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Influence of epidemiological factors on asthma phenotypes among asthma patients attending a tertiary care center – A cross-sectional study

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ABSTRACT

Background: Asthma is a heterogeneous disease presenting with various phenotypes usually characterized by chronic airway inflammation. **Aims and Objectives:** The aim of the study was to identify epidemiology of bronchial asthma phenotypes in patients attending to tertiary care center. **Materials and Methods:** This was a descriptive cross-sectional study involving 69 patients who are suspected to have asthma according to the Global Initiative for Asthma guidelines. The data of each patient were collected on a proforma specially designed for this study on demographic data, socioeconomic status, body mass index, exposure to environmental factors, and other history-related questions. Sputum cytology and absolute eosinophil count were also examined using coGuide and $P<0.05$ was considered significant. Asthma phenotypes were the primary outcome and association of environmental factors with asthma phenotypes was the secondary outcome. **Results:** Out of 69, majority 26 (37.7%) were aged 31–40 years; 35 (50.7%) males and 34 (49.3%) were females. Fifty-eight (84.1%) were illiterate and 46 (66.7%) belonged to low class. Sputum cytology report showed that majority of them, 66.7%, had eosinophilia. The majority, 48 (69.6%), of the participants were triggered by weather and 55.1% had a history of respiratory tract infections. Thirty-three (47.83%) participants had obesity associate asthma, 34 (49.28%) had allergic asthma, and 20 (28.99%) had smoking-related asthma phenotypes. The difference in the proportion of few environment factor was significant for allergic and smoking-related asthma ($P>0.005$). **Conclusion:** The factors associated with the asthma phenotypes will help us further in anticipating the clinical course and better management of the specific phenotypes.

Key words: Asthma; Biomarkers; Phenotypes; Respiratory tract infections; Tobacco smoking

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INTRODUCTION

Asthma is the most common non-communicable disease globally that is ranked 16th among the primary reason for years lived with disability and 28th among the main reason for increased burden of disease, as calculated by disability-adjusted life years.[1] Three hundred million people worldwide are affected by the disease (around 4.5% of adults and 11% of children worldwide).[2] Moreover, it is likely that by 2025, a further 100 million may be affected as its prevalence has more than doubled in the past decades throughout the globe.[3]

According to the current evidence, asthma is a complex multifactorial disorder, and its etiology is increasingly attributed to interactions between genetic susceptibility, host factors, and environmental exposures. Mainly, the IL1 family gene and its members participate in inflammation and are considered to be having a well-established biological plausibility for asthma or related phenotypes.[4,5]

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Recently, there has been an increase in many studies which are trying to identify asthma phenotypes based on non-invasive Type 2-markers, and more recent work has shown that this model explains the global asthma patterns and time trends.[6,7] Phenotypes were classified into T-helper-2 high and T-helper-2 low subtypes. Cluster analysis were used to recognize the phenotypes.[8] About <50% of asthma cases are attributable to eosinophilic airway inflammation that has been demonstrated by studies using induced sputum.[9] The most common approaches to identify asthma phenotypes include clinical, trigger-related, demographic, and pathological factors, statistical methods such as latent class analysis.[10] Allergic multimorbidity phenotypes have been identified based on eczema, rhinitis, and asthma in the large Mechanisms of the Development of ALLergy study.[11]

The previous studies reported that asthma is a heterogeneous disease presenting various phenotypes.[12] There are very few studies reported in India. Therefore, we conducted a study aimed to determine the epidemiological factors associated with the different phenotypes of bronchial asthma.

Aims and objectives

The objectives of the study are as follows:

1. To study the epidemiological factors affecting bronchial asthma phenotypes among asthma patients attending tertiary care centers
2. To assess the influence of environmental exposure on the prevalence and expressions of various bronchial asthma phenotypes.

MATERIALS AND METHODS

Study design

This was a descriptive and cross-sectional study.

Source population

Patients attending the pulmonary outpatient department of tertiary care hospital.

Study population

The study involves patients with symptoms suggestive of bronchial asthma who are diagnosed to have asthma according to the Global Initiative for Asthma (GINA). A patient is suspected of having asthma if he or she has any one of the following symptoms as advocated by GINA guidelines 2015.[3]

1. Wheeze, shortness of breath (dyspnoea), chest tightness, or cough
2. Variable expiratory airflow limitation
3. Symptoms frequently worsen at night or early morning
4. Symptoms vary over time and in intensity
5. Symptoms are triggered by viral infections, exercise, allergen exposure, changes in weather, or exposure to irritants.

Sample size and sampling procedure

All 69 subjects diagnosed with asthma according to the GINA guidelines were selected using convenient sampling for the feasibility of the study.

Calculation

Based on the previous study, prevalence value is 2.38%. Based on Aggarwal et al.,[13]

n=4pq/L2 where p=2.38, q=100-2.38=97.62.

L is precision error = 4% (absolute precision error).

n=4×2.38×97.62/42

=989.34/16

=62.08 (10% non-response error)

=62.08+5.80

=68.88≈69.

Study duration

The duration of the study was for a period of 2 years from March 2018 to March 2020.

Inclusion criteria

The following criteria were included in the study:

i. Patients aged more than 14 years but <60 years
ii. Patients with dyspnea, wheeze, chest tightness, or cough
iii. Patients with significant post-bronchodilator reversibility
iv. Patient with no significant lung lesions.

Exclusion criteria

The following criteria were included in the study:

The following criteria were excluded from the study:

i. Patients with bronchiectasis, chronic bronchitis, and emphysema
ii. Patients with acute exacerbation of asthma
iii. Patients who cannot perform spirometry
iv. Patients with no significant post-bronchodilator reversibility.

Ethical and informed consent

Institutional Ethical Committee approval was obtained before the commencement of the study from the corresponding hospital. After explaining the study, informed written consent from each participant was obtained, and confidentiality of the data was maintained.

Data collection

The data of each patient were collected on a proforma specially designed for this study on demographic data, occupation, socioeconomic status, body mass index, birth order, history of allergy/atopy, presence of disease in family members, exposure to farm products in childhood, exposure to allergens, chemicals at workplace, history of sensitivity to aspirin/NSAIDs (non-steroidal anti-inflammatory drugs) exposure to environmental tobacco

smoke, food habits, family size, history of recurrent respiratory tract infections, history of gastroesophageal reflux disorder, changes in climatic conditions, history of stress and emotional conditions, knowledge about the disease, diagnosis, and mode of treatment.

Investigations

The study investigations are spirometry, sputum cytology, and absolute eosinophil count.

Statistical methods

Demographic parameters, environmental factors, specific environmental factors, and knowledge of diagnosis and symptom control were considered as the primary outcome variable. Absolute eosinophil count (elevated eosinophilia vs. normal eosinophilia) was considered as a primary explanatory variable.

Descriptive analysis

Quantitative variables were represented as mean and standard deviation, and frequency and proportion for categorical variables were represented as frequency and proportion. Inferential statistics: The association between explanatory variables and categorical outcomes was assessed by cross-tabulation and comparison of percentages using the Chi-square test. P<0.05 was considered statistically significant. coGuide version V.1.0 was used for statistical analysis.[14]

RESULTS

A total of 69 subjects were included in the final analysis.

Table 1: Among the study population, the majority 26 (37.7%) were aged between 31 and 40 years; 35 (50.7%) were male, and the remaining 34 (49.3%) were females. Most of them, 58 (84.1%), were illiterate and 46 (66.7%) belonged to the low class. The majority (52.50%) of the participants were normal. The proportion of 40.6% was obese according to the BMI.

Table 2: The mean absolute eosinophil count was 470.29 cells/μL in the study population. Sputum cytology report showed that the majority 66.7% had eosinophilia. Forty-nine (71%) participants knew diagnosis and 60 (87%) knew symptom control.

Table 3: Out of 69 participants, 33 (47.83%) participants had obesity associate asthma, 34 (49.28%) had allergic asthma, and 20 (28.99%) had smoking-related asthma phenotypes. The majority 48 (69.6%) of the participants were triggered by weather. The most of the participants 55.1% had a history of respiratory tract infections, 49.3% had a history of atopy, 47.8% had hereditary history, 39.1% were in exposure to products of smoke, 31.9% were in exercise, 30.4% were in stress, and 29% were in the history of smoking;

Table 1: Summary of the demographic parameter in the study population (n=69)

Parameter	Summary n (%) or Mean±S.D
Age (in years) mean±S.D	33.88±10.01 (ranged 15–35)
Gender	
Male	35 (50.7%)
Female	34 (49.3%)
Level of education	
Illiterate	58 (84.1%)
Literate	11 (15.9%)
Socioeconomic status	
Middle	23 (33.3%)
Low	46 (66.7%)
BMI	
Normal	36 (52.2%)
Over weight	28 (40.6%)
Birth order	
Obese	5 (7.2%)
First order	40 (58%)
Second order	29 (42%)
Age of onset	
Early onset	54 (78.3%)
Late onset	15 (21.7%)
Allergen exposure	
Animals	12 (17.4%)
Environmental dust	7 (10.1%)
Food	15 (21.7%)
Nil	35 (50.7%)
Hereditary pattern	
Father	9 (13%)
Mother	24 (34.8%)
Not applicable	36 (52.2%)
Family size	
Large	30 (43.5%)
Small	39 (56.5%)

Table 2: Summary of clinical parameters in the study population (n=69)

Parameter	Summary
Absolute eosinophil count cells/μL (Mean±S.D)	470.29±203.11 (ranged 140–990)
Elevated eosinophilia	46 (66.7%)
Normal eosinophilia	23 (33.3%)
Sputum cytology	
Basophilic	1 (1.4%)
Eosinophilic	46 (66.7%)
Lymphocytic	1 (1.4%)
Mixed	6 (8.7%)
Neutrophilic	12 (17.4%)
Knowledge of diagnosis and symptom control	
Wedge diagnosis	49 (71%)
symptom control	60 (87%)

Table 4: The difference in the proportion of history of environment factor between obesity associate asthma was statistically not significant (P>0.005) whereas, it was significant between a few environmental factors and allergy and smoking-related asthma phenotype.

Table 5: The difference in the proportion of environmental factors between obesity associate asthma was statistically

not significant (P>0.05) whereas, it was significant between a few environmental factors and allergy and smoking-related asthma phenotype.

DISCUSSION

To date, the present study is one of its kind to characterize a clinically difficult patient group, and the findings of the present study may improve future asthma care in hospitals in India. Out of 69, the majority 26 (37.7%) were aged between 31 and 40 years; 35 (50.7%) were male, and the remaining 34 (49.3%) were females. The most of the 58 (84.1%) were illiterate, and 46 (66.7%) belonged to the low socioeconomic class. Sputum cytology report showed that the majority 66.7% had eosinophilia. The majority 48 (69.6%) of the participants were triggered by weather. The most of the participants 55.1% had a history of respiratory tract infections, 49.3% had a history of atopy, 47.8% had a hereditary history, 33 (47.83%) participants had obesity associate asthma, 34 (49.28%) had allergic asthma, and 20 (28.99%) had smoking-related asthma phenotypes. The difference in the proportion of a few environmental factors was significant for allergic and smoking-related asthma (P>0.005).

Table 3: Summary of asthma phenotypes and environment factors in the study population (n=69)

Parameter	Summary (%)
Asthma phenotypes	
Obesity associate asthma	33 (47.83)
Allergic asthma	34 (49.28)
Smoking related asthma	20 (28.99)
Environment factors	
History of smoking	20 (29)
Exposure to farm products	12 (17.4)
Exposure to products of smoke	27 (39.1)
History of atopy	34 (49.3)
Hereditary history	33 (47.8)
History of Aspirin	6 (8.7)
History of Respiratory tract infections.	38 (55.1)
History of Gastro esophageal reflux disease.	10 (14.5)
Weather	48 (69.6)
Stress	21 (30.4)
Exercise	22 (31.9)

About 51% of the study subjects were in the age group of 10–40 years, 11 (15.9%) were in between 10 and 21 years, 14 (20.3%) were 21–30 years, and 26 (37.7%) were aged between 31 and 40 years which denotes a higher prevalence of asthma in the first, second, and third decade of life which is in comparison to a study by Damodaran et al.,[15] which reported 65% of subjects in the age group of 21–40 years. The ratio of males and females was almost similar in the present study, which is in contrast to a study by Carra et al.,[16] where 75% of the subjects were females. In the present study, 36 (52.2%) had normal BMI, which is similar to a study by Renthlei et al.,[17] where the maximum number of patients was in the normal range of BMI, that is 18.5–22.9.

However, no biomarkers predict asthma exacerbations accurately to date, but an increased eosinophil count in sputum and blood is considered a high risk of asthma exacerbation and hospitalization and is easy to assess in clinical practice. A cutoff of ≥400/mm^3 was strongly associated with the future uncontrolled asthma based on the PREDUNA study[18] (a retrospective and cohort study that examined the relationship between blood eosinophil count at baseline and asthma exacerbations in the following 12 months). In the present study, the mean absolute blood eosinophil count was 470.29 cells/μL in the study

Table 4: Comparison of history of environmental factors with asthma phenotypes (n=69)

Parameter	H/o smoking (n=20)	H/o atopy (n=34)	H/o aspirin (n=6)	H/o RTI (n=38)	H/o GERD (n=10)
Obesity Associate Asthma	12 (60%)	15 (44.12%)	3 (50%)	16 (42.11%)	7 (70%)
P-value	0.062	0.543	1.000	0.292	0.177
Allergic asthma	4 (20%)	31 (91.18%)	0 (0%)	34 (89.47%)	0 (0%)
P-value	**0.006**	**<0.001**	*	*	*
Smoking related asthma	–	4 (11.76%)	2 (33.33%)	4 (10.53%)	0 (0%)
P-value		**0.002**	1.000	**<0.001**	*

*No statistical test was applied -due to o subjects in the cells

Table 5: Comparison of environmental factors with asthma phenotypes (n=69)

Parameter	ETFP (n=12)	ETPS (n=27)	Hereditary history (n=33)	Weather (n=48)	Stress (n=21)	Exercise (n=22)
Obesity Associate Asthma	3 (25%)	16 (59.26%)	14 (42.42%)	20 (41.67%)	12 (57.14%)	12 (54.55%)
P-value	0.082	0.307	0.390	0.121	0.305	0.445
Allergic asthma	11 (91.67%)	21 (77.78%)	25 (75.76%)	31 (64.58%)	0 (0%)	13 (59.09%)
P-value	**<0.001**	**<0.001**	**<0.001**	**<0.001**	*	0.265
Smoking related asthma	2 (16.67%)	3 (11.11%)	5 (15.15%)	9 (18.75%)	13 (61.9%)	8 (36.36%)
P-value	0.486	**0.029**	**0.015**	**0.005**	**<0.001**	0.355

*No statistical test was applied -due to o subjects in the cells

population, and the sputum cytology report showed that the majority 66.7% had eosinophilia. However, this finding was in contrast to a study by Pola-Bibian et al.,[19] where they found no association between blood eosinophil count and the presence of exacerbation of asthma.

The present study reported a history of allergy/atopy, presence of disease in family members, exposure to farm products in childhood, exposure to allergens, chemicals at workplace, history of sensitivity to aspirin/NSAIDs, exposure to environmental tobacco smoke, food habits, family size, history of recurrent respiratory tract infections, history of gastroesophageal reflux disorder, changes in climatic conditions, history of stress and emotional conditions, knowledge about the disease, diagnosis, and mode of environmental treatment factors. Custovic et al.,[20] reported in detail about all these exacerbations/triggering factors for asthma at reported European Academy of Allergy and Clinical Immunology in 2013 summit.

In the present study, though we followed GINA guidelines, we did not consider the characteristics associated with asthma that can differentiate asthma from chronic obstructive pulmonary disease (COPD) and asthma COPD overlap syndrome (ACOS). This is in contrast to a study reported by Daniel et al.,[21] where the magnitude of ACOS among patients diagnosed with asthma and COPD was studied.

The authors identified three types of asthma phenotypes based on clinical symptoms. Out of 69 participants, 33 (47.83%) participants had obesity associate asthma, 34 (49.28%) had allergic asthma, and 20 (28.99%) had smoking-related asthma phenotypes. Our study has shown a significantly higher prevalence of allergy associated with asthma. This reflects the substantial interaction between the lower and upper airways and, in particular, between the underlying etiological mechanisms of airways pathology. A similar interaction is also found in a study by Philpott et al.,[22] where the prevalence of asthma-related allergy was high. Obesity and asthma were reported by Peters et al.,[23] where he reported that obese asthma syndrome is complex and multifactorial. Potential underlying mechanisms include a shared genetic component, dietary and nutritional factors, alterations in the gut microbiome, systemic inflammation, metabolic abnormalities, and changes in lung anatomy and function. Kumar et al., reported the high odds of smoking-related asthma[24] and suggested that the burden of asthma can be reduced overall by providing education, cessation in smoking, and schemes like Pradhan Mantri Ujjwala Yojana, by providing clean fuel (LPG) to poor and vulnerable households.

There exists a considerable spatial variation between environmental and ecological factors and asthma. These findings signify the need to prioritize various public health interventions for the health and well-being of the population in India. The prevalence of asthma and bronchitis in India and minimizing the risk factors for lung disorders can be minimized by following some of the ongoing interventions in India like the Swatch Bharat Mission that enhances awareness and practices to improve hygiene and sanitation and MPOWER (Monitoring tobacco consumption and the effectiveness of preventive measures, protect people from tobacco smoke, offer help to quit tobacco, warn about the dangers of tobacco, enforce ban on tobacco, advertising, promotion, and sponsorship, and raise taxes on tobacco) strategies to strengthen the tobacco control program.[25]

Limitations of the study

The present study, however, contains some limitations. The study results may be affected by variables like tobacco use and weather, leading to confounding bias and lead information bias given by participants on history. The present study is a cross-sectional study with a small sample size from a single-center and a tertiary hospital. This may not truly represent the phenotypes in the community or other health-care settings. The overlap between asthma and COPD/chronic bronchitis was overlooked. Hence, we recommend further multicenter and prospective studies with large sample size and new diagnostic tools to identify asthma phenotypes.

CONCLUSION

This cross-sectional analysis concluded that asthma is a disease of all general population irrespective of age, socioeconomic factors, and other environmental factors. The sample in our study was representative of real-life practice in our geographic area and specified that a major number of asthmatic patients might get unnoticed, receive low-grade care, or even go untreated. Phenotyping of asthma helps as a mechanism in developing a practice of personalized treatment for asthma that would reduce the likelihood of prescribing wrong drugs to the wrong patients. It will also decrease the number of heterogeneous inflammatory diseases in a community.

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VV- Concept and design of the study and prepared first draft of manuscript; **SBM and JV-** Interpreted the results, reviewed the literature, and manuscript preparation; **RR and NT-** Concept, coordination, statistical analysis, and interpretation; and **SBM, JV, RR, VV, and NT-** Preparation of manuscript and revision of the manuscript.

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