COMMENTARY

A Custodian Cured the Doctor!

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ABSTRACT. Opinions expressed in commentaries are those of the authors and not necessarily those of the American Academy of Pediatrics or its Committees. Commentaries are not peer-reviewed. Pediatrics 2000;105(5). URL: http://www.pediatrics.org/cgi/content/full/105/5/e71; hypersensitivity pneumonitis.

ABBREVIATIONS. HP, hypersensitivity pneumonitis; IgG, immunoglobulin G.

In this month’s Pediatrics electronic pages, Sarvaas et al1 report on a family with extrinsic allergic alveolitis, also known as hypersensitivity pneumonitis (HP), to pigeon serum proteins. HP is an interstitial lung disease caused by an abnormal immunologic response to any of a wide variety of antigens of chemical or biologic nature, most commonly inhaled antigens of fungi, bacteria, and animal proteins of avian, bovine, porcine, or murine origin.2,3 The symptoms are usually intense and include nonproductive cough, dyspnea, chills, fever, myalgia, and malaise. End-expiratory rales over the lower lung fields are commonly noted. Chest radiograph usually shows parenchymal nodular densities, reticulation, and coarse bronchovascular markings. Exacerbations are often associated with prostration and marked leukocytosis and thus mimic acute pulmonary infection. The immunologic reaction affects primarily the interstitium and alveoli and comprises both the cell-mediated (T cells) and humoral immunity (specific immunoglobulin G [IgG] antibodies in immune complexes). The diagnosis is supported by the demonstration of high titers of serum-precipitating antibodies specific to the causative antigen. Systemic corticosteroid therapy brings rapid symptomatic relief. Early detection and total avoidance of the offending agent are associated with complete recovery. Chronic low-grade exposure might be associated with minimal symptoms but can slowly lead to pulmonary fibrosis and irreversible loss in lung function.

Most cases of HP are related to heavy occupational exposure. HP caused by inhaled avian serum proteins in bird droppings is commonly called bird-breeder’s lung or bird-fancier’s disease. Of special interest in the report by Sarvaas et al1 is that the antigen source was wild city pigeons nesting outside the family’s house. The initial unawareness of such information resulted in the misdiagnosis and rapid death of the first affected family member—the mother. Within a few months of the mother’s death, her 5 children developed severe respiratory illness that required hospitalization. Antibiotics did not ameliorate the symptoms and extensive evaluation did not reveal the nature of their illness. Inspection of the family’s house did not reveal any source of toxic or infectious agents. It was striking, however, to observe the abundance of pigeons nesting on the back of the house. Only then did the children mention that they often helped their mother manually clean the fire escape of bird droppings and feathers. Testing the children’s sera showed high titers of IgG antibodies to pigeon proteins, supporting the diagnosis of HP. Systemic corticosteroid therapy resulted in striking recovery and the pigeon nests were eliminated.

The report by Sarvaas et al1 highlights the difficulty in suspecting HP outside the occupational setting and the potential gravity of missing the diagnosis. In my experience, for example, it was easy to clinically suspect and serologically confirm HP in a patient who had multiple hospitalizations for acute pulmonary illness and his environmental history revealed the presence of 58 birds of various species in his house. Only after repeated counseling did his wife (reluctantly) give away 52 birds and keep 6! On the other hand, HP was not suspected in a colleague of mine, an allergist, who for several months had a distressing cough, the cause of which was not clear, and the chest radiograph showed minimal findings. He did not respond to multiple courses of various antibiotics. His symptoms tended to occur more at work, were worse on Mondays, and markedly improved after he went on vacation for 2 weeks. He seriously considered moving to another job. His symptoms were especially exacerbated whenever he entered a particular room in the clinic that has a full-blowing air conditioner vent. Inspection of the air conditioning unit, which was on the roof just above our clinic area, revealed the presence of several pigeon nests at the intake vent. It was only after the custodian had exterminated those nests and cleaned the vents that my colleague’s symptoms rap-
idly improved. Testing his serum for precipitating antibodies to avian serum proteins showed high titer to pigeon serum albumin. It is quite possible that the abundance of wild pigeons in certain cities might be causing HP more than is being realized. Another unusual case of HP, that was difficult to recognize, was that caused by inhaled soybean antigens in a scientist who was working on developing a veterinarian diet using soybean flour as one of the ingredients.  

The corollary is that it would be prudent to consider HP in patients who have pulmonary disease of unknown cause. Young age or the lack of history of exposure to a conventional source of an organic inhalant should not deter the physician from pursuing detailed information about the patient’s environment, which may include inspection. A high index of suspicion might lead to the culprit. It would be prudent to institute a trial of avoidance of the suspected factors while arranging to perform the relevant tests. The importance of expertise in carrying out those tests cannot be overemphasized.  

REFERENCES
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/content/105/5/e71.full.html