Case Report

Starling-Induced Hypersensitivity Pneumonitis: Minimal but Persistent Antigen Exposure

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Introduction

Bird fancier’s lung is a disease which is a subset of a condition known as hypersensitivity pneumonitis (HP) or extrinsic allergic alveolitis.1-3 It is triggered in susceptible individuals by inhalation of avian antigens, which can be found in the serum, feathers, droppings or secretions of birds. These particles, mainly after activating macrophages and T lymphocytes, lead to a type III inflammatory response by immune complexes, while type I and IV responses can also coexist. The end result is a lymphohistiocytic inflammatory reaction or a response from monocytes and macrophages in the peripheral airways and the surrounding interstitium.4 Classically, the clinical presentation is divided into 3 forms: acute, subacute and...
chronic, depending on the intensity and frequency of exposure to the causative agent.

Generally speaking, persons with this condition have usually come into contact with birds in their work or leisure time. Less frequently, it is a result of inhalation of feathers from the inside of quilts, cushions or other objects. It is important to diagnose the disease in its early stages as it can lead to fibrosis or chronic obstructive disease and even severe chronic respiratory failure.

The importance of this case lies in the realisation that the disease can be caught outside the home or occupation and after persistent, but not mass, antigen load. Indeed, the etiology was the inhalation of starling droppings in a public park near her house in the city centre. This exposure, so common in cities, has to our knowledge not been described until now as a cause of NH.

**Clinical Observation**

A 51-year old female nursing director, without any known toxic habits, and with a history of allergy to iodinated contrast and positive Mantoux from 10 years earlier. The patient consulted due to the progressive development over 6 months of watery rhinorrhea, a nonproductive cough, grade II dyspnoea and fatigue. The patient was visited due to an increase in dyspnoea. She was afebrile, with blood pressure of 120/80mm Hg, a heart rate of 70bpm, a respiratory rate of 32rpm, with no respiratory noises or other pathological findings detected in the examination.

The chest radiograph showed a predominantly ground glass pattern in the lower area. A chest CT showed a micronodular pattern of centrilobular location (fig. 1). The haemogram results were 6,300 leukocytes/dL (54% neutrophils, 2% eosinophils, 34% lymphocytes and 9% monocytes), ESR was 21mm/hr and the rheumatoid factor was 48IU/mL. The rest of the laboratory tests, the ACE, C-reactive protein and procalcitonin levels were normal.

Serum levels of IgG in commercial avian antigens (dove, parakeet, chicken and parrot) and fungi were below those levels considered positive. The prick test (immediate hypersensitivity skin test) carried out to a number of pneumoallergens was negative. Respiratory function tests revealed a restrictive defect with reduced DLCO, FVC of 2,730 (66%), FEV1/FVC of 86, FEF 25%-75% of 2,870 (111%), RV of 990 (54%); TLC of 3,390 (65%), DLCO of 13.1 (54%) and KCO of 4.14 (81%), a PI max of 73 (90%) and PE max of 83 (88%). The SO2 with a FiO2 of 21% was 98%. The 6-minute walk test showed a desaturation after 600m (at the end of the test, the SO2 was 88% and heart rate was 134/min).

Fiberoptic bronchoscopy showed no abnormalities at macroscopic scale. The BAL cell count showed 85% macrophages, 12% lymphocytes and less than 3% neutrophils. No determination of lymphocyte subpopulations was performed due to the absence of a clearly high lymphocyte count. As no definitive diagnosis of interstitial disease was found, a videothoracoscopy was performed and biopsies showed lymphohistiocytomatous pneumonitis with non-necrotising granulomas, which were highly suggestive of HP (fig. 2).

Extracts were prepared from serum, feathers and starling droppings according to the protocol described. The immediate hypersensitivity skin test to starling feathers was positive, as well as the specific IgG for starling feathers, which was 0.812UA at 450 nm (normal value: 0.634UA at 450 nm). The specific IgG for starling droppings extract was negative (0.370UA at 450 nm [normal: 0.530 UA at 450 nm]). A specific bronchial provocation test was performed according to the technique described above2 with a 1/10 dilution of the starling feather extract which was positive, giving a temperature increase of 0.7C after 4hr, a 16% decrease of DLCO and 12% decrease of FVC after 8hr.

Treatment with prednisone (30mg/24hr) was started and maintained for one month, with later gradual reduction, which improved clinical and lung function. After stopping the corticoids, the cough and dyspnoea reappeared 3 months after stopping treatment. Therefore, a maintenance dose of 10mg/day was required and a move away from the area was recommended.

**Discussion**

Traditionally, bird fancier’s disease has been found in pigeon fanciers, but it can also affect keepers of other birds, such as doves, pheasants, parrots, parakeets, canaries, budgerigars and other exotic birds.

Diagnosis of NH is sometimes difficult, as there may be no apparent relationship to a specific agent in the workplace or at home, despite performing a very detailed medical history. There have been several schemes with diagnostic criteria, but none has been universally accepted. Recently, a group of experts developed a diagnostic protocol based on predictor variables, which has proven useful in acute and subacute patients.

The patient lived in a house with windows, adjacent to a public park populated by starlings which left a lot of droppings on the ground. She had to cross this park at least twice a day on her journey to and from work, and this walk triggered coughing without any other respiratory or systemic symptoms. NH symptoms depend on the type, intensity and duration of exposure to the antigen. A daily but non-passive, intermittent exposure in an open environment could be responsible for a subacute or chronic presentation, with no acute symptoms, as well as a restriction in lung function and moderately high BAL lymphocytosis. In this case, the CT findings and especially the VATS biopsy were diagnostic of NH. Although it was not necessary to identify the antigen for diagnosis of the disease, the absence of an etiological diagnosis favoured the persistence of exposure to an antigen and eventual progression to chronic forms of the disease. Evidence of antigenic exposure requires skin testing and precipitins for the suspected antigen. It should be noted that in many cases a specialist laboratory is required, as there are no strong commercial extracts for some antigens, as in this case. Finally, it may be necessary to perform a specific bronchial provocation test in those cases where diagnostic doubts persist, where a definitive diagnosis in the workplace is required or if a new etiological agent appears, as described here.

Regarding the etiological agent, in Spain there are 2 varieties of starlings: *Sturnus unicolor* and *Sturnus vulgaris*. Their population has increased remarkably due to their high reproductive capacity, the decline of natural predators and the remains of food in fields near cities (vegetables, cereals, fruits and small insects). They have become
a pest in many cities and are becoming a health hazard. At dusk, thousands of starlings come from the country in flocks and spend the night in trees or the roofs or cavities of houses. They damage rooftops and street furniture, as well as making excessive noise and producing large amounts of malodorous acidic droppings which dirty the buildings and parks where they live.

Given the significant presence of starlings in many southern European cities and the large number of people exposed to them in parks, public gardens and adjacent buildings, this type of non-mass exposure may condition NH and subsequent pulmonary fibrosis or chronic obstructive pulmonary disease of unknown etiology in susceptible people. Given the not infrequent findings of non-mass exposure in the etiology of NH, clinicians must be very careful to study the etiology of interstitial lung diseases, especially those that are diagnosed as idiopathic pulmonary fibrosis, non-specific interstitial pneumonia and cryptogenic organising pneumonia, which are all forms related to previously having NH.

References