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Original Research Article.

Study of Evaluation of Clinico-pathological Profile of Patients with Hypersensitivity Pneumonitis

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ABSTRACT

Background: Hypersensitivity pneumonitis (HP) is a respiratory condition characterized by symptoms such as shortness of breath and cough, which arise from the inhalation of an antigen to which the individual has previously been sensitized. Hence; the present study was conducted for evaluation of clinico-pathological profile of patients with hypersensitivity pneumonitis.

Materials & methods: A cohort of 50 patients diagnosed with hypersensitivity pneumonitis was recruited for the study. Comprehensive demographic and clinical information were collected for each participant. Patients suspected of having hypersensitivity pneumonitis underwent a thorough symptom assessment, which included detailed exposure history, general and respiratory physical examinations, routine blood analyses, sputum evaluations, connective tissue serology, complete pulmonary function tests (PFT), as well as transbronchial lung biopsy (TBLB) and endobronchial lung biopsy (EBLB) for selected individuals.

Results: A total of 50 HP patients were evaluated. The mean age of the patients was 51.8 years. 58 percent of the patients were females. Mean duration of symptoms was 15.3 months. Cough, Dyspnea, Fever, Crackles and Digital clubbing were seen in 98 percent, 90 percent, 24 percent, 78 percent, and 20 percent of the patients respectively. BAL lymphocytosis > 30%, bronchocentric inflammation with ill-defined granuloma and bronchocentric inflammation with lymphocytic inflammation was seen in 52 percent, 56 percent and 44 percent of the patients respectively.

Conclusion: HP is classified as an occupational disease; however, it is more accurately described as an exposure-related environmental disease, given that it can manifest in the absence of any occupational exposure. A multidisciplinary approach is the most effective method for diagnosis, necessitating the collaboration of at least a pulmonologist and a pathologist.

Key words: Hypersensitivity, Pneumonitis.

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INTRODUCTION

Hypersensitivity pneumonitis (HP) is a respiratory condition characterized by symptoms such as shortness of breath and cough, which arise from the inhalation of an antigen to which the individual has previously been sensitized. The acute and subacute forms of hypersensitivity pneumonitis are considered the most active manifestations of the disease, with the potential to progress to a chronic state. In some cases, the condition may advance to end-stage lung disease. Diagnosis of hypersensitivity pneumonitis typically depends on a combination of nonspecific clinical symptoms and signs observed in a relevant clinical context, alongside the identification of interstitial patterns on chest radiographs, the presence of serum antibodies against specific antigens, lymphocytic alveolitis detected through bronchoalveolar lavage (BAL), and/or granulomatous changes observed in lung biopsy specimens.[1-3]

HP, similar to many interstitial lung diseases, is classified as a rare condition. A population-based investigation indicated an estimated annual incidence of interstitial lung disease at 30 cases per 100,000 individuals. Within this research, HP represented less than 2% of the newly identified cases. The study took place in New Mexico, a region characterized by a dry climate that is generally unfavorable for the emergence of various types of HP. In the HP Study, 30% of the 661 participants enrolled in this prospective multi-center cohort were diagnosed with HP.[3-6]

Hence; the present study was conducted for present study was conducted for evaluation of clinico-pathological profile of patients with hypersensitivity pneumonitis.

MATERIALS & METHODS

The present study was conducted for evaluation of clinico-pathological profile of patients with hypersensitivity pneumonitis. A cohort of 50 patients diagnosed with hypersensitivity pneumonitis was recruited for the study. Comprehensive demographic and clinical information were collected for each participant. Patients suspected of having hypersensitivity pneumonitis underwent a thorough symptom assessment, which included detailed exposure history, general and respiratory physical examinations, routine blood analyses, sputum evaluations, connective tissue serology, complete pulmonary function tests (PFT), as well as transbronchial lung biopsy (TBLB) and endobronchial lung biopsy (EBLB) for selected individuals. All the results were recorded in Microsoft excel sheet and were subjected to statistical analysis using SPSS software.

RESULTS

A total of 50 HP patients were evaluated. The mean age of the patients was 51.8 years. 58 percent of the patients were females. Mean duration of the symptoms was 15.3 months. Cough, Dyspnea, Fever, Crackles and Digital clubbing were seen in 98 percent, 90 percent, 24 percent, 78 percent, and 20 percent of the patients respectively. BAL lymphocytosis > 30%, Bronchocentric inflammation with ill-defined granuloma and Bronchocentric inflammation with lymphocytic inflammation was seen in 52 percent, 56 percent and 44 percent of the patients respectively.

Table 1: Demographic details

Variable	Number	Percentage
Mean age (years)	51.8	
Males	21	42
Females	29	58
Mean duration of symptoms	15.3 months	

Table 2: Clinical profile

Clinical profile	Number	Percentage
Cough	49	98
Dyspnea	45	90
Fever	12	24
Crackles	39	78
Digital clubbing	10	20

Table 3: Bronchoscopic and histopathological findings

Findings	Number	Percentage
BAL lymphocytosis > 30%	26	52
Bronchocentric inflammation with ill-defined granuloma	28	56
Bronchocentric inflammation with lymphocytic inflammation	22	44

DISCUSSION

HP also referred to as extrinsic allergic alveolitis, is characterized as a granulomatous inflammatory condition of the lungs resulting from the inhalation of antigenic organic particles or vapors. The clinical presentation of this disease can manifest as acute, subacute, or chronic forms. Typically, acute and subacute episodes of HP resolve upon the removal of the offending antigen. In contrast, chronic HP may progress to a state that is irreversible and can lead to significant fibrotic lung disease. Recent research indicates that the pathogenesis of HP is associated with both type III and type IV hypersensitivity reactions, which are mediated by immune complexes and Th1 T cells, respectively. The activation of alveolar macrophages by proinflammatory cytokines and chemokines leads to an influx of CD8+ lymphocytes into the pulmonary system, facilitating granuloma formation and contributing to the onset of pulmonary fibrosis. Interferon-gamma plays a crucial role in the development of HP, while interleukin-10 appears to influence the severity of the disease. Additionally, tumor necrosis factor-alpha and transforming growth factor-beta have been linked to the pulmonary fibrosis observed in chronic HP. Studies have demonstrated that individuals, such as pigeon fanciers, who develop HP exhibit an increased prevalence of the HLA-DRB1*1305 and HLA-DQB1*0501 alleles, a reduced frequency of the HLA-BRB1*0802 allele, and a higher occurrence of the TNF-2 (-308) polymorphism in the TNF-alpha promoter gene.[7-9] Hence; the present study was conducted for assessment clinical and pathological profile of patients with Hypersensitivity pneumonitis.

A total of 100 HP patients were evaluated. The mean age of the patients was 51.8 years. 58 percent of the patients were females.

Mean duration of the symptoms was 15.3 months. Cough, Dyspnea, Fever, Crackles and Digital clubbing were seen in 98 percent, 90 percent, 24 percent, 78 percent, and 20 percent of the patients respectively. BAL lymphocytosis > 30%, Bronchocentric inflammation with ill-defined granuloma and Bronchocentric inflammation with lymphocytic inflammation was seen in 52 percent, 56 percent and 44 percent of the patients respectively. 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. HP was first reported among metal machining workers in 1995. In the following years, additional outbreaks were reported. The most recent outbreak was reported in 2007 from an engine manufacturing facility in England. HP results from an exaggerated immune response, leading to the onset of symptoms similar to acute or progressive lung damage, sometimes irreversible.[10- 12] Morell, Ferran et al evaluated chronic hypersensitivity pneumonitis in patients diagnosed with idiopathic pulmonary fibrosis. 20 of the 46 (43%, 95% CI 29–58) patients with IPF according to 2011 guidelines had a subsequent diagnosis of chronic hypersensitivity pneumonitis: nine patients had positive bronchial challenge testing (eight of whom were also IgG positive and six of these patients also had surgical lung biopsy showing a pattern consistent with chronic hypersensitivity pneumonitis); seven were IgG positive plus had histopathology on surgical lung biopsy that was consistent with hypersensitivity pneumonitis; one was IgG positive plus had greater than 20% lymphocytes in bronchoalveolar lavage fluid; and three had findings on surgical lung biopsy that were consistent with subacute hypersensitivity pneumonitis (and IgG positive). Altogether, 29 of 46 patients diagnosed with IPF who had met the 2011 criteria had lung tissue available for histopathology (surgical lung biopsy in 28 patients and explanted lung in two patients, one of whom also had surgical biopsy) during the study period, and 16 of the 20 patients with chronic hypersensitivity pneumonitis had histopathological features on surgical lung biopsy that were consistent with this diagnosis. 26 of the 46 patients remained with a diagnosis of IPF.[13]

CONCLUSION

HP is classified as an occupational disease; however, it is more accurately described as an exposure-related environmental disease, given that it can manifest in the absence of any occupational exposure. A multidisciplinary approach is the most effective method for diagnosis, necessitating the collaboration of at least a pulmonologist, a radiologist, and a pathologist.

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