Wood Worker’s Lung - A Case Report

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Abstract: Hypersensitivity pneumonitis (HP), also referred to as extrinsic allergic alveolitis, is a pulmonary disease that occurs due to inhalational exposure to a variety of antigens leading to an inflammatory response of the alveoli and small airways. Wood dust is one of the oldest and one of the most common occupational exposures in the world. Here I present a case of 57 year old male non smoker, no previous history of tuberculosis whose occupation was stripping bark from maple logs for more than 10 years presented as lung fibrosis.

Keywords: Hypersensitivity pneumonitis: Wood worker’s lung

1. Introduction

HP is an immune-mediated condition that occurs in response to inhaled antigens that are small enough to deposit in distal airways and alveoli. Most cases of hypersensitivity pneumonitis develop only after many years of inhaling allergens. In general, among those exposed to an allergen, approximately 5%–15% develop hypersensitivity pneumonitis. The disease frequency probably relates to several issues, including the amount of allergen inhaled, the duration of exposure, the nature of the antigen, and host factors. Without proper treatment and antigen avoidance, it may progress to lung fibrosis, hypoxemic respiratory failure and lung cancer.

2. Case Report

Mr. Raji 57 years male non smoker presented with fever for 6 months intermittent associated with chills and progressive dyspnoea, fatigue for 15 days. History of weight loss, clubbing of digits were present. His labour entailed stripping bark from maple logs or shovelling it for more than 10 years. And he discontinued his job 8 months back due to his persistent illness. No history of tuberculosis. On auscultation he had right supraclavicular and infraclavicular vocal resonance decreased with occasional crackles. Chest X ray PA view shows no abnormalities. CT thorax shows patchy fibrosis, small subpleural nodules, patchy honeycombing in right lung upper lobe. With this history, examination and investigation the patient was diagnosed as hypersensitivity pneumonitis due to exposure of wood. Patient was started on prednisone 0.5-1 mg/kg/day for 2 weeks, followed by a taper over next 4 weeks. Patient was symptomatically better after 6 weeks and he was on regular follow up.

3. Discussion

Hypersensitivity pneumonitis (HP) is a complex pulmonary syndrome mediated by the immune system and caused by inhalation of a wide variety of antigens to which the individual has been previously sensitized. The pathobiology of the disease is not fully understood, but in addition to the triggers that initiate the disease, host/genetic factors are likely to be important, as only a minority of exposed individuals develop HP. Sensitization to an inhaled antigen as manifested by specific circulating IgG antibodies is necessary for the development of HP.

Most patients (80%–95%) with hypersensitivity pneumonitis are nonsmokers. It is divided into acute, subacute, and chronic types. Acute HP usually manifests itself 4–8 h following exposure to the inciting antigen, often intense in nature. Systemic symptoms, including fevers, chills, and malaise, are prominent and are accompanied by dyspnoea. Symptoms resolve within hours to days if no further exposure to the offending antigen occurs. In subacute HP resulting from ongoing antigen exposure, the onset of respiratory and systemic symptoms is typically more gradual over the course of weeks. A similar presentation may occur as a culmination of intermittent episodes of acute HP. Although respiratory impairment may be quite severe, antigen avoidance generally results in resolution of the symptoms, but with a slower time course, on the order of weeks to months, than that seen with acute HP.

Chronic HP can present with an even more gradual onset of symptoms than subacute HP, with progressive dyspnoea, cough, fatigue, weight loss, and clubbing of the digits. The insidious onset of symptoms and frequent lack of an antecedent episode of acute HP make diagnosing chronic HP a challenge. Unlike with the other forms of HP, there can be an irreversible component to the respiratory impairment that is not responsive to removal of the responsible antigen from the patient’s environment.
The histopathological changes in the lung were centrilobular fibrosis and emphysema, the presence of intra alveolar basophilic particles which had excited a histiocytic and foreign body reaction. Chest radiographs obtained in many patients with hypersensitivity pneumonitis are normal. High-resolution CT has greatly improved the radiologic diagnosis of hypersensitivity pneumonitis: abnormalities are seen in more than 90% of patients. The radiologic findings include diffuse ground-glass opacification, centrilobular ground-glass opacities, air trapping, fibrosis, lung cysts, and emphysema. The histologic and radiologic features in some cases may resemble those of usual interstitial pneumonia or nonspecific interstitial pneumonia.

Differentiating fibrotic HP from the idiopathic interstitial pneumonias can be a challenge. In HRCT Fibrotic ILD features include septal thickening, traction bronchiectasis, and honeycomb cysts. In patients with radiologic fibrosis, specific HRCT findings prompting consideration of fibrotic or chronic HP include distribution of abnormality in a patchy or geographic pattern, often with upper lung predominance, ground-glass opacities, and centrilobular nodules, mosaic attenuation, and air trapping. HRCT findings plus BAL lymphocytosis greater than 30% (or 20% for current smokers) as the “gold standard” criteria for HP developed a prediction rule for identifying “active” HP.

The disease progression of chronic HP to lung fibrosis and hypoxemic respiratory failure can mirror that seen in idiopathic pulmonary fibrosis (IPF). Fibrosis, emphysema and lung cancer are the common complication of HP. The presence and extent of histopathologic fibrosis are consistently associated with worse survival in HP.

Occupational exposure contributes 5 to 15% risk of developing lung cancer. Studies suggest there is sufficient evidence in humans for the carcinogenicity of wood dust on nasal cavity, paranasal sinuses and nasopharynx. squamous cell carcinoma, large cell carcinoma and adenocarcinoma were common among occupational exposure.

Due to the lack of a diagnostic gold standard, the diagnosis of HP is not straightforward and relies on the integration of a number of factors, including history of exposure, precipitating antibodies to the offending antigen, clinical features, bronchoalveolar lavage, radiological and pathologic features. Treatment includes avoiding the allergen, if possible, and, in severe cases, systemic corticosteroids. The long-term prognosis is usually good, but some patients develop severe respiratory insufficiency, and a few die of the disease.

4. Conclusion

Hypersensitivity pneumonitis is reported 56 – 68% every year as chronic stage. So Careful history taking, early detection and intervention to prevent disease progression to irreversible fibrosis and lung cancer is needed.

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