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Bird fanciers' lung induced by exposure to duck and goose feathers

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Data Interpretation D
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Patient: Female, 60
Final Diagnosis: Bird fanciers' lung
Symptoms: Cough productive • hypoxia • short of breath • substernal chest pain
Medication: —
Clinical Procedure: —
Specialty: —

Objective: Rare disease

Background: Hypersensitivity pneumonitis (HP) is a group of inflammatory interstitial lung diseases caused by hypersensitivity reactions from repeated insults of inhalation of fine particulate organic dusts derived from environmental sources. Bird fanciers' lung (BFL) is the most common form of HP, with an estimated prevalence of 0.5–7.5% and is observed in individuals who develop a hypersensitivity response to avian droppings or antigens on bird feathers.

Case Report: A 60-year-old woman presented to our care with shortness of breath with exertion. She was hypoxic with oxygen saturation of 70% on room air. The CTA of the chest revealed a diffuse bilateral ground glass density in the lung parenchyma with a mosaic attenuation pattern. On further questioning she explained that she collected many duck and goose feathers she found on the ranch and placed them in a vase at home. Transbronchial lung biopsy revealed non-caseating granulomas, aggregates of epithelioid macrophages, and patchy mononuclear cell infiltration with lymphocytes and fibrotic tissue. The patient clinically improved and was discharged home on the 6th hospital day with prednisone 20 mg daily, with clinical improvement noted on subsequent follow up visits.

Conclusions: There is no specific clinical manifestation; abnormal laboratory test results help establish a definitive diagnosis. The best diagnostic tool is the correlation of symptom onset with the environmental exposure. The prognosis is excellent after a single episode of HP, but continuous re-exposure carries the risk of progressive pulmonary impairment.

MeSH Keywords: Bird Feathers • Alveolitis, Extrinsic Allergic • Bird Fancier's Lung

Full-text PDF: http://www.amjcaserep.com/download/index/idArt/890184

Background

Hypersensitivity pneumonitis (HP) is a group of inflammatory interstitial lung diseases that result from hypersensitivity reactions from repeated inhalation insults of fine particulate organic dusts derived from environmental sources [1]. HP manifests by inflammatory alveolitis with lymphocytic infiltration with the presence of poorly defined non-caseous epithelioid cell granulomas in the peripheral airways [1]. Exposure to avian proteins triggers a type-III immune complex-mediated hypersensitivity reaction with activation of alveolar macrophages and T cell lymphocytes [2].

Bird fanciers' lung (BFL) is the most common form of HP, with an estimated prevalence of 0.5–7.5% [3]. It has been reported that the prevalence of pigeon breeder's disease among pigeon breeders is between 0.5% and 22% [3]. BFL is observed in individuals who develop a hypersensitivity response to avian droppings or antigens on bird feathers [4]. Feather duvet lung (FDL) is a rare subgroup of BFL, caused by inhalation of organic dust from duck or goose feathers in duvets or pillows. Exposure to a range of bird antigens can initiate an immune response that causes inflammatory alveolitis [4]. The products of various kinds of birds, such as pigeons, parrots, canaries, doves, turkeys, chickens, geese, and ducks, are responsible for this disease [5].

Excreted intestinal mucin and immunoglobulins (Ig) IgA and IgG from bird droppings and bloom are highly antigenic, and are the likely major sources for inhalant bird antigen [6]. Pigeon dropping and bloom contain pigeon intestinal mucin (PIM), which is a large glycoprotein with a large amount of carbohydrate content that is resistant is biochemical breakdown [7]. PIM stimulates the production of antibodies in patients with exposure to pigeons. The reactive IgG antibodies bind to the inhaled pigeon antigen, leading to an immune complex formation that accumulates in the lungs. This eventually leads to inflammation, tissue damage, and functional loss. We present a unique case of BFL caused by overexposure to goose and duck feathers.

Case Report

A 60-year-old Caucasian woman presented to our care with shortness of breath with exertion, which was relieved by rest for 2 weeks. She initially went to her primary care physician, who noted that the patient was hypoxic with oxygen saturations at 73%. She was therefore sent to the emergency department for further evaluation. Other complaints included intermittent substernal chest pain of moderate intensity with radiation to the back of the neck and a productive cough with white phlegm, but no blood was noted. She denied any fever, chills, nausea, vomiting, headaches, dizziness, abdominal pain, or diarrhea. Her comorbidities included hypertension and hypothyroidism. Home medications were levothyroxine 112 mcg tab daily and hydrochlorothiazide 12.5mg tab daily. She quit smoking tobacco about 3 years ago but previously had smoked ½ pack per day for 10 years.

Table 1. Initial laboratory work-up.

White blood cell count	7.25×10³ uL (4.5–11.0×10³/UL)
Hemoglobin	13.8 g/dL (12.0–15.0 g/dL)
Hematocrit	43.2% (36.0–47.0%)
Platelet count	403×10³/uL (150–450×10³/UL)
Sodium	138 mmol/L (135–145 mmol/L)
Potassium	5.1 mmol/L (3.5–5.1 mmol/L)
Chloride	103 mmol/L (98–107 mmol/L)
CO_2	23 mmol/L (21–32 mmol/L)
Serum glucose	100 mg/dL (70–100 mg/dL)
BUN	12 mg/dL (7–22 mg/dL)
Creatinine	0.31 mg/dL (0.60–1.30 mg/dL)
Calcium	8.9 mmol/L (8.5–10.1 mmol/L)
Albumin	3.4 g/dL (3.4–5.0 g/dL)
Protein	8.1 g/dL (6.4–8.2 g/dL)
AST	218 IU/L (15–37 IU/L)
ALT	308 IU/L (12–78 IU/L)
Alk. phosphatase	94 IU/L (50–136 IU/L)
BNP	112 pg/mL (0–100 pg/mL)
Troponin	<0.02 ng/mL (0.00–0.08 ng/mL)
TSH	3.97 MIU/L (0.35–4.0 MIU/L)

Initial vital signs were significant for hypertension (blood pressure: 155/81) and hypoxia (oxygen saturation was 70% on room air). She was then placed on 3 liters of oxygen through nasal cannula; oxygenation then increased to 94%. The only significant physical examination findings were bilateral diffuse wheezing upon auscultation of the lungs. The initial laboratory workup (Table 1) was only significant for elevated liver enzymes. A chest radiograph (Figure 1) showed mild cardiomegaly with increased parahilar vascularity suggestive of pulmonary edema. Computed tomography (CT) angiogram (Figure 2) of the chest was ordered to rule out pulmonary embolism. It revealed a diffuse bilateral ground glass density in the lung parenchyma with intervening normal appearing lung parenchyma having a mosaic attenuation pattern. Diffuse micronodules

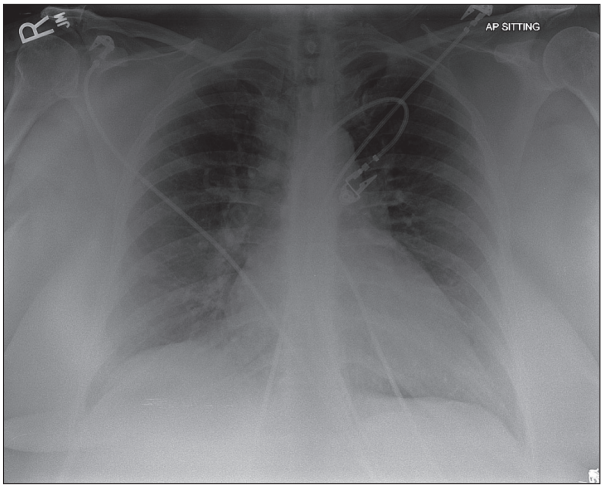

Figure 1. CXR: mild cardiomegaly with increased parahilar vascularity.

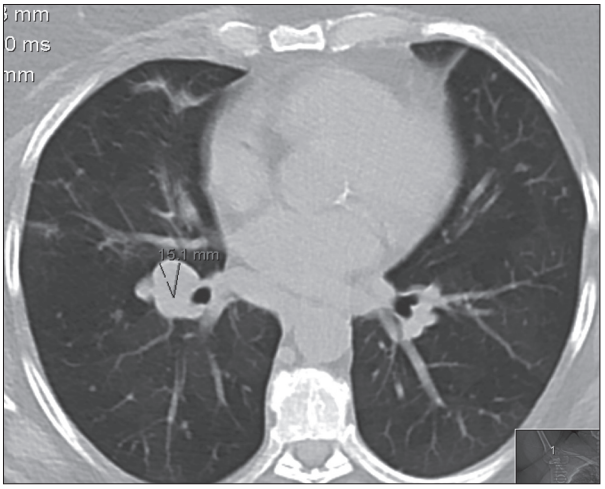

Figure 2. Chest CT: diffuse bilateral ground glass density in the lung parenchyma with intervening normal appearing lung parenchyma having a mosaic attenuation pattern.

throughout both lungs were also noted. 2D echocardiogram showed a borderline left atrial enlargement and left ventricle ejection fraction of 70±5%.

Our differential diagnosis after our initial assessment and imaging studies was hypersensitivity pneumonitis, sarcoidosis, miliary tuberculosis, bacterial pneumonia, and lung malignancy. We suspected hypersensitivity pneumonitis as the most likely diagnosis but were unsure of the cause. On further questioning, the patient explained that she worked as a server at a restaurant located on a ranch approximately 35 miles east of El Paso, Texas. This ranch has several different species of farm animals, including birds (ducks and geese), cattle, and horses. She explained that she did not have actual close exposure to the animals or the birds. However, she had been collecting the feathers of the ducks and geese nearby the restaurant for

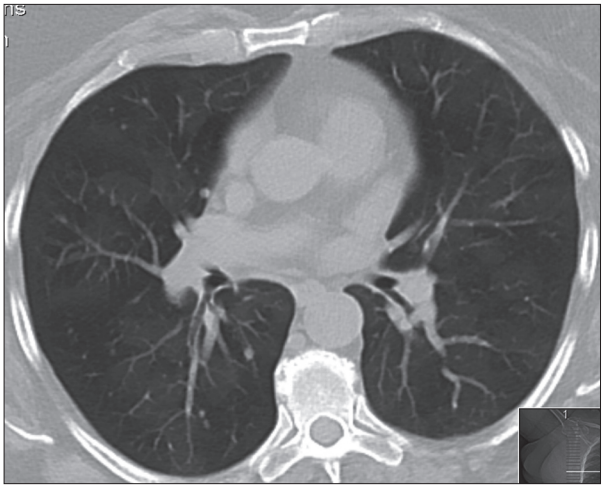

Figure 3. Chest CT: diffuse bilateral geographic mosaic attenuation and bilateral centrilobular/peribronchial pulmonary nodules.

the past 6 months and placed them in a vase on her kitchen table at home. She did not have any pet birds or birds living outside the house. No other possible environmental exposures were noted.

On the 3rd hospital day, a bronchoscopy was done that showed anatomically normal airways with diffusely erythematous mucosa and clear secretions. Bronchoalveolar lavage (BAL) of the right middle lobe was negative for malignancy, pneumocystic carinii, fungi, or granulomas. The BAL fluid was hazy and red, the cell analysis included a red blood cell count of 5000 cells per mm^3 and white blood cell count was 1968 cells per mm^3 with neutrophils accounting for 90%, lymphocytes of 6% and monocytes 4%. Cultures of BAL were negative for any microorganism growth, acid fast bacilli or fungi. The pulmonary function tests (PFTs) showed a proportional decrease in both FEV_1 and FVC, leading to a decreased FEV_1/FVC ratio. The carbon monoxide diffusing capacity (DL_{CO}) was also decreased, suggesting a restrictive lung disease process. The human immunodeficiency virus (HIV) test result was negative. The absolute CD4 was 1272 cells/mcL (490–1740 cells/mcL), absolute CD8 count was 652 cells/mcL (180–1170 cells/mcL), and the CD4/CD8 ratio was 1.95 (0.86–5.0). Transbronchial lung biopsy revealed non-caseating granulomas, aggregates of epithelioid macrophages, and patchy mononuclear cell infiltration with lymphocytes and fibrotic tissue.

The patient clinically improved and was discharged home on the 6th hospital day with prednisone 20 mg daily. At her 1-month follow-up with the pulmonologist after discharge, she still worked at the restaurant on the ranch, but had minimal exposure to the birds and no longer collected bird feathers. The chest CT (Figure 3) findings were unchanged from previous imaging evaluation. The chest CT findings were diffuse bilateral

geographic mosaic attenuation and bilateral centrilobular/peribronchial pulmonary nodules, likely representing granulomas, measuring up to 7 mm and scattered throughout both lungs. There was also subcentimeter bilateral upper/lower paratracheal, and subcarinal and hilar lymph node enlargement. She was on home oxygen and prednisone 20 mg daily. She explained that she no longer had any shortness of breath, but on physical examination mild diffuse wheezing was present bilaterally. The PFTs still had a proportional decrease in both FEV_1 and FVC with a decreased FEV_1/FVC ratio, but improved from the last PFTs and the DL_{CO} was also improved. Since the patient was doing well, we initiated a prednisone taper over the course of 1 month. Additional chest CT and PFTs were performed at the 6-month follow-up visit, without any interval improvement. At this time she was no longer using the home oxygen and was tapered off the prednisone. She was asymptomatic and no wheezing was noted on auscultation of the lungs.

Discussion

Clinical features include increasing dyspnea on exertion, fatigue, anorexia, cough, and weight loss [8]. Physical findings include tachypnea and rales without wheezing. The clinical presentation is divided into acute, subacute, and chronic forms, depending on the amount of inhaled antigen and repeated exposure [9]. The acute form occurs on exposure to a massive amount of antigens on a single occasion and symptoms appear 4–6 hours afterwards. The chronic form is less intense but results from a prolonged continuous exposure to a low dose of antigen [9]. There is no specific clinical manifestation; abnormal laboratory test results are pathognomonic and help establish a definitive diagnosis. However, the best diagnostic tool is the correlation of symptom onset with the environmental exposure.

Lung function abnormalities include low diffusing capacity of the lung (DLCO) and restrictive pattern on pulmonary function tests (PFT). The characteristic findings of computed tomography (CT) of the chest in BFL are bilateral diffuse centrilobular nodules, ground glass opacities, and consolidations in the lungs [9]. BFL can eventually progress to respiratory failure secondary to chronic obstructive pulmonary disease (COPD) or pulmonary fibrosis [10]. Bronchoscopy with bronchial alveolar lavage (BAL) can help establish a histological confirmation of HP. Lung biopsy confirmed the histological triad: interstitial pneumonia with predominance of lymphocytes and peribronchiolar distribution, non-necrotizing granuloma, and foci of obliterative bronchiolitis [11]. Lung biopsies can strongly support the diagnosis but are recommended only if the diagnosis is uncertain.

Conclusions

The prognosis in BFL depends on a number of factors: duration of exposure, histopathologic changes, presentation (acute, subacute, or chronic), age, intensity of exposure, and lung function abnormality. The recommended and most effective treatment for BFL is immediate removal of the birds in the environment and avoidance of re-exposure to bird antigens [12]. Resolution of symptoms following the avoidance of antigen can support but not confirm the diagnosis. The prognosis is excellent after a single episode of HP but continuous re-exposure carries the risk of progressive pulmonary impairment. This present case report highlights the importance of a careful assessment of environmental exposure [13]. Physicians should be aware of this possible source of antigenic material when investigating patients with suspected hypersensitivity pneumonitis [14].

Disclosures

All participating authors in this study declare no financial, professional, or personal conflicts.
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All authors were involved in manuscript preparation and literature review.

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