Case Report |  WVMJ OA

Pigeon Breeder’s Lung: A case report of hypersensitivity pneumonitis

Racine Gue, MD  
West Virginia University, Radiology Resident

Zalak Patel, MD  
West Virginia University, Visiting Clinician

Lana Winkler, MD  
West Virginia University, Assistant Professor, Section of Cardiothoracic Imaging

Corresponding Author: Lana Winkler, MD, PO Box 9235 Morgantown, WV 26506. Email: lwinkler@hsc.wvu.edu.

Abstract

Hypersensitivity pneumonitis (HP) is an inflammatory disease caused by repeated inhalation of antigens. HP can mimic a variety of clinical processes from life-threatening anaphylaxis to insidious malignancy, and thus is often misdiagnosed. It is crucial for the clinician to correlate relevant exposure to onset of symptoms, and for the radiologist to provide appropriate differential diagnoses for prompt treatment. Radiological findings, when present, may include ground glass opacities and mosaic attenuation on CT. Clinical improvement upon removal of antigen helps cement diagnosis and facilitate the treatment of antigen avoidance and corticosteroids. We present an interesting case of hypersensitivity pneumonitis, particularly Pigeon Breeder’s lung, in a young man exposed to birds in a research laboratory whose symptoms occurred much sooner than the usual timeframe reported in the literature.

Case presentation

A 28 year-old male presented to the emergency department with dyspnea, cough, intermittent fever and 20 lb unintentional weight loss over the past 5 months. Significant past medical history included moderate persistent asthma for which he was previously well-controlled with budesonide/formoterol, montelukast and albuterol as needed. Of late, his anti-asthmatic medications failed to provide any symptomatic relief. For social history, patient is a lifelong nonsmoker, working as a PhD student in a lab with pigeons and rats for two years. Clinical exam was remarkable for oxygen saturation of 92% on room air. Labs showed normal blood counts, normal metabolic panel, and a total serum immunoglobulin E level of 2.6 IU/ml (normal <100.0 IU/mL). Spirometry suggested a restrictive lung process. CXR (figure 1) did not identify any significant abnormality. However, CT chest (figures 2,3,4,5) demonstrated diffuse ground glass opacities with upper lobe predominant centrilobular nodules and mosaic perfusion. With thorough investigation and careful history taking, it was determined that patient suffered from hypersensitivity pneumonitis due to pigeon exposure. He was prescribed a tapering course of prednisone over 2 months and advised to wear a PAPR (powered air purifying respirator) whenever working in the lab. On follow-up visits, the patient showed marked symptomatic improvement with resolution of shortness of breath, cough, wheezing, and fever. Repeat spirometry showed significant improvement in restrictive pattern.

Discussion

The current case highlights one of the most common causes of hypersensitivity pneumonitis and provides the opportunity to discuss the pathophysiology, diagnosis and challenges thereof. In general, hypersensitivity pneumonitis is a respiratory syndrome caused by...
an allergic reaction to an antigen. Repeated inhalation of tiny particles (< 5 μm) allows the antigen to reach the lung parenchyma and evoke a hypersensitive immune response. The most common antigens causing HP are microbes and plant or animal proteins. This particular case was likely caused by near daily exposure to proteins found within the serum, feces, or feathers of pigeons. In fact, bird fancier’s lung (also called pigeon-breeder’s lung) is reported to be one of the most common types of hypersensitivity pneumonitis.

Clinical presentation of HP can range from acute, subacute, or chronic, and anywhere in between. Symptoms of all stages are vague and nonspecific, creating challenges for clinicians with regards to prompt diagnosis. Thorough occupational, social, and environmental histories with subsequent correlation of exposure and onset of symptoms is paramount in diagnosing HP in all stages. In acute hypersensitivity pneumonia, cough, dyspnea, and a flu-like syndrome occur approximately 4-6 hours after exposure. Symptoms gradually decrease but may recur with re-exposure. Understandably, affected patients are frequently misdiagnosed with acute bacterial or viral illness. Subacute HP is characterized by a more insidious onset of dyspnea, fatigue, and cough that develops over weeks to a few months, caused by repeated low-level exposure to inhaled antigens. Unrecognized and untreated acute/subacute episodes may eventually evolve to chronic HP. Patients then present with a slowly progressive chronic respiratory disease characterized by progressive dyspnea, cough, fatigue, malaise, and weight loss. In most cases, it takes months to years of inhalational exposure to result in this disease, with the average exposure to symptom onset of 9 years in patients with bird fancier’s. Hypersensitivity pneumonitis develops in sensitized individuals with repeat exposure to a disease-causing antigen. In patients with the exposure, however, only a minority develops HP causing some to hypothesize that there can be a genetic predilection for HP. Also, perhaps counterintuitively, HP is less frequent in smokers than in nonsmokers. In our case, the family history was noncontributory and the patient was a lifelong nonsmoker.

As symptoms of HP are nonspecific, differential diagnosis can include many other interstitial lung diseases. Laboratory studies can be helpful but results are often as nonspecific as the clinical symptoms. Specific antibody detection can be seen as a marker of disease, but the assay demonstrates a propensity for false negatives in acute and chronic HP. Spirometry can demonstrate a restrictive lung disease pattern, with moderate to severe reduction of the carbon monoxide diffusing capacity (DLco). Bronchoalveolar lavage will show increased inflammatory cells. Re-exposure of the patient to the suspected inciting agent after a period of avoidance can reveal a relationship between exposure and symptoms (antigenic challenge), thus supporting a diagnosis of HP. Sometimes, lung biopsy is necessary for confident diagnosis and may show cellular bronchiolitis, diffuse lymphocytic interstitial infiltration, and noncaseating granulomas.

Radiographical investigation typically includes chest radiographs and, sometimes, HRCT. The majority of patients with HP have normal chest radiographs. Some abnormal radiographic findings observed in patients could include numerous poorly defined small opacities throughout both lungs, with a pattern of fine reticulation. Distribution of abnormalities varies temporally from patient to patient. When fibrosis develops, chest radiographs show a reticular pattern.
with upper lobe predominance. HRCT, while not first-line imaging modality, has revolutionized the radiologic diagnosis of HP as abnormalities are seen in the vast majority of patients. Airspace disease in HP as can be seen as patchy ground-glass opacities and/or centrilobular nodules. In patients with extensive bronchiolar obstruction, shunting of blood away from poorly ventilated regions of lung manifests as mosaic attenuation. Air trapping is diagnosed when the hypoattenuating areas persist on expiratory CT scans. In the setting of fibrosis, CT scans demonstrate reticulation, honeycombing, and traction bronchiectasis.

Treatment is as simple as avoidance of exposure and corticosteroids.

Conclusion

Hypersensitivity pneumonitis can occur with repeated exposure to a number of antigens. Thorough history taking and correlation of exposure to symptoms remains crucial for diagnosis. Laboratory findings and chest radiographs are, more often than not, normal. HRCT can show patchy ground glass opacities, indicating airspace disease. Clinical symptoms range in severity and acuity and generally resolve with avoidance of antigen and corticosteroids. A familiarity with the syndromes associated with certain antigenic exposures and their diagnosis can help achieve early treatment and resolution of symptoms.

References