Case Report

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Hypersensitivity Pneumonitis Caused by Esparto Dust in a Young Plaster Worker: A Case Report and Review of the Literature

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Key Words
Hypersensitivity pneumonitis · Esparto dust · *Aspergillus fumigatus*

Abstract
We report a case of a 25-year-old, white, male plaster worker who started developing fever, severe dyspnea and cough during the manipulation of esparto fibers. The functional lung study showed restrictive lung disease and decreased single-breath carbon monoxide transfer lung capacity. High-resolution computed tomography revealed a diffuse 'ground-glass' pattern. The histopathological findings were interstitial inflammation with a marked predominance of lymphocytes and microgranulomas. Bronchoalveolar lavage showed a significant predominance of lymphocytes, with an increase in the level of CD8. Serum precipitins against fungal antigens confirmed that *Aspergillus fumigatus* was the cause of the patient's hypersensitivity pneumonitis.

Introduction

Hypersensitivity pneumonitis or extrinsic allergic alveolitis may be defined as an immunologic pulmonary disease caused by a variety of factors, such as inhaled biologic dust, low-molecular-weight chemicals and medications. The inhaled organic or inorganic dusts are derived from different sources, although they are usually occupational. Esparto grass *(Stipa tenacissima)* is a gramineous plant 60–100 cm in height, widely found in the southeastern Iberian Peninsula (especially in the Almeria, Albacete, Murcia, Granada, Alicante and Valencia zones), south of France and Italy, Greece, Turkey, Lebanon and Israel, and northern zones of the North African countries. It is usually used for the manufacture of ropes, hemp sandals, rush mats and baskets; for decorative stucco plates, used on walls and ceilings, and for decorative work in the building industry. We present a case of extrinsic allergic alveolitis secondary to esparto grass.

Case Report

We present the case of a 25-year-old man who worked as a plasterer for the previous 6 years. During the last 2 years, he had recurrent episodes of fever, cough, dyspnea and chest pain, requiring emergency treatment on five occasions.

The patient associated these symptoms with the manipulation of esparto fibers, and he showed marked relief by avoiding exposure to this material. Physical and analytical examinations were normal.

Serological investigations of influenza A and B, *Mycoplasma, Chlamydia, Coxiella, Legionella*, HIV and autoantibodies were also normal. Arterial blood gas determinations revealed a pH of 7.38, a $PaCO_2$ of 38 mm Hg and a PaO_2 of 53 mm Hg. A chest roentgenogram showed bilateral reticulonodular infiltrates with a diffuse pat-

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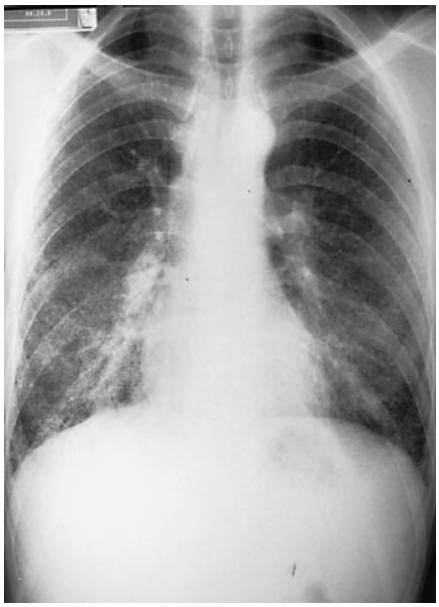

Fig. 1. Chest posterior-anterior roentgenogram showed bilateral reticulonodular infiltrates with a diffuse pattern.

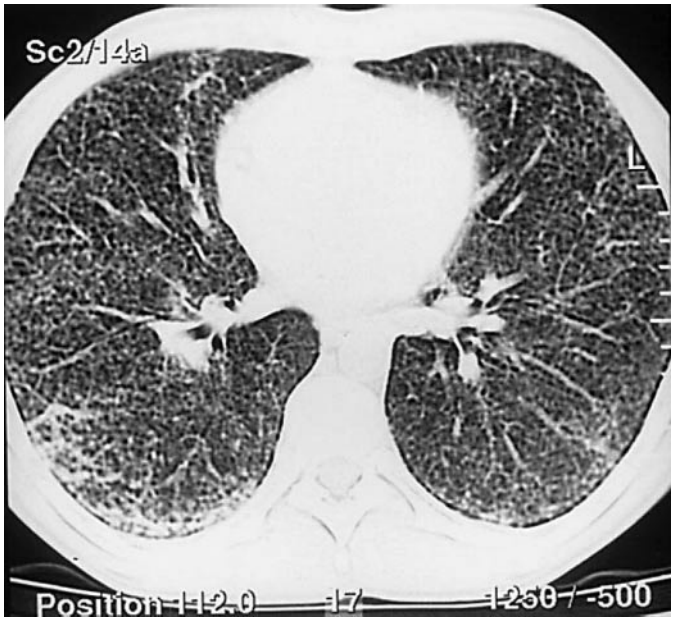

Fig. 2. High-resolution computed tomography revealed a diffuse 'ground-glass' pattern.

tern (fig. 1). Spirometry revealed a forced vital capacity (FVC) of 3,850 ml (67% of predicted value), FEV_1 of 3,330 ml (69%) and FEV_1/FVC of 86%, a moderately restrictive lung pattern. Total lung capacity, as determined by plethysmography, was 5,500 ml (70%). Single-breath carbon monoxide transfer lung capacity was decreased, at 16.62 ml/min/mm Hg (42%). High-resolution computed tomography revealed a diffuse 'ground-glass' pattern and some little mediastinic lymphatic nodes (fig. 2). Histopathological findings showed interstitial inflammation with a marked predominance of lymphocytes. Bronchoalveolar lavage showed a significant predominance of lymphocytes (50%), with 65% CD8 and 16% CD4. Serum precipitating antibodies against *Alternaria, Mucor, Cladosporium* and *Penicillium* were negative, but positive to *Aspergillus*. Total and specific IgE using the UniCAP technique for *Penicillium notatum, Cladosporium herbarum*, *Aspergillus fumigatus, Mucor racemosus* and *Alternaria alternata* were also negative.

Discussion

Rodriguez-Adrados [1] made a connection between esparto dust and an occupational respiratory disease for the first time in 1961 and coined the term espartosis. In 1969, Jiménez-Díaz et al. [2] suspected that this entity could be an immunologic disease. Dantin Gallego et al. [3], in 1969, described espartosis as a byssinosis-like disorder. In 1984, Hinojosa et al. [4] demonstrated for the first time the existence of precipitin IgG antibodies in the serum of patients exposed to esparto, using a double immunodiffusion test, and they also obtained a positive reaction to the pulmonary inhalation provocation test. The histopathologic changes were typical of hypersensitivity pneumonitis. They included espartosis in extrinsic allergic alveolitis disease [4]. In 1996, Hinojosa et al. [5] isolated *A. fumigatus* from esparto fibers and detected IgG antibodies against this organism in sera from five patients with hypersensitivity pneumonitis caused by esparto dust. In 1998, Quirce et al. [6] demonstrated an immune response to *A. fumigatus,* and reproduced the symptoms and laboratory abnormalities of hypersensitivity pneumonitis by pulmonary inhalation provocation test to this fungus alone. In the acute form of hypersensitivity pneumonitis, nonproductive cough, dyspnea, fever up to 104° F, chills, myalgia and malaise may persist for 18 h; recovery is spontaneous. With frequent episodes, additional symptoms of anorexia, weight loss and progressive dyspnea may be prominent. In all forms, levels of all isotypes of immunoglobulin are elevated, except for IgE. The immunologic finding characteristic of hypersensitivity pneumonitis is serum precipitating antibodies against the specific offending organic dust antigen. Chest X ray may be normal if the acute attacks are widely spaced. More commonly, there are fine, sharp nodulations and reticulation. End-stage disease may appear as diffuse fibrosis. High-resolution computed tomographic scan of

the chest can show centrilobular nodules as scattered small, round, ground-glass opacities, as well as emphysema in a bronchocentric pattern rather than following the bronchovascular bundles. Blood gases show hypoxemia and respiratory alkalosis.

Abnormalities in pulmonary function consist of a restrictive pattern. Decreased diffusion capacity and compliance also occurs. Arterial oxygen tension drops with exercise. Bronchoalveolar lavage shows increases in CD8 T lymphocytes, activated lung macrophages, mast cells and natural killer cells.

In the histopathological study in acute disease, we found alveolar and interstitial inflammation with a marked predominance of lymphocytes, activated macrophages, plasma cells and neutrophils. Macrophages with foamy cytoplasm surrounded by large numbers of mononuclear cells, which are likely lymphocytes, appear to be unique to this disease. As the disease progresses, the interstitial infiltrative process becomes fibroblastic, with obliteration of the alveolar spaces.

Pharmacological measures should be used if the offending antigen cannot be avoided or if the disease appears to be progressing even with avoidance. Oral corticosteroids (40–80 mg/day) are the mainstay of therapy in patients with hypersensitivity pneumonitis [7].

On the basis of the association between exposure to esparto and the symptoms, the restrictive spirometric pattern, the gasometric hypoxemia results, the low carbon monoxide transfer, and the findings on the high-resolution computed tomography and in bronchoalveolar lavage, we conclude that our patient was suffering from a hypersensitivity pneumonitis caused by esparto fibers. He improved quickly with corticosteroids and actually has been asymptomatic since he abandoned his occupation as plasterer.

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