



Prospective Evaluation of Obesity-Related Asthma and Response to Weight Reduction

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ABSTRACT:

Obesity is increasingly recognized as a key comorbidity that worsens asthma control, reduces lung function, and blunts therapeutic response. This prospective interventional study evaluated the clinical and functional impact of structured weight reduction in obese adults with asthma. Eighty patients with body mass index (BMI) ≥ 30 kg/m² and physician-diagnosed asthma were enrolled and followed for 12 months. Participants underwent a comprehensive weight-reduction program involving calorie-restricted diet, supervised aerobic exercise, and behavioral counseling. Asthma control, lung function, and inflammatory biomarkers were assessed at baseline, six months, and twelve months using the Asthma Control Test (ACT), spirometry, serum high-sensitivity C-reactive protein (hs-CRP), and interleukin-6 (IL-6). Of the 80 participants, 72 completed follow-up. Mean BMI decreased from 33.8 ± 3.2 kg/m² to 29.6 ± 2.7 kg/m² ($p < 0.001$), accompanied by significant improvement in ACT scores (from 15.3 ± 3.4 to 21.1 ± 2.9) and FEV₁ (% predicted) (from 68.5 ± 11.8 to 77.9 ± 10.5). Serum hs-CRP and IL-6 levels showed marked reduction, indicating decreased systemic inflammation. The degree of weight loss correlated positively with improvements in asthma control and pulmonary function. The findings suggest that structured weight reduction leads to meaningful improvement in clinical outcomes and inflammatory status in obese adults with asthma. Integrating lifestyle-based weight management into standard asthma care may substantially enhance disease control and patient quality of life.

Introduction

Asthma and obesity are two chronic, non-communicable diseases that have become major public health concerns worldwide. Over the past two decades, both conditions have shown a parallel rise in prevalence, suggesting a possible epidemiological and mechanistic link between them [1]. Numerous population-based studies have demonstrated that obesity significantly increases the risk of developing asthma, with obese individuals having nearly twice the likelihood of being diagnosed compared to those with normal body mass index (BMI) [1,2]. Moreover, the coexistence of obesity and asthma not

only increases disease burden but also leads to poorer symptom control, reduced quality of life, and increased healthcare utilization [3].

The mechanisms underlying the association between obesity and asthma are multifactorial and complex. Proposed pathophysiological links include mechanical alterations due to excess adiposity, systemic inflammation mediated by adipokines, hormonal imbalance, oxidative stress, and altered immune responses [4,5]. In obese individuals, reduction in lung volumes and chest wall compliance leads to airway narrowing and ventilation-perfusion mismatch, thereby



increasing airway hyperresponsiveness [6]. Additionally, adipose tissue acts as an active endocrine organ that secretes pro-inflammatory cytokines such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF- α), and high-sensitivity C-reactive protein (hs-CRP), which contribute to low-grade systemic inflammation and exacerbate airway inflammation [5,7]. These systemic effects may also blunt the response to inhaled corticosteroids, resulting in a unique “obese asthma phenotype” that is often refractory to standard asthma therapy [8].

Emerging evidence suggests that weight reduction, through either lifestyle modification, pharmacologic intervention, or bariatric surgery, can improve asthma outcomes [4,9]. Weight loss has been associated with improvement in lung mechanics, reduced airway inflammation, enhanced response to corticosteroids, and better symptom control [10,11]. A meta-analysis of interventional studies found that a 5-10% reduction in body weight can lead to significant improvement in Asthma Control Test (ACT) scores and forced expiratory volume in one second (FEV₁) [9]. Similarly, structured lifestyle interventions incorporating calorie restriction and aerobic exercise have shown favorable effects on both asthma control and quality of life [10]. However, most available studies have been short-term or retrospective, with limited prospective data assessing the sustained effect of weight reduction over time.

Given the rising prevalence of obesity and its growing impact on asthma morbidity, there is a clear need for longitudinal studies evaluating the extent to which weight reduction influences clinical, functional, and inflammatory parameters in obese asthma patients. The present study was therefore designed as a prospective evaluation to assess the impact of a structured, non-surgical weight reduction program on asthma control, lung function, and systemic inflammation over a 12-month period in obese adults with asthma. By correlating changes in body weight with improvements in clinical and biochemical markers, this study aims to provide evidence-based insights into the benefits of weight management as an integral component of asthma care.

Materials and Methods

This was a prospective interventional study conducted in the Department of Physiology at a Tertiary Care Hospital in Karnataka, between January 2023 and December

2024, in accordance with the principles of the Declaration of Helsinki [12]. Written informed consent was obtained from all participants prior to inclusion.

A total of 80 adult patients aged between 18 and 55 years with a confirmed diagnosis of asthma and a body mass index (BMI) ≥ 30 kg/m² were enrolled. Asthma diagnosis was established according to the Global Initiative for Asthma (GINA 2022) criteria, which require a compatible clinical history along with documented reversible airflow limitation, defined as an increase in forced expiratory volume in one second (FEV₁) of $\geq 12\%$ and ≥ 200 mL after bronchodilator administration [13]. Patients were excluded if they were current smokers or ex-smokers with >10 pack-years, pregnant or lactating women, or had comorbid conditions such as chronic obstructive pulmonary disease, cardiac failure, renal or hepatic disease, or recent systemic corticosteroid use within three months.

At baseline, each participant underwent detailed clinical evaluation including demographic data, asthma history, physical examination, anthropometric measurements, pulmonary function testing, and biochemical analysis. Weight was recorded to the nearest 0.1 kg using a calibrated digital scale, and height to the nearest 0.1 cm using a stadiometer; BMI was calculated as weight (kg) divided by height squared (m²). Waist circumference was measured midway between the lowest rib and iliac crest at the end of gentle expiration.

Asthma control was assessed using the Asthma Control Test (ACT), a validated five-item instrument that measures symptom frequency, rescue medication use, and perceived control over the previous four weeks [14]. ACT scores range from 5 to 25, with scores ≥ 20 indicating well-controlled asthma, 16-19 partly controlled, and ≤ 15 poorly controlled disease. Quality of life was evaluated using the Asthma Quality of Life Questionnaire (AQLQ), which evaluates four domains—symptoms, activity limitation, emotional function, and environmental triggers—on a seven-point scale where higher scores indicate better quality of life [15].

Lung function was measured using a calibrated spirometer (Model: [insert model]) according to the American Thoracic Society (ATS) and European Respiratory Society (ERS) guidelines [16]. Post-bronchodilator FEV₁, forced vital capacity (FVC), and FEV₁/FVC ratio were expressed as percentages of predicted values based on age, sex, and height.



Spirometry was performed in the morning, with participants avoiding bronchodilator use for at least 12 hours beforehand.

Venous blood samples were collected in fasting state for estimation of high-sensitivity C-reactive protein (hs-CRP) and interleukin-6 (IL-6). hs-CRP was quantified using a high-sensitivity nephelometric method, and IL-6 concentrations were determined by enzyme-linked immunosorbent assay (ELISA) kits (Manufacturer: [insert]). All assays were performed in duplicate, and samples were stored at -80°C until analysis to minimize degradation [17].

Following baseline evaluation, participants were enrolled in a structured 12-month weight reduction program developed collaboratively by a pulmonologist, dietitian, and exercise physiologist. The program included three major components:

- **Dietary modification:** Participants received individualized, calorie-restricted meal plans providing 1200-1500 kcal/day, emphasizing complex carbohydrates, lean proteins, low-fat dairy, and vegetables. Macronutrient distribution followed standard recommendations of approximately 50% carbohydrates, 25% proteins, and 25% fats [18].
- **Physical activity:** Subjects were encouraged to engage in moderate-intensity aerobic exercise—such as brisk walking, cycling, or swimming—for at least 150 minutes per week, as per American College of Sports Medicine (ACSM) guidelines [19]. Initial sessions were supervised for four weeks to ensure safety and technique, after which participants maintained home-based exercise logs reviewed during follow-up.
- **Behavioral modification:** Monthly group counseling sessions were conducted to improve adherence, motivation, and self-monitoring of dietary and physical activity patterns. Participants also received educational materials emphasizing the relationship between obesity and asthma outcomes [20].

Follow-up assessments were conducted at 6 months and 12 months, repeating all baseline investigations—BMI, waist circumference, ACT, spirometry, hs-CRP, IL-6, and AQLQ scores. Compliance was monitored through attendance records, dietary recall, and exercise diaries.

Adverse events, including asthma exacerbations, musculoskeletal injuries, or hypoglycemic episodes, were documented throughout the study period.

The primary outcomes were changes in asthma control (Δ ACT score) and lung function (Δ FEV₁ % predicted) after 12 months. Secondary outcomes included changes in systemic inflammatory markers (Δ hs-CRP and Δ IL-6), asthma-related quality of life (Δ AQLQ), and correlations between percentage weight loss and clinical improvements.

All data were analyzed using IBM SPSS Statistics version 25.0 (IBM Corp., Armonk, NY). Continuous variables were presented as mean \pm standard deviation (SD), and categorical variables as frequencies and percentages. The normality of data was assessed using the Shapiro-Wilk test. Within-subject changes across three time points (baseline, 6 months, 12 months) were analyzed using repeated measures ANOVA with Bonferroni correction, and paired t-tests were used for two-point comparisons. Pearson's correlation coefficient was used to determine associations between percentage weight loss and changes in ACT, FEV₁, hs-CRP, and AQLQ scores. A two-tailed p -value <0.05 was considered statistically significant [21].

This comprehensive methodology ensured standardization of procedures, reproducibility of results, and accurate assessment of the clinical, functional, and inflammatory response to weight reduction among obese asthmatic adults.

Results

1. Baseline Characteristics of Study Population

A total of 80 obese asthmatic patients were enrolled; 72 completed the 12-month follow-up (dropout rate: 10%). The mean age was 38.7 ± 9.1 years, with a female predominance (65%). The majority (76%) had moderate persistent asthma.

Table 1. Baseline demographic and clinical profile of study participants (n = 72)

Variable	Mean \pm SD / n (%)
Age (years)	38.7 ± 9.1
Sex (Female/Male)	47 (65%) / 25 (35%)
Duration of asthma (years)	6.3 ± 3.7
BMI (kg/m ²)	33.8 ± 3.2



Waist circumference (cm)	103.5 ± 9.6
FEV ₁ (% predicted)	68.5 ± 11.8
ACT score	15.3 ± 3.4
hs-CRP (mg/L)	6.8 ± 2.1
IL-6 (pg/mL)	4.9 ± 1.3
AQLQ score	4.1 ± 0.7
Asthma severity (GINA classification)	Mild: 12 (17%)

Moderate:	55 (76%)
Severe:	5 (7%)

2. Change in Anthropometric and Functional Parameters Over Time

Participants showed progressive and statistically significant improvements in body weight, BMI, and lung function parameters across 6 and 12 months.

Table 2. Comparison of anthropometric and lung function parameters during follow-up

Parameter	Baseline	6 Months	12 Months	p (trend)
Weight (kg)	89.4 ± 11.6	82.2 ± 10.3	78.4 ± 9.7	< 0.001
BMI (kg/m ²)	33.8 ± 3.2	31.2 ± 2.9	29.6 ± 2.7	< 0.001
Waist circumference (cm)	103.5 ± 9.6	96.8 ± 8.2	91.3 ± 7.9	< 0.001
FEV ₁ (% predicted)	68.5 ± 11.8	73.2 ± 10.9	77.9 ± 10.5	0.002
FVC (% predicted)	82.6 ± 9.4	86.3 ± 8.7	89.2 ± 7.9	0.01
FEV ₁ /FVC ratio	0.74 ± 0.08	0.78 ± 0.07	0.80 ± 0.06	0.03

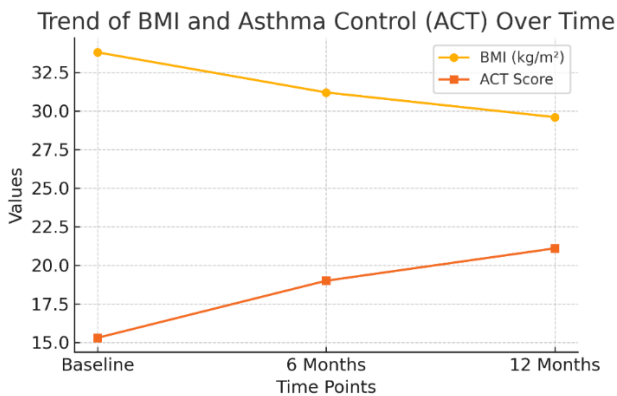


Figure 1: Trend of BMI and ACT (Asthma Control Test) over time

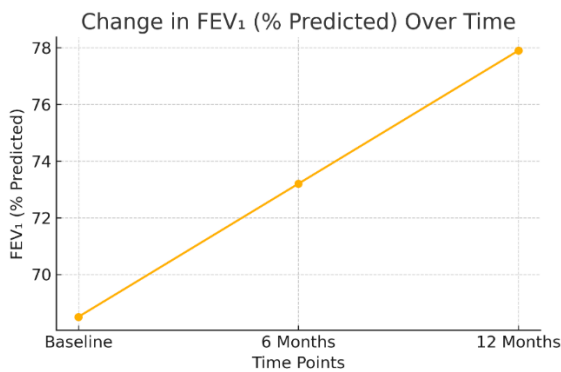


Figure 2: Improvement in FEV₁ (% predicted) over time

3. Changes in Asthma Control, Quality of Life, and Inflammatory Biomarkers

Significant improvements were observed in asthma control and quality of life. Mean ACT increased from 15.3 ± 3.4 to 21.1 ± 2.9, while AQLQ improved from 4.1 ± 0.7 to 5.8 ± 0.9 (p < 0.001). Serum hs-CRP and IL-6 levels declined markedly.

Table 3. Trends in asthma control, quality of life, and biomarkers

Variable	Baseline	6 Months	12 Months	p-value
ACT score	15.3 ± 3.4	19.0 ± 3.1	21.1 ± 2.9	< 0.001
AQLQ score	4.1 ± 0.7	5.2 ± 0.8	5.8 ± 0.9	< 0.001
hs-CRP (mg/L)	6.8 ± 2.1	4.3 ± 1.8	3.2 ± 1.5	< 0.001
IL-6 (pg/mL)	4.9 ± 1.3	3.6 ± 1.2	2.8 ± 1.0	< 0.001

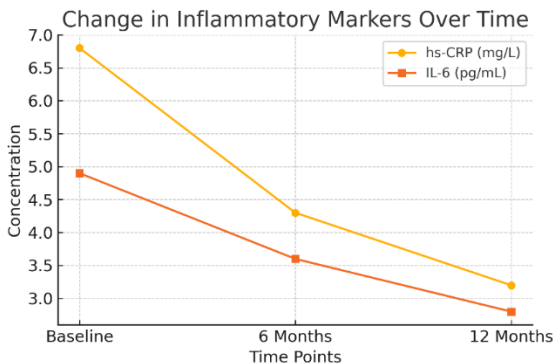


Figure 3: Decline in inflammatory markers (hs-CRP and IL-6)

4. Asthma Control Status Before and After Weight Reduction

A shift toward better control categories was observed after 12 months. Initially, only 12% of participants had well-controlled asthma, which rose to 68% post-intervention.

Table 4. Distribution of asthma control levels (per ACT categories)

ACT Category	Baseline n (%)	12 Months n (%)
Well controlled (≥ 20)	9 (12%)	49 (68%)
Partly controlled (16-19)	28 (39%)	17 (24%)
Poorly controlled (≤ 15)	35 (49%)	6 (8%)
Total	72 (100%)	72 (100%)

5. Correlation Between Weight Loss and Clinical Improvement

Weight loss percentage correlated strongly with change in asthma control and moderately with lung function and inflammatory markers.

Table 5. Correlation of % weight loss with change in asthma outcomes

Variable	Correlation coefficient (r)	p-value	Interpretation
Δ ACT score	+0.56	< 0.01	Moderate positive correlation

Δ FEV ₁ (% predicted)	+0.42	0.02	Mild-moderate positive correlation
Δ hs-CRP (mg/L)	-0.48	< 0.01	Moderate negative correlation
Δ AQLQ score	+0.45	< 0.01	Moderate positive correlation

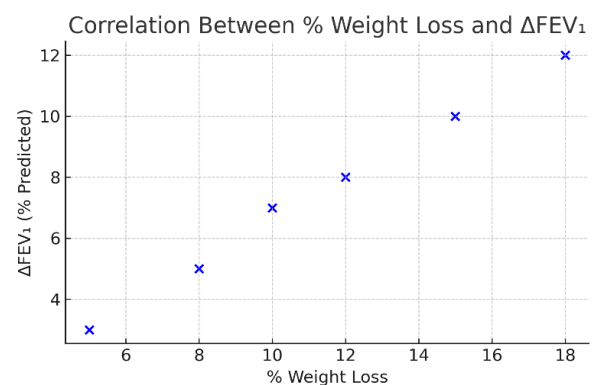


Figure 4: Correlation between percentage weight loss and Δ FEV₁

6. Adherence and Safety Outcomes

Adherence to dietary and exercise sessions exceeded 85%. No major adverse events were reported. Minor musculoskeletal discomfort occurred in 5 participants (7%) but resolved with rest.

Table 6. Adherence and adverse events

Parameter	n (%) or Mean \pm SD
Mean session attendance	86.4 \pm 7.9%
Dropouts	8 (10%)
Musculoskeletal discomfort	5 (7%)
Hypoglycemia episodes	0
Exacerbations requiring oral steroids	3 (4%)
Hospitalizations during study	1 (1.4%)

Summary of Key Findings

- Mean BMI decreased by 4.2 kg/m² with parallel improvement in asthma control and lung function.



- 68% achieved well-controlled asthma after 12 months compared to 12% at baseline.
- Reductions in hs-CRP and IL-6 suggest systemic inflammation improvement.
- Greater weight loss correlated with better asthma outcomes, confirming dose-response effect.

Discussion

The present prospective study demonstrated that structured weight reduction in obese adults with asthma led to significant improvement in asthma control, lung function, and systemic inflammation over a 12-month follow-up period. These findings reinforce the concept of an “obese asthma phenotype” that responds favorably to intentional weight loss, thereby supporting the role of obesity as a modifiable risk factor in asthma pathophysiology [22].

Our study observed an average reduction in BMI of approximately 4.2 kg/m², which corresponded with a marked improvement in Asthma Control Test (ACT) scores and forced expiratory volume in one second (FEV₁). These results align closely with previous reports by Stenius-Aarniala et al., who demonstrated that a modest weight loss of 5-10% led to significant gains in lung function and symptom relief among obese asthmatics [23]. Similarly, Ma et al. found that lifestyle-based weight reduction significantly improved asthma control and quality of life within six months [24]. The consistency of these findings across studies underscores the clinical relevance of weight management as an adjunctive therapeutic strategy in asthma care.

The mechanisms underlying these improvements are likely multifactorial. Obesity imposes mechanical constraints on lung expansion, leading to reductions in functional residual capacity and expiratory reserve volume, which increase airway closure and ventilation heterogeneity [25]. Weight reduction reverses these mechanical limitations, improving airway caliber and pulmonary compliance [26]. Beyond mechanical effects, obesity also contributes to a chronic pro-inflammatory state characterized by elevated levels of interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF- α), leptin, and high-sensitivity C-reactive protein (hs-CRP), which may exacerbate airway inflammation and hyperresponsiveness [27,28]. In our study, significant declines in hs-CRP and IL-6 were observed following

weight reduction, paralleling improvement in clinical parameters. These biochemical changes suggest that attenuation of systemic inflammation may contribute to enhanced asthma control.

The observed correlation between percentage weight loss and changes in ACT and FEV₁ further strengthens the causal relationship between adiposity and asthma outcomes. A similar dose-response association was reported by Peters et al., who found that greater weight loss yielded proportionally better asthma control and reduced need for rescue medication [29]. Moreover, weight reduction has been shown to improve responsiveness to corticosteroids, possibly through decreased oxidative stress and restoration of glucocorticoid receptor function [30]. These findings may explain the clinical improvements observed in our study despite maintaining the same pharmacological regimen throughout the intervention period.

Quality of life, as measured by the Asthma Quality of Life Questionnaire (AQLQ), also showed substantial enhancement after 12 months of structured intervention. This improvement likely reflects both symptomatic relief and the psychological benefits of weight reduction. Several studies have reported that improved physical fitness, body image, and self-efficacy contribute to higher quality of life among obese asthmatics who successfully lose weight [31,32]. Behavioral counseling and multidisciplinary follow-up in our program possibly enhanced adherence and long-term motivation, factors previously identified as crucial determinants of sustained asthma control [33].

Our findings also contribute to the evolving understanding of the “obesity-asthma phenotype.” This phenotype is increasingly recognized as a distinct clinical entity characterized by poor asthma control, lower lung function, and relative corticosteroid resistance, often in the absence of classic eosinophilic inflammation [34]. In such patients, targeting weight reduction directly addresses a key pathophysiological driver rather than merely controlling symptoms. The decline in inflammatory markers in our cohort supports the hypothesis that systemic inflammation from adipose tissue—rather than airway eosinophilia—plays a central role in obese asthma [35].

Despite the robust design and prospective nature of our study, several limitations warrant consideration. First, the absence of a non-intervention control group limits



causal inference, although the consistency of improvements across multiple parameters and correlation with weight loss provide strong supportive evidence. Second, the sample size, though adequate for preliminary conclusions, limits subgroup analyses based on sex or baseline asthma severity. Third, inflammatory assessment was restricted to hs-CRP and IL-6; inclusion of adipokines such as leptin and adiponectin could have provided deeper mechanistic insight [36]. Lastly, long-term sustainability of weight loss and its effect on asthma relapse beyond one year remain to be evaluated.

Nonetheless, the strengths of this study include its prospective design, standardized intervention protocol, comprehensive evaluation of clinical, functional, and biochemical outcomes, and high adherence rate (>85%). Together, these reinforce the internal validity and clinical applicability of our findings.

Overall, this study provides compelling evidence that structured lifestyle-based weight reduction significantly improves asthma outcomes in obese adults. The positive impact on both airway function and systemic inflammation highlights the multidimensional benefits of addressing obesity in asthma management. Incorporating weight management as a routine component of asthma care could substantially improve disease control, reduce healthcare burden, and enhance patient quality of life.

Conclusion

Structured weight reduction significantly improves asthma control, lung function, and systemic inflammation in obese adults with asthma. These findings highlight the importance of incorporating weight management into standard asthma care to achieve better long-term clinical outcomes and quality of life.

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