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BMH Medical Journal 2016;3(2):55-58 **Case Report**

Hypersensitivity Pneumonitis Due To Domestic Mold

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Introduction

Hypersensitivity pneumonitis (HP) is a syndrome caused by repeated inhalation of specific antigens from environmental exposure in sensitized individuals. It is considered a granulomatous interstitial disease of the lungs. HP is the result of a cell-mediated immune response of the lung to a wide variety of inhaled antigens. The mainstay of diagnosis and management is a careful exposure history and further avoidance of the causative agent if it is identified.

Case Report

A 45 years old female presented with cough, progressive dyspnea, weight loss of 1.5 years duration. Her cough was principally dry in nature, progressive with no diurnal or seasonal variation. Her dyspnea was gradually progressive. (Grade 1 to 3 MMRC) over a period of 1.5 years. H/o weight loss ~ 10 kg over a period of 1.5 years. No h/o chest pain, palpitation, orthopnea, paroxysmal nocturnal dyspnea, pedal edema, wheezing or hemoptysis. No symptoms suggestive of connective tissue disorder like joint pain, rashes, photosensitivity or Raynaud's phenomena. No major medical illness found in her past as well as in her family.

All her symptoms started after shifting to their relative's old house, which was unoccupied for a long time. Her vitals were normal except that she was tachypneic. Her oxygen saturation (SPo2) was 88% without oxygen and with oxygen 2 L / min via nasal prongs it was 94%. She had clubbing, bilateral basal fine end-inspiratory crackles and squeak all over lung field. Other systemic examination was unremarkable. Routine blood investigations were unremarkable. Serology for HIV and HBsAg were Negative. Sputum for AFB was negative and so was Tuberculin skin test. ECG was normal. Her chest radiograph (**Figure 1**) showed bilateral reticulo-nodular opacities.

On the basis of available data the working diagnosis interstitial lung disease (ILD) was made with differential diagnoss of HP and sarcoidosis. Her angiotensin converting enzyme (ACE) was 48.00 U/L (Reference Interval: 8.00-65.00), ANA Screening was Equivocal (1.20) and ANA Profile was negative. Ophthalmologic examination was unremarkable. Echocardiogram was normal. Ultrasonogram of abdomen showed borderline hepatosplenomegaly. Spirometry showed severe restrictive abnormality and DLCO: 36.9 %, moderately reduced.

HRCT thorax (**Figure 2**) showed mosaic attenuation with patchy areas of ground glass opacities in mid and lower lobes, interlobular and intralobular interstitial thickening, and traction bronchietasis. The combination of ground glass opacities and mosaic perfusion constitutes the Headcheese sign. Bronchoscopy was normal and bronchial washing was negative for AFB. Trana-bronchial biopsy (TBB) was suggestive off non specific inflammation with lymphocytic infiltration.

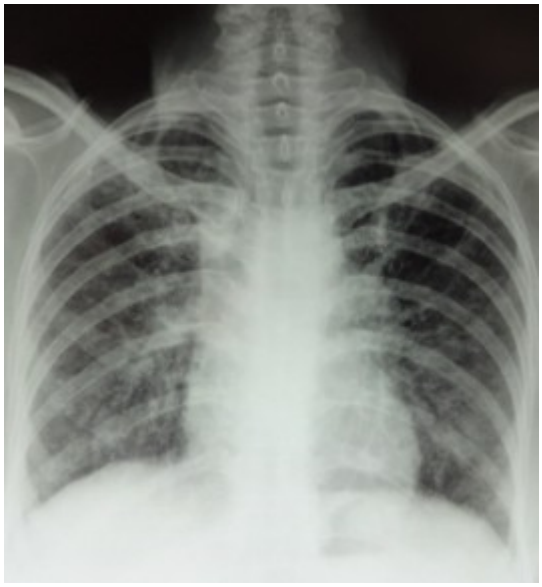

Figure 1: Chest radiograph

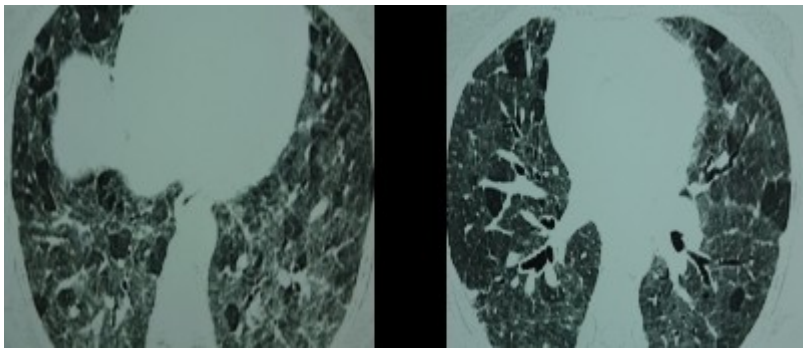

Figure 2: HRCT Thorax

Review of her history, clinical and radiological data were done and as she mentioned that her symptoms started after shifting to old house so with strong suspicion of any environmental exposure, house visit was done and found to have heavy mold infestation in patient's room (**Figure 3**). Air sampling was done by using Burkard spore trap showed multiple fungal spores (**Figure 4**). Thus a diagnosis of hypersensitivity pneumonitis was made and treated with oral glucocorticosteroids and advised to remove mold infestation completely and repaint the room. At present patient is doing well with treatment and is under our regular follow up.

Discussion

Hypersensitivity pneumonitis (HP), also known as extrinsic allergic alveolitis, results from immunologically induced inflammation in response to inhalation of chemical antigens (or haptens). It may be noted that HP is not an atopic disease and there is no increase IgE levels or eosinophil count [1].

Initial 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 [2]. The diagnosis of HP requires a combination of clinical, radiographic, physiologic, pathologic, and immunologic criteria, each of which is rarely pathognomic alone [3]. In general, the environmental history often provides a clue as to the potential etiologic agent. In this case, episodes of recurrent fever with breathlessness, cough, and malaise that follow a home environmental exposure are typical of extrinsic allergic alveolitis. The chest roentgenogram showed bilateral reticulo-nodular opacities, CT showed mosaic attenuation with patchy areas of ground glass opacities in mid and lower lobes, interlobular and intralobular interstitial thickening, and traction bronchietasis and spirometry showed restrictive ventilatory impairment with decreased diffusion capacity. Moreover, the pathologic findings of a specimen obtained by transbronchial lung biopsy showed interstitial lymphocytic infiltration. All of these characteristics are consistent with the diagnosis of HP.

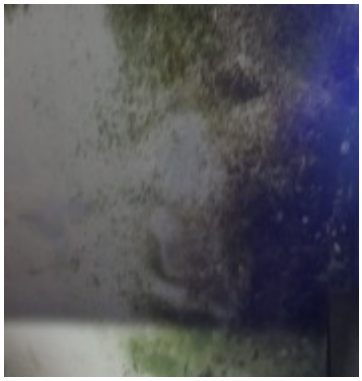

Figure 3: Heavy mold infestation in patient's room

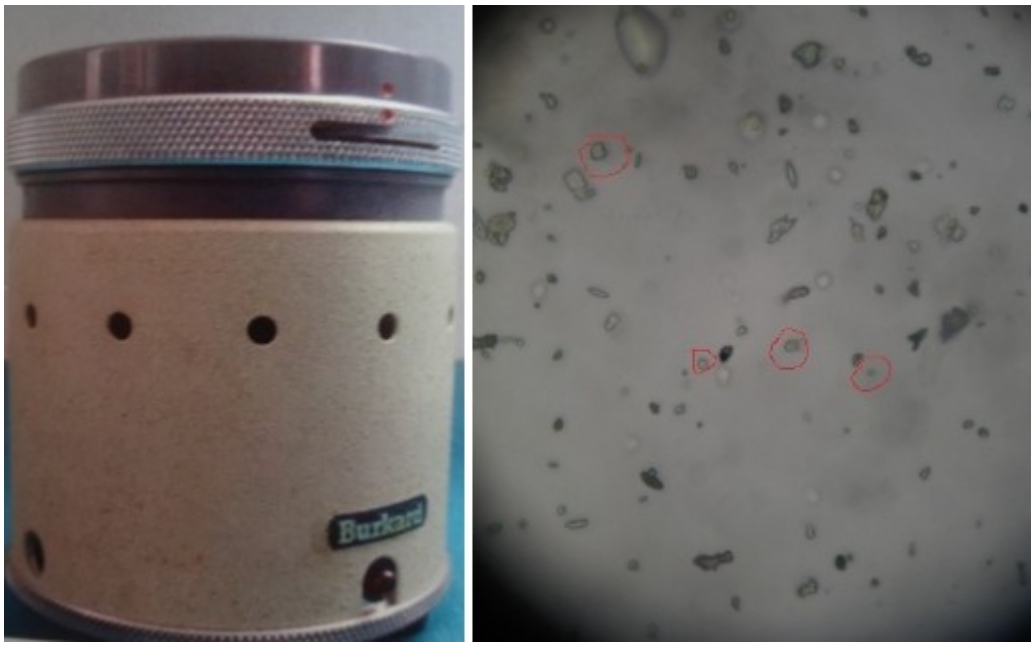

Figure 4: Burkard spore trap and fungal spores

Inhaled mold spores could provoke production of specific antibodies. The detection of these antibodies plays an important role in the diagnosis of HP. Although Ouchterlony's double immunodiffusion is the classical test to investigate specific antibodies, other studies such as counter immune electrophoresis, radioimmunoassay, ELISA, crossed radioimmuno electrophoresis, or immunoblotting can also be used [4]. But in this case it was not done due to financial concerns. This study describes a patient having clinical and immunological features of HP resulting from mold exposure in her home environment. Although we neither performed an inhalation challenge nor

identified the fungal spores, but many elements of the history and laboratory findings indicate the diagnosis.

Systemic glucocorticosteroids are usually required to treat severely symptomatic patients, although there is no formal evidence that such treatment is associated with long-term abatement of symptoms or radiologic or pulmonary function test abnormalities. The usual treatment is prednisone or prednisolone, 40 to 60 mg a day for 2 weeks, followed by a gradual decrease over 2 to 4 weeks. If patients are removed from exposure before there are permanent radiologic or physiologic abnormalities, the prognosis is excellent, with little evidence of long-term ill effects. If removal from exposure is impossible, the use of efficient masks during exposure can result in prevention of acute HP and an excellent prognosis [1,5].

Conclusion

The mainstay of diagnosis and management of HP is a careful exposure history and further avoidance of the causative agent if it is identified. Thus, prompt identification of inciting allergen and its removal will help in complete clearance of lesion and near total recovery of lung function. We present this case to emphasize the importance of history of exposure to any substances and the need for environmental study in suspected cases of HP as a diagnostic method.

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