



Association of Body Mass Index with Severity, Inflammatory Markers, and Clinical Outcomes in Bronchial Asthma: A Hospital-Based Observational Study.

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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, control, and outcomes. However, limited data exist from Indian populations evaluating the combined impact of body mass index (BMI), inflammatory markers, and clinical severity of asthma. Aim of the study was to evaluate the association between BMI and the severity of bronchial asthma, and to assess the relationship of obesity with inflammatory markers (C-reactive protein) and morbidity among asthma patients.

Material and Methods: This hospital-based observational study was conducted at a tertiary care center in Telangana, India, over 18 months. A total of 100 patients aged 20–40 years with clinically and spirometrically confirmed bronchial asthma were included. Patients were categorized based on BMI (normal, overweight, obese) and asthma severity according to GINA guidelines (mild intermittent to severe persistent). Clinical evaluation, spirometry (FEV₁/FVC), oxygen saturation (SpO₂), and laboratory investigations including C-reactive protein (CRP) were performed. **Results:** The mean age of participants was 30.3 ± 6.39 years, with a nearly equal gender distribution. Obesity was observed in 51% of patients. A highly significant association was found between BMI and asthma severity (p < 0.001), with all moderate and severe cases occurring in obese individuals. Mean BMI increased progressively with severity. Physiological parameters showed worsening trends with increasing severity, including reduced SpO₂ and declining FEV₁/FVC ratios (p < 0.001). Elevated CRP levels were observed exclusively in obese patients and were significantly associated with higher BMI (p < 0.001). Morbidity indicators, including life-threatening events and missed workdays, were predominantly seen in obese patients with severe asthma. **Conclusion:** Increasing BMI is strongly associated with greater severity of bronchial asthma, impaired lung function, reduced oxygenation, and elevated systemic inflammation. Obesity also contributes to increased morbidity in asthma patients. These findings highlight the importance of weight management as an integral component of asthma care to improve clinical outcomes.

Keywords: Bronchial asthma; Body mass index; Obesity; Asthma severity; C-reactive protein; Spirometry; SpO₂; Inflammation; Morbidity; GINA guidelines.



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INTRODUCTION

Bronchial asthma is a chronic inflammatory airway disorder characterized by variable airflow obstruction, bronchial hyper-responsiveness, and recurrent respiratory symptoms, contributing substantially to global morbidity and healthcare burden, with over 260 million individuals affected worldwide and a rising prevalence in India due to urbanization and environmental factors (1,2). Parallel to this, obesity has emerged as a global epidemic, defined by increased body mass index (BMI), and is particularly concerning in Asian populations where metabolic risks occur at lower BMI thresholds (3,4). Increasing evidence over the past two decades suggests a strong association between elevated BMI and both the incidence and severity of bronchial asthma (5,6). Obese asthmatics are more likely to experience frequent exacerbations, reduced responsiveness to inhaled corticosteroids, increased hospitalization rates, and poorer quality of life compared to non-obese individuals (7,8). The mechanistic link between obesity and asthma is multifactorial, involving both mechanical and inflammatory pathways. Reduced lung volumes, decreased functional residual capacity, and impaired chest wall compliance contribute to airway narrowing, while adipose tissue-mediated systemic inflammation—through cytokines

such as TNF- α and IL-6 and altered adipokine balance—further exacerbates airway inflammation (9,10). This has led to the recognition of an obesity-associated asthma phenotype, often characterized by late onset, increased severity, and altered inflammatory profiles (11).

Despite the established association, variability exists in the strength and nature of this relationship across different populations. Several international studies, including large cohort and cross-sectional analyses, have consistently demonstrated a positive correlation between BMI and asthma severity, as well as poorer asthma control and lung function parameters (5–8). However, regional differences influenced by genetic predisposition, environmental exposures, dietary habits, and healthcare access may significantly modify this relationship. In the Indian context, available studies remain limited and often lack standardized assessment using global guidelines such as those proposed by the Global Initiative for Asthma (GINA), particularly in evaluating severity classification alongside objective measures like spirometry and biomarkers (12,13). Furthermore, while some studies have explored the role of systemic inflammation in obese asthmatics, there is insufficient data correlating BMI with inflammatory markers such as C-reactive protein (CRP) and their impact on disease morbidity in Indian populations.

This highlights a critical research gap: the lack of comprehensive, region-specific studies that simultaneously evaluate BMI, clinical severity of asthma, inflammatory markers, and morbidity using standardized methodologies. Addressing this gap is essential for improving risk stratification and guiding integrated management strategies, especially considering that weight reduction has been shown to improve asthma control and lung function outcomes (14). Therefore, the present study aims to evaluate the clinical correlation between BMI and the severity of bronchial asthma, while also examining the association of obesity with inflammatory markers (C-reactive protein) and morbidity among patients attending a tertiary care hospital. This integrated approach is expected to provide clinically relevant insights for optimizing asthma management in the context of the growing obesity burden.

MATERIALS AND METHODS

Study Design and Setting

This was a hospital-based, single-centre observational study conducted to evaluate the association between body mass index (BMI) and the severity of bronchial asthma. The study was carried out at Mamata Medical College and General Hospital, Khammam, Telangana, India, over a period of 18 months from February 2024 to January 2026 after obtaining Institutional Ethics Committee approval.

Study Population and Sample Size

The study population included patients diagnosed with bronchial asthma attending the outpatient and inpatient departments of General Medicine. A total of 100 subjects who fulfilled the eligibility criteria were enrolled in the study after obtaining informed consent.

Inclusion Criteria

- Patients diagnosed with bronchial asthma
- Age between 20–40 years
- Patients fulfilling spirometric criteria (post-bronchodilator increase in FEV₁ $\geq$ 12% and $\geq$ 200 mL from baseline)

Exclusion Criteria

- Patients with chronic obstructive pulmonary disease (COPD)
- Smokers
- Age <20 years or >40 years
- Pregnant women
- Patients with comorbid conditions such as ischemic heart disease, congestive cardiac failure, or valvular heart disease
- Known cases of obstructive sleep apnea

Study Tool

- Structured clinical history and examination proforma
- Spirometry for pulmonary function assessment
- BMI calculation based on Indian classification
- Laboratory investigations including:
 - o Complete blood count (CBC)
 - o Renal function test (RFT)
 - o Liver function test (LFT)
 - o C-reactive protein (CRP)

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- Imaging and supportive investigations:
 - o Chest X-ray
 - o Electrocardiogram (ECG)

Data Collection

- Eligible patients were enrolled after obtaining written informed consent
- Detailed clinical history and examination findings were recorded
- Pulmonary function tests were performed to confirm diagnosis and assess severity
- Asthma severity was classified according to GINA guidelines into:
 - o Mild intermittent
 - o Mild persistent
 - o Moderate persistent
 - o Severe persistent
- BMI was calculated and categorized (Indian standards):
 - o Normal (18.5–22.9 kg/m²)
 - o Overweight (23–24.9 kg/m²)
 - o Obese (≥25 kg/m²)
- Laboratory parameters including CRP were measured to assess inflammatory status
- Correlation between BMI categories and asthma severity was analyzed

Statistical Analysis

Data were entered into Microsoft Excel and analyzed using SPSS version 26.0. Quantitative variables were expressed as mean ± standard deviation, while qualitative variables were presented as frequencies and percentages. Appropriate statistical tests were applied to assess associations between BMI and asthma severity. A p-value <0.05 was considered statistically significant.

RESULTS

Table 1: Baseline Demographic Characteristics of Study Population

Variable	Category	n (%)
Age (years)	20–25	33 (33%)
	26–30	17 (17%)
	31–35	24 (24%)
	36–40	26 (26%)
Gender	Male	51 (51%)
	Female	49 (49%)
Mean Age (years)	—	30.3 ± 6.39

The present study included 100 patients with bronchial asthma, predominantly belonging to the younger adult age group. The majority of participants were aged 20–25 years (33%), followed by 36–40 years (26%), 31–35 years (24%), and 26–30 years (17%). The overall mean age of the study population was 30.3 ± 6.39 years, indicating that most subjects were in the third decade of life. Gender distribution was nearly equal, with males accounting for 51% and females for 49% of the study population.

Table 2: BMI Distribution and Anthropometric Characteristics

Variable	Category	n (%)
BMI (kg/m ²)	Normal (18.5–22.9)	30 (30%)
	Overweight (23–24.9)	19 (19%)
	Obese (≥25)	51 (51%)
Mean BMI (kg/m ²)	—	26.3 ± 4.42

In the present study, a significant proportion of patients were found to have elevated body mass index. More than half of the study population (51%) were classified as obese (BMI ≥25 kg/m²), while 19% were overweight (BMI 23–24.9 kg/m²), and only 30% had a normal BMI (18.5–22.9 kg/m²). The mean BMI of the study population was 26.3 ± 4.42 kg/m², which falls within the obese range according to the applied classification. These findings indicate a high prevalence of overweight and obesity among patients with bronchial asthma, suggesting a potential role of increased body weight in the disease profile.

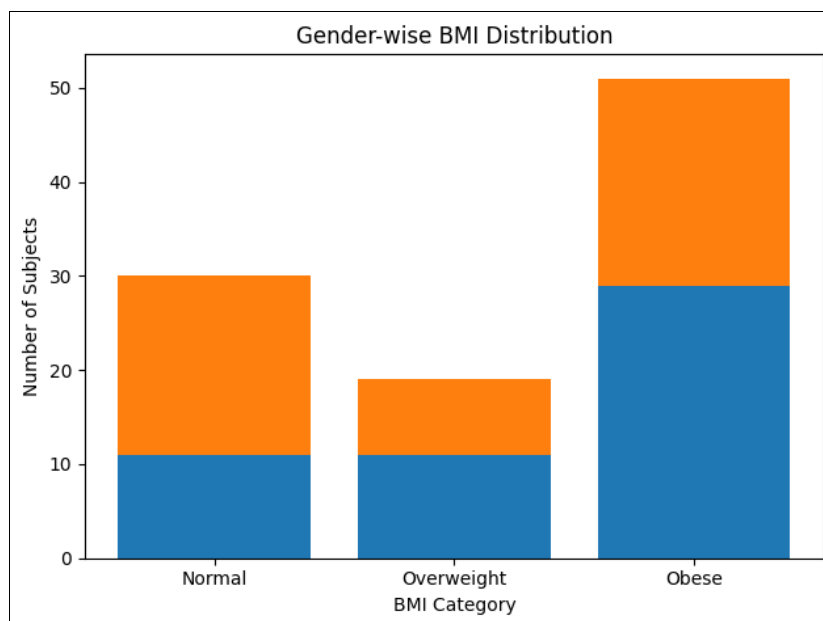


Figure 1: Gender-wise Distribution of BMI

The gender-wise distribution of body mass index (BMI) in the present study demonstrates a notable variation across BMI categories. Among individuals with normal BMI, females constituted a higher proportion (63.3%) compared to males (36.7%). In contrast, overweight and obese categories showed a male predominance, with males accounting for 57.9% and 56.9%, respectively, while females comprised 42.1% and 43.1% in these groups. Overall, these findings indicate that higher BMI categories were more prevalent among males, whereas females were more commonly observed within the normal BMI range, suggesting a gender-based difference in anthropometric profiles among patients with bronchial asthma.

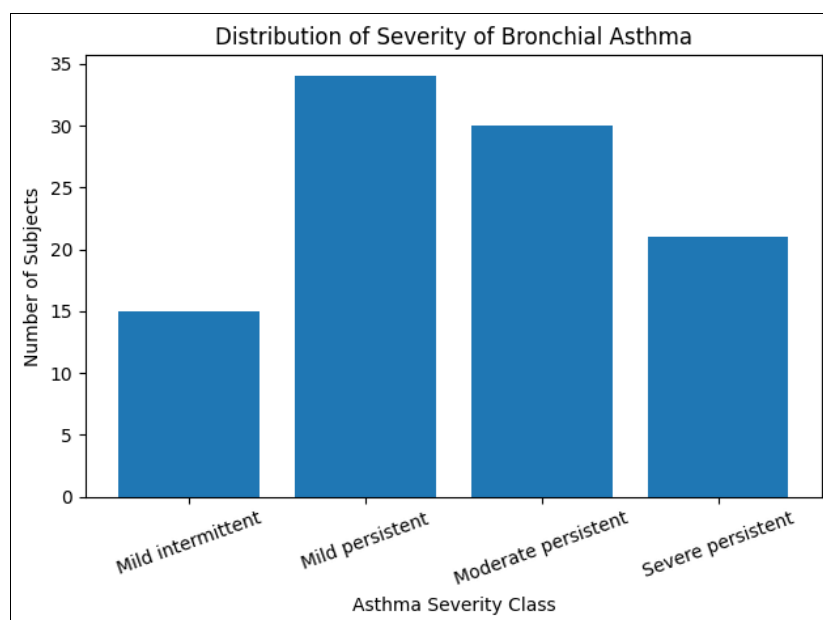


Figure 2: Distribution of Severity of Bronchial Asthma

The distribution of bronchial asthma severity in the present study reveals that the majority of patients were classified as having mild persistent asthma (34%), followed by moderate persistent asthma (30%). Severe persistent asthma accounted for 21% of cases, while mild intermittent asthma constituted the smallest proportion at 15%. Notably, more than half of the study population (51%) belonged to the moderate to severe categories (Class III and IV), indicating a substantial burden of advanced disease. These findings suggest that a significant proportion of patients present with persistent forms of asthma, emphasizing the need for early identification and appropriate long-term management strategies.

Table 3: Association Between BMI and Severity of Bronchial Asthma

BMI Category	Class I	Class II	Class III	Class IV	Total
Normal	15 (100%)	15 (44.1%)	0	0	30
Overweight	0	19 (55.9%)	0	0	19
Obese	0	0	30 (100%)	21 (100%)	51
Total	15	34	30	21	100
Mean BMI (kg/m²)	20.9 ± 0.93	23.3 ± 0.96	27.5 ± 1.30	33.1 ± 2.13	p-value: <0.001

The present study demonstrates a strong and statistically significant association between body mass index (BMI) and the severity of bronchial asthma ($p < 0.001$). All patients with mild intermittent asthma (Class I) were found to have normal BMI, indicating that milder forms of asthma were predominantly seen in individuals with normal body weight. Among patients with mild persistent asthma (Class II), 44.1% had normal BMI while 55.9% were overweight, with no obese individuals in this category. Notably, all patients with moderate (Class III) and severe persistent asthma (Class IV) belonged exclusively to the obese category, accounting for 100% of cases in these severity groups.

Furthermore, a progressive increase in mean BMI was observed with increasing asthma severity, rising from 20.9 ± 0.93 kg/m² in Class I to 33.1 ± 2.13 kg/m² in Class IV. This clear gradient suggests a dose-response relationship between BMI and asthma severity. These findings strongly indicate that higher BMI is associated with more severe forms of bronchial asthma, highlighting obesity as a significant risk factor influencing disease progression and clinical severity.

Table 4: Physiological Parameters According to Severity of Bronchial Asthma (SpO₂)

SpO ₂ (%)	Class I	Class II	Class III	Class IV
<90%	0	12 (35.3%)	23 (76.7%)	21 (100%)
90–95%	6 (40%)	17 (50%)	7 (23.3%)	0
96–98%	9 (60%)	5 (14.7%)	0	0
Mean SpO₂ (%)	96.8 ± 1.2	93.9 ± 2.1	89.8 ± 2.3	87.1 ± 1.9
p-value: <0.001				

The present study demonstrates a clear and statistically significant association between peripheral oxygen saturation (SpO₂) levels and the severity of bronchial asthma ($p < 0.001$). Patients with mild intermittent asthma (Class I) maintained normal oxygenation, with none exhibiting SpO₂ levels below 90%, and the majority (60%) having SpO₂ between 96–98%. In mild persistent asthma (Class II), a decline in oxygen saturation was observed, with 35.3% of patients showing SpO₂ <90% and only 14.7% maintaining levels between 96–98%.

In moderate persistent asthma (Class III), a substantial proportion of patients (76.7%) had SpO₂ levels below 90%, indicating significant hypoxemia, while none had normal oxygen saturation. This trend was further pronounced in severe persistent asthma (Class IV), where all patients (100%) exhibited SpO₂ <90%, reflecting marked oxygen desaturation.

The mean SpO₂ values showed a progressive decline with increasing severity, decreasing from $96.8 \pm 1.2\%$ in Class I to $87.1 \pm 1.9\%$ in Class IV. These findings highlight a strong inverse relationship between oxygen saturation and asthma severity, suggesting that SpO₂ can serve as an important objective indicator of disease progression and severity in bronchial asthma patients.

Table 5 : Physiological Parameters According to Severity of Bronchial Asthma (FEV₁/FVC Ratio)

FEV ₁ /FVC	Class I	Class II	Class III	Class IV
<0.5	0	0	0	7 (33.3%)
0.5–0.7	9 (60%)	23 (67.6%)	16 (53.3%)	7 (33.3%)
>0.7	6 (40%)	11 (32.4%)	14 (46.7%)	7 (33.3%)
Mean FEV₁/FVC	0.69 ± 0.076	0.67 ± 0.092	0.70 ± 0.089	0.63 ± 0.150
p-value: <0.001				

The present study demonstrates a statistically significant association between the FEV₁/FVC ratio and the severity of bronchial asthma ($p < 0.001$), indicating worsening airflow obstruction with increasing disease severity. All patients with a severely reduced FEV₁/FVC ratio (<0.5) were observed exclusively in the severe persistent asthma group (Class IV), highlighting marked airway obstruction in advanced disease.

The majority of patients across all severity classes had FEV₁/FVC values in the range of 0.5–0.7, with the highest proportion seen in Class II (67.6%) and Class III (53.3%), suggesting moderate airflow limitation in these groups. Patients with relatively preserved lung function (FEV₁/FVC >0.7) were more commonly seen in Class I (40%) and Class III (46.7%), though they were present across all categories.

The mean FEV₁/FVC ratio showed a declining trend with increasing severity, decreasing from 0.69 ± 0.076 in Class I to 0.63 ± 0.150 in Class IV. These findings indicate that lower FEV₁/FVC ratios are associated with higher severity of bronchial asthma, reflecting progressive airflow limitation and worsening pulmonary function as the disease advances.

Table 6: Association of BMI with C-Reactive Protein (CRP) Levels

CRP Levels	Normal Weight (n=30)	Overweight (n=19)	Obese (n=51)	Total (n=100)	Mean BMI (kg/m ²)
Increased CRP	0 (0%)	0 (0%)	37 (72.5%)	37 (37%)	30.6 ± 3.43
Normal CRP	30 (100%)	19 (100%)	14 (27.5%)	63 (63%)	23.7 ± 2.59
Total	30 (100%)	19 (100%)	51 (100%)	100 (100%)	26.3 ± 4.41
Mean CRP (mg/L)	2.3 ± 1.08	2.9 ± 0.74	7.2 ± 2.22	4.9 ± 2.94	—

The present study demonstrates a strong and statistically significant association between body mass index (BMI) and C-reactive protein (CRP) levels ($p < 0.001$), indicating a close link between obesity and systemic inflammation. Elevated CRP levels were observed exclusively among obese individuals, with 72.5% of patients in the obese category exhibiting increased CRP, while none of the subjects with normal weight or overweight had elevated CRP levels. Conversely, all individuals with normal BMI and overweight had normal CRP levels, whereas only 27.5% of obese individuals maintained normal CRP levels.

The mean BMI was significantly higher among patients with increased CRP (30.6 ± 3.43 kg/m²) compared to those with normal CRP (23.7 ± 2.59 kg/m²), further reinforcing the association between higher body weight and inflammation. Additionally, mean CRP levels showed a progressive increase across BMI categories, rising from 2.3 ± 1.08 mg/L in normal-weight individuals to 7.2 ± 2.22 mg/L in obese patients, with an overall mean of 4.9 ± 2.94 mg/L.

Table 7: Morbidity According to Severity of Bronchial Asthma and BMI

Morbidity	Class III	Class IV	Obese (BMI III)	Total
Previous life-threatening events	5 (45.5%)	6 (54.5%)	11 (100%)	11
Missed working days	19 (33.3%)	38 (66.7%)	57 (100%)	57

The present study highlights a significant association between morbidity, severity of bronchial asthma, and obesity. All patients who experienced major morbidity outcomes, including previous life-threatening events and missed working days, belonged exclusively to the obese category (BMI Class III), accounting for 100% of cases in both groups.

Among patients with previous life-threatening events, a higher proportion was observed in severe persistent asthma (Class IV) (54.5%) compared to moderate persistent asthma (Class III) (45.5%). Similarly, missed working days were more frequent in Class IV (66.7%) than in Class III (33.3%), indicating greater functional impairment with increasing disease severity.

DISCUSSION

The present study was conducted to evaluate the clinical correlation between body mass index (BMI) and the severity of bronchial asthma, along with its association with inflammatory markers and morbidity. The findings demonstrate a strong and statistically significant relationship between increasing BMI and worsening asthma severity, supported by objective physiological parameters and inflammatory markers.

In the current study, the majority of participants were young adults with a mean age of 30.3 ± 6.39 years, and a nearly equal gender distribution (51% males, 49% females). This aligns with observations from previous epidemiological studies, which report asthma prevalence across early and middle adulthood with no strong gender bias in adult populations (15). However, some studies have reported female predominance in obesity-associated asthma phenotypes, particularly in later life (11), indicating possible hormonal or metabolic influences.

A key finding of this study was that more than half of the patients (51%) were obese, with a mean BMI of 26.3 ± 4.42 kg/m². Similar trends have been reported in Indian and global populations, reflecting the rising burden of obesity among patients with chronic respiratory diseases (16,17). The increasing prevalence of obesity in asthma patients has been attributed to sedentary lifestyles, dietary changes, and urbanization.

The most significant observation in the present study was the strong association between BMI and asthma severity ($p < 0.001$). All patients with mild intermittent asthma had normal BMI, whereas all patients with moderate and severe asthma belonged exclusively to the obese category. Additionally, mean BMI increased progressively with severity, from 20.9 kg/m² in mild cases to 33.1 kg/m² in severe cases. These findings are consistent with studies by Beuther and Sutherland (5) and Peters-Golden et al. (7), which demonstrated that obesity is associated with increased asthma severity, poor control, and reduced response to treatment. Similarly, a study by Lessard et al. (18) showed that obese asthmatics have significantly worse symptom burden and airflow limitation compared to non-obese individuals.

The physiological parameters assessed in this study further support the relationship between obesity and asthma severity. A significant decline in SpO₂ levels was observed with increasing severity, with all severe asthma patients exhibiting hypoxemia (SpO₂ <90%). This finding is in agreement with studies that have demonstrated impaired gas exchange in severe asthma due to ventilation-perfusion mismatch and airway obstruction (19). Similarly, the FEV₁/FVC ratio showed a declining trend with increasing severity, indicating worsening airflow limitation. These results are comparable with findings by Salome et al. (9), who reported that obesity contributes to reduced lung volumes and impaired pulmonary mechanics.

An important strength of the present study is the evaluation of systemic inflammation using C-reactive protein (CRP). It was observed that elevated CRP levels were exclusively seen in obese individuals, with mean CRP significantly higher in this group (7.2 ± 2.22 mg/L). This supports the concept that obesity is associated with a chronic low-grade inflammatory state, which may exacerbate airway inflammation in asthma. Similar findings have been reported by Shore (10) and Sutherland et al. (20), who demonstrated that inflammatory mediators released from adipose tissue contribute to asthma pathogenesis and severity.

The morbidity outcomes in the present study further highlight the clinical impact of obesity in asthma. All patients with severe morbidity indicators, such as previous life-threatening events and missed working days, belonged to the obese category and were predominantly in moderate to severe asthma classes. This is consistent with previous studies showing that obese asthmatics experience higher rates of hospitalization, absenteeism, and reduced quality of life (21).

The findings of the present study also demonstrate that age is significantly associated with asthma severity ($p < 0.001$), with more severe disease observed in relatively older individuals within the study group. This trend has been reported in earlier studies, suggesting cumulative exposure to risk factors and progressive airway remodeling with age (22). However, gender was not found to be significantly associated with severity ($p = 0.506$), which is consistent with some studies but contrasts with others that suggest gender-specific differences in asthma phenotype (23).

Despite the strong associations observed, variability in findings across studies may be attributed to differences in genetic predisposition, environmental exposures, and lifestyle factors. In the Indian context, limited studies have comprehensively evaluated BMI, inflammatory markers, and asthma severity together, making the present study particularly relevant.

Overall, the present study clearly demonstrates that obesity is not only a risk factor for asthma but also plays a significant role in determining disease severity, physiological impairment, systemic inflammation, and morbidity. These findings emphasize the importance of incorporating weight management strategies into routine asthma care.

CONCLUSION

The present study establishes a strong and statistically significant association between increasing body mass index and the severity of bronchial asthma. Obese individuals were found to have more severe disease, poorer pulmonary function, reduced oxygen saturation, and higher levels of systemic inflammation as indicated by elevated CRP. Additionally, obesity was associated with increased morbidity, including life-threatening events and reduced functional capacity. These findings highlight the critical role of obesity as a modifiable risk factor in asthma progression. Integrating weight reduction strategies along with standard pharmacological management may significantly improve clinical outcomes and quality of life in patients with bronchial asthma.

REFERENCES

1. World Health Organization. Global surveillance, prevention and control of chronic respiratory diseases. WHO; 2022.
2. GBD Chronic Respiratory Disease Collaborators. Global burden of asthma. *Lancet Respir Med.* 2020;8(6):585–596.
3. World Health Organization. Obesity: preventing and managing the global epidemic. WHO; 2000.
4. Misra A, Khurana L. Obesity and metabolic syndrome in developing countries. *J Clin Endocrinol Metab.* 2008;93(11):S9–S30.
5. Beuther DA, Sutherland ER. Overweight, obesity, and incident asthma. *Am J Respir Crit Care Med.* 2007;175(7):661–666.
6. Camargo CA Jr, et al. Prospective study of BMI and asthma risk. *Arch Intern Med.* 1999;159(21):2582–2588.
7. Peters-Golden M, et al. Influence of obesity on asthma severity. *J Allergy Clin Immunol.* 2006;118(6):1313–1319.
8. Taylor B, et al. Obesity and asthma outcomes. *Thorax.* 2008;63(1):14–20.
9. Salome CM, et al. Effects of obesity on lung function. *Eur Respir J.* 2010;35(4):761–768.
10. Shore SA. Obesity and asthma: possible mechanisms. *J Allergy Clin Immunol.* 2008;121(5):1087–1093.
11. Wenzel SE. Asthma phenotypes: the evolution from clinical to molecular approaches. *Nat Med.* 2012;18(5):716–725.
12. Global Initiative for Asthma (GINA). Global strategy for asthma management and prevention. 2023.
13. Sin DD, et al. Obesity is a risk factor for dyspnea but not airflow obstruction. *Arch Intern Med.* 2002;162(13):1477–1481.
14. Dixon AE, et al. Effects of weight loss on asthma control. *J Allergy Clin Immunol.* 2011;128(3):508–515.

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15. To T, Stanojevic S, Moores G, et al. Global asthma prevalence in adults. *Lancet Respir Med.* 2012;**1**(1):1–8.
16. Ng M, Fleming T, Robinson M, et al. Global, regional, and national prevalence of overweight and obesity. *Lancet.* 2014;**384**(9945):766–781.
17. Luhar S, Mallinson PAC, Clarke L, et al. Trends in obesity in India. *PLoS Med.* 2020;**17**(5):e1003169.
18. Lessard A, Turcotte H, Cormier Y, et al. Obesity and asthma: a specific phenotype. *Chest.* 2008;**134**(2):317–323.
19. O'Donnell DE, et al. Mechanisms of dyspnea in asthma. *Chest.* 2006;**130**(2):523–532.
20. Sutherland ER, et al. Systemic inflammation in asthma and obesity. *J Allergy Clin Immunol.* 2008;**121**(2):314–320.
21. Schatz M, Zeiger RS, et al. Obesity and asthma outcomes. *J Allergy Clin Immunol.* 2005;**115**(3):557–563.
22. Busse WW, Lemanske RF. Asthma pathophysiology and aging. *N Engl J Med.* 2001;**344**(5):350–362.
23. Almqvist C, Worm M, Leynaert B. Gender impact on asthma. *Eur Respir J.* 2008;**31**(3):671–677.