

RECURRENT PNEUMONITIS IN A 10 – YEAR – OLD GIRL WITH PIGEON BREEDER'S DISEASE*

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SUMMARY: Dilber E, Özçelik U, Göçmen A, Mısırlıgil Z, Kiper N. (Chest Disease Unit, Department of Pediatrics, Hacettepe University Faculty of Medicine, Ankara, Turkey). Recurrent pneumonitis in a 10-year-old girl with pigeon breeder's disease. Turk J Pediatr 1997; 39: 541-545.

Hypersensitivity pneumonitis is an allergic disease resulting from sensitization to inhaled organic dust, usually manifest with recurrent bouts of cough, dyspnea and fever. Most of the reported cases concern the adult population. The disease is rarely seen in the pediatric age group, as stated by most of the reviews on the subject. In this article, a case with hypersensitivity pneumonitis is described. Precipitating antibodies to avian protein were demonstrated in serum from a girl with recurrent bouts of pneumonitis which was otherwise unexplained. *Key words: hypersensitivity pneumonitis, pigeon breeder's disease, precipitating antibodies.*

Hypersensitivity pneumonitis or extrinsic allergic alveolitis is an allergic disease resulting from the sensitization and recurrent exposure to any of a wide variety of inhaled organic dusts¹. Pigeon breeder's disease is the most common form^{2, 3}. Although it is rarely encountered in the pediatric age group, it should be considered in any child with recurrent or persistent and unexplained respiratory symptoms^{2, 4}.

Case Report

A 10-year-old girl with a history of recurrent pneumonitis was referred to our hospital from another center. She had no serious health problems until three months previously when she had developed pneumonitis with fever, dry cough, dyspnea, tachypnea and congestive heart failure. Chest x-ray, taken at the time of pneumonitis, disclosed diffuse nodular infiltrates with patchy infiltration along the peripheral areas (Fig. 1). She was then hospitalized in a local institute, treated with antibiotics, and was discharged upon full recovery. One month later, her symptoms recurred and she was hospitalized in the same institute for a second time. During the second attack, a computerized tomography (CT) scan of the

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thorax was done which revealed diffuse miliary infiltrates in addition to several patchy consolidations (Fig. 2). The patient once again responded to the nonspecific management with antibiotics and supportive therapy with full recovery. After her recovery from the second bout, she was sent to us for the etiological investigation of severe recurrent pneumonitis which seemed to be quite unexplained.

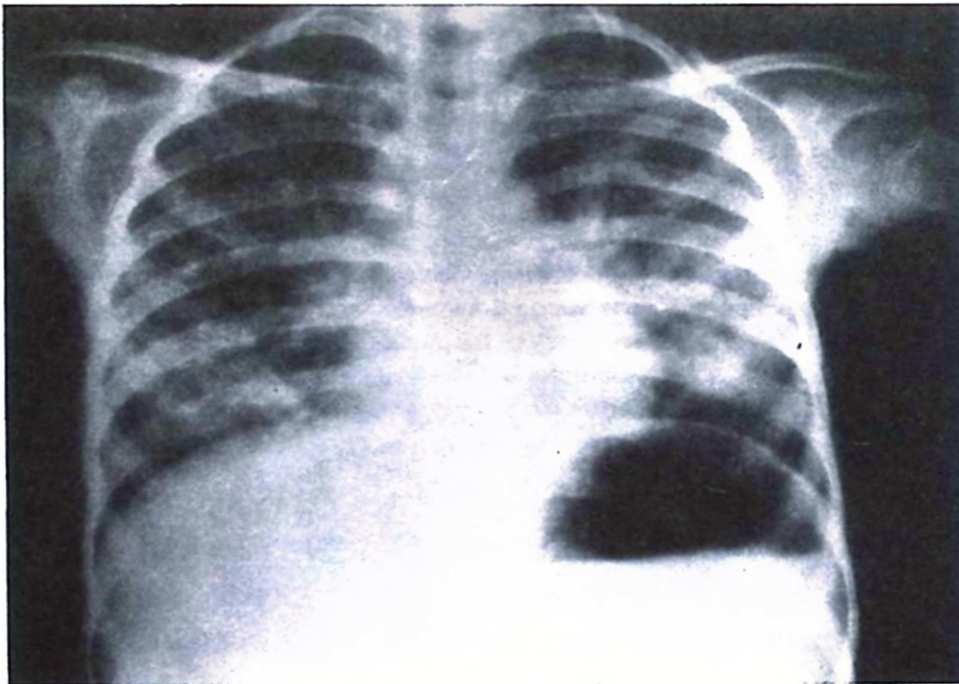


Fig. 1: Widespread nodular infiltrates and patchy infiltration prominent at the lung base.

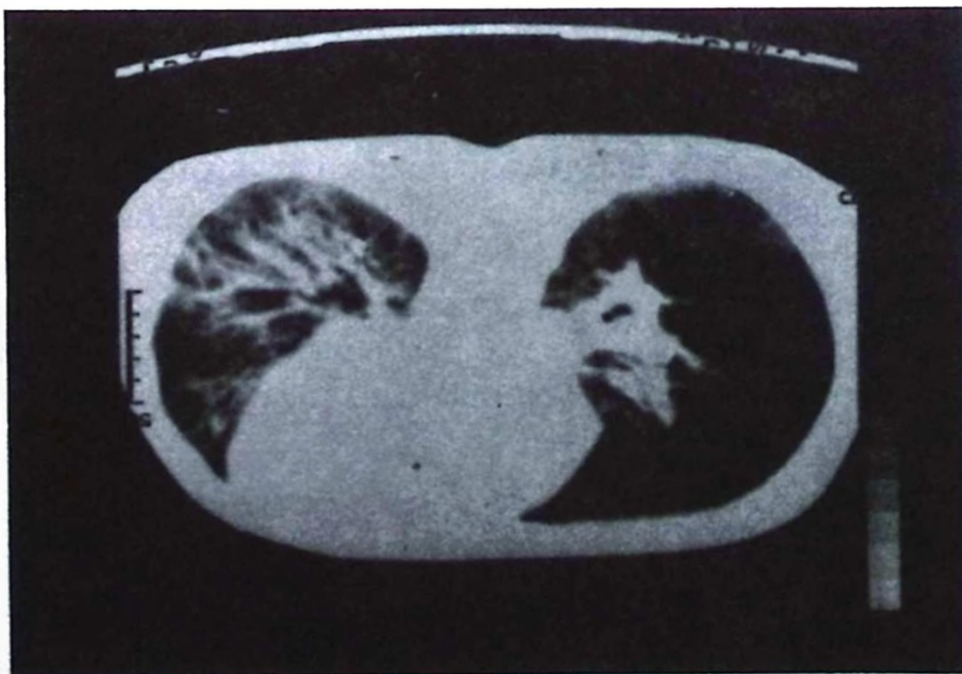


Fig. 2: Coronal section of thoracic computed tomography (CT). Diffuse miliary infiltrates are present, especially on the right side, with patchy consolidation.

Upon admission at our hospital she was in good health and was without symptoms. Her chest x-ray had reverted to normal (Fig. 3). Results of pulmonary function tests and serum immunoglobulin levels were all within normal limits. Upon inquiry, the family reported a history of pigeon breeding in the attic, dating back six months before the initiation of the patient's symptoms. Although the patient normally did not have direct contact with the birds as part of her daily activities, she did report having direct contact three to four hours prior to her symptoms on both occasions. Precipitating antibodies to avian protein were present in her serum (Fig. 4). The patient has been well since the removal of the birds from her house (12 months before the writing of this report).



Fig. 3: Chest x-ray of the patient after referral to us.

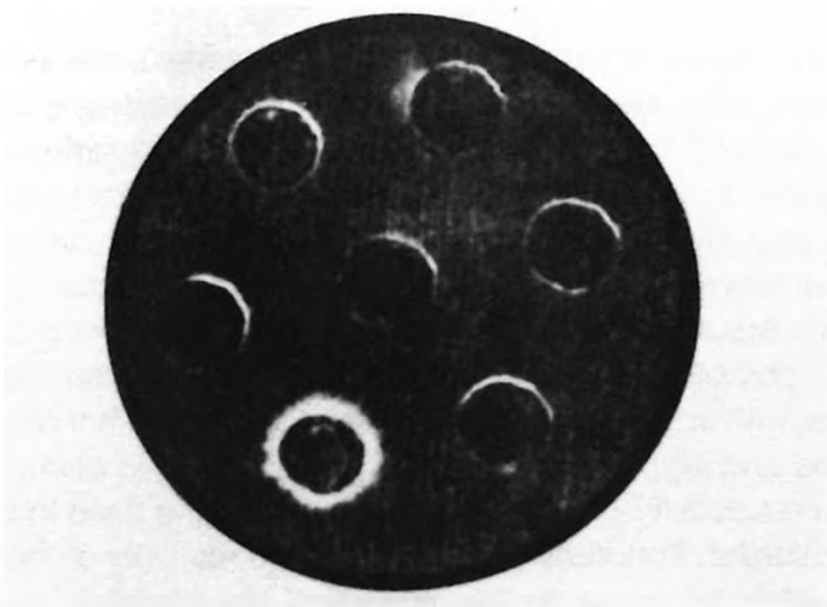


Fig. 4: Ouchterlony plate showing precipitating antibodies to avian protein in the patient's serum.

Discussion

Pigeon breeder's lung in children was first described by Reed et al.⁵ Exposure to a wide range of pigeon-derived materials, including extracts from droppings, serum, egg white, and feathers, induces a hypersensitivity pneumonitis. The disease is a diffuse, predominantly mononuclear inflammation of the lung parenchyma, particularly the terminal bronchioles, interstitium, and alveoli. The inflammation often organizes into granulomas and may progress to fibrosis^{1, 3, 4}.

It appears to be two distinct syndromes^{1, 2, 4-6}. In the acute form, chills, fever, malaise, dry cough and dyspnea usually begin four to six hours after exposure to the causative organic dust. Physical findings include rapid respiration and crepitant rales, usually without wheezing. In the chronic form, due to relatively less intense and continuous exposure to the offending dust, cough and dyspnea may be progressive without acute episodes. Dyspnea on exertion, anorexia, weight loss and fatigue may occur without fever, and positive findings may be limited to fine basilar rales and, rarely, clubbing of the fingers. With the above history and clinical findings, we determined our patient had an acute and recurrent form of the disease.

No single clinical feature or laboratory test is diagnostic of the disease. Usually the diagnosis is based on a combination of characteristic symptoms, physical findings, chest x-ray abnormalities, pulmonary function, and immunologic tests^{1, 2, 6}. The demonstration of precipitating antibodies to the pigeon antigens raises the suspicion of hypersensitivity pneumonitis, although it may not be diagnostic per se⁷. Reynaud et al.⁸ found a high sensitivity (86%), specificity (93%) and accuracy (92%) of precipitins in the diagnosis of pigeon breeder's disease. In our patient, precipitating antibodies to avian antigen were demonstrated in the serum.

As in other allergic disorders, the therapy in pigeon breeder's disease is immediate removal of the bird from the environment and careful avoidance of re-exposure to causative antigens^{1, 4, 6}. Steroids may be used for the treatment of acute and fulminating lung disease, as they may help subside the ongoing inflammation in the bronchioles and alveoli^{2, 6}. It is also reported that a fatal course may ensue in some cases presenting with an acute fulminant clinical picture, if steroids are withheld. Steroids are also believed to prevent the development of fibrosis, which is a late chronic complication in some cases. Our patient, who was admitted to the local hospital with severe dyspnea and heart failure, did not receive steroids, as her symptoms and signs were attributed to a nonspecific pulmonary infection rather than hypersensitivity pneumonitis. This is often the case in such children with the same disorder. Fortunately, she had a good recovery on both occasions, which may be attributed more to the removal of the offending antigens during her hospital stay rather than to the antibiotic regimen.

Considering the facts stated above, hypersensitivity pneumonitis in children should be borne in mind in the appropriate clinical setting, and the patient's history carefully checked, in order to prevent subsequent morbidity due to a delayed diagnosis.

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