### ORIGINAL RESEARCH

# Prospective Analysis of Burden of Hypersensitivity Pneumonitis at a Tertiary Care Hospital

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## **ABSTRACT**

Background: Establishing whether patients are exposed to a 'known cause' is a key element in both the diagnostic assessment and the subsequent management of hypersensitivity pneumonitis (HP).

Objective: The aim of the study is to determine the burden of hypersensitivity pneumonitis at a respiratory center.

Methods: 100 patients had been enrolled to the study. Demographic, clinical, radiological and aetiological data were collected with a random identification code for each patient in view of maintaining the confidentiality during data collection.

Results: Out of 100 patients, there were 65 females and 35 males. Mean age of the population was 55.50 years (SD = 10.50) years. 50 patients were less than 65 years of which 30 were aged between 45 to 60 years while 20 were aged <5 years.

Conclusion: In general, patients with acute disease, if correctly and timely diagnosed and treated, have a good prognosis, and patients usually improve. By contrast, patients with subacute/chronic HP (in particular those with bird fancier's disease) often progress to irreversible pulmonary fibrosis and may die within a few years after diagnosis.

Keywords: Hypersensitivity Pneumonitis, Prognosis, Chronic.

## **INTRODUCTION**

Hypersensitivity pneumonitis (HP) is a complex syndrome resulting from repeated exposure to a variety of organic particles. HP may present as acute, subacute, or chronic clinical forms but with frequent overlap of these various forms. Hypersensitivity pneumonitis (HP) is a complex syndrome caused by exposure to a wide variety of organic particles small enough to reach the alveoli (<5 mm). In susceptible individuals, these antigens provoke an exaggerated immune response of the small airways and lung parenchyma. The causative antigens include fungi; bacterial, protozoal, animal, and insect proteins; and low–molecular-weight chemical compounds. HP may occur in a variety of occupational, home, and recreational environments. The prevalence varies considerably around the world, depending on disease definition, diagnostic methods, type and intensity of exposure, geographical conditions, agricultural and industrial practices, and host risk factors. Furthermore, the definite prevalence of HP is uncertain, primarily because cases may go undetected or are misdiagnosed. In addition, there

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is no consistent, standardized epidemiological approach for assessing the various forms of HP. High attack rates may be found among exposed individuals during sporadic outbreaks and in occupational settings. Studies on incidence are scanty. In a large, general-population—based study, the incidence of HP was approximately 1 per 100,000 in the UK population. The disease is uncommon in children, and a recent report in Denmark showed an incidence of 2 per year and a prevalence of 4 per 1,000,000 children.

HP is seen worldwide, and the most commonly implicated antigens are thermophilic actinomycete species, fungi, and bird proteins. Thermophilic actinomycete (i.e., Saccharopolyspora rectivirgula) and a variety of fungi (i.e., Aspergillus species and Penicillium species) are implicated in HP in a variety of occupations, such as farming, but also may be responsible for the disease acquired in home environments.<sup>4</sup>

Even though the diagnosis may be straightforward given the right clinical and radiological evidence, the management would be challenging at times in the absence of overt evidence for underlying aetiology.<sup>5</sup>

The aim of the study is to determine the burden of hypersensitivity pneumonitis at a respiratory center.

#### **METHODS**

The present study included participants with clinical radiological and pathological, characteristics consistent with the diagnosis of hypersensitivity pneumonitis. 100 patients had been included in the study. Demographic, clinical, radiological and aetiological data were collected with a random identification code for each patient in view of maintaining the confidentiality during data collection.

Data was analysed by a separate person who didn't involve with the data collection using SPSS (26 version). Population characteristics studied were described using descriptive statistics such as mean, median and standard deviation and frequency.

#### **RESULTS**

Out of 100 patients, there were 65 females and 35 males. Mean age of the population was 55.50 years (SD = 10.50) years. 50 patients were less than 65 years of which 30 were aged between 45 to 60 years while 20 were aged <5 years. Shortness of breath 65%) and Cough (57%) were the most common symptoms on presentation.

Mean duration of symptoms was 6 months (SD = 2.4 months). 40 patients had severe restrictive (FVC less than 50% of predicted) lung disease by presentation, while another 35 had moderate restriction (FVC of 50% to 80% of predicted). Moderate to Severe fibrosis were evident in almost 70% patients while only 30% having mild or no fibrosis. (Table 1,2,3)

Table 1: Distribution of patients according to age

Age group	N
<40	20
45-60	30
>60	50
Total	100

Table 2: The distribution of patients according to the degree of restrictive lung disease

	n
Mild restriction	25%
<b>Moderate restriction</b>	35%
Severe restriction	40%

Table 3: Distribution of patients in relation to the degree of pulmonary fibrosis in HRCT scan

Mild or no fibrosis	30%
Moderate or severe fibrosis	70%

#### DISCUSSION

Hypersensitivity pneumonitis (HP) is a common form of interstitial lung disease (ILD), with widely varying causes and clinical outcomes. Differentiating HP from other forms of fibrotic ILD is a common diagnostic dilemma for multidisciplinary teams (MDTs), resulting in a low level of diagnostic agreement internationally.

There had been a substantial patient delay in seeking medical help for their condition in the studied population.

Most of the affected were having moderate to severe disease by presentation both clinically and radiologically. This could have been partly accounted by the substantial delay in their presentation to the medical service. Previous studies have shown that repeated exposure to the offending agent long term leads to progressive decline in lung function.<sup>9</sup>

Although consensus agreement was not reached, 54% of participants agreed that they commonly attribute HP to be an 'idiopathic disease'. It is not possible from the survey results to determine whether this term is used to reflect the difficulties clinicians encounter in identifying the cause, or a true belief that HP can occur spontaneously (ie, without there being a cause to identify). Notably, 43% of participants recognised that BAL lymphocytosis in HP can persist following cessation of exposure, and in some cases, it may therefore be impossible to identify the cause, if it is no longer present in the work or home environment. Causation in HP is likely to vary between countries due to a wide range of factors, including differences in geography, climate, housing and industry. In terms of identifying possible causes, GB HP survey participants reported that they more commonly attribute the disease to domestic exposures in the home or garden, than occupational exposures in the workplace. For domestic HP, the most commonly suspected exposures are to birds, bird droppings or feathers. This is in keeping with the majority of studies from other countries, where avian exposure has been the most commonly identified cause, accounting for 17%–66% of all cases. 10,11,12

## **CONCLUSION**

In conclusion, the GB HP survey has demonstrated national variation in the utilisation of invasive diagnostic tests in HP, but consensus opinion for some of the key aspects of practice relating to establishing causation. Prevalence in females and people of working age, Delayed presentation, established fibrosis and advanced disease on presentation could have potentially influenced the outcome of hypersensitive pneumonitis in the studied population. Public awareness regarding the disease is essential in terms of minimizing its burden on the community.

## **REFERENCES**

- 1. Selman M, Buendía- Roldán I, Navarro C, Gaxiola M. Hypersensitivity pneumonitis. In Pulmonary Hypertension and Interstitial Lung Disease 2017 (pp. 145-164). Springer, Cham.
- 2. Solaymani-Dodaran M, West J, Smith C, Hubbard R. Extrinsic allergic alveolitis: incidence and mortality in the general population. Journal of the Association of Physicians. 2007 Apr 1;100(4):233-7.
- 3. Buchvald F, Petersen BL, Damgaard K, Deterding R, Langston C, Fan LL, Deutsch GH, Dishop MK, Kristensen LA, Nielsen KG. Frequency, treatment, and functional outcome

- in children with hypersensitivity pneumonitis. Pediatric pulmonology. 2011 Nov;46(11):1098-107.
- 4. Selman M, Lacasse Y, Pardo A, Cormier Y. Hypersensitivity pneumonitis caused by fungi. Proceedings of the American Thoracic Society. 2010 May 15;7(3):229-36.
- 5. Wild LG, Lopez M. Hypersensitivity pneumonitis: a comprehensive review. Journal of investigational allergology & clinical immunology. 2001 Jan 1;11(1):3-15.
- 6. Kouranos V, Jacob J, Nicholson A, Renzoni E. Fibrotic hypersensitivity pneumonitis: key issues in diagnosis and management. Journal of clinical medicine. 2017 Jun;6(6):62.
- 7. Jo HE, Corte TJ, Moodley Y, Levin K, Westall G, Hopkins P, Chambers D, Glaspole I. Evaluating the interstitial lung disease multidisciplinary meeting: a survey of expert centres. BMC pulmonary medicine. 2016 Dec;16(1):1-6.
- 8. Walsh SL, Wells AU, Desai SR, Poletti V, Piciucchi S, Dubini A, Nunes H, Valeyre D, Brillet PY, Kambouchner M, Morais A. Multicentre evaluation of multidisciplinary team meeting agreement on diagnosis in diffuse parenchymal lung disease: a case-cohort study. The lancet Respiratory medicine. 2016 Jul 1;4(7):557-65.
- 9. Schmidt CD, Jensen RL, Christensen LT, Crapo RO, Davis JJ. Longitudinal pulmonary function changes in pigeon breeders. Chest. 1988 Feb 1;93(2):359-63.
- 10. Hanak V, Golbin JM, Ryu JH. Causes and presenting features in 85 consecutive patients with hypersensitivity pneumonitis. In Mayo Clinic Proceedings 2007 Jul 1; 82(7): 812-6.
- 11. Pérez ER, Swigris JJ, Forssén AV, Tourin O, Solomon JJ, Huie TJ, Olson AL, Brown KK. Identifying an inciting antigen is associated with improved survival in patients with chronic hypersensitivity pneumonitis. Chest. 2013 Nov 1;144(5):1644-51.
- 12. Lacasse Y, Selman M, Costabel U, Dalphin JC, Ando M, Morell F, Erkinjuntti- Pekkanen R, Muller N, Colby TV, Schuyler M, Cormier Y. Clinical diagnosis of hypersensitivity pneumonitis. American journal of respiratory and critical care medicine. 2003 Oct 15;168(8):952-8.