Hypersensitivity pneumonia in a schoolchild admitted to the hospital's asthma clinic

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Bird fancier's lung (BFL), also called bird-breeder's lung or pigeon-breeder's disease (PBD), is a hypersensitivity pneumonitis (HP) that is rare in children. A 9-year-old male patient complained of cough, dyspnea and chest pain, in his examination his lips were cyanosed and his SaO2 was 86% at room air. Bilateral crepitant rales and sibilant rhochi were also detected. In his thoracic computerized tomography, the ground glass areas were noted in both lungs. In his medical history, it was discovered that he lived above an office in which birds and bird manure were merchandised. Precipitant antibody level was determined. Since the levels of precipitant antibody were high and symptoms reappeared with natural provocation, our patient was diagnosed with PBD. The case diagnosed as BFL was presented in this study as an HP which should be considered in the differential diagnosis of children with respiratory distress syndrome and asthma clinic.

Key words: asthma, child, hypersensitivity pneumonitis, pigeon-breeder's disease.

Hypersensitivity pneumonia (HP) is a hypersensitivity reaction that develops as a result of an inhalation of various chemical substances or organic dust particles. It is also known as an extrinsic allergic alveolitis[1-4]. Over 200 antigens leading to HP were defined[2–5, 6]. Bird and animal proteins cause HP types, such as pigeon-breeder's disease (PBD) and furrier's lung. Though pigeons are the most frequent type of bird that causes HP, it can also form with parrot, lovebird, chicken, duck, goose, and turkey proteins[7]. PBD is uncommon in children. Morell et al.[8] reported that the rate of children younger than 15 made up 8% in their study, which is the broadest serial with 86 cases. The case of a school age patient admitted to our asthma clinic but diagnosed as PBD is presented below.

Case Report

A nine-year-old male who had a cough, dyspnea on exertion, and chest pain complaints for four months was evaluated as an asthma patient since he had partially benefited from an inhaled steroid and a pollen susceptibility was determined in his skin test. However, he was admitted to our center when his complaints continued. In his examination, his lips were cyanosed and SaO2 was at 86% at room air. Crepitant rales and bilateral sibilant rhonchi were heard in his two lungs. Patchy nodular infiltration was determined in the patient's chest radiograph. In his thoracic computerized tomography, the ground glass areas were present in both lungs (Fig. 1). Pulmonary hypertension was not revealed in the echocardiography. An inhaled steroid treatment was initiated, and his hypoxia partly recovered, beginning from the seventh day of the treatment. In his detailed medical history, it was discovered that he lived above an office in which birds and bird manure were merchandised. A further examination was demanded in order to determine the precipitant antibodies. The patient, whose symptoms regressed, was discharged from the hospital providing that he would come for a

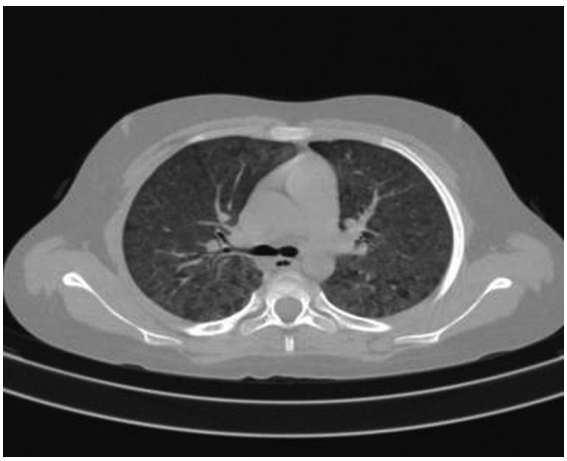

Fig. 1. Ground glass areas in both of the lungs as seen in the thoracic computerized tomography

control once a week while his antibody results were awaited. Although he was warned, the patient went back to the house where a bird lived and he was taken to the hospital with respiratory distress.

Pigeon-breeder's disease was considered due to the reappearance of the auscultation symptoms and the decrease in the pulmonary function tests (PFT) after the exposure. The precipitant antibody levels for pigeons and budgerigars were found to be high (pigeon IgG>200 [0–38] and budgerigar IgG >200 [0–8]). Since the antibodies were positive and the symptoms reappeared through natural provocation, our patient was diagnosed with PBD. His treatment was 60 mg/day of systemic steroids. The dosage of the steroid was decreased after the symptoms got under control (Table 1). Regression was observed in the patchy nodular filtrates located in the chest x-ray taken on day 42 of the patient's follow-up. The follow-up and the treatment of the patient are continuing.

Discussion

Hypersensitivity pneumonia is developed through exposure to various antigens, and it develops through type III and type IV allergic reactions in the bronchi and alveoli[4,6,9]. Though the immune mechanisms explain the pathogenesis of the disease, why the disease does not develop in everybody who is exposed to the antigen remains a mystery[10,11]. HP is clinically classified as acute, subacute, and chronic[3,4,6,8]. The acute form develops in susceptible people through the intermittent exposure to high amounts of the antigen. The symptoms occur 4–8 hours after the exposure and mimics an acute viral infection. The most common symptoms are high fever, chills, malaise, myalgia, a nonproductive cough, and dyspnea. The subacute form develops in susceptible people who continuously inhale low concentrations of the antigen. The symptoms progress deviously. In the absence of acute symptoms, the progressive cough and dyspnea exist for several weeks and months. In the acute and subacute forms, the symptoms are resolved by moving the person away from the environment[3,12].

Hypertension and cor pulmonale may develop in chronic form. Our patient with a repeated exposure history had come to us with chest pain on exertion and coughing complaints continuing for the past four months. Bilateral sibilant rhonchi, rales, and hypoxemia were present in the application, but the pulmonary

Table I. Treatment Plan and PFT Results of the Patient

	SaO2(%)	FVC(%)	FEV1(%)	FEV1/FVC	PEF(%)	Starting treatment
Appllication	86	58	68	114	86	Inhaled steroid
7.day	93	56	66	114	86	Inhaled steroid
14. day (Natural provocation)	90	54	60	108	86	Oral steroid (60 mg/day)
21. day	94	68	79	114	100	Oral steroid(30 mg/day)
42. day	97	74	86	113	110	Oral steroid(15 mg/day)
84. day	98	82	85	104	109	Oral steroid(7.5mg/day)
120. day	99	84	92	106	100	Oral steroid discontinued Inhaled steroid continued

SaO2: Arterial blood oxygen saturation, FVC: Forced vital capacitiy, FEV1: Forced expiratory volume in one second, FEV1/FVC: Forced expiratory volume in one second / Forced vital capacitiy (The ratio of the two volumes), PEF: Peak expiratory flow.

hypertension did not exist. As a result, we thought that our patient was in a subacute form[3,12]. In the chronic form, there is reversible lung damage. Progressive dyspnea, cough, restlessness, and weight loss are the main symptoms. Pulmonary hypertension and cor pulmonale may also develop. Above all, the symptoms are not resolved by discontinuing the exposure to the antigen[3,15].

There is still no specific test or biomarker for the diagnosis of this disease. There are multiple diagnostic criteria for HP. The criteria is: appropriate exposure, exertional dyspnea, inspiratory crackles, and lymphocytic alveolitis. Lymphocytic alveolitis is not required providing that at least two of the following criteria are met: recurrent febrile episodes, infiltrates on the chest radiograph, decreased diffusing capacity of the lung for carbon monoxide (DLCO), or an improvement away from the exposure[13]. One of the characteristic symptoms of HP is the existence of precipitant antibodies. The pigeon and budgerigar precipitant antibody levels in the serum of our patient, who was exposed to a variety of birds, were found to be high.

Pigeon-breeder's disease in children is rare compared to adults and as in the form of case reports. The youngest case reported is at age 4.4 years. In the publication, which reported two more patients younger than seven years of age, along with this patient, it was emphasized that the anamnesis was deepened since the existence of a long-lasting cough brings this disease to mind[14]. Up to the present, there have been three childhood cases reported in Turkey. One case was at age 10; after 6 years, another case was at age 5.5 years. These cases were reported from the same center[15,16]. Ceviz et at.[17] reported the other Turkish case. The same disease is detected in the symptomatic mother and asymptomatic sibling of the child patient with a PBD diagnosis who came with pulmonary hypertension. This indicated to us the necessity of examining all of the members of the family in which an index case is detected.

The most significant step of the treatment is the elimination of the antigen. The steroids also have a key position in the treatment. There is no consensus about the duration of the treatment. However, there are cases reported with treatment durations as long as one year[14]. While we had a clinical response with an inhaled steroid at the beginning, we had to continue with a systemic steroid after the reexposure of the patient. We think we have been able to decrease the dosage of the systemic steroid owing to the usage of the inhaled steroid.

In conclusion, the environmental exposure should be examined and HP should be kept in mind when the applicant children with an asthma profile also have serious hypoxia and in the presence of radiological findings.

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