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Case Report

A woman and her breathtaking jewelry

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A 45-year-old woman presented at our outpatient pulmonology clinic with complaints of progressive dyspnea on mild exertion and a dry cough since three weeks. Suspecting a respiratory infection, her general practitioner prescribed a course of amoxicillin-clavulanate, without improvement of symptoms. She had no personal or familial medical history, nor did she take any chronic medication. She had been an active smoker for 30 years (30 pack years). For the last 11 years, she had been working in a jewelry polishers' company, cleaning mainly silver jewelry. Physical examination did not reveal any abnormalities (oxygen saturation of 98 percent on room air, normal heart and breath sounds). Biology showed a slightly elevated C-reactive protein (13.9 mg/L – reference value: 0-5 mg/L) without leukocytosis. Pulmonary function testing was compatible with small airway obstructive disease (FEF75%: 57% predicted) in the presence of air trapping (RV/TLC 192% predicted), a normal total lung capacity (103% predicted) and a mildly reduced diffusing capacity (DLCO: 69% predicted). High resolution thoracic CT scan showed dense homogeneously distributed centrilobular nodules in both lungs, as well as some tree in bud pattern, alveolar consolidations and some ground glass opacities. Bronchoscopy with broncho-alveolar lavage (BAL) was normal (no endobronchial abnormalities, normal BAL lymphocyte counts as well as CD4/CD8 ratio and cultures, including mycobacterial cultures, remained negative). After multidisciplinary consultation, a VATS (video assisted thoracoscopic surgery) lung biopsy was scheduled. Microscopy revealed multiple groups of epithelioid histiocytes filled with Perls' iron stain positive dust as well as mild chronic inflammatory infiltrates in the absence of significant fibrosis (figure 2). What is your diagnosis?

Discussion

Pulmonary siderosis, also known as Welder's siderosis is a form of benign pneumoconiosis caused by inhalation of iron oxide, that is usually seen in welders and grinders. It can also occur in silver polishers since iron oxide can be inhaled when using rouge (ferric oxide Fe_2O_3) during silver polishing. Therefore, silver polisher's lung is accepted as a synonym of pulmonary siderosis.

Although pulmonary siderosis is mainly an asymptomatic disease, symptoms can occur in current smokers and in patients with associated silicosis. In a recent retrospective study in seven cases, two patients had symptoms (cough and chest pain) and both were smokers [1], as was also the case in our patient. The reason why symptoms are more frequent in smokers, is not known to date. Pulmonary function tests usually are normal. In this case, the small airway disease and associated air trapping as well as the reduced diffusion capacity could be explained by the active smoking, rather than by pulmonary siderosis.

Chest X-ray and mainly high-resolution chest CT scan are of utmost importance to diagnose the disease. Generally, a reticulonodular pattern is seen. The dense nodules are widespread, small, ill-defined and present in a centrilobular distribution. The nodules present collections of dust-laden macrophages aggregated along the perivascular and peribronchial lymphatic vessels [2]. Interestingly all these chest CT findings can disappear over time in patients with pulmonary siderosis, when exposure to iron oxide is removed [1].

HRCT scan alone is insufficient to diagnose the disease in most cases. Usually a lung biopsy will be required to exclude other diagnoses [1]. The differential diagnosis of pulmonary siderosis consists of respiratory diseases with presence of a centrilobular reticulonodular pattern like hypersensitivity

pneumonitis, infectious diseases (including endobronchial tuberculosis) as well as other pneumoconiosis (like coal workers pneumoconiosis) and sarcoidosis. Multidisciplinary consultation in the presence of pulmonologist and radiologist (specialized in occupational diseases) as well as pathologist is important to decide whether a biopsy is necessary. Pathological examination is characterized by Perls' stain positive iron loaded macrophages, localized in the perivascular and peribronchial area's [3]. Benign forms of pneumoconiosis do, by definition, not cause fibrosis. However, if fibrosis is present in the lung biopsy of a suspected patient, this can be caused by concomitant inhalation of silica or asbestos (e.g. in welders and grinders) [3, 4].

Removal of the causative exposure is the only therapeutic measure one can take. This has an enormous impact on the patient's life, because this implies changing profession. Preventive measures can be taken by employers. This implies that the rooms are adequately ventilated, and employees must wear respiratory equipment. Smokers should be advised to stop smoking. In Belgium, pulmonary siderosis is recognized as an occupational disease and compensation and reimbursement can be requested from the fund of occupational diseases.

This case illustrates the importance of inquiring occupational history in all our patients, regardless of their symptoms, as well as the need for multidisciplinary consult in patients suspected of having interstitial lung disease.

Statements

All papers must contain the following statements:

Acknowledgement

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Statement of Ethics

The patient gave written approval to publish her case.

Disclosure Statement

The authors have no conflict of interest to declare.

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Author Contributions

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Vincken Stefanie: co- author, critical revision and correction.

Hanon Shane: co-author, critical revision and correction.

Vanderhelst Eef: co-author, critical revision and correction.

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Figure Legends

Fig. 1. Coronal CT image: interstitial micro-nodular pattern with centrilobular distribution, without distinct cranio-caudal gradient. In some areas of the lungs, the tree in bud pattern, as well as alveolar consolidations and some ground glass opacifications are present.

Fig. 2. Lung biopsy - microscopy: foreign body granuloma, multiple groups of epithelioid histiocytes filled with Perls' iron stain positive dust in peribronchial and perivascular areas, as well as a mild chronic inflammatory infiltrate in the absence of significant fibrosis.