Bird Fancier's Disease Due to Exposure to Birds Via a Desert Cooler

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ABSTRACT

Hypersensitivity pneumonitis (HP) is an immunologically mediated pulmonary disease caused by various organic particles and some non-organic chemicals. HP is mostly associated with some occupations such as farming and bird breeding. We report a case of hypersensitivity pneumonitis in a housewife without any prior history of occupational exposures. The disease was developed due to indirect exposure of the patient to doves antigens via a desert cooler ducts.

Keywords: Bird fanciers' disease, housewife, hypersensitivity pneumonitis, organic particles

Case Report

INTRODUCTION

Hypersensitivity pneumonitis (HP) or extrinsic allergic alveolitis which is also named by such other names as farmers' lung, bird fanciers' lung or humidifier's lung is an immunologically mediated pulmonary disease caused by various factors (mostly organic particles and some non-organic chemicals) from various sources.[1-6] Despite extensive studies, the exact immunologic mechanisms of HP are not fully known.[7] This disease is characterized by production of specific IgG and proliferation of CD_8^+ lymphocytes.[1,2] Some non-allergic, inflammatory reactions are considered as the differential diagnoses of this disease, naming some of them: Inhalation fevers, toxic alveolitis, and organic dust toxic syndrome.[8-10] HP is mostly associated with some occupations such as farming, bird breeding, wood working, etc.[11] It is also reported that HP may be associated with air conditioning and humidification systems.[6,12] In this type of HP, usually thermophilic actinomycetes are responsible.[7]

One of the forms of HP is caused by exposure to the antigens of birds. The products of many kinds of birds such as pigeons, canaries, parrots, doves, chickens, turkeys, ducks, and geese are responsible for this disease.[13]

There are multiple diagnostic criteria for HP. One of these criteria is as following: Appropriate exposure, exertional dyspnea, inspiratory crackles, and lymphocytic alveolitis which is not required provided that at least two of the following criteria are met: Recurrent febrile episodes, infiltrates on chest radiograph, decreased DLCO or improvement away from the exposure.[14]

We report a case of hypersensitivity pneumonitis in a housewife without any prior history of occupational exposures.

CASE REPORT

A 57-year-old female referred to Imam Khomeini hospital with chief complaint of exertional dyspnea. She lived in the southern parts of Tehran. Her dyspnea which began about 4 months before her admission to hospital was gradually progressive and was accompanied by non-purulent cough. At the time of her admission she had some problems for performing activities of daily living. Dyspnea did not occur during resting and sleep and was not related to the time of eating. She didn't complain of orthopnea and paroxysmal nocturnal dyspnea (PND). The history of hemoptysis and exposure to tuberculous patients was negative.

In her past medical history, she was under treatment for hyperlipidemia (atorvastatine) and hypothyroidism (levothyroxine). She didn't mention the history of any other respiratory or cardiovascular diseases. She didn't have any hypersensitivity to drugs or nutritious substances and the history of seasonal allergy was negative as well.

Her family history was negative for respiratory and cardiovascular diseases. History of smoking, alcohol consumption and drug abuse was negative. She was a housewife without any specific occupational exposures. Her husband was a farmer who had been died due to cardiovascular diseases 9 years ago. She lives in the first floor of a 3-storey buildingin Tehran (She didn't use to live in her husband's farm). Her house was not near any industrial plants.

In the physical examination, she was completely alert. Her vital signs included: Pulse rate: 78/min, respiratory rate: 20/min, temperature: 37.1°C, blood pressure: 110/75 mmHg. Her body mass index was 35.5 kg/m^2. Lung field auscultation revealed inspiratory crackles in the bases of both lungs, especially right lung and to a lesser intensity in the mid-zones of both lungs.

In arterial blood gas analysis, O_2 saturation was 78%, and other parameters were normal. The results of complete blood count included:

WBC: 10900/mm^3
RBC: 6.66×10^6 ml/mm^3
Hemoglobin: 18.6 g/dl
HCT: 58.9%
MCV: 88 fl
MCHC: 28 pg
Plt: 230000/mm^3
MPV: 8.5 fl
RDW: 15.1%
Poly: 63.3%
Lymph: 28.3%
Mono: 4.6%
Eos: 3.3%
Baso: 0.5%
Anisocytosis: Slight

The result of spirometry showed a severe restrictive pattern (FVC = 1 L, 47.3% of predicted) which was approved by body plethysmography (TLC = 2.97 lit, 76% of predicted). Diffusing capacity (DLCO) was decreased (73.9%). She showed a significant response to bronchodilator administration hence a 21% and 17.7% increase in FVC and FEV_1, respectively. The purified protein derivative (PPD) test for TB was negative.

Exercise stress test was performed but not completed because of her dyspnea. The result of transthoracic echocardiography was as following:

PAPs: 40 mmHg, LVEF = 55%, Normal RV size and systolic function, diastolic dysfunction grade 2, trivial MR, and mild TR

Chest X ray showed reticulo-nodular pattern in both lower lung fields (despite a normal chest X ray 6 months before). Figure 1 shows two chest X rays before and after onset of disease.

High-resolution computed tomography (HRCT) scan results in inspiration and expiration was as following: Generalized ground glass opacities in both lung fields and hypo-dense areas due to focal emphysema. Multiple lobular, patchily dispersed areas of air trapping throughout pulmonary fields, on expiratory views, mostly accompanied by mosaic perfusion on the corresponding inspiratory phase views. Interlobular septa were unremarkable. Neither bronchiectasis nor bronchiolectasis was noted, on either side. Interlobular and peribronchchovascular interstitium were ordinary depicted. The main pulmonary artery was dilated. Cardiomegaly was depicted [Figure 2].

After observing the results of paraclinic tests, because of high suspicion of hypersensitivity pneumonitis, another history with details was

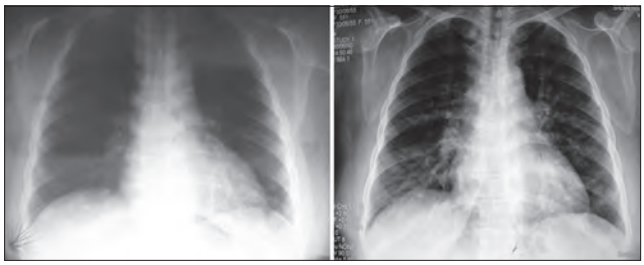

Figure 1: Chest X ray before (left) and after (right) onset of disease

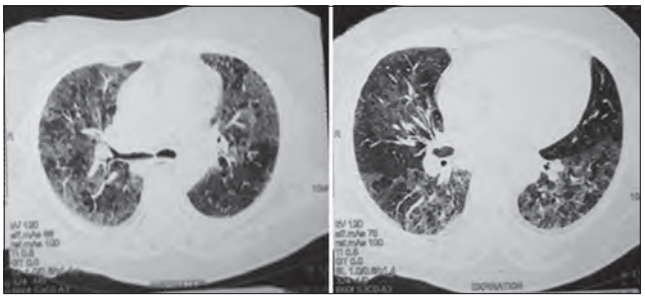

Figure 2: HRCT of the patient in inspiration (left) and expiration (right)

obtained from her. She mentioned that her son keeps a considerable number of birds (doves) on the roof of the building and their cage was beside the desert cooler of the building. So she fulfilled the criteria of HP and after removing the exposure her symptoms subsided and her pulmonary function improved significantly hence FVC = 1.79l, 82% of predicted and FEV_1 = 1.42l, 79% of predicted.

A written informed consent was obtained from patient to publish her case report with her radiographic images. This consent is available (in Persian) for the journal editorial.

DISCUSSION

We report a case of hypersensitivity pneumonitis due to the indirect exposure to organic particles of a kind of bird which is native in Iran and is called laughing dove. The disease was diagnosed in a housewife without any direct exposure to birds although further investigations showed an indirect exposure via the ducts of a desert cooler.

The clinical symptoms of the patient (inspiratory crackles and exertional dyspnea) as well as pulmonary function tests (especially a mild reduction in DLCO) and the radiological findings suggested the diagnosis of HP; so two kinds of HPs were suspected for her: Humidifiers lung and bird fanciers disease.

In Iran, most buildings are cooled by a desert cooler. This device is located on the roof and forces cool air via some ducts to the building. They were using this desert cooler for more than 10 years but from 6 months ago (in winter) the doves cages were transformed to the roof beside the desert cooler, and in the spring when the desert cooler was working, the particles were transformed to the building inside.

The disappearance of the symptoms and improvement of the pulmonary functions after eliminating the exposure (transforming doves cages to another place) confirmed the diagnosis of Bird Fancier's Disease (BFD). In the literature, there are some case reports of bird fanciers' disease mostly in occupational settings and due to exposure to pigeons, hens, canaries, and some native birds such as finches and siskins.[3,8,13] We couldn't find any case reports of BFD due to doves and especially laughing dove.

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