Bird Fanciers Lung: Clinical and Pathological View

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Abstract

Two female patients aged 53 and 44 years were admitted to hospital with back pain, chronic cough and malaise. Both patients had radiological findings of interstitial lung disease and both had a history of exposure to budgerigar antigens for a number of years. Pathological examination of their lung tissues were compatible with hypersensitivity pneumonitis (HP). Based on these findings, a diagnosis of "Bird Fanciers' Lung" (BFL) was made in these two patients. Since breeding birds at home is quite frequent in Turkey, it is important to be aware of health problems which may arise from this type of hobby.

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Introduction

Hypersensitivity pneumonitis (HP) or extrinsic allergic alveolitis are terms which designate a group of lung diseases caused by the inhalation of a wide variety of different materials that are usually organic and always antigenic (1). Affected individuals have an abnormal sensitivity to the antigen, which, in contrast to asthma, involves primarily the alveoli (2).

Bird Fanciers' Lung (BFL), which is a subset of HP, is thought to be related to exposure to avian proteins present in the dry dust of the droppings and sometimes in the feathers of a variety of birds. Birds such as pigeons, budgerigars (parakeets), parrots, turtle doves, turkeys and chickens have been implicated (3).

Besides the type of the antigen, patterns and duration of exposure seem to play an important role in the development of the hypersensitivity process. Those who look after many birds experience intermittent exposure to high concentrations of antigen at intervals of 1 or 2 weeks while cleaning the lofts or cages and typical acute disease may develop in these individuals. Those who keep one or two birds are exposed more or less continuously to low concentrations of antigen and these individuals develop a gradual and insidious disease. This insidious onset is marked by progressive dyspnea on effort. Severe fibrosis is likely to be established by the time the patient is first seen (3). After a heavy exposure, acute symptoms begin 4 to 6 hours later with malaise,

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chills, fever, non-productive cough and chest discomfort. In the chronic form there is usually anorexia and weight loss besides dyspnea and progressive interstitial fibrosis is the most disabling feature of the disease (4). This condition is occasionally fatal within 2 to 3 years of the onset (5).

On auscultation of the lung, loud, dry crackling rales are heard at the bases. Pulmonary function tests show reduced lung volumes and impaired gas transfer (4). Immunological reactions are thought to have a critical role in the pathogenesis of the disease. Tests for IgG antibody to the offending agent are strongly positive either by qualitative double diffusion in agarose or by quantitative ELISA tests (4) However, precipitins against particular antigenic components may mediate reactions in one individual but not in another. Thus, they are often found in healthy (asymptomatic) persons who have had similar exposure as well as in those with disease (3).

Lymphocytes are the predominant cells in bronchoalveolar lavage (BAL) fluid, with CD8+ cells in greater numbers than CD4+ cells, although activated macrophages and increased numbers of mast cells are also present (4). In the transbronchial parenchymal biopsy specimens of the lung in the acute disease, initially there is edema of the lungs with a predominantly lymphocytic infiltration. Within the first two weeks, as edema subsides, numerous caseating, sarcoid type epitheloid granulomas with Langhans' giant cells develop. The granulomas sometimes contain birefringent bodies. Granulomas occur in alveolar walls and in the walls of terminal and respiratory bronchioles, which they may almost obliterate. They resolve in about 3 or 4 months and are replaced by a substantial lymphocytic infiltration of alveolar walls and scattered lymphoid follicles with germinal centers. In the chronic disease there is collagenous fibrosis of alveolar walls around the terminal and respiratory bronchioles and perivascular zones. Granulomas are absent but a few may still be found in the chronic period. Foreign body giant cells containing birefringement material of uncertain identity may also be present (3).

Radiograms of the chest during the acute phase may be normal but characteristically show bilateral patchy 1 to 2 cm pneumonic infiltrates in the mid lung fields. In the chronic form, radiograms are often normal in the early years of the disease, but later typically show micronodular infiltrates and reticulation. After years of repeated exposures, the appearance is one of end-stage pulmonary fibrosis and honeycombing. In cases in which the chest radiogram is normal, high resolution computed tomography (HRCT) may be needed to demonstrate the pulmonary pathology (4).

Table 1. Diagnostic criteria of hypersensitivity pneumonitis

Major Criteria

1. Symptoms compatible with HP
2. Evidence of exposure to appropriate antigen by history or detection in serum and/or BALF antibody
3. Findings compatible with HP on chest radiogram or HRCT

Minor Criteria

1. Bibasilar rales
2. Decreased diffusing capacity
3. Arterial hypoxemia, either at rest or during exercise
4. BALF lymphocytosis (if BAL performed)
5. Pulmonary histological changes compatible with HP (if lung biopsy performed)
6. Positive "natural challenge" reproduction of symptoms and laboratory abnormalities after exposure to suspected environment

The prevalence of BFL was found to be between 0.5 and 7.5% among budgerigar fanciers in a survey from Britain (6) and it was found to be between 8 and 30% among members of pigeon- breeding clubs in other surveys (1). Different prevalence figures have been reported in different countries. Although this disease entity has usually been reported in risk groups, sporadic cases can also be detected, especially in housewives who are responsible for the cleaning of the cages. HP can rarely have a serious outcome. Lethal cases were reported, one of which was from Japan (7).

Symptoms compatible with HP, evidence of exposure to appropriate antigen by history or detection of antibody in serum and/or broncho-alveolar lavage fluid (BALF) and radiological clues of HP on chest radiograms or HRCT constitute the major diagnostic criteria, along with other mentioned minor criteria (1) (Table 1). For the diagnosis of HP in suspected patients, all of the major criteria and at least four of the minor criteria must be fulfilled and other diseases with similar symptoms such as sarcoidosis or idiopathic pulmonary fibrosis must be ruled out (1).

HP causes chronic inflammatory changes in lungs necessitating corticosteroid therapy and has a risk of ending in overall fibrosis. Systemic corticosteroids are sometimes required to treat severe disease (1). But the most important therapy of BFL is total avoidance of bird exposure, the natural antigen. This measure may decrease the need for corticosteroid therapy.

Case Report

Case 1

A 53-year-old woman was admitted to hospital with symptoms of cough, high fever and back pain especially predominant over the left scapula, which had been present for 2 months. The patient did not have a history of smoking. On auscultation, end inspiratory rales were heard bilaterallly in the basilar areas the lungs. There were no other specific

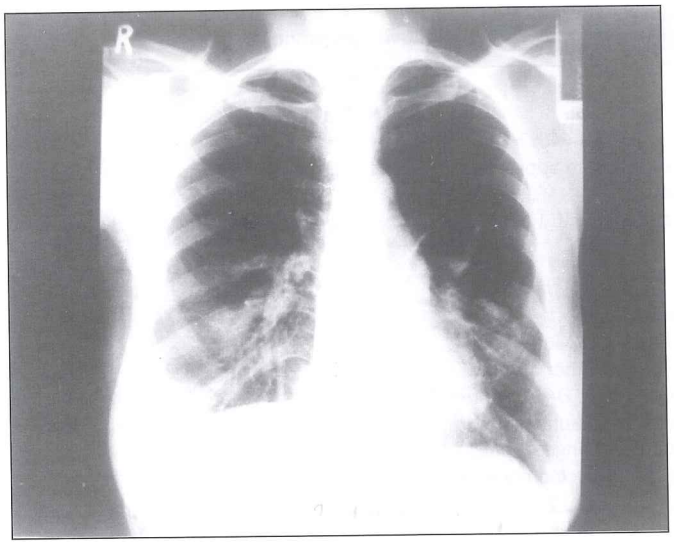

Figure 1. *Bilateral patchy infiltrations in lung parenchyma (Postero-anterior chest X-ray).*

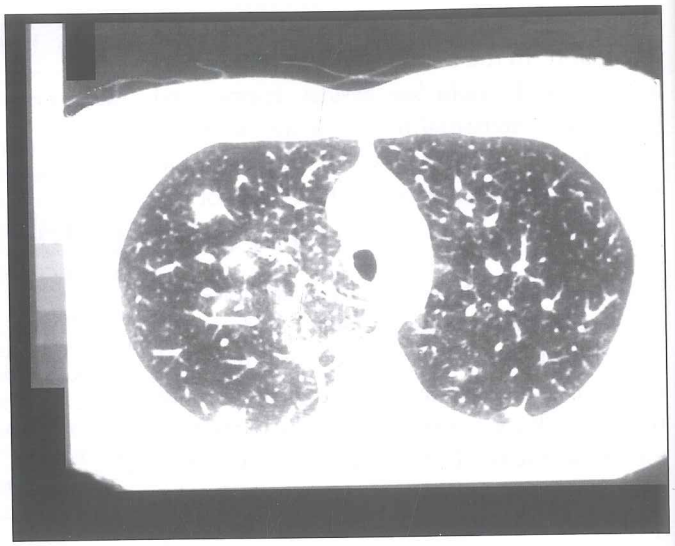

Figure 2. *Bilateral patchy infiltrations in lung parenchyma (thorax HRCT).*

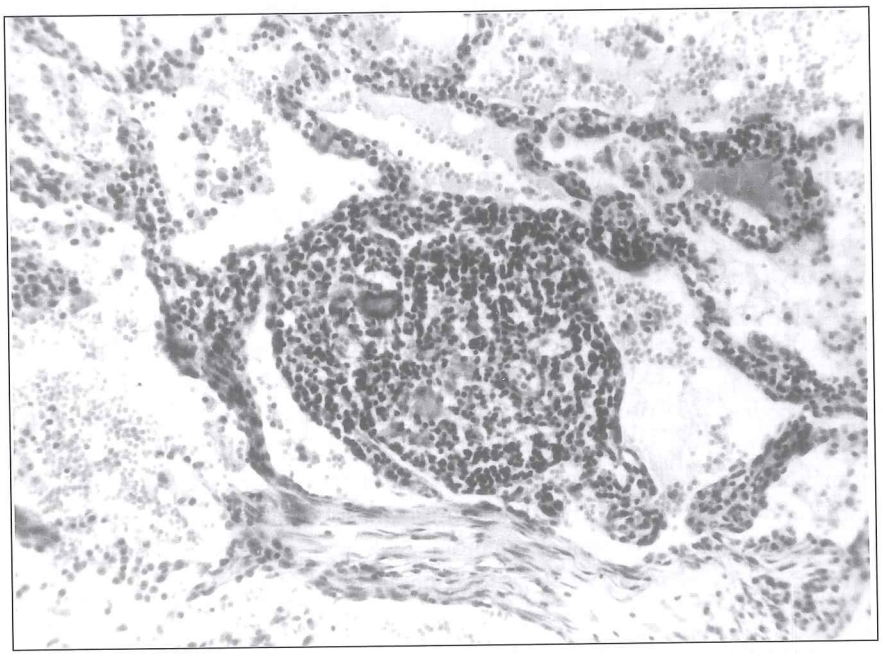

Figure 3. *Inflammation in alveolar septae with formation of a loose granulomatous structure including giant cell.*

findings on physical examination. The only abnormality in the laboratory parameters was a high erythrocyte sedimentation rate (72mm/h). Tests for collagen vascular disease were negative. Arterial blood gases revealed no hypoxemia. Results of diffusion capacity tests were: DLCO=12.4 ml/min/mmHg (56%) and DLCO/VA=2.93 1/min/mmHg (48%). Fiberoptic bronchoscopy was performed. There was no endobronchial patology and in BALF cytology there were 68.8% lymphocytes, 15.6% polymorphonuclear leucocytes, 14.5% eosinophils and 1.1% alveolar macrophages. There were bilateral patchy infiltrations in lung parenchyma visible both in the chest radiograms and in HRCT of the thorax. (Picture 1,2)

Further evaluation of the history of the patient revealed exposure to budgerigar antigens at home. The patient was a housewife and had been breeding a budgerigar at home for four years. Repeated chest radiograms showed that the interstitial densities change places. Video assisted thoracoscopic surgery was performed to the nodular lesions in the left lung. Microscopic evaluation of the lung tissue showed inflammation in alveolar septae with formation of a loose granulomatous structure which included giant cells (Picture 3).

The patient's complaints ended and the radiological infiltrates decreased after starting oral corticosteroid therapy and sending the bird away.

Case 2

A 44-year-old woman was admitted to hospital with long lasting cough, sputum, malaise and anorexia. She also had dyspnea on exertion and sharp pain on the left side of her chest when breathing deeply. She did not have a smoking history. The patient was known to be breeding a budgerigar at home for five years. There were end inspiratory rales on both lung bases. No abnormality was found in her routine blood tests and arterial blood gases. Diffusion test was not performed. The chest roentgenogram showed bilateral diffuse micronodular densities which were also evident in HRCT. No endobronchial pathology was present in fiberoptic bronchoscopy but there was lymphocytosis in BAL fluid (50% lymphocytes, 20% alveolar macrophages, 10% polymorphonuclear leucocytes and 10%

eosinophils). The microscopic examination of the transbronchial parenchymal biopsy specimen revealed bronchiolitis, interstitial inflammation and fibrosis with histiocyte clusters. After avoidance of exposure to offending antigens by sending the bird away and starting corticosteroid therapy, the patient became free of symptoms. All these findings led us to a diagnosis of BFL.

Discussion

Different types of work areas and different antigens related to HP have been reported from all around the world. In Turkey, pigeon antigens, mould fungi and thermophilic actinomycetes have been reported to be associated with HP in malt workers and housewives. (8,9,10,11). However, there are probably other areas and hobbies to be investigated in relation to their similar harmful effects to lungs.

In our cases there were prominent clinical, radiological and histological clues for the diagnosis of HP. In both of these housewives, all of the major criteria (symptoms compatible with HP, evidence of exposure to appropriate antigen by history and findings compatible with HP on chest radiograms and HRCT) for diagnosis were present. Of the minor criteria, bilateral basilar rales, BALF lymphocytosis and pulmonary histological changes on biopsy compatible with HP were present. Pathological evaluation showed findings compatible with the chronic phase of the disease. Diffusion capacity was decreased in the first patient but was not investigated in the second. Natural challenge test was not performed and arterial hypoxemia was not present in either patient. Arterial hypoxemia is thought to be a later manifestation of the disease. Thus, four minor criteria were present in our first patient and three minor criteria were in our second patient. With the exclusion of any other pathologies, we made a diagnosis of BFL in both patients and planned our treatment accordingly. Avoidance of bird exposure and corticosteroid therapy led to significant improvement in both patients.

It is known that in these patients, when exposure is avoided before permanent radiological or physiological abnormalities develop, the prognosis is excellent, with little evidence of long term ill effects. On the other hand, if exposure persists, some patients will progress to diffuse pulmonary fibrosis with resultant cor pulmonale and death (12). Although our cases were exposed continuously to low concentrations of antigen at home and developed a gradual and insidious disease, they were in a relatively earlier stage and showed no severe effects such as end stage lung disease or cor pulmonale.

It is important to note that in the past few years, the hobby of breeding birds at home became more popular in Turkey. There are also an increasing number of individuals who are exposed to bird antigens in bird-breeders' clubs or in their own workplaces such as pet shops. This situation obviously leads to HP as a health problem. It should be pointed out that the chronic form of HP may be misdiagnosed as idiopathic pulmonary fibrosis if a good history is not taken and immunological and BALF testing is not evaluated (13). Therefore it is important to take a more detailed history of hobbies especially in patients with prominent clinical and radiological signs of interstitial lung disease and consider HP as a not too far-fetched entity in the diagnostic work-up.

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