

lesion. In the literature reviewed, we found four reported cases of FDE to fluoroquinolones: two were induced by ofloxacin (3, 4), one by ciprofloxacin and ofloxacin (5), and one by ciprofloxacin and norfloxacin (2). In contrast to this last case, ciprofloxacin was well tolerated by our patient. The explanation might lie in the multiple metabolic pathways of fluoroquinolones. Although norfloxacin and ciprofloxacin have a similar chemical structure (Fig. 1), they do not give rise to the same metabolites (1). After oral administration of norfloxacin, formyl piperazine substitutions, and amino and acetyl-amino open-ring metabolites are recovered in urine. These metabolites are not detected in urine after oral ciprofloxacin (1). It seems plausible that one (or more) of these norfloxacin metabolites induced the immunologic response leading to FDE in our patient, explaining the good tolerance of ciprofloxacin.

Key words: ciprofloxacin; fixed drug eruption; fluoroquinolones; norfloxacin.

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Hypersensitivity pneumonitis induced by exposure to the legume algarroba

WE PRESENT a case of hypersensitivity pneumonitis (HP) due to inhalation of a previously undescribed agent, algarroba (*Vicia monanthos*), a legume used as a livestock feed.

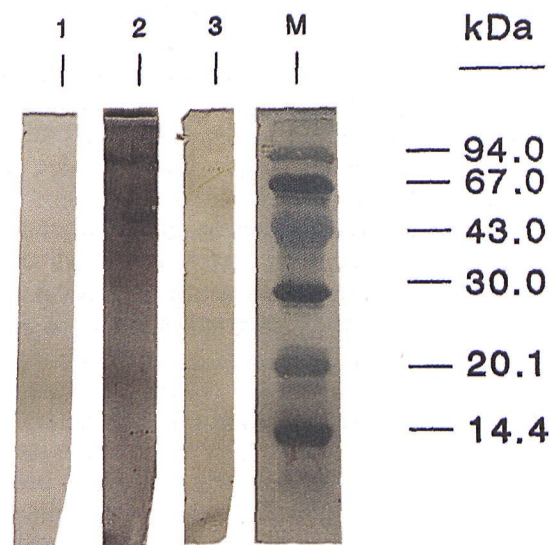


FIGURE 1 SDS-PAGE algarroba immunoblot analysis. Membranes developed with anti-IgE (lane 1), anti-IgG (lane 2), and anti-IgM (lane 3). Lane M shows known molecular weight markers, developed with black amide. Only lane developed with anti-IgG fixed seven different lanes of algarroba proteins.

■ The patient is a 75-year-old woman who had always lived in a farming community. A lifelong nonsmoker, she underwent a hysterectomy and extirpation of a mucinous ovarian cystadenoma in 1980, but had no history of asthma or atopy. Some 30-40 years ago, the patient began to experience intense dyspnea, asthenia, and myalgia after some 2-6 h of contact with algarroba meal. The symptoms persisted for hours and then remitted spontaneously. There were no accompanying nasojunctival symptoms. At first, the symptoms appeared only after handling algarroba for several hours, but finally, the patient's mere presence in the place where the algarroba was stored or short visits to the homes of neighbors who used algarroba for feed were sufficient to bring on an attack.

This patient had IgG antibodies in SDS-PAGE and immunoblotting to seven protein bands of algarroba (Vicia monanthos)

At present, the patient suffers dyspnea on exertion with no signs of bronchial hyperreactivity. On physical examination, bilateral crackles were detected in the mid- and lower lung fields. Laboratory test results were as follows: hemogram: normal; ESR 107 mm/h; arter-

ial gasometry: P_{O_2} 64.6, P_{CO_2} 45.4 (mmHg), pH 7.4; IgG 1970 mg/dl, IgA 211 mg/dl, IgE 2 IU/ml, IgM 7350 mg/dl; kappa 810, lambda 115 (mg/dl). Absence of light chains in urine; angiotensin-converting enzyme level 33.8 U/l. Precipitins (by double immunodiffusion) were negative against *Thermoactinomyces vulgaris*, *Mycropolyspora faeni*, *Candida albicans*, *Aspergillus fumigatus*, and pigeon, parakeet, and chicken sera and feces. Chest roentgenogram: diffuse micronodular pattern, more acute in middle fields and the base of lungs. Pulmonary function tests: TLC 79%, FEV₁ 62%, DLCO 56% (not reversible with β_2 -agonists). Transbronchial biopsy: interstitial lymphoplasmocytic infiltrates with some histiocytoid cells, predominantly located at the peribronchiolar level. Bronchoalveolar lavage fluid was negative for mycobacteria and aerobic germs. Bone marrow biopsy: compatible with reactive lymphoplasmocytosis, with no evidence of tumor. Abdominal-pelvic CT: heterogeneous increase in density at the epiploon and mesentery level. Round, lobule-like masses in right iliac fossa and in both inguinal areas. Precipitins (by double immunodiffusion and counterimmunoelectrophoresis) were positive against algarroba extracts. Two pools of control and atopic serum revealed no precipitins. Immunoblotting with algarroba extract (using anti-IgG, anti-IgE, and anti-IgM) revealed seven lanes (Fig. 1) with anti-IgG antiserum, with the following molecular weights: 83.2, 66.1, 56.3, 49, 43.7, 38, and 19.5 kDa. There were no lanes with anti-IgM or anti-IgE.

Laparotomy revealed the presence of a peritoneal pseudomyxoma.

■ This is the case of a patient with a long history of respiratory symptoms compatible with HP that appeared always, and exclusively, on inhalation of algarroba meal. Her degree of sensitivity increased gradually, so that finally the inhalation of very small quantities of the antigen was sufficient to trigger an episode.

In addition to the clinical history, the patient's physical examination, pulmonary biopsy, pulmonary function tests, and chest roentgenogram were compatible with a chronic form of HP (1-3).

In this case, we were able to demonstrate the presence of specific IgG against seven different algarroba protein fractions and the absence of specific precipitins against the antigens most commonly implicated in the etiology of HP (4).

Although the diagnosis could be definitely confirmed by bronchoprovocation test, the age and clinical status of the patient counseled against it. However, the most important step in the diagnosis is the recognition of occupational and environmental exposure (5), which in our patient was evident.

The patient presented a raised monoclonal IgM concentration that did not meet the macroglobulinemia criteria of Waldestrom, and was interpreted as a reactive form in the presence of a chronic inflammatory stimulus such as her peritoneal pseudomyxoma.

In our review of the literature, we were unable to find a single case of HP attributable to algarroba.

Algarroba (*V. monanthos*) is an annual herbaceous plant of the papilionaceous family. It is grown within a small area (4628 hectares) of Spain, but, given the gradual disappearance of algarroba as a crop, we feel it unlikely that new cases attributable to this legume will arise.

Key words: algarroba; hypersensitivity pneumonitis; *Vicia monanthos*.

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