Hypersensitivity Pneumonitis: A Case of Suspected Humidifier Lung

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INTRODUCTION

Hypersensitivity pneumonitis (HP) results in lung parenchymal inflammation caused by immune response to the inhalation of a wide variety of typically innocuous antigens. The nonspecific clinical presentation and rarity of the syndrome challenges clinicians in recognizing HP early and differentiating it from more commonly seen respiratory illnesses.

CASE

A 70-year-old otherwise healthy female presented to her internist clinic with a 1 week history of dyspnea, dry cough, and fatigue. She was tachycardic, tachypneic, and hypoxic with saturations in the low 80s on room air. She was sent to the ED on 5 L oxygen. Upon admission, physical exam was notable for bilateral wheezing with bibasilar rales. Labs were notable for mild leukocytosis with neutrophil predominance. CXR was unremarkable. The patient was started on antibiotics for possible pneumonia. Given concern for pulmonary embolism (PE), CT chest (Figure 1) was ordered, which revealed no PE, but rather diffuse mosaic perfusion involving bilateral lung parenchyma. While infectious work-up and HP panel were negative, subsequent transbronchial biopsies revealed interstitial inflammation and ill-defined epitheliod granulomas consistent with HP. The patient was started on prednisone 40 mg PO daily and titrated down to 4 L oxygen prior to discharge. Further investigation revealed the use of an old humidifier that began 1 month prior to admission as the source of likely microbial antigen exposure, of which thermophilic actinomycetes, Klebsiella, or Acanthamoeba species are more typically found. She was discharged home on oxygen and prednisone. At 1 month follow-up, the patient reported improvement of symptoms and no longer required supplemental oxygen. PFT done 2 months after revealed a moderate obstructive defect with a mildly reduced diffusion capacity.

DISCUSSION

HP is primarily the result of types III and IV hypersensitivity, affecting 0.1-0.4 per 100,000/year. Diagnosis of HP is made difficult due to its nonspecific presentation and rarity. Patients typically present with +/- fever/chills, dyspnea, fatigue, and may have dry cough, wheezing, or rales on physical exam. Cyanosis and digital clubbing may be seen in chronic disease.

Diagnosis (Figure 3) requires a detailed history aided by elevation of immunoglobulin precipitins, centrilobular ground-glass or mosaic perfusion patterns on CT, and/or poorly formed non-necrotizing granulomas on biopsy. PFT is used to determine physiologic abnormalities and associated impairment. HP is classified as either acute, subacute, or chronic largely based on timing and degree of signs/symptoms and histologic and radiologic findings. The most effective treatment of HP is antigen avoidance. Therefore, when HP is suspected or diagnosed, an effort should be made to identify the inciting agent. Corticosteroids are often used in patients with severe illness to hasten recovery in the acute setting, but have not been shown to influence long-term recovery. Prognosis in most patients is good, especially in those who avoid antigen exposure, however in a minority of patients, disease will progress to pulmonary fibrosis.

REFERENCES