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COMPARATIVE STUDY ON PREVALENCE, CLINICAL PRESENTATION AND PULMONARY FUNCTION TEST IN BRONCHIAL ASTHMA IN OBESE AND NONOBESE PATIENT.

Dr. B Mohan[1]*, Dr.Anusha Gorapalli[2], Dr.K. Surendar Reddy[3].

[1]Assistant Professor, Department of Respiratory Medicine, Surabhi Institute of Medical Sciences, Mittapally(v), Siddipet (M)&(Dt), Telangana, INDIA.

[2]Assistant Professor, Department of Respiratory Medicine, SVIMS - SPMC -W, Alipiri Road, Tirupati, Andhra Pradesh, INDIA.

[3]Professor and HOD, Department of Respiratory Medicine, ESIC Medical College & Hospital, Sanath Nagar, Hyderabad, Telangana, INDIA.

Correspondence:
Dr. B Mohan.
Assistant Professor, Department of Respiratory Medicine, Surabhi Institute of Medical Sciences, Mittapally(v), Siddipet (M)&(Dt), Telangana, INDIA.

Email:
mohanmbbs.007@gmail.com

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Abstract

Background: Bronchial asthma is a chronic inflammatory airway disease with increasing global prevalence. Obesity has emerged as an important risk factor influencing asthma severity, clinical presentation, and pulmonary function. ***Aim:*** To compare the prevalence, clinical presentation, and pulmonary function test parameters in bronchial asthma among obese and non-obese patients. ***Methods:*** This cross-sectional comparative study was conducted in a tertiary care hospital over a period of one year. A total of 41 patients diagnosed with bronchial asthma were included and categorized into obese (n=6) and non-obese (n=35) groups based on body mass index. Clinical features were recorded, and pulmonary function tests were performed using spirometry before and after bronchodilator administration. Statistical analysis was carried out using Student's t-test and Chi-square test, with $p < 0.05$ considered significant. ***Results:*** Obese patients showed a higher mean age and longer duration of asthma, though not statistically significant. Clinical symptoms such as cough, expectoration, and shortness of breath were more frequent in obese patients. Pulmonary function tests revealed significantly lower FEV1, FVC, and FEF 25–75% values in obese patients compared to non-obese patients ($p < 0.05$). A higher proportion of obese patients had uncontrolled and moderate-to-severe asthma, though without statistical significance. ***Conclusion:*** Obesity is associated with increased symptom burden, impaired pulmonary function, and poorer asthma control. Weight management should be considered an integral component in the management of bronchial asthma.

Keywords: Bronchial asthma; Obesity; Pulmonary function test.

INTRODUCTION

Bronchial asthma is a chronic, heterogeneous inflammatory disorder of the airways characterized by variable airflow obstruction and bronchial hyperresponsiveness. It is clinically manifested by recurrent episodes of wheeze, shortness of breath, chest tightness, and cough, which vary in intensity and frequency over time. The disease burden of asthma is significant, affecting approximately 300 million individuals worldwide, with projections suggesting a further increase in prevalence in the coming decades. The variability in clinical presentation and severity is influenced by multiple factors including genetic predisposition, environmental exposures, and comorbid conditions.[1]

Obesity has emerged as a major global health concern, with its prevalence rising dramatically due to sedentary lifestyles, dietary changes, and urbanization. According to the World Health Organization (WHO), obesity is defined as a body mass index (BMI) ≥ 30 kg/m^2. In India, the prevalence of obesity has been steadily increasing, contributing to the rising incidence of non-communicable diseases. Increasing evidence suggests a strong association between obesity and bronchial asthma, with obesity not only increasing the risk of asthma but also influencing its severity, control, and response to treatment.[2] The relationship between asthma and obesity is complex and multifactorial. Mechanical factors such as reduced chest wall compliance, decreased lung volumes (especially expiratory reserve volume and functional residual capacity), and increased airway resistance play a crucial role. Additionally, obesity is associated with systemic inflammation mediated by adipokines like leptin and cytokines, which may contribute to airway inflammation and hyperresponsiveness. Obese asthmatics often present with more severe symptoms, reduced quality of life, and poorer disease control compared to non-obese individuals.[3]

Pulmonary function tests (PFTs), particularly spirometry, are essential tools in the diagnosis and assessment of asthma. Parameters such as Forced Expiratory Volume in one second (FEV1), Forced Vital Capacity (FVC), and FEV1/FVC ratio help in evaluating airflow limitation and its reversibility. Studies have shown that obese individuals may exhibit altered pulmonary function due to both mechanical restriction and airway changes, which may complicate the interpretation of spirometric findings.[4]

Clinical presentation of asthma may also differ between obese and non-obese patients. Obese patients often report increased dyspnea and exercise intolerance, which may not always correlate with the degree of airway obstruction. Furthermore, comorbidities such as gastroesophageal reflux disease (GERD), obstructive sleep apnea (OSA), and metabolic disorders are more prevalent in obese individuals and may further exacerbate asthma symptoms.[5]

AIM

To compare the prevalence, clinical presentation, and pulmonary function test parameters in bronchial asthma among obese and non-obese patients.

OBJECTIVES

1. To compare the clinical presentation of bronchial asthma between obese and non-obese patients.
2. To evaluate pulmonary function test parameters in obese and non-obese asthmatic patients.
3. To assess the prevalence and severity of asthma in relation to body mass index.

MATERIALS AND METHODS

Source of Data

The data were collected from patients attending the outpatient and inpatient departments of Respiratory Medicine in a tertiary care hospital. Patients diagnosed with bronchial asthma based on clinical features and spirometry were included in the study.

Study Design

The study was a cross-sectional comparative analytical study conducted to evaluate differences between obese and non-obese asthmatic patients.

Study Location

The study was conducted in the Department of Respiratory Medicine of a tertiary care teaching hospital.

Study Duration

The study was carried out over a period of 12 months.

Sample Size

A total of 41 patients were included in the study, comprising:

- Obese group: 6 patients
- Non-obese group: 35 patients

Inclusion Criteria

1. Patients aged between 10–80 years.
2. Patients diagnosed with bronchial asthma based on clinical symptoms and spirometry (reversibility ≥12% and 200 ml).
3. Patients willing to participate in the study.
4. Patients with normal chest X-ray findings.

Exclusion Criteria

1. Patients aged <10 years or >80 years.
2. Smokers or ex-smokers.
3. Patients with other chronic respiratory diseases.
4. Patients with abnormal chest X-ray findings.
5. Patients with history of pulmonary tuberculosis.
6. Patients unwilling to participate.

Procedure and Methodology

Patients fulfilling the inclusion criteria were recruited into the study. A detailed history was taken, including symptoms such as cough, wheeze, breathlessness, and chest tightness, along with past medical and family history of allergy or asthma. Clinical examination was performed thoroughly.

Body mass index (BMI) was calculated using the formula:

BMI = Weight (kg) / Height (m^2).

Patients were categorized into obese and non-obese groups based on WHO classification. Pulmonary function tests were performed using spirometry. Baseline spirometry was conducted, followed by administration of bronchodilator (salbutamol 200–400 mcg). Post-bronchodilator spirometry was repeated after 10–15 minutes to assess reversibility.

Sample Processing

Spirometry readings were recorded using standardized equipment. At least three acceptable and reproducible flow-volume curves were obtained for each patient. The best values of FEV1, FVC, and FEV1/FVC ratio were selected for analysis.

Statistical Methods

Data were entered into Microsoft Excel and analyzed using statistical software (SPSS).

- Categorical variables were expressed as frequency and percentage.
- Continuous variables were expressed as mean ± standard deviation.
- Chi-square test was used to assess association between categorical variables.
- Student's t-test was used to compare means between two groups.
- A p-value <0.05 was considered statistically significant.

Data Collection

Data were collected using a structured proforma including demographic details, clinical presentation, BMI, comorbidities, and pulmonary function test parameters. All data were recorded systematically and verified for accuracy before analysis.

RESULTS

TABLE 1: Baseline Characteristics and Prevalence Comparison

Variable	Obese (n=6) n(%) / Mean±SD	Non-Obese (n=35) n(%) / Mean±SD	Test of Significance	95% CI	P value
Age (years)	48.5 ± 10.2	42.3 ± 12.4	t = 1.21	-3.8 to 16.2	0.23
Female	4 (66.7%)	22 (62.9%)	$\chi^2 = 0.03$	OR: 1.18 (0.18–7.5)	0.86
Male	2 (33.3%)	13 (37.1%)			
Duration of asthma (years)	8.2 ± 3.5	5.6 ± 2.8	t = 1.78	-0.4 to 5.6	0.08
Family history of asthma	2 (33.3%)	11 (31.4%)	$\chi^2 = 0.01$	OR: 1.09 (0.16–7.3)	0.92
Prevalence proportion	6 (14.6%)	35 (85.4%)			

Table 1 shows the baseline characteristics of the study population comparing obese and non-obese asthmatic patients. The mean age was higher in the obese group (48.5 ± 10.2 years) compared to the non-obese group (42.3 ± 12.4 years), although this difference was not statistically significant (t = 1.21, p = 0.23). Female predominance was observed in both groups, with 66.7% females in the obese group and 62.9% in the non-obese group, with no significant association (p = 0.86). The mean duration of asthma was longer in obese patients (8.2 ± 3.5 years) compared to non-obese patients (5.6 ± 2.8 years), but this difference did not reach statistical significance (p = 0.08). Family history of asthma was comparable between both groups (33.3% vs 31.4%, p = 0.92).

TABLE 2: Clinical Presentation Comparison

Clinical Feature	Obese (n=6) n(%)	Non-Obese (n=35) n(%)	Test of Significance	95% CI	P value
Cough	5 (83.3%)	27 (77.1%)	$\chi^2 = 0.11$	OR: 1.45 (0.14–14.8)	0.74
Expectoration	4 (66.7%)	18 (51.4%)	$\chi^2 = 0.52$	OR: 1.89 (0.32–11.0)	0.47
Shortness of breath	6 (100%)	30 (85.7%)	$\chi^2 = 1.03$		0.31
Wheeze	5 (83.3%)	29 (82.9%)	$\chi^2 = 0.00$	OR: 1.02 (0.10–10.4)	0.98
Chest tightness	2 (33.3%)	21 (60.0%)	$\chi^2 = 1.41$	OR: 0.33 (0.05–2.0)	0.23
Allergic history	2 (33.3%)	17 (48.6%)	$\chi^2 = 0.52$	OR: 0.53 (0.09–3.1)	0.47

Table 2 compares the clinical features of bronchial asthma between obese and non-obese patients. Cough was slightly more common in obese patients (83.3%) compared to non-obese patients (77.1%), though the difference was not statistically significant (p = 0.74). Expectoration was also higher in obese patients (66.7% vs 51.4%), but without statistical significance (p = 0.47). All obese patients (100%) presented with shortness of breath compared to 85.7% in the non-obese group (p = 0.31). The prevalence of wheeze was nearly identical in both groups (83.3% vs 82.9%, p = 0.98). Chest tightness was more commonly reported in non-obese patients (60.0%) compared to obese patients (33.3%), though this difference was not statistically significant (p = 0.23). Similarly, allergic history was more frequent in the non-obese group (48.6%) than in the obese group (33.3%), but without statistical significance (p = 0.47).

TABLE 3: Pulmonary Function Test Comparison

Parameter	Obese (n=6) Mean±SD	Non-Obese (n=35) Mean±SD	Test of Significance	95% CI	P value
FEV1 (% predicted)	61.2 ± 8.5	72.8 ± 10.2	t = 2.63	-20.3 to -2.9	0.012*
FVC (% predicted)	68.4 ± 7.9	78.5 ± 9.4	t = 2.41	-18.4 to -1.8	0.020*
FEV1/FVC (%)	69.8 ± 5.6	74.2 ± 6.3	t = 1.62	-9.9 to 1.1	0.11
Post-BD FEV1 improvement (%)	14.2 ± 3.1	16.8 ± 4.2	t = 1.54	-6.2 to 0.9	0.13
FEF 25–75 (%)	48.6 ± 9.2	58.3 ± 10.8	t = 2.02	-19.4 to -0.2	0.049*

(*Significant)

Table 3 demonstrates the comparison of pulmonary function test parameters between obese and non-obese asthmatic patients. The mean FEV1 (% predicted) was significantly lower in obese patients (61.2 ± 8.5) compared to non-obese patients (72.8 ± 10.2), indicating worse airflow limitation in the obese group (p = 0.012). Similarly, FVC (% predicted) was also significantly reduced in obese patients (68.4 ± 7.9) compared to non-obese patients (78.5 ± 9.4) (p = 0.020). The FEV1/FVC ratio was lower in obese patients (69.8 ± 5.6) compared to non-obese patients (74.2 ± 6.3), but this difference was not statistically significant (p = 0.11). Post-bronchodilator improvement in FEV1 was slightly lower in obese patients (14.2 ± 3.1%) compared to non-obese patients (16.8 ± 4.2%), though not statistically significant (p = 0.13). However, mid-expiratory flow rate (FEF 25–75%) was significantly reduced in obese patients (48.6 ± 9.2) compared to non-obese patients (58.3 ± 10.8) (p = 0.049), suggesting greater small airway involvement.

TABLE 4: Asthma Severity vs BMI

Severity	Obese (n=6) n(%)	Non-Obese (n=35) n(%)	Test of Significance	95% CI	P value
Controlled	1 (16.7%)	8 (22.9%)	$\chi^2 = 0.12$	OR: 0.68 (0.07–6.3)	0.73
Partially controlled	1 (16.7%)	12 (34.3%)	$\chi^2 = 0.68$	OR: 0.38 (0.04–3.4)	0.41
Uncontrolled	4 (66.7%)	15 (42.9%)	$\chi^2 = 1.18$	OR: 2.65 (0.46–15.3)	0.27
Moderate–Severe asthma	4 (66.7%)	13 (37.1%)	$\chi^2 = 1.63$	OR: 3.37 (0.56–20.1)	0.20

Table 4 illustrates the association between asthma severity and body mass index. Controlled asthma was observed in a slightly lower proportion of obese patients (16.7%) compared to non-obese patients (22.9%), though this difference was not statistically significant (p = 0.73). Partially controlled asthma was less frequent in obese patients (16.7%) compared to non-obese patients (34.3%) (p = 0.41). A higher proportion of obese patients had uncontrolled asthma (66.7%) compared to non-obese patients (42.9%), although this difference did not reach statistical significance (p = 0.27). Similarly, moderate-to-severe asthma was more prevalent among obese patients (66.7%) compared to non-obese patients (37.1%), but without statistical significance (p = 0.20).

DISCUSSION

In the present study, obese asthmatic patients constituted 14.6% of the study population, while non-obese patients constituted 85.4%. The mean age was higher among obese patients (48.5 ± 10.2 years) compared to non-obese patients (42.3 ± 12.4 years), although the difference was not statistically significant (p = 0.23). Female predominance was observed in both groups, with 66.7% females in obese and 62.9% in non-obese patients. Similar female predominance and higher asthma burden among obese individuals were reported by Schiffers et al.(2023)[8], who demonstrated increased asthma prevalence in females and highlighted obesity as a contributing factor to asthma phenotypes. Likewise, Kim et al.(2022)[9] reported a stronger association between obesity and lung function impairment in adult asthmatic women, emphasizing gender-related differences. The duration of asthma was longer in obese patients (8.2 ± 3.5 years) compared to non-obese patients (5.6 ± 2.8 years), suggesting that obesity may be associated with more chronic asthma morbidity, although the

difference was not statistically significant. This finding was comparable with Mamilla et al.(2024)[1] and Singh et al.(2023)[3], who observed longer disease duration and increased severity among obese asthmatics.

Regarding clinical presentation, cough, expectoration, and shortness of breath were more frequent among obese patients. Cough was present in 83.3% obese and 77.1% non-obese patients, while expectoration was seen in 66.7% obese and 51.4% non-obese patients. Shortness of breath was present in all obese patients (100%) compared to 85.7% non-obese patients. These findings suggest that obese asthmatics had greater symptom burden, although the differences were statistically not significant. Similar findings were reported by Arslan et al.(2025)[5], who observed that obese asthmatics experienced increased symptom severity, poorer quality of life, and reduced asthma control. Reyes Noriega et al.(2023)[6] also explained that dyspnea in obese individuals may be related to mechanical restriction of the chest wall, systemic inflammation, and altered respiratory physiology. In contrast, wheeze was almost similar in both groups (83.3% vs 82.9%), while chest tightness and allergic history were more common among non-obese patients. This supports the concept that obese asthma may represent a distinct phenotype, less driven by atopy and more influenced by metabolic and inflammatory mechanisms.

Pulmonary function test parameters showed significant impairment among obese asthmatic patients. Mean FEV1 was significantly lower in obese patients (61.2 ± 8.5%) compared to non-obese patients (72.8 ± 10.2%) ($p = 0.012$). Similarly, FVC was significantly reduced in obese patients (68.4 ± 7.9%) compared to non-obese patients (78.5 ± 9.4%) ($p = 0.020$). FEF 25–75% was also significantly lower in obese patients (48.6 ± 9.2%) compared to non-obese patients (58.3 ± 10.8%) ($p = 0.049$), indicating small airway involvement. These findings are consistent with Alqarni et al.(2023)[2], who reported significant reductions in spirometry parameters among overweight and obese asthmatic patients. Arismendi et al.(2020)[4] also demonstrated that obesity affects lung function through airway dysanapsis and reduced lung volumes. Furthermore, Tashiro et al.(2022)[7] found that obesity significantly impairs pulmonary function in asthmatic patients, while having minimal effect in non-asthmatics. Additionally, Al Khathlan et al.(2023)[10] highlighted the role of inflammatory cytokines in obese individuals contributing to reduced lung function and airway inflammation.

In the present study, asthma control was poorer among obese patients. Uncontrolled asthma was observed in 66.7% obese patients compared to 42.9% non-obese patients, while moderate-to-severe asthma was also more frequent in obese patients (66.7% vs 37.1%). Although these differences were statistically not significant, probably due to the small sample size, the clinical trend suggests a strong association between obesity and poor asthma control. Similar findings were reported by Arslan et al.(2025)[5], who found that obesity is associated with poor asthma control and increased disease severity. Kim et al.(2022)[9] also reported that increasing BMI negatively influences asthma control and lung function outcomes..

CONCLUSION

The present study aimed to compare the prevalence, clinical presentation, and pulmonary function test parameters in bronchial asthma among obese and non-obese patients. The findings of this study demonstrate that obesity has a significant influence on the clinical profile and pulmonary function of patients with bronchial asthma.

The prevalence of obese asthmatic patients in this study was lower compared to non-obese patients; however, obese individuals exhibited a trend toward more severe disease characteristics. Although baseline characteristics such as age, gender distribution, and family history of asthma were comparable between the two groups, obese patients showed a relatively longer duration of illness, suggesting a chronic and possibly progressive disease course.

In terms of clinical presentation, symptoms such as cough, expectoration, and shortness of breath were more pronounced in obese patients, indicating a higher symptom burden. However, these differences were not statistically significant. Chest tightness and allergic history were more common in non-obese patients, supporting the concept that obesity-related asthma may represent a distinct phenotype that is less driven by atopy and more influenced by mechanical and inflammatory factors.

Pulmonary function test analysis revealed that obese asthmatic patients had significantly lower FEV1, FVC, and FEF 25–75% values compared to non-obese patients, indicating impaired lung function and possible small airway involvement. These findings highlight the impact of obesity on respiratory mechanics, including reduced lung volumes and decreased chest wall compliance.

Furthermore, obese patients showed a higher proportion of uncontrolled and moderate-to-severe asthma compared to non-obese patients, although the association did not reach statistical significance. This suggests that obesity may contribute to poorer asthma control and increased disease severity.

Overall, the study concludes that obesity adversely affects bronchial asthma by increasing symptom severity, impairing pulmonary function, and contributing to poorer disease control. These findings emphasize the importance of addressing obesity as a modifiable risk factor in the comprehensive management of asthma.

LIMITATIONS OF THE STUDY

1. The sample size was small, particularly in the obese group, which may limit the statistical power of the study.
2. The study was conducted at a single center, limiting generalizability to the wider population.
3. Cross-sectional design prevents establishing causal relationships between obesity and asthma severity.
4. Potential selection bias due to hospital-based sampling.
5. Confounding factors such as diet, physical activity, and socioeconomic status were not assessed.
6. Comorbidities like GERD and obstructive sleep apnea were not evaluated in detail.
7. Long-term follow-up and disease progression were not studied.
8. Inflammatory biomarkers and adipokines were not measured.
9. Spirometry interpretation may be influenced by patient effort variability.
10. Severity classification relied on clinical parameters without advanced phenotyping.

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