must be generated from a detailed history to guide the diagnostic workup and eventual discovery of pulmonary talcosis. In this patient CASE, we demonstrate that a comprehensive review of exposure history may have prevented a delay in diagnosis and unnecessary immunosuppression leading to a fungal infection and liver dysfunction. While talcosis is a rare cause of pneumoconiosis, it should be included when eliciting patients' medical history.

4. Conclusion

1. Talc is found in widely-available household products and an overlooked cause of pneumoconiosis that can cause clinically significant lung disease.
2. There is wide variability in time of exposure and disease onset among patients with pulmonary talcosis.
3. A comprehensive medical history including past exposures can prevent delays in diagnosis and treatment that can be harmful to patients.

Declaration of competing interest

There is no competing interest for any author.

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