

RESEARCH

Open Access



# Factors influencing the clinical presentation of hypersensitivity pneumonitis in pigeon breeders in Minia governorate: an Egyptian experience

Nezar R. Mohamed<sup>1,2</sup>, Eman Ramadan Ghazawy<sup>1,2</sup> and Zainab H. Saeed<sup>1,2\*</sup>

## Abstract

**Background:** Bird fanciers' lung (BFL) is a very famous type of hypersensitivity pneumonitis (HP) produced by airborne exposure to avian antigens. Immunological response and duration of exposure to the avian protein increase the risk of developing BFL. The current study investigates the risk factors and the clinical characteristics of BFL in pigeon breeders. This study aimed to determine the risk factors and clinical characteristics of HP in pigeon breeders.

**Results:** This cross-sectional observational study included 67 hypersensitivity pneumonitis patients with a history of pigeon breeding. Patients were subjected to history taking (age, smoking history and duration, and type of exposure to birds), clinical examination, chest X-ray, high-resolution computed tomography (HRCT), oxygen saturation, and spirometry.

Most of BFL patients were females (83.6%) and non-smokers (86.6%). Breathlessness, cough, fever, and crackles were the most common findings. Sweeping for birds was associated with more reduction of forced vital capacity (FVC) ( $p$  value 0.02). Patients who were exposed in closed places had a rapid onset of symptoms ( $p$  value 0.01).

**Conclusion:** In this study, most of the patients with BFL are females and non-smokers. Sweeping for birds and exposure in closed areas are important risk factors of HP in BFL.

**Keywords:** Hypersensitivity, Pneumonitis, Pigeon, Bird breeders

## Background

Hypersensitivity pneumonitis (HP) is an induced, non-immunoglobulin E (IgE)-mediated immunologic lung disease resulting from recurrent exposure to a variety of inhaled organic dust [1].

Bird fanciers' lung (BFL) is one of the common causes of hypersensitivity pneumonitis [2]. It is an immunologically mediated lung disease caused by inhalation of bird dropping extracts and antigens in feathers [3]. A

clinical prediction rule can help accurate diagnosis of active HP [4].

Immunological response and duration of exposure to the avian protein increase the risk of developing BFL [5].

HP is less frequent in smokers than in nonsmokers [6]. There was a significant relation between heavy exposure and case deterioration [7].

Avian antigens are complex proteins found in the feathers, droppings, and serum of turkeys, chickens, geese, ducks, parakeets, parrots, pigeons, doves, and love birds. Immunoglobulins are released from both birds' feathers and intestinal mucin which is largely produced by flying birds such as pigeons and parakeets [8].

\* Correspondence: [Chestawya@yahoo.com](mailto:Chestawya@yahoo.com)

<sup>1</sup>Department of Chest, Minia University, Minia, Egypt

<sup>2</sup>Department of Public Health and Preventive Medicine, Faculty of Medicine, Minia University, Minia, Egypt

Although bird breeding at houses in Egypt is a common practice, yet there is a paucity of studies that assess the bird fanciers' lung among Egyptians particularly in Upper Egypt. Therefore, the objective of this study was to determine the risk factors and clinical characteristics of hypersensitivity pneumonitis in pigeon breeders in Egypt.

## Methods

### Study design

This cross-sectional observational study enrolled 67 patients with HP randomly selected from patients who sought medical advice in the outpatient clinics of the Chest Department of Minia Cardiothoracic University Hospital during the period from May to November 2018.

The study was approved by the Faculty of Medicine Ethics Committee, Minia University. All patients provided written informed consent.

### Study population

The study participants were patients consulting the chest outpatients' clinic in Minia University Hospital throughout the study period. The total number of patients during the study period was 70 hypersensitivity pneumonitis patients, of whom 3 patients refused to participate in the study, leaving 67 patients with a response rate of 95.7%.

The inclusion criteria in the study were patients with a history of breeding of birds and confirmed diagnosis of hypersensitivity pneumonitis according to these criteria [9]: evidence of exposure to a provocative antigen, HRCT scan shows "classic" features (e.g., small centrilobular nodules, ground-glass attenuation, and lobular areas of decreased attenuation and vascularity [10]) and bronchoalveolar lavage (BAL) lymphocytosis > 40%.

Exclusion criteria were as follows:

1. Patients with significant exposures, e.g., those who work in stone cutting, cement industries, or any other exposures known to affect the lung parenchyma.
2. Chronic obstructive pulmonary disease.
3. Patients with significant clinical or laboratory evidence of connective tissue diseases.
4. Patients with typical usual interstitial pneumonia (UIP) pattern in HRCT especially basal sub-pleural honeycomb appearance.

A structured interview questionnaire was designed and included inquiries about sociodemographic characteristics of the participants, smoking history, detailed history of breeding of birds, and general and respiratory symptoms.

Patients were subjected to clinical examination. Chest X-ray, high-resolution chest computed tomography (HRCT), and oxygen saturation (O<sub>2</sub> sat) were done.

Spirometry and bronchodilator reversibility were performed using a spirometer (ZAN 300, Germany) according to the American Thoracic Society Standard [11].

Forced expiratory volume in one second (FEV<sub>1</sub>)% predicted, forced vital capacity (FVC)% predicted, and FEV<sub>1</sub>/FVC% were measured.

### Statistical analysis

Data entry and analysis were all done using software called SPSS for windows version 22.

Quantitative data were presented by mean and standard deviation, while qualitative data were presented by frequency distribution. Chi-square test was used to compare between proportions. Student *t* test was used to compare between means of two groups.

Pearson correlation analysis was used to describe the association between numerical variables within each group.

A statistically significant level was considered when *p* value was less than 0.05.

## Results

This study includes 67 patients, whose ages ranged between 15 and 65 years with a mean of  $38.12 \pm 13.07$  years. Almost 83.6% of the patients were females, and the majority of them, 86.8%, were non-smokers. Over 90% of the patients had no family history of HP. About 71.6% of patients were sweeping for pigeons with a mean duration of exposure of  $53.92 \pm 66.1$  months.

The mean interval from initiation of exposure until symptoms was  $16.41 \pm 10.8$  months (Table 1).

Cough and dyspnea were the most common clinical symptoms (97% and 94% respectively), and nearly 76.1% had grade II dyspnea at diagnosis. On clinical examination, basal crepitations were heard on more than half (52.2%) of patients (Table 2). Restrictive ventilatory impairment was the most frequent functional pattern (86.6%) (Table 2).

The most frequent radiologic finding was basal veiling in 4/40 (35%) of patients. Common chest CT features were ground-glass areas and a mosaic pattern (61%) (not shown in tables).

Patients who had a history of exposure to smoking had a better FVC ( $61.6 \pm 16.9$ ) compared to those of whom had never exposed ( $48.7 \pm 17.8$ ) (Table 3).

When comparing the pattern of exposure, it was found that patients who were exposed in closed places had a more rapid onset of symptoms after exposure than those who were breeding in open areas ( $11.39 \pm 10.9$  vs  $22.8 \pm 20.21$ ) respectively. However, there were no statistically significant

**Table 1** Demographic and epidemiological data

Variables	Number (%)
<b>Age</b>	15–65, 38.12 ± 13.07
<b>Gender</b>	
Male	11 (16.4%)
Female	56 (83.6%)
<b>Smoking status</b>	
None smoker	58 (86.6%)
Ex-smoker	9 (13.4%)
Current smoker	0 (0%)
<b>Biomass exposure</b>	
No	29 (43.3%)
Previous exposure	30 (44.8%)
Current exposure	8 (11.9%)
<b>Passive smoking exposure</b>	
No	45 (67.2%)
Yes	22 (32.8%)
<b>Family history of HP</b>	
Yes	4 (6%)
No	63 (94%)
<b>Duration of exposure to birds (month)</b>	3–240, 53.92 ± 66.1
<b>Place of exposure</b>	
Closed place	28 (41.8%)
Open place	39 (58.2%)
<b>Sweeping for pigeons and birds</b>	
Yes	48 (71.6%)
No	19 (28.4%)
<b>Other species of birds in association</b>	
Yes	56 (83.6%)
No	11 (19.4%)
<b>Interval from initiation of exposure until symptoms (months)</b>	1–60, 16.41 ± 10.8

Data presented as no.%, range and mean ± SD  
 HP hypersensitivity pneumonitis

differences between the two patterns of exposure as regards FVC, grades of dyspnea, and O<sub>2</sub> saturation (Table 4).

Regarding sweeping, it was found that patients who sweep had a more rapid onset of symptoms after exposure than those who not sweep (12.6 ± 4.4 vs 16.3 ± 6.4) respectively. Additionally, patients who had a history of sweeping for birds had a significant reduction in their FVC compared to those who never sweep for birds (53.18.8 ± 18.8 vs 60.3 ± 2.31). There were statistically significant differences between the two groups as regards the grades of dyspnea (Table 5).

**Table 2** Symptoms, physical examination, and pulmonary function tests

Variable	Number (%)
<b>Symptoms</b>	
Cough	65 (97%)
Dyspnea	63 (94%)
Flu-like symptoms	43 (64.2%)
Wheezing	44 (65.7%)
Fever	14 (20.9%)
Loss of weight	6 (9%)
<b>Physical examination</b>	
Basal crepitation	35 (52.2%)
Diffuse crepitation	4 (6%)
Wheezes	6 (9%)
Digital clubbing	4 (6%)
<b>Pulmonary function</b>	
Restrictive pattern	58 (86.6)
Normal	9 (14.3)

**Discussion**

Reed et al. were the first to diagnose pigeon breeder’s lung [12].

A noninvasive testing like antigen exposure, recurrent symptoms after exposure, inspiratory crepitations, and weight loss could have a high probability of 98% of diagnosing HP, so BAL or lung biopsy would be unnecessary for the confirmation especially with consistent HRCT findings [13].

In the present study, all the diagnosed subjects gave a significant history of exposure to pigeons. The mean age was 38.12 ± 13.07 which is younger than most previous studies. In a large Spanish study of 86 BFL [5], the mean age was 47 years.

Moreover, Selman et al. [14] reported similar results where the mean age of BFL patients was 49 years.

**Table 3** Effect of smoking and biomass exposure on FVC and FEV1

	FVC	FEV1
<b>Exposure to smoking</b>		
No	48.7 ± 17.8	54.8 ± 18.5
Yes	61.6 ± 16.9	63.7 ± 15.9
<i>p</i> value	0.02	0.1
<b>Biomass exposure</b>		
No	56.8 ± 16.3	60.0 ± 18.2
Yes	51.87 ± 19.6	56.86 ± 17.4
<i>p</i> value	0.3	0.6

Data presented as mean ± SD  
 FVC forced vital capacity, FEV1 forced expiratory volume in first second

**Table 4** Effect of the pattern of exposure on different clinical and functional parameters

Variables	Pattern of exposure		p value
	Closed place	Open place	
<b>Duration of symptoms (months)</b>	51.6 ± 67.56	30.7 ± 57.27	0.2
<b>FVC</b>	48.13 ± 20.94	57.8 ± 15.47	0.09
<b>Oxygen saturation</b>	95.5 ± 2.35	96.1 ± 2.26	0.4
<b>Interval from initiation of exposure until symptoms (months)</b>	11.39 ± 10.9	22.8 ± 20.21	0.01
<b>Dyspnea grade</b>			
Grade 2	24 (85.7%)	27 (77.1%)	0.4
Grade 3	4 (14.3%)	6 (17.1%)	
Grade 4	0 (0%)	2 (5.7%)	

FVC forced vital capacity

The close contact to birds of which sweeping is the most prominent feature in Egypt could be considered one prominent factor of rapid and early onset of the disease, so the presentation occurs at younger age.

High proportion of disease among females is anticipated as they spent more time at home. In confirmation of that, the current study found that 56 (83.6%) of patients were females. Several studies found the same finding [5, 10, 14]. Also, in our community, women are usually more concerned with caring and sweeping for birds as a part of their daily activities.

As regards smoking history, 58 (86.6%) of our patients were nonsmokers. This could be attributed to the predominance of females in the current study where smoking habit is rare in Egyptian community. Similarly, Selman et al. reported that 83% of BFL were never-smokers [13]. Also, in a large Spanish study, 78% of BFL patients were non-smokers [5]. The incidence of HP in non-smokers is higher than smokers, as smokers had a lower level of expression of immunostimulatory molecules such as peripheral membrane protein B7 on their alveolar macrophages [15].

As shown in the results, most patients were not exposed to biomass fuel and this can be explained in two

ways, first is the decreased use of biomass fuel in general in our community and the other may be the same biologic effect of smoking on macrophages. Hirschmann et al. [16] reported that heredity may play an important role in HP, with families positive for HLA-DR7, HLA-B8, and HLA-DQw3 showing a stronger predisposition.

We also found that 7.7% of patients had a positive family history of HP. As regards the clinical manifestation of HP in our study, 63 patients (94%) presented with dyspnea, and 65 (97%) patients presented with cough. Physical examination revealed crackles in 58.2% of patients. Finger clubbing was observed in 6% of patients. In the same line, Morell et al. [5] found that 98% of patients presented with dyspnea, 82% presented with cough, and only 7% had finger clubbing. Contrary, Sansores et al. [17] found a larger percentage (51%) of clubbing in BFL.

A restrictive pulmonary function is almost the case in BFL, but sometimes, patients had normal pulmonary functions after resolution of acute stage [18].

In the present study, 9 (14.3%) had normal pulmonary functions.

Tsutsui et al. [19] found a more rapid deterioration in clinical and functional state in BFL cases exposed to a

**Table 5** Effect of the sweeping on different clinical and functional parameters

Variables	Sweep		p value
	Yes	No	
<b>Duration of symptoms (months)</b>	41.4 ± 63.4	6.89 ± 1.4	0.02
<b>FVC</b>	53.6 ± 18.8	60.3 ± 2.31	0.02
<b>Oxygen saturation</b>	95.7 ± 2.2	98.0 ± 0.2	0.01
<b>Interval from initiation of exposure until symptoms (months)</b>	12.6 ± 4.4	16.3 ± 6.4	0.008
<b>Dyspnea grade</b>			
Grade 2	32 (72.7%)	19 (100%)	0.04
Grade 3	10 (22.7%)	0 (0%)	
Grade 4	2 (4.5%)	0 (0%)	

FVC forced vital capacity

higher concentration of avian antigens collected from their household environments. This gives a clear explanation for two significant findings in the present study which are as follows: first, the more rapid onset of symptoms after the onset of exposure in patients who were exposed to pigeons in closed areas than those breeding in open areas ( $p$  value 0.01), and second, the significant reduction in FVC%, O<sub>2</sub> saturation, increase in dyspnea grade, and more rapid onset of symptoms after exposure in patients who had a history of sweeping for birds ( $p$  value 0.02, 0.01, 0.04, and 0.008 respectively) which is also a common habit in our community.

## Conclusion

In a study of HP in bird breeders in Minia governorate, Egypt, most of the patients were young-aged females and non-smokers.

Sweeping for birds which is a common habit among Egyptian females especially in upper Egypt is an important risk factor of developing more rapid, early symptoms, long duration of symptoms, and functional deterioration of HP in Egyptian bird fanciers.

Our study is limited by its small number of patients. An accurate quantification of avian antigens is required for the future management of BFL.

## Supplementary information

**Supplementary information** accompanies this paper at <https://doi.org/10.1186/s43168-020-00019-w>.

**Additional file 1:** STROBE checklist.

## Abbreviations

BFL: Bird fanciers lung; HP: Hypersensitivity pneumonitis; HRCT: High-resolution computed tomography; FVC: Forced vital capacity; IgE: Immunoglobulin E; BAL: Bronchoalveolar lavage; O<sub>2</sub> sat: Oxygen saturation; FEV<sub>1</sub>: Forced expiratory volume in one second

## Acknowledgements

N/A

## Authors' contributions

NM collected the patients' data and revised the methods and results. ZS conceived the publication design and prepared the manuscript. EG performed the statistical component. All authors have read and approved the final manuscript.

## Funding

Nil

## Availability of data and materials

Not applicable

## Ethics approval and consent to participate

This study was approved by the hospital's research ethics board of Minia University, and informed consent was obtained from either patients themselves or their relatives. Committee's reference number not available.

## Consent for publication

Not applicable

## Competing interests

No competing interests

Received: 5 May 2020 Accepted: 15 July 2020

Published online: 29 July 2020

## References

- Alberts W. Hypersensitivity pneumonitis. In: ACCP Pulmonary Med Brd Rev, 25th ed., 2009. pp.63–72.
- Chan A, Juarez M, Leslie K, Ismail H, Albertson T (2012) Bird fancier's lung: a state-of-the-art review Clin. Rev. Allergy Immunol. 43:69–83
- Moss J, Maniaci M, Johnson M (2010) 73-year-old woman with progressive shortness of breath Mayo Clin. Proc. 85:95–98
- Lacasse Y, Selman M, Costabel U, Dalphin J, Ando M, Morell F et al (2003) Clinical diagnosis of hypersensitivity pneumonitis. Am.J.Respir.Crit. Care Med. 168:952–958
- Morell F, Roger A, Reyes L, Cruz MJ, Murio C, Munoz X. Bird fancier's lung: a series of 86 patients. Medicine(Baltimore). 2008;87:110-30.
- Selman MCA, Pardo A, King TE (2012) Hypersensitivity pneumonitis. American Journal of Respiratory and Critical Care Medicine. 186(4):314–324
- Annals of the American Thoracic Society. Jul 01,2015 Vol:12, No.7.
- Karen C. Patterson MD, Cecile S. Rose MD, MPH, in Murray and Nadel's textbook of respiratory medicine (Sixth Edition); 2016
- Julie Morisset , Kerri A. Johansson , Kirk D. Jones , Paul J. , Harold R. Collard , Simon L. F. Walsh , Brett Ley , and the HP Delphi Collaborators. Wolters Identification of diagnostic criteria for chronic hypersensitivity pneumonitis. An International Modified Delphi Survey. AJRCCM. 2017; Volume 197:Issue 8
- Kumar R, Singh M (2015) Bird fancier's lung: clinical-radiological presentation in 15 cases. Pneumonol Alergol Pol. 83(1):39–44
- ATS/ ERS, standardization of spirometry, 2005.
- Reed C, Barbee R (1965) Pigeon-breeders' lung: a newly observed Interstitial pulmonary disease. JAMA. 193:261–265
- Demedts M, Wells AU, Anto JM, Costabel U, Hubbard R, Cullinan P, et al. Interstitial lung diseases: an epidemiological overview. Eur Respir J 200; Suppl32:2 s–16 s.
- Selman M, Lacasse Y, Pardo A, Cormier Y (2010) Hypersensitivity pneumonitis caused by fungi. Proc Am Thorac Soc. 7:229–236
- Carrillo T, Rodriguez de Castro F, Cuevas M, Diaz F, Cabrera P (1991) Effect of cigarette smoking on the humoral immuneresponse in pigeon fanciers. Allergy. 46:241–244
- Hirschmann J, Pipavath S, Godwin J (2009) Hypersensitivity pneumonitis: a historical, clinical, and radiologic review. Radiographics. 29(7):1921–1938. <https://doi.org/10.1148/rg.297095707>
- Sansores R, Salas J, Chapela R, Barquin N, Selman M (1990) Clubbing in hypersensitivity pneumonitis. Its prevalence and possible prognostic role. Arch Intern Med. 150:1849–1851
- Sharma O (1989) Hypersensitivity pneumonitis: a clinical approach. Prog Respir Res. Basel. Karger 23:49–50. <https://doi.org/10.1159/000417418>
- Tsutsui T, Miyazaki Y, Kuramochi J, Uchida K, Eishi Y, Inase N (2015) The amount of avian antigen in household dust predicts the prognosis of chronic bird-related hypersensitivity pneumonitis. Ann Am Thorac Soc. 12(7):1013–1021

## Publisher's Note

Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.