Case Report

Rare case of acute hypersensitivity pneumonitis caused by moldy hay of sugarcane

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Abstract

Introduction: Hypersensitivity pneumonitis(H.P.) OR Extrinsic allergic alveolitis (EAA) is resulting from immunological induced inflammation in response to inhaled organic or inorganic **Matarials:** It is relatively rare disease, constituting 2% of cases of interstitial lung disease. The authors present the case of a patient 45 years old female farmer, born in rural Maharashtra, mostly growing sugarcane in the field observed at the Emergency Room (ER) of the hospital in june 2014, with complaints of abrupt onset dyspnoea (MRC GRADE 3), weakness, easy fatigability for moderate exertion, exertional dyspnea, nonproductive cough. The study pointed to the initial diagnosis of sarcoidosis with severe restrictive syndrome, but further study revealed that it was of hypersensitivity pneumonitis by pressed moldy hay inhalation of sugarcane. The patient underwent treatment with prednisone with good response, showing clinical and radiologic improvement. The authors present this case to emphasize the importance of history of exposure and early identification, a lack of specificity of clinical manifestations, the need for invasive diagnostic methods and, sometimes, poor response to therapy.

Keywords: Hypersensitivity pneumonitis, Ground glass appearance.

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Received Date: 03/11/2014 Accepted Date: 14/11/2014

Access this article online	
Quick Response Code:	Website: www.statperson.com
	DOI: 15 November 2014

INTRODUCTION

The hypersensitivity pneumonitis (HP) or extrinsic allergic alveolitis (EAA) is the paradigm of lung response to inhaled organic material, either in the form of particles or of organic protein, or even of simple chemicals, organic and inorganic. Given its etiologic multiplicity and diversity of clinical, HP has been considered as a syndrome, with highly variable and difficult to assess prevalence. Clinically, can be considered three forms of presentation - acute, subacute and chronic - depending on the nature of the aggressor agents, their physical characteristics, their concentration, the intensity of individual exposures and individual variability[1]. The acute response after inhalation is a nonspecific diffuse pneumonitis with inflammatory cell infiltration of the bronchioles, alveoli, and interstitium. In the subacute and chronic stages, loosely formed, noncaseating, epithelioid cell granulomas may be dispersed in the interstitium. In a population study, the estimated annual incidence of interstitial lung diseases was 30 per 100,000 individuals[2]. In that study, the HP was less than 2% of cases. Several diagnostic criteria for HP proposals have been published, the most used is by Richerson *et al*[3]. Corticotherapy given a picture of a single determinant hp, its eviction is mandatory. The treatment is based on, despite the absence of long-term benefit evidence[4]

CASE REPORT

The authors present the case of a patient 45 years old female farmer, born in rural Maharashtra, mostly growing sugarcane in the field observed at the Emergency Room (ER) of the hospital in June 2014, with complaints of abrupt onset dyspnoea (MRC GRADE 3)weakness, easy fatigability for moderate exertion, exertional dyspnea, nonproductive cough. She had no family or personal history of pathological relevance. She denied smoking,

How to site this article: Praveen S Chabukswar, Pravin N Soni. Rare case of acute hypersensitivity pneumonitis caused by moldy hay of sugarcane. *International Journal of Recent Trends in Science and Technology* November 2014; 13(1): 127-129 http://www.statperson.com (accessed 15 November 2014).

exposure for wood smoke present for about 4 years. She was not on any kind of medication and epidemiological history was irrelevant. On physical examination the patient was alert, oriented., afebrile tachypnoeic with a respiratory rate of 26 per minute using her accessory muscles of respiration, blood pressure 130/76 mmHg and pulse oximetry 87% on room air. The pulmonary auscultation revealed a bibasilar dry rales. The cardiac auscultation and abdominal examination showed no changes. She had no palpable lymphadenopathy or peripheral edema, and the remaining physical examination was normal. The patient had some complementary diagnostic exams performed on an outpatient basis such as hemogram and routine lab which was essentially normal. Chest x-ray done suggestive of ground glass appearance predominantly mid and lower zones.

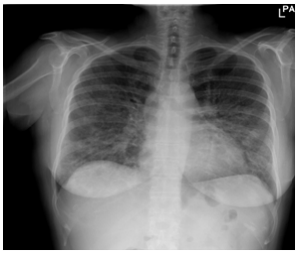

Figure 1: Chest x ray s/o b/l ground glass opacities

The chest CT revealed areas of thickening of interlobular septa in both lung bases and at the apical segments of upper lobes, some areas of ground glass appearance in both lower lobes and multiple enlarged mediastinal lymph nodes (Fig.2).

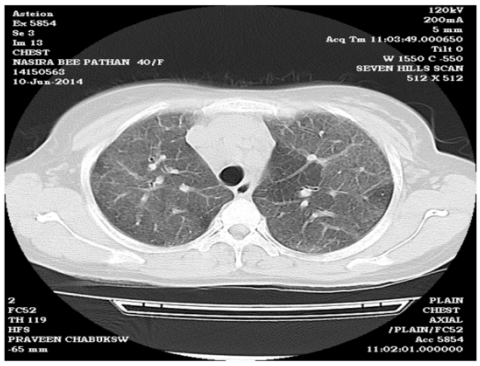

Figure 2

The imaging tests identified lung fibrosis. The clinical setting associated with it leads us to think this as a disease associated with fibrosis: a) an occupational disease causing interstitial pneumonitis (extrinsic allergic asthma), b) infectious disease, including pulmonary tuberculosis, c) sarcoidosis or systemic autoimmune disease, d) idiopathic pulmonary fibrosis. In this context, the patient was then hospitalized at the Pulmonary Medicine Department for further study. From the exams carried out during hospitalization we highlight a hypoxemia of 65.3 mmHg in arterial blood gas analysis.. HIV and Mantoux tests were negative. The following tests were performed after treatment The optic bronchoscopy and bronchoalveolar lavage (BAL) done after treatment showed no changes had a CD4/CD8 ratio of 3.12. Respiratory function tests (RFT) performed after treatment showed a severe restrictive syndrome with decreased alveolocapillary diffusion by carbon monoxide Cultures of biological products, including BAL, bronchial aspirates, blood and urine were negative, so as antinuclear antibodies, ANCA and anti-glomerular basement membrane antibodies(samples were collected before giving methylprednisolone). At this stage it seemed as most likely diagnosis the occupational interstitial lung disease (chronic granulomatous disease of unknown etiology professional, including hypersensitivity pneumonitis) but the possibility of sarcoidosis could not definitively excluded. During hospitalization was treated with pulse methylprednisolone 1gm with clinical improvement achieved in a week and the patient was discharged to the outpatient service, keeping corticotherapy (oral 1 mg/kg/day prednisolone/day in tapering doses). Patient achieved clinical improvement in a week, radiological clearance of shadows In a month.

DISCUSSION

Hypersensitivity pneumonitis, also known as extrinsic allergic alveolitis, is a syndrome caused by repeated inhalation of specific antigens from occupational or environmental exposure in sensitized individuals. Hypersensitivity pneumonitis is considered a granulomatous interstitial disease of the lungs[5]. Analysis of BAL fluid reveals lymphocytosis of predominantly $CD8^+$ T cells with a decrease in the $CD4^+/CD8^+$ratio. However, BAL findings may vary depending on clinical presentation, the timing of the most recent antigen exposure and the attainment of BAL fluid. A predominant accumulation of $CD8^+$ T cells seems to be a feature in acute or subacute HP, whereas a prevalent elevation of $CD4^+$ T cells is found in the chronic form of the disease[1,6-7]. The first cases of HP were described at the beginning of the twentieth century, in farmers exposed to hay or straw. Since then it has been attributed to inhalation of various antigens found in the environment[1] HP results from an exaggerated immune response, leading to the onset of symptoms similar to an acute or progressive lung damage, sometimes irreversible[1]. The clinical features and severity of symptoms vary according to the frequency and intensity of exposure. A history of exposure to potential agents or changes in the domestic

and other environments (or both) is essential to diagnosis and treatment. The interval between exposure to the antigen and clinical manifestations of lung disease is unknown, although symptoms can occur as soon as 4 to 12 hours after exposure. In more chronic and low-level exposures, however, the onset is insidious. Our case represents the lack of specificity of the complaints and emphasize the importance of history of exposure (moldy pressed sugar cane). Further avoidance of exposure to the antigen or antigens and treatment with corticosteroids are important if improvement is to be obtained. Continued exposure to the unidentifiable antigens or prolonged exposure to antigens, or both, have led to chronic hypersensitivity pneumonitis and irreversible fibrosis that may not respond to any treatment regimen. Hypersensitivity pneumonitis is an under diagnosed entity; therefore its exact prevalence is unknown. In addition, the diagnosis is sometimes particularly difficult, since the clinical picture is not specific. In our case it was possible to identify the acute episode suggestive of this type of pathology, particularly during the period that he had occupational exposure to the allergen in question. However, there is reference to some patients in whom prolonged and continuous exposure to small amounts of antigen resulted in irreversible lung damage without acute illness[8]. In these situations, pulmonary fibrosis becomes prominent and only in advanced stages[9] arise progressive dyspnea, coughing, malaise, anorexia and significant weight loss, without fever, with bibasal crackles and no digital clubbing. Also relevant in this case report was the necessity to assess the history, clinico-radiologic correlation and to keep possibility of acute hypersensitivity pneumonitis in mind. Knowledge of each HP evolution is essential, particularly in the professional level, with legal and economic implications. However, it is not easily predictable, depending on several factors, from the nature and intensity of aggression to the characteristics of each individual. Although there are no controlled clinical trials regarding the treatment of chronic HP, it is recommended that patients with severe or progressive chronic HP be treated with trial corticosteroids[10,11].

ACKNOWLEDGMENT

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Source of Support: None Declared
Conflict of Interest: None Declared