



Dyspnea following intravenous drug use



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1. Case presentation

A 41-year-old man was referred to the outpatient clinic of our respiratory care department presenting with the symptoms of progressive cough, exertional dyspnea and tachycardia. The symptoms had started about 18 months earlier and had worsened continuously during the past year. At presentation the patient complained of dyspnea at the slightest exertion.

An external chest radiograph showed shadowing with a bilateral perihilar “butterfly” distribution consistent with sarcoidosis and silicosis.

The medical history revealed a history of intravenous drug abuse with cocaine for a period of three years until twelve years before presentation. The patient continued to smoke cigarettes (cumulatively 40 pack years) and to use methamphetamines and cannabis. The patient has been suffering from bronchial asthma and

pollinosis since the age of 15.

There was a history of immune complex glomerulonephritis five years earlier, with arterial hypertension and a significantly reduced renal function. The patient had been treated with dipyridamol and acetylsalicylic acid for several years.

2. Findings

On physical examination the patient showed bronchial breathing sounds and dyspnea on minimal exertion with no further pathological findings as well as no further signs of respiratory infection or systemic inflammation. Pulmonary function testing (bodyplethysmography) showed severe obstruction of the airways and significant hyperinflation. The diffusing capacity for CO (TLCO studied by single breath technique) was markedly reduced. The arterial blood gas analysis demonstrated hypoxemic respiratory failure.

Computed tomography (CT) of the thorax, performed without a contrast agent due to chronic renal insufficiency, revealed bilateral fibrosis with bronchial distortion and reticular opacities in all pulmonary lobes. It also demonstrated advanced pulmonary emphysema (Fig. 1).

Transthoracic echocardiography showed hypertrophy of the right ventricle, minor tricuspid regurgitation, but normal myocardial contractility. The left ventricle showed good systolic pumping function. Systolic pulmonary arterial pressure estimated from tricuspid regurgitation was slightly elevated. A bronchoscopy was performed and transbronchial biopsies revealed slight interstitial chronic inflammation, pulmonary granulomas with foreign-body giant cells and excessive granular foreign material strongly positive in polarization microscopy (Fig. 2).

3. Discussion

A clinical diagnosis of pulmonary talcosis due to repetitive intravenous administration of cocaine cut with talc was made.

Pulmonary talcosis is a rare disease, which was first described over 100 years ago [1]. It is usually caused by inhalation of talc dust and results in pulmonary fibrosis with chronic dry cough and

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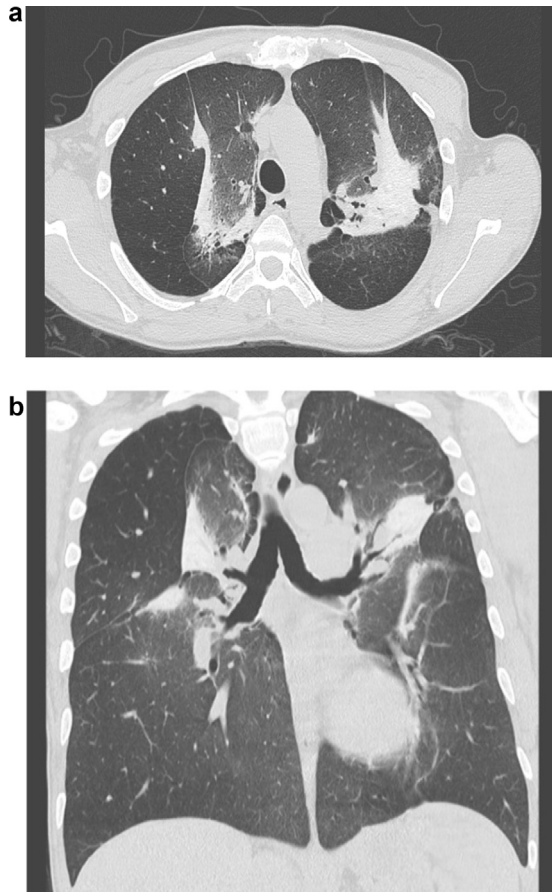


Fig. 1. a: CT-Scan, lung window. b: CT-Scan, coronal reconstruction.

dyspnea [2].

In the 1960s a new form of pulmonary talcosis evolved as a consequence of intravenous injection of dissolved oral medication leading to interstitial foreign-body granulomas in the lungs [3,4]. On imaging, the earliest findings are small nodules, generally <1 mm in diameter, evenly distributed throughout the lungs. They then can enlarge and coalesce to produce masses, usually bilateral, in the perihilar region [5].

Many tablets contain talc as structural binders and drugs, such as cocaine, are often cut with talc to increase their mass and consequently their street value. Injecting such foreign substances can cause foreign-body granulomatous reactions. Generally, the total amount of injected talc has to be very high to produce symptomatic disease, typically consisting of thousands of injections.

Since drugs are mainly administered intravenously and the first capillary filters for foreign material are the lungs, it is here that these foreign-body granulomas are most likely to accumulate. Talc-containing foreign bodies have also been described in the liver, retina, cardiac tissue, mediastinal lymph nodes, spleen, muscle, kidney, skin, pancreas and bone marrow [2,6,7]. The findings on fundoscopic examination can provide an important clue to the diagnosis of pulmonary talcosis. These show numerous tiny yellowish-white glistening particles in both the retinal arteries and veins, concentrated around the macula, where blood flow is highest and vasculature densest [8].

In regard to the lungs, talc injected intravenously not only leads to interstitial pulmonary fibrosis, due to development of granulomas in the interstitium, but may also lead to talc granulomas in

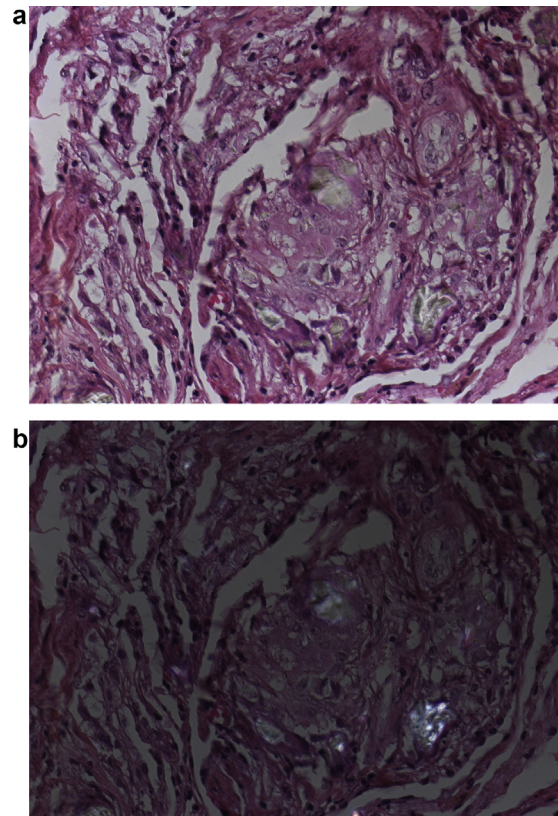


Fig. 2. a: Histology of transbronchial biopsies (HE, 200 \times). Large granulomas with foreign-body giant cells and foreign material in the granulomas. b: Histology of transbronchial biopsies (polarization microscopy, 200 \times).

and around pulmonary arteries (intravascular talcosis) [7,9,10]. Both sequelae, pulmonary interstitial talcosis fibrosis and pulmonary intravascular talcosis, may lead to pulmonary hypertension.

The low incidence makes pulmonary talcosis a diagnostic challenge. This case illustrates the importance of a complete medical history. Although the extensive formation of pulmonary foreign-body granulomas in the lungs as seen in this patient is rare, it is a typical complication of intravenous use of drugs containing talc. The timely recognition of symptoms, a complete medical history and knowledge about this disease and its radiological appearance are critical to establishing the diagnosis.

As there is no specific causal therapy for pulmonary talcosis, symptomatic treatment was initiated with bronchodilators. The patient was advised to begin physiotherapy, breathing exercises and physical endurance training. A long term oxygen was initiated and smoking cessation was recommended.

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