Original Research

Assessment of Clinico-pathological Profile of 50 Patients with Hypersensitivity Pneumonitis: An Institutional Based Study

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Abstract

Background: Hypersensitivity pneumonitis (HP), also called extrinsic allergic alveolitis, is a respiratory syndrome involving the lung parenchyma and specifically the alveoli, terminal bronchioli, and alveolar interstitium, due to a delayed allergic reaction. Hence; the present study was conducted for assessing the clinical and pathological profile of 50 patients with Hypersensitivity pneumonitis

Materials & Methods: A total of 50 patients were enrolled. Complete demographic and clinical details of all the patients were obtained. HP suspected patients underwent a symptom screening with special exposure history, general and respiratory physical examination, chest X-ray, routine blood tests, sputum examination, connective tissue serology complete pulmonary function test (PFT). Only confirmed cases of HP were included as study group. A Performa was made and detailed clinical and pathological findings of all the painters were recorded. Bronchoalveolar lavage (BAL) findings were recorded separately. Histopathologic findings were analyzed separately. All the results were recorded in Microsoft excel sheet and were subjected to statistical analysis using SPSS software.

Results: On histopathological analysis, BAL lymphocytosis > 30% was seen in 46 percent of the patients while Broncho-centric inflammation with ill-defined granuloma, Broncho-centric inflammation with lymphocytic inflammation and Ill-defined granuloma with lymphocytic inflammation was seem om 26 percent, 26 percent and 18 percent of the patients respectively.

Conclusion: It is believed that HP is an occupational illness. In all suspected instances, a lung biopsy and bronchoscopy with BAL should be performed to confirm the diagnosis.

Key words: Hypersensitivity Pneumonitis, Bronchoalveolar Lavage.

INTRODUCTION

Hypersensitivity pneumonitis (HP), also called extrinsic allergic alveolitis, is a respiratory syndrome involving the lung parenchyma and specifically the alveoli, terminal bronchioli, and alveolar interstitium, due to a delayed allergic reaction. Such reaction is secondary to a repeated and prolonged inhalation of different types of organic dusts or other substances to which the patient is sensitized and hyper responsive, primarily consisting of organic dusts of animal or vegetable origin, more rarely from chemicals.[1-3] A study carried out in Wisconsin on 1400 individuals estimated HP prevalence at 4.2%, while other data estimates HP affecting from 0.5 to 19.0% of exposed farmers. In Europe, according to ILD registries, HP affects from 4 to 15% of all ILD cases, even if disease prevalence widely varies in different

countries and within the same country due to geographic, seasonal, and climatic factors. In a study of 431 incident cases in central Denmark, HP was the third most common ILD (7%), after idiopathic pulmonary fibrosis (28%) and connective tissue diseases (14%). In a Brazilian database including 3168 cases of ILD, prevalence of HP was 15%, the second place after connective tissue diseases 17%.[4- 9]Hence; the present study was conducted for assessing the clinical and pathological profile of 50 patients with Hypersensitivity pneumonitis.

MATERIALS & METHODS

The present study was conducted to assess the clinical and pathological profile of 50 patients with Hypersensitivity pneumonitis. A total of 50 patients were enrolled. Complete demographic and clinical details of all the patients were obtained. HP suspected patients underwent a symptom screening with special exposure history, general and respiratory physical examination, chest X-ray, routine blood tests, sputum examination, connective tissue serology complete pulmonary function test (PFT). Only confirmed cases of HP were included as study group. A Performa was made and detailed clinical and pathological findings of all the painters were recorded. Bronchoalveolar lavage (BAL) findings were recorded separately. Histopathologic findings were analyzed separately. All the results were recorded in Microsoft excel sheet and were subjected to statistical analysis using SPSS software. Univariate analysis was done for assessment of level of significance.

RESULTS

The mean age of the patients was 42.8 years. 60 percent of the patients were females while mean duration of symptoms was 15.3 months. Cough, dyspnea, fever, crackles and digital clubbing were seen in 100 percent, 96 percent, 20 percent, 72 percent and 16 percent of the patients respectively. On histopathological analysis, BAL lymphocytosis > 30% was seen in 46 percent of the patients while Broncho-centric inflammation with ill-defined granuloma, Broncho-centric inflammation with lymphocytic inflammation and Ill-defined granuloma with lymphocytic inflammation was seem om 26 percent, 26 percent and 18 percent of the patients respectively.

Table 1: Clinical and demographic variables

Variable	Number	Percentage
Mean age (years)	42.8	
Males	20	40
Females	30	60
Mean duration of symptoms (months)	15.3	
Cough	50	100
Dyspnea	48	96
Fever	10	20
Crackles	36	72
Digital clubbing	8	16

Table 2: Histopathological findings

Histopathologic findings	Number	Percentage
BAL lymphocytosis > 30%	23	46
Broncho-centric inflammation with ill-defined granuloma	13	26
Broncho-centric inflammation with lymphocytic inflammation	13	26
Ill-defined granuloma with lymphocytic inflammation	9	18

DISCUSSION

Hypersensitivity pneumonitis (HP), also known as extrinsic allergic alveolitis, 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. 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, and radiological and pathologic features. However, in the appropriate setting, a high index of suspicion is critically important and may obviate the need for more invasive tests. Clinical presentation and natural history vary widely. Acute forms generally resolve without

sequelae, while chronic forms, which are caused by persistent low-grade exposures, are associated with poor prognosis. Corticosteroids may be useful in acute episodes for symptomatic relief or in chronic and progressive disease, but their long-term efficacy has never been validated in prospective clinical trials. Ideally, patients with HP should be referred to centers with expertise, as the overlap with other forms of interstitial lung disease may be substantial.[8- 10]

Mean age of the patients was 42.8 years. 60 percent of the patients were females while mean duration of symptoms was 15.3 months. Cough, dyspnea, fever, crackles and digital clubbing was seen in 100 percent, 96 percent, 20 percent, 72 percent and 16 percent of the patients respectively. On histopathological analysis, BAL lymphocytosis > 30% was seen in 46 percent of the patients while Broncho-centric inflammation with ill-defined granuloma, Broncho-centric inflammation with lymphocytic inflammation and Ill-defined granuloma with lymphocytic inflammation was seem om 26 percent, 26 percent and 18 percent of the patients respectively. Specific antibodies analysis can be useful as supportive evidence. The results of the HP Study demonstrate that positive serum antibodies are a significant predictor of HP. Antigens available for testing in most centers included pigeon and parakeet sera, dove feather antigen, Aspergillus sp, Penicillium, Saccharopolyspora rectivirgula, and Thermoactinomyces viridans. These antigens cover most cases of HP including pigeon breeder's disease, bird fancier's lung, farmer's lung, and humidifier lung. The antigen Trichosporoncutaneum is also available in Japan for cases of summer-type HP. The selection of antigens to be tested often needs to be determined locally according to the prevalent antigens. In Eastern France, by using a panel of antigens really responsible for farmer's lung and not a classical standardized panel, serological tests showed a high rate of sensitivity and specificity. Several methods for determination of precipitins or total IgG antibodies (immunodiffusion, immunoelectrophoresis, enzyme-linked immunosorbant assays [ELISA]) and different antigen preparations have been described. ELISA is usually the preferred method. Unfortunately, even the ELISA technique lacks standardization.[11- 17] Bronchoalveolar lavage fluid can be useful in supporting a diagnosis of HP as well as excluding other etiologies such as infection or hemorrhage. Increased bronchoalveolar lavage fluid cellularity with a lymphocytosis >50% is strongly suggestive of HP in the appropriate clinical context, although its absence does not exclude the diagnosis, particularly in smokers and in patients with chronic HP. Transbronchial biopsies are rarely helpful to rule in chronic HP or identify possible alternative diagnoses, although a normal transbronchial biopsy is also insufficient to exclude HP. Surgical lung biopsies may show bronchiolocentric chronic interstitial pneumonia with poorly formed noncaseating granulomas, often with cholesterol clefts in the interstitium.[18, 19] Histologic findings of HP have been recently revised. The diagnosis of chronic nonfibrosing HP is best made on a wedge biopsy, but the diagnosis can occasionally be suggested on a transbronchial biopsy with appropriate clinical correlation, when peribronchiolar chronic inflammation and small granulomas or giant cells are found. Histopathological features comprise chronic bronchiolocentric inflammation and poorly formed nonnecrotizing granulomas. Bronchiolitis is common and can be cellular, follicular, or obliterans. Peribronchiolar metaplasia is a prominent finding, especially in airway-centered interstitial fibrosis (ACIF) and should point to diagnosis. The granulomas characteristic of HSP are small, loose, and epithelioid and may be difficult to find. In fact, up to 30% of biopsies obtained from patients with clinically documented HP lack granulomas. The granulomas and giant cells are typically located in the bronchiolar wall or interstitium, but can also be found in alveolar spaces.[20- 22]

CONCLUSION

It is believed that HP is an occupational illness. In all suspected instances, a lung biopsy and bronchoscopy with BAL should be performed to confirm the diagnosis.

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