

Bird Fancier's Disease in a 50 Year Old Woman

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Abstract

Background: Although considered a rare disease, bird fancier's disease (BFD) is actually the most common type of hypersensitivity pneumonitis. It is a disease that is historically misdiagnosed and therefore the most common sequelae often include years of suffering and side effects, as in the case of this particular patient. **Case Report:** This is the presentation of a 50-year-old owner of a cockatoo, who for 25 years thought that she had persistent asthma. However, after being away from her bird while on a business trip, she experienced a reprieve of her symptoms followed by their exacerbation on returning home. This led to her being correctly diagnosed with BFD. **Conclusion:** Asthmatic patients should be asked about environmental exposures to potential allergic antigens, especially when the asthma symptoms are persistent and chronic in nature.

Keywords: Alveolitis, Animals, Antigens, Asthma, Cockatoos, Environmental Exposure.

Introduction

According to the American Lung Association, hypersensitivity pneumonitis is defined as “a disease of the lungs in which your lungs become inflamed as an allergic reaction to dust, fungus, molds or chemicals” [1]. Bird fancier's disease (BFD) is triggered specifically by antigens within bird feces or feathers [2]. According to a population-based study in the UK, the disease is rare and under diagnosed with an incidence of approximately one in 100,000 [3]. The case of a 50-year-old woman who was initially treated for asthma for 25 years but who was diagnosed as BFD will be presented. She experienced reprieve of her symptoms after being away from her cockatoo while on a business trip followed by an acute exacerbation of her symptoms on returning home.

Case Report

A 50-year-old female patient presented with exacerbation of respiratory symptoms for the last

two months. She first complained of the acute onset of chills, fever, and fatigue followed by a cough productive of thick green sputum that started upon her return home from a business trip. She had multiple visits to her physician and was treated with a course of azithromycin with prednisone, then a subsequent course of oral levofloxacin, followed by intravenous (IV) antibiotics and steroids through an outpatient urgent care center. Despite these interventions, no improvement in symptoms occurred so she went to the emergency department.

The patient has a past medical history of gastroesophageal reflux disease, mitral valve prolapse, peptic ulcer disease, and hypertension. She stated that she has had asthma for 25 years which has been progressive in nature and gets “bronchitis” at least twice a year. The patient lives in a house with her husband, her two dogs, and her pet cockatoo that has been in her care for the past 25 years. Her current medications include estradiol vaginal cream when necessary (PRN), albuterol PRN, pantoprazole 40 mg oral daily,

and montelukast 10 mg orally every bed time. She denied any use of tobacco, alcohol, or illicit drugs.

On physical examination her vital signs were temperature of 36.9°C, pulse of 74/min, respiratory rate of 18/min, and blood pressure of 157/90 mm Hg. Her ear, nose, and throat examination was normal. She had no carotid bruits or thyroid enlargement. Her lungs had diminished breath sounds with a prolonged expiratory phase with diffuse wheezing. There were no other abnormalities noted. She had a normal blood panel consisting of a complete blood count (CBC), comprehensive metabolic profile, blood urea nitrogen, creatinine, and Troponin I with the exception of a mild hypokalemia ($K^+= 3.3$). She also had a negative urinalysis, a negative blood culture, and a negative D-dimer in the emergency department. She was screened for a variety of autoimmune diseases and was negative for Scl-70, anti-SSA, anti-SSB, anti-Smith, and anti-dsDNA antibodies. Her anti-neutrophil cytoplasmic antibody (ANCA), myeloperoxidase, and proteinase 3 antibodies were also negative. However, she was positive for speckled ANA at a 1:320 dilution. She was tested for fungal antibodies and was negative for coccidioides, blastomycosis, histoplasma and aspergillus. Chest X-ray was significant for mild bilateral interstitial lower lobe nodular infiltrates. She was admitted to the hospital and started on IV vancomycin and piperacillin-tazobactam. While admitted, a bronchoalveolar lavage revealed benign bronchial epithelial cells, many histiocytes with vacuoles, and neutrophils in her right lower lobe and right upper lobe. At this time, she was diagnosed with hypersensitivity pneumonitis secondary to her pet cockatoo, and was given IV salmeterol and albuterol nebulizers. Her condition quickly improved and after 4 days a chest X-ray showed significant clearing of the nodules present upon admission.

The differentials that were considered were asthma exacerbation, pulmonary infectious processes, autoimmune processes, pulmonary embolism, and congestive heart failure. Her normal

CBC, negative fungal antibodies, and negative blood cultures ruled out an infectious process. Her autoantibodies were negative with the exception of speckled ANA which is incredibly non-specific. A negative D-dimer ruled out pulmonary embolism. She had no signs of fluid overload and her chest X-ray was not suggestive of any cardiac involvement.

The patient's chest X-ray and bronchoalveolar fluid analysis findings suggested that she was suffering from something more than just a mere exacerbation of her existing asthma. Characteristics of asthma on chest X-ray include bronchial thickening, hyperinflation, and focal atelectasis [4]. When bronchoalveolar fluid is obtained from a patient experiencing an acute asthma exacerbation, one would expect to find an elevated number of eosinophils and neutrophils [5]. Because the findings in this patient were inconsistent with the findings in asthma, hypersensitivity pneumonitis was considered as a diagnosis since the patient had reported that she had a cockatoo at home. In hypersensitivity pneumonitis, a micronodular or diffuse interstitial pattern is common on chest X-ray [6]. Additionally, Reynolds (2001) described the typically broncho-alveolar lavage findings in patients with hypersensitivity pneumonitis. At baseline, one can expect the fluid from these patients to have an elevated cell count comprised of approximately 70% lymphocytes and the majority of the remainder cells to be alveolar macrophages. When the patients are given a reprieve from exposure to the antigen and are then re-exposed to the antigen, total cell count will double and there will be a much higher predominance of alveolar macrophages to lymphocytes [7]. These findings were very consistent with the findings in our patients and combined with the rapid improvement of X-ray findings on admission (and removal of antigen exposure), lead to the diagnosis of bird fancier's disease.

She was discharged after 5 days. In the meantime, the patient and her husband were able

to find a new home for their cockatoo and deep clean their home. The patient had a follow up appointment with her internist three months later and stated her symptoms had resolved completely. In fact, she was able to cease all of her asthma medications.

Discussion

BFD is one type of hypersensitivity pneumonitis caused by the antigens of several different types of birds. The antigens are most commonly found in bird droppings but can also be found on feathers [3]. The pathogenesis of the disease is not completely understood, but is thought to be due to an exaggerated IgG response with subsequent proliferation of CD8+ lymphocytes that cause an inflammatory response in the alveoli, terminal bronchi, and interstitial lung areas [8]. The disease is extremely rare and many researchers believe it occurs via a "two hit" mechanism, in which the first hit is a genetic predisposition or environmental exposure and the second hit is exposure to the antigen itself [3]. Many individuals with the disease do not go on to develop symptoms. This is thought to be due to immune tolerance, where the regulatory T cells suppress the response of Th1 and Th2 cells [8].

BFD can present as acute, sub-acute, or chronic. Patients with acute disease present acutely ill with fever, shortness of breath, and malaise in the hours after exposure to the antigen [9]. Subacute cases develop after repeated low dose exposure to the antigen. Chronic cases are result of long term exposure and can result in irreversible damage in the form of pulmonary fibrosis [9]. The patient in this case presentation had a history, symptoms, and physical findings that suggest that she likely had an overlap of these multiple types. She was exposed to the allergens for 25 years and had low grade symptoms. After some time away from her bird, she developed symptoms of acute disease upon her return.

Diagnosing BFD can be quite challenging. The condition is rare and it mimics many other respiratory diseases such as alpha 1-antitrypsin deficiency, asthma, and sarcoidosis. Acute BFD will show ground glass opacities with micro-nodules on CT scan and lower lobe reticulo-nodular infiltrates on chest X-ray [9]. Patients with sub-acute or chronic disease may have findings suggestive of fibrosis [9].

Conclusion

The most important lesson to take home from this case is the importance of a detailed and comprehensive history and physical examination. Considering factors such as a patient's job occupation or environmental exposures can tell us a great deal about potential diagnoses. Additionally, it is important to search for a secondary cause of symptoms when diagnostic study findings are inconsistent with findings that are to be expected in patients with asthma. Any patient with a new or existing diagnosis of asthma should be asked about environmental exposures to potential allergic antigens, especially when the asthma symptoms are persistent and chronic in nature.

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