Pulmonary Intravascular Talcosis Due to Intravenous Drug Use: A Case Report

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Abstract

Foreign body granulomatosis has many etiologies, including the injection of oral medications intravenously. The insoluble filler materials that are used in the medications can lodge in pulmonary arterioles and capillaries, which can trigger foreign body giant cell reaction, chronic inflammation, thrombosis, and fibrosis. On imaging, this may present with multiples micronodules. Here, we present a case of 65 years old patient, with history of heavy smoking, intravenous drug abuse and intravascular talcosis diagnosed by biopsy; presented to the emergency room for respiratory distress with fever and productive cough, A thoracic CT was requested, it showed an alveolar consolidation of left lower lobar with centrilobular periarteriolar micronodules. The majority of the nodules was calcified, related to her disease (talcosis IV).

Keywords: Intravascular talcosis, Intravenous drug use, imaging, CT scan.

INTRODUCTION

Intravascular pulmonary talcosis is a rare granulomatosis of foreign body due to the intravenous injection of oral medication. This form of talcosis called intravascular talcosis (talcosis IV) is different from talcosis secondary to inhalation of talc, which is more similar to pneumoconiosis [1, 2]. It is caused by intravenous (IV) injection of crushed pharmaceutical tablets intended for oral administration. These tablets typically contain insoluble binding agents, such as talc (hydrated magnesium silicate), microcrystalline cellulose, and crospovidone, in the lungs; they become irreversibly trapped and produce angiocentric foreign body granulomatous inflammation [3]. We present a case of this rare disease in a patient admitted to the emergency room for respiratory distress with a long history of addiction.

CASE REPORT

A 65-year-old patient, who visited Morocco on vacation, presented to the emergency room for respiratory distress with fever and productive cough. The patient had a history of heavy smoking 15 pack/year, intravenous drug abuse and unprotected sex; she also was diagnosed with pulmonary intravascular talcosis 10 years ago by a pulmonary biopsy.

The patient’s vitals on admission were stable except for low saturation on a pulse oximetry of 86% on room air. On physical examination, the patient was tachypnic and had rhonchi in the bilateral lung fields on auscultation.

Laboratory admission results showed an elevated CRP (129) and hyperleukocytosis 32,000 (predominantly neutrophilic) with positive PCR in favor of staphylococcal infection, liver and renal function tests was normal. A chest radiograph showed left basal alveolar syndrome (Fig 1).

The Geneva Score (Revised) was calculated at 6 (intermediate risk); A thoracic CT was requested in search of a pulmonary embolism, The CT was negative for pulmonary embolism, however, it showed an alveolar consolidation of left lower lobar, the upper margin of the consolidation was limited by the fissure, with innumerable tiny, well-defined, centrilobular periarteriolar micronodules, the majority of the micronodules was calcified, related to her disease (talcosis IV) (Fig 2 and 3).

The patient was put on antibiotic therapy with good clinical evolution and beginning of radiographic resolution (Fig 4).
Fig-1: A front chest x-ray showing an alveolar

Fig-2: A thoracic contrast-enhanced computed tomography in axial sections and mediastinal window showing an alveolar consolidation of left lower lobar, with innumerable tiny and calcified nodules related to her disease (Intravascular talcosis). Consolidation of left lower lobar

Fig-3: A thoracic computed tomography in axial (A and B) and frontal (C) sections, in lung window, in maximum-intensity-projection (MIP) (B and C) showing an alveolar consolidation of left lower lobar with centrilobular periarteriolar micronodules, making a discreet tree-in-bud pattern in some places, related to her disease (Intravascular talcosis).
Intravascular pulmonary talcosis is still an underrecognized condition, although the first description dates back to the early 1960s [4]. The term “talcosis” refers to talc deposition from inhalation as well as from injection. Whereas injection leads to intravascular pulmonary talcosis, inhalation leads to pneumoconiosis. Workers may inhale talc in mining or in manufacturing cosmetics, textiles, rubber, and insecticides. Inhaled talc is usually accompanied by other dusts, such as silica or asbestos, and these other dusts may predominate in causing pneumoconiosis. Inhaled talc particles, which are typically less than 5 μm, deposit in distal bronchioles and alveolar ducts, whereas injected talc particles, which are usually larger than 10 μm, deposit in arterioles and perivascular tissues [5]. Talc acts as a filler and lubricant in tablets containing oral medications. It is important both as a binding agent and as a material to prevent the tablet from sticking to the manufacturing equipment. When oral drug tablets containing talc are crushed, melted, dissolved in water, and intravenously injected, numerous tiny particles of talc become lodged in the pulmonary vessels resulting chronic inflammation, thrombosis, and fibrosis. This can result in pulmonary hypertension and progressive shortness of breath, which is, potentially, fatal. In time, microcrystalline cellulose and talc particles will form foreign bodies, which can provoke a histiocytic and foreign body giant cell reaction. Although, compared with talc, the microcrystalline cellulose particles are larger and are less likely to pass through the lungs to other organs [6, 3]. In our patient, there was extensive lung involvement with nodular densities, but there was no evidence of other organ involvement.

Pulmonary foreign body granulomatosis tends to, most often, affect males in their fourth decade of life [6]. Presentations of patients with pulmonary foreign body granulomatosis can range from asymptomatic to fulminant. Symptomatic patients typically present with varying nonspecific complaints including progressive exertional dyspnea and dry cough. Physical examination is typically unremarkable although hypoxia may be noted. More severe cases may result in adult respiratory distress syndrome or progressive massive fibrosis [3].

A number of different lesions in the lungs of individuals exposed to talc in both inhalational and intravenous forms have been described. These are formation of granulomas, interstitial fibrosis, and progressive massive fibrosis [7], on radiography; the typical findings of talcosis IV are innumerable micronodules (up to 2 mm in diameter) in the lungs, corresponding to perivascular granulomas. The micronodules are better depicted by CT, especially by maximum-intensity-projection (MIP). The nodules are distributed along pulmonary arterioles, which are centrilobular [5].

Thus, the micronodules largely spare the lobular septa, fissures, and subpleural interstitium. Although centrilobular nodules usually reflect bronchiolar disease, in the setting of talcosis IV, they reflect arteriolar disease. Centrilobular periarteriolar micronodules can even create a tree-in-bud pattern, further mimicking bronchiolar disease. Over time, talc micronodules may coalesce into perihilar conglomerate masses, resembling progressive massive fibrosis from silicosis or coal worker’s pneumoconiosis. The conglomerate masses in talcosis IV may contain high-attenuation material, likely talc or calcium [8, 5].

Other findings include enlargement of pulmonary arteries from pulmonary hypertension and signs of right ventricular strain. Ground-glass opacities can be present, probably reflecting confluent micronodules. Hilar and mediastinal lymphadenopathy are not major findings. Calcification of nodes is uncommon [5], both centrilobular and panacinar emphysema patterns have been reported in intravenous drug users, with lower lobe panacinar pattern being the predominant finding. These emphysematous changes can resemble bullous sarcoidosis or alpha-1 antitrypsin deficiency [9].

The differential diagnosis based on imaging findings is broad and includes miliary tuberculosis, interstitial lung disease, sarcoidosis, pneumoconiosis, endemic fungal infections, opportunistic infections, cytomegalovirus pneumonia, and neoplasms, such as bronchoalveolar carcinoma and lymphoid malignancy [3, 10].

Laboratory data are not helpful although angiotensin-converting enzyme levels may be elevated as a sign of granuloma formation [11]. The diagnosis is
confirmed with transbronchial or lung biopsy and demonstration of peri- and intravascular collections of exogenous material collocated within foreign body–type granulomas. These granulomas have giant cells that can be visualized as birefringent talc crystals with polarized light interestingly, starch is considered a marker of recent drug injection because it is metabolized and disappears with time [3, 12].

There are no established guidelines for the treatment of foreign body granulomatosis, and the approach to management typically depends on the stages and pattern of disease and the severity of symptoms. The most important first step is the cessation of smoking and IV drug abuse [13]. Success with steroids has been reported, but most authors believe that there are no benefits from using steroids and immunosuppressants. Associated pulmonary hypertension should be treated with vasodilators. Lung transplantation is considered a viable option in the treatment of talcosis. It is reserved as a last resort for patients with end-stage disease. Organ transplantation in substance abusers is controversial because of the possibility of relapse, even in those after long-term abstinence. A history of drug abuse is often considered a contraindication to transplantation in the context of limited donor resources [14].

CONCLUSION

Intravenous injection of oral medication causing foreign body granulomatosis of the lung is a complication that can be overlooked. Correct interpretation of the CT patterns and association of clinical data and imaging findings can be very useful to the radiologist in making the correct diagnosis. The use of a bronchoscopy with a biopsy confirms the diagnosis of this disease. The treatment is not well established, and the main treatment is supporting and modulating the immune system.

REFERENCES