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The Dual Burden of Obesity and Asthma: Implications for Respiratory Health and Asthma Control in Adults with Asthma

Obezite ve Astımın İkili Yüku: Astımlı Yetişkinlerde Solunum Sağlığı ve Astım Kontrolü Üzerindeki Etkileri

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Abstract: Asthma and obesity are both highly prevalent conditions with significant public health implications, and obesity has been identified as an aggravating factor in the pathogenesis of asthma. The present study aims to investigate differences in pulmonary function, lung diffusion capacity, functional exercise capacity, and asthma control between obese and non-obese adults with asthma. Fifty asthma patients were divided equally into obese and non-obese groups. Participants performed pulmonary function test (PFT), lung diffusion capacity, six-minute walk test (6MWT), and Asthma Control Test (ACT). No significant differences were found among groups in terms of PFT, lung diffusion capacity, and ACT ($p > 0.05$). Significant differences were observed between groups in end-test SpO_2 ($p = 0.045$), baseline and end-test systolic blood pressure ($p = 0.017$ and $p = 0.002$), end-test respiratory rate ($p = 0.002$), perceptions of dyspnea and fatigue at both baseline and end-test (all $p < 0.05$), end-test leg pain ($p = 0.045$), and total walking distance ($p = 0.038$). A moderate and statistically significant negative correlation was found between body mass index (BMI) and the 6MWT distance ($r = -0.592$, $p = 0.028$). This study reveals that functional capacity is significantly impaired in patients with mild asthma, and obesity further aggravates this limitation. These results highlight the need for early functional evaluation and reinforce the importance of incorporating structured exercise and weight control into comprehensive asthma rehabilitation programs.

Keywords: Asthma, Asthma control, Functional exercise capacity, Lung diffusion capacity, Obesity, Pulmonary function test

Özet: Astım ve obezite, her ikisi de önemli halk sağlığı sorunları olan yaygın durumlardır ve obezitenin astımın patogeneğinde kötüleştirici bir faktör olduğu belirlenmiştir. Bu çalışma, astımı olan obez ve obez olmayan yetişkinler arasında akciğer fonksiyonları, akciğer diffüzyon kapasitesi, fonksiyonel egzersiz kapasitesi ve astım kontrolü açısından farklılıkları araştırmayı amaçlamaktadır. Elli astım hastası eşit şekilde obez ve obez olmayan iki gruba ayrılmıştır. Katılımcılara solunum fonksiyon testi (SFT), akciğer diffüzyon kapasitesi testi, altı dakikalık yürüme testi (6DYT) ve Astım Kontrol Testi (AKT) uygulanmıştır. Gruplar arasında SFT, akciğer diffüzyon kapasitesi ve AKT açısından anlamlı bir fark bulunmamıştır ($p > 0.05$). Gruplar arasında test sonu SpO_2 ($p = 0.045$), başlangıç ve test sonu sistolik kan basıncı ($p = 0.017$ ve $p = 0.002$), test sonu solunum hızı ($p = 0.002$), hem başlangıç hem de test sonunda nefes darlığı ve yorgunluk algısı (tüm $p < 0.05$), test sonu bacak ağrısı ($p = 0.045$) ve toplam yürüme mesafesi ($p = 0.038$) açısından anlamlı farklar gözlemlenmiştir. Vücut kitle indeksi (VKİ) ile 6DYT mesafesi arasında orta düzeyde ve istatistiksel olarak anlamlı negatif bir korelasyon bulunmuştur ($r = -0.592$, $p = 0.028$). Bu çalışma, hafif astımı olan hastalarda fonksiyonel kapasitenin belirgin şekilde bozulduğunu ve obezitenin bu kısıtlamayı daha da kötüleştirdiğini ortaya koymaktadır. Bu sonuçlar, erken fonksiyonel değerlendirme gerekliliğini vurgulamakta ve yapılandırılmış egzersiz ile kilo kontrolünü içeren kapsamlı astım rehabilitasyon programlarının önemini pekiştirmektedir.

Anahtar Kelimeler: Akciğer Difüzyon Kapasitesi, Astım, Astım kontrolü, Fonksiyonel egzersiz kapasitesi, Obezite, Solunum Fonksiyon Testi

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1. Introduction

Asthma is a heterogeneous, chronic inflammatory airway disease marked by variable airflow obstruction and bronchial hyperresponsiveness. Clinically, it presents with symptoms such as dyspnea, wheezing, chest tightness, and cough, which tend to worsen at night or in the early morning hours (1). The global burden of asthma continues to rise, currently affecting approximately 339 million people, with prevalence rates varying between 1% and 20% depending on geographic and environmental conditions (2).

Obesity has been identified as a significant modifiable factor influencing asthma development and progression. An asthma-obesity phenotype has been described, characterized by more severe symptoms, reduced response to standard therapies and asthma control, and lower quality of life, and increased healthcare utilization (3). Mechanistically, obesity contributes to systemic inflammation, airway remodeling, and altered immune responses, all of which may exacerbate asthma severity and frequency of exacerbations (4, 5). Furthermore, obesity-related comorbidities such as gastroesophageal reflux disease, obstructive sleep apnea, and metabolic syndrome may further contribute to the worsening of asthma symptoms (6).

Several studies have reported an inverse association between body mass index (BMI) and pulmonary function, functional capacity, and asthma control, suggesting that increased adiposity negatively affects respiratory health (7, 8). Excess weight has been linked to increased airway resistance, reduced lung compliance, and physical inactivity, which may amplify systemic inflammation and respiratory symptoms (9). However, findings across the literature remain inconsistent regarding the magnitude and nature of these effects, particularly concerning lung diffusion capacity and exercise capacity (10).

Given these ambiguities, further research is warranted to clarify the impact of obesity on key physiological and clinical parameters in asthma. The present study aims to investigate differences in pulmonary function, lung diffusion capacity, functional exercise capacity, and asthma control between obese and non-obese adults with asthma, thereby contributing to a more nuanced understanding of the asthma-obesity phenotype.

2. Materials and Methods

Study design and subjects

This study was conducted as a prospective and cross-sectional study. Fifty patients who met the inclusion criteria among 58 asthma patients referred from the Department of Chest Diseases of a university hospital between January 2024 and June 2024 were included in the study. The criteria for selecting the subjects were as follows: being diagnosed with asthma, age at more than 18 years old, and being able to read and understand written and spoken language. Subjects were excluded if they were unable to complete the tests or exercises due to diagnosed comorbid conditions, had experienced a disease exacerbation in the past 8 weeks, were currently involved in, or had participated in a regular exercise training program within the past year. After the initial assessments, patients were divided into two groups: either the obese ($n=25$) or the non-obese ($n=25$) group. BMI was calculated as weight in kilograms divided by height in meters squared (kg/m^2). According to the World Health Organization (WHO) criteria (11), individuals with a BMI of $30 \text{ kg}/\text{m}^2$ or higher were classified as obese, while those with a BMI below $30 \text{ kg}/\text{m}^2$ were considered non-obese.

The study was conducted by the tenets of the Declaration of Helsinki and approved by the Ethics Committee of Atlas University (date: 09.10.2023, protocol number: 08.07). All participants provided written informed consent.

Outcome measures

Asthma Severity

Asthma severity was assessed using the GINA criteria, which categorize patients into four groups—mild intermittent, mild persistent, moderate persistent, and severe persistent—based on clinical symptoms and spirometric measurements reflecting the degree of airway obstruction (12).

Asthma Control Test

The Asthma Control Test (ACT) is a five-item questionnaire that evaluates asthma control based on symptoms, medication use, and daily functioning. Each item is scored on a 5-point scale, yielding a total score between 5 and 25.

Scores ≥ 20 indicate well-controlled asthma, while scores < 20 suggest poor control (13).

Pulmonary Function Test (PFT)

Pulmonary function was assessed with a spirometer (COSMED Pony FX) following American Thoracic Society (ATS) and European Respiratory Society (ERS) standards (14). Parameters such as forced vital capacity (FVC), forced expiratory volume in one second (FEV₁), FEV₁/FVC ratio, peak expiratory flow (PEF), and forced expiratory flow between 25% and 75% (FEF₂₅₋₇₅) were recorded and expressed as percentages of predicted values.

Lung Diffusing Capacity

Lung diffusing capacity for carbon monoxide (DLCO) was measured using the single-breath technique with an automated device (CareFusion, Hochberg, Germany). Participants first breathed normally for 4–5 breaths, then exhaled fully to residual volume. They were then instructed to inhale a test gas mixture rapidly to total lung capacity, hold their breath for 4 seconds, and exhale steadily back to residual volume (15).

Functional Exercise Capacity

The 6-Minute Walk Test (6MWT), recommended by the ATS to evaluate functional exercise capacity (16), was conducted along a 30-meter straight corridor. Participants were instructed to walk as fast as possible at their own pace without running for 6 minutes. Physiological parameters including oxygen saturation (SpO₂), heart rate (HR), systolic (SBP) and diastolic blood pressure (DBP), respiratory rate (RR), dyspnea, fatigue (via the Modified Borg Scale), and leg pain (via the Visual Analogue Scale) were measured before and after the test. The total walking distance covered in 6 minutes was recorded in meters (m).

Statistics and sample size

Statistical analyses were performed using the SPSS 16.0 software package. The normality of data distribution was assessed using the Shapiro-Wilk test. Categorical variables were analyzed using the Chi-square (χ^2) test. For comparisons between groups, the Independent Samples T-test was applied when data followed a normal distribution; otherwise, the Mann-Whitney U test was used. The One-Sample T-test was conducted to compare results with reference values from a healthy population. Correlations between variables were assessed using either Pearson or

Spearman correlation tests, depending on data distribution. Correlation strength was categorized based on the correlation coefficient (r) as follows: weak ($r < 0.3$), moderate ($r = 0.3–0.5$), and strong ($r = 0.5–1.0$). A p -value of < 0.05 was considered statistically significant.

G*Power 3.1 software was used to calculate the sample size. (University of Düsseldorf, Germany). Sava et al. (17) reported a significant difference in functional exercise capacity between obese and non-obese patients with COPD in which an effect size was 0.843 (342 ± 79 m vs 407 ± 75 m). We calculated that at least 24 participants per group would be required to detect this difference with %80 power and 95% confidence in the study.

3. Results

Fifty-eight asthma patients were assessed for eligibility; a total of eight patients were excluded for not meeting the inclusion criteria or refusing to participate. Twenty-five patients for each group (obese and non-obese) were included in the study (Figure 1). The demographic and clinical characteristics of the patients are shown in Table 1. No significant difference was found between the two groups' demographic and clinical data except for BMI.

There was no statistically significant difference between the two groups in terms of PFT parameters and lung diffusion capacity (Table 2).

The comparison of 6MWT results between obese and non-obese asthma patients is presented in Table 3. A significant difference was observed in end-test SpO₂ levels, with the non-obese group having higher oxygen saturation compared to the obese group ($p = 0.045$). Both baseline and end values of systolic blood pressure (SBP) were significantly higher in the obese group ($p = 0.017$ and $p = 0.002$, respectively). Additionally, the respiratory rate at the end of the test was significantly elevated in the obese group ($p = 0.002$). Perceptions of dyspnea and fatigue were significantly more pronounced in the obese group at both the beginning ($p = 0.004$ and $p = 0.018$, respectively) and at the end of the test ($p = 0.010$ and $p = 0.026$, respectively). End-test leg pain was also significantly higher among obese patients ($p = 0.045$). Furthermore, the total walking distance was significantly shorter in the obese group compared to the non-obese group ($p = 0.038$), indicating reduced functional exercise capacity.

The relationship between BMI and PFT, lung diffusion capacity, and functional exercise

capacity is presented in Table 4. A moderate and statistically significant negative correlation was

found between BMI and the 6MWT distance ($r = -0.592, p = 0.028$).

Table 1. Demographic and clinical characteristics of the groups

	Obese group (n=25)	Non-obese group (n=25)	p value
Age (years)	42.84±13.13	38.88±12.69	0.218
Gender			
Female	17 (68%)	15 (60%)	0.556
Male	8 (32%)	10 (40%)	
Body Composition			
Weight (kg)	95.08±21.33	68.53±11.21	<0.001*
Height (cm)	166.60±8.36	168.28±8.65	0.600
BMI (kg/m ²)	34.25±7.52	24.24±3.04	<0.001*
Disease duration (year)	7.60±9.48	4.84±6.01	0.434
Smoking history (pack-years)	20.85±14.47	19.07±10.29	0.903
Number of asthma attacks in the previous year	1.48±0.65	1.68±0.69	0.270
Drugs, number of users, n (%)			
Inhaled corticosteroids	15 (60%)	21 (84%)	0.269
β ₂ agonists	6 (24%)	4 (16%)	
GINA classification			
Mild intermittent	13 (52%)	14 (56%)	0.833
Mild persistent	5 (20%)	3 (12%)	
Moderate persistent	6 (24%)	6 (24%)	
Severe persistent	1 (4%)	2 (8%)	
ACT	20.17±3.78	21.25±2.72	0.930

Data are presented as mean ± standard deviation or n (%). * $p \leq 0.05$.

Abbreviations: BMI: Body mass index; cm: centimeter; kg: kilogram., GINA: Global Initiative for Asthma; ACT: Asthma Control Test

Table 2. Comparison of pulmonary function and lung diffusion capacity between the groups

	Obese group (n=25)	Non-obese group (n=25)	p value
Pulmonary function			
FVC (% predicted)	91.80±15.49	90.96±11.96	0.831
FEV ₁ (% predicted)	84.52±13.30	84.72±13.62	0.763
FEV ₁ / FVC (%)	78.35±8.06	79.01±9.20	0.808
PEF (% predicted)	75.28±15.81	72.64±17.35	0.884
FEF ₂₅₋₇₅ (% predicted)	64.80±26.09	69.92±28.69	0.515
Lung Diffusion Capacity	5.68±1.93	5.72±2.13	0.907
DLCO (% predicted)	81.68±16.66	79.04±15.61	0.554

Data are presented as mean ± standard deviation or n (%). * $p \leq 0.05$.

Abbreviations: FVC: forced vital capacity; FEV₁: forced expiratory volume in 1s; PEF: peak expiratory flow. FEF₂₅₋₇₅: forced expiratory flow between 25% and 75; DLCO: diffusing capacity for carbon monoxide.

Table 3. Comparison of 6MWT results between the groups

	Time	Obese group (n=25)	Non-obese group (n=25)	p value
HR (beats/min)	Rest	94.40±14.86	89.92±14.09	0.203
	End	116.56±22.07	111.48±18	0.197
SpO ₂ (%)	Rest	97.44±1.28	96.48±1.89	0.400
	End	95.92±4.74	96.68±2.86	0.045*
SBP (mmHg)	Rest	12±0.57	11.40±0.91	0.017*
	End	13.08±1.32	12.04±1.48	0.002*
DBP (mmHg)	Rest	8.60±1.29	8.16±1.34	0.080
	End	9.68±1.34	8.92±1.15	0.058
RR (breaths/min)	Rest	15.76±1.92	14.92±1.70	0.099
	End	21.60±3.08	19.20±2.85	0.002*
Dyspnoea (M. Borg)	Rest	0.35±0.61	0.010±0.020	0.004*
	End	2.17±1.65	0.98±0.95	0.010*
Fatigue (M. Borg)	Rest	0.27±0.65	0.01±0.01	0.018*
	End	2.33±1.93	1.15±1.45	0.026*
Leg pain (VAS)	Rest	0.01±0.01	0	0.153
	End	1.12±0.08	0.42±0.94	0.045*
6MWT distance (m)		414.16±71.81	459.52±67.86	0.038*

Data are presented as mean ± standard deviation or n (%). * $p \leq 0.05$.

Abbreviations: HR: heart rate; min: minute; SpO₂: Peripheral Capillary Oxygen Saturation; SBP: systolic blood pressure; DBP: diastolic blood pressure; RR: respiratory rate; M. Borg: Modified Borg Scale; VAS: Visual Analogue Scale; 6MWT: 6 minute walk test; m: meter.

Table 4. Correlation of the BMI with pulmonary function, lung diffusion capacity, and functional exercise capacity

	BMI	
	r	p
Pulmonary function		
FVC (% predicted)	-0.064	0.659
FEV ₁ (% predicted)	-0.077	0.594
FEV ₁ /FVC (%)	-0.098	0.497
PEF (% predicted)	0.057	0.692
FEF ₂₅₋₇₅ (% predicted)	-0.156	0.279
Lung diffusion capacity		
DLCO (% predicted)	0.199	0.165
Functional exercise capacity		
6MWT distance (m)	-0.592	0.028*

Abbreviations: BMI: body mass index; FVC: forced vital capacity; FEV₁: forced expiratory volume in 1s; PEF: peak expiratory flow. FEF₂₅₋₇₅: forced expiratory flow between 25% and 75; DLCO: diffusing capacity for carbon monoxide; 6MWT: 6 minute walk test; m: meter. * $p \leq 0.05$.

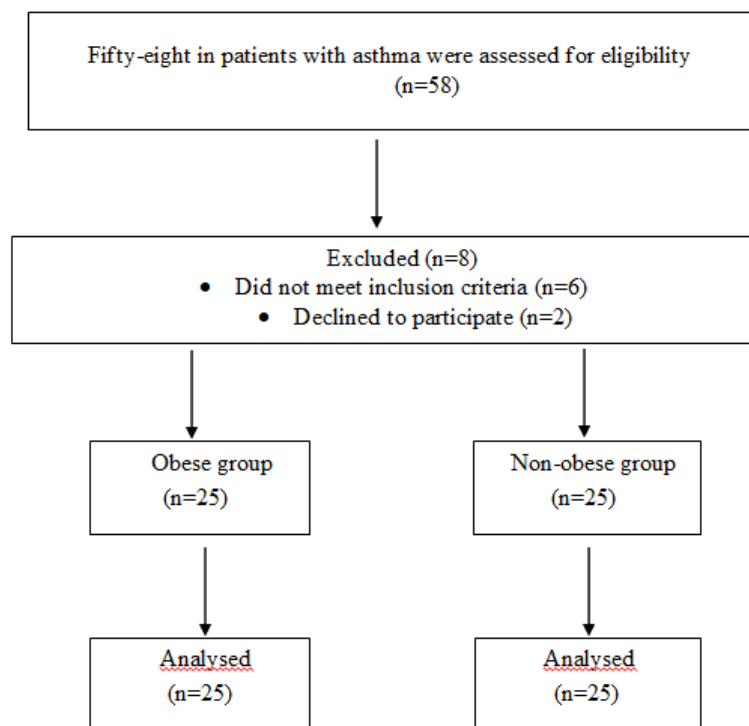


Figure 1. Flow diagram of study design.

4. Discussion

This study set out with the aim of investigating the impact of obesity on pulmonary function, lung diffusion capacity, functional exercise capacity, and asthma control in adults with asthma, involving 50 participants aged 18–65 years, including 25 obese and 25 non-obese individuals. Significant differences—favoring the non-obese group—were identified between the groups in several 6MWT variables. Specifically, end-test SpO₂ levels were significantly lower in obese individuals compared to non-obese individuals. Both baseline and end-test systolic blood pressure (SBP) values were significantly higher in the obese group. The respiratory rate at the end of the test, as well as perceived dyspnea and fatigue at both the beginning and end of the test, were also significantly elevated in obese participants. In addition, leg pain at the end of the test was greater, and total walking distance was shorter in the obese group. These findings collectively indicate impaired functional performance and increased perceived exertion in obese asthma patients.

Obesity is known to negatively affect the respiratory system, primarily through the mechanical compression resulting from increased adipose tissue in the thoracic and abdominal regions. This mechanical burden can impair the mobility of the

chest wall and diaphragm, subsequently reducing lung volumes and respiratory capacity (3). In patients with asthma, excess body weight may further compromise diaphragmatic function. Previous studies have demonstrated that obesity leads to both a reduction in diaphragm muscle fiber number and size, as well as increased pulmonary blood flow. Consequently, diaphragmatic activity is elevated in obese individuals compared to those of normal weight, reflecting a compensatory mechanism due to inefficient ventilation (18). Another mechanism through which obesity may influence asthma involves changes in airway smooth muscle. The increase in fat mass may alter respiratory mechanics and impair contractile properties of airway muscles, leading to disrupted actin-myosin interactions, weakened respiratory muscle strength, and compromised pulmonary function (19).

However, the literature presents conflicting evidence on whether obesity further impairs pulmonary function in individuals with asthma. Several studies have reported a negative correlation between increased BMI and parameters such as functional residual capacity (FRC) and expiratory reserve volume (ERV) in patients with asthma (20). Rastogi et al. observed that normal-weight individuals with

asthma had significantly higher predicted values of FEV₁, FEV₁/FVC, and FEF₂₅₋₇₅ compared to their obese counterparts (21). Similarly, studies have shown negative correlations between BMI and predicted FVC and FEV₁/FVC values in mildly and moderately obese individuals, reinforcing these findings (22). Schachter et al., in a study involving 1971 healthy participants, found that obese individuals had significantly lower FEV₁ and FVC values compared to those with normal BMI (23). These results were further supported by Steier et al., who reported statistically significant differences in FEV₁, FVC, and FEV₁/FVC between obese and non-obese participants (24). In contrast, some studies have reported no significant changes in pulmonary function following weight loss in obese asthmatic individuals (25), and others found no difference in PFT parameters between obese and non-obese asthmatic patients (26). Ghabashi et al. reported no correlation between BMI and any PFT parameter (27). In our study, consistent with these findings, no significant differences were found in any pulmonary function parameters between the obese and non-obese asthmatic groups. Pulmonary function decline in patients with asthma is often associated with disease severity, symptom burden, and poor asthma control (28). Grzelewska-Rzymowska et al. reported that well-controlled asthma was associated with FEV₁ values remaining within the expected range, whereas disease duration longer than 20 years, particularly in cases of moderate persistent asthma, was linked to a decline in pulmonary function (29). In our study, consistent with these findings, no significant differences were found in any pulmonary function parameters between the obese and non-obese asthmatic groups. This result may be explained by the fact that the majority of participants in both the obese (52%) and non-obese (56%) groups were classified as having mild intermittent asthma. Additionally, similar ACT scores and comparable numbers of asthma exacerbations in the past year between the two groups may have contributed to the absence of statistically significant differences. In addition to similar asthma severity and control scores, no significant differences were observed between the obese and non-obese groups in smoking history, number of asthma attacks in the previous year, or medication use (including inhaled corticosteroids and β_2 agonists). This clinical similarity likely contributed to the absence of differences in pulmonary function and asthma control. Supporting these findings, previous studies have emphasized that asthma-related outcomes such as lung function and symptom control are more strongly influenced by disease severity and treatment adherence than by BMI alone, particularly in populations with similar medication regimens and

exacerbation history (30,31). Moreover, asthma severity classification based on GINA criteria did not differ significantly between the obese and non-obese groups, as shown in Table 1. This clinical similarity likely contributed to the comparable pulmonary function outcomes observed in our study.

DLCO plays a significant role in the early diagnosis of various pulmonary diseases, including emphysema, cystic fibrosis, pulmonary embolism, and COPD, where it is typically reduced. Although its primary use in asthma is limited, DLCO measurements can be particularly helpful in differentiating asthma from COPD (32). In COPD patients, especially those with emphysema, DLCO values are often decreased, whereas in asthma, values are generally normal or even increased in early stages. This increase has been linked to increased pulmonary blood flow and elevated cardiac output, commonly seen in asthma. However, in elderly patients with asthma or in severe disease, ventilation-perfusion mismatching may lead to decreased DLCO values (33). While some clinical studies have reported increased DLCO in asthma and obesity, others have presented contradictory results (34). While DLCO is typically decreased in patients with severe obstruction compared to those with mild obstruction, other studies found no significant differences in DLCO across groups with varying degrees of obstruction (35). Viegi et al. observed elevated DLCO values in individuals with FEV₁/FVC ratios between 65% and 75%, suggesting only a weak association between DLCO and the degree of airway obstruction (36). Schultz et al. reported decreased DLCO in asthmatic patients with FEV₁ below 40%, supporting the notion that DLCO remains largely unaffected in mild to moderate obstruction (37). Similarly, in a study by Kanat et al., which included 91 asthmatic patients and 47 healthy controls without any comorbid conditions affecting DLCO or smoking history, no statistically significant difference in DLCO was found between the groups (38). In line with the existing literature, our study also found no significant difference in DLCO between the obese and non-obese asthma groups. A possible explanation for this result is that the majority of patients in both groups were classified as having mild intermittent asthma and exhibited only mild airway obstruction (mean FEV₁ % predicted: 84.52 in the obese group vs. 84.72 in the non-obese group), suggesting that DLCO remains stable in cases of mild disease severity and mild airflow obstruction.

Previous literature has demonstrated that individuals with asthma generally exhibit lower functional exercise capacity compared to healthy controls. This

limitation is likely due to exercise-induced bronchospasm, heightened dyspnea perception, medication side effects, asthma-related symptoms, kinesiophobia, and subsequent physical inactivity (39). In our study, functional exercise capacity was assessed using the 6MWT, a validated and routinely applied field test in asthma populations (40). The mean 6MWT distance was 414.14 m in the obese group and 459.52 m in the non-obese group. Notably, the literature reports an average 6MWT distance of approximately 627.8 m in healthy adults aged 18–70 years (41). Statistical analysis confirmed that both asthma groups demonstrated significantly lower than normative values 6MWT distances compared to healthy norms (One-sample t-test; $p < 0.001$). Recent findings suggest that overweight and obese adults are more likely to identify exercise as an asthma trigger and therefore engage in avoidant behaviors more frequently than normal-weight individuals (42). A study reported a stepwise decline in 6MWT distances according to weight classification: 613.4 ± 45.9 m in underweight, 532.3 ± 62.7 m in obese, and 462.8 ± 68.2 m in morbidly obese participants. These differences were statistically significant between underweight individuals and the other two groups (43). In line with these findings, our study revealed a statistically significant difference in 6MWT distances between obese and non-obese asthma groups. Supporting prior studies, our results suggest that BMI may be a primary determinant of reduced functional capacity in asthmatic individuals (44, 45). For instance, Santuz et al. found no association between BMI and asthma severity or FEV₁, but did identify a correlation between BMI and functional capacity in obese asthma patients (46). A lack of significant association between PFT and 6MWT performance in our study may be explained by factors such as the low number of asthma exacerbations in the past year and the classification of patients within the partially controlled asthma category based on ACT scores. Additionally, the homogeneity in medication uses and clinical characteristics between groups enabled a more accurate assessment of the independent effect of obesity on functional capacity, separate from PFT parameters.

In the context of asthma, obesity is associated with a complex, multifactorial phenotype involving diverse pathophysiological mechanisms and subphenotypes (47). Numerous physiological alterations linked to obesity can negatively influence cardiopulmonary responses during physical activity. Studies consistently report that obese individuals exhibit greater increases in heart rate and blood pressure

during exercise, as well as heightened perceptions of fatigue and dyspnea, contributing to lower functional capacity compared to non-obese individuals (48). One study reported a positive correlation between BMI and both pre- and post-test SBP and HR during the 6MWT. Individuals with the highest BMI experienced more pronounced fatigue and dyspnea and exhibited resting heart rates approximately 12% higher than those with lower BMI. These participants also showed greater increases in perceived exertion and fatigue (49).

Our findings further confirmed that resting HR values were significantly and consistently elevated in the obese asthma group relative to their non-obese counterparts. Lindgren et al. further emphasized the prognostic implications of elevated resting HR and SBP, suggesting these variables, when coupled with high BMI and low cardiorespiratory fitness, may indicate an increased risk of future heart failure and all-cause mortality (50). The interaction between higher BMI and reduced 6MWT performance has been reinforced by several studies, which attribute this reduction to factors such as a sedentary lifestyle, and increased perception of fatigue, dyspnea, and pain during walking (51). Similar physiological limitations have also been reported in studies involving obese patients with chronic obstructive pulmonary disease (COPD), indicating shorter walking distances compared to patients with normal BMI during exercise testing (52). Further supporting these findings, Seres et al. (53) reported that decreased exercise capacity in morbidly obese individuals was associated with increased oxygen consumption, heart rate, systolic blood pressure, and minute ventilation. They concluded that the increased energy demand required to mobilize excess body mass may underlie the reduced physical performance in this population (53). Likewise, another study demonstrated that even well-controlled asthmatic individuals with obesity had lower functional capacity and greater leg fatigue compared to normal-weight asthmatics, likely due to the elevated metabolic demand required to move heavier limbs during activity (54). Although research on oxygen saturation changes during exercise in obese asthmatic individuals is limited, studies in healthy populations provide relevant insight. For instance, recent research in healthy adults reported that those with higher BMI experienced greater declines in oxygen saturation following the 6MWT compared to those with normal BMI (55). Additionally, a study involving children aged 5–9 years showed that overweight participants had higher SBP and lower oxygen saturation both at

rest and end-6MWT compared to their normal-weight counterparts (56). Consistent with the literature, the present study revealed statistically significant differences between obese and non-obese asthma groups in multiple post-exercise physiological parameters. These included lower end-of-test SpO₂, higher pre- and post-test SBP, elevated end-test RR, as well as notably greater perceived dyspnea, more intense leg fatigue, and significantly increased leg pain in obese participants. These findings support prior research indicating that obesity contributes to an altered physiological and perceptual response to exercise, ultimately impairing functional capacity in individuals with asthma.

An increasing number of studies suggest a potential link between obesity and asthma, although the exact nature of this relationship remains unclear. Several studies have demonstrated that higher BMI is linked to poorer asthma control, increased symptom burden, and greater medication use, independent of asthma severity (57-59). Additionally, Boulet and Franssen found reduced responsiveness to inhaled corticosteroids in obese asthmatic patients compared to their non-obese counterparts (60). In contrast, our study found no significant difference in asthma control between obese and non-obese groups. One likely explanation for this discrepancy is the lower prevalence of class II and III obesity in our study population. In addition to comparable ACT scores, both groups also shared similar asthma severity profiles, with no statistically significant difference in GINA-based asthma staging. Supporting our findings, multiple studies have also reported no association between obesity and asthma control, severity, or symptom expression (61-63). These include both cross-sectional and cohort studies assessing symptom frequency, medication use, exacerbations, and pulmonary function. One possible explanation is that similar clinical characteristics across BMI groups, such as disease severity, treatment regimen, and exacerbation history, may diminish observable differences. These factors likely minimized confounding effects and enabled a clearer interpretation of obesity's impact on asthma control in a relatively homogeneous sample.

Our study has several limitations. First, body composition was assessed solely through height and weight measurements, without evaluating more specific parameters such as basal metabolic rate, body fat percentage, or fat mass. This limited the exploration of more nuanced relationships between obesity and asthma-related outcomes. Second, although previous studies have demonstrated that physical activity level is a significant determinant of pulmonary function, functional capacity, and asthma control, we did not assess participants' physical activity levels using either subjective or objective methods. The absence of this data may have prevented us from accounting for an important confounding variable that could influence the study outcomes. Subsequent research could incorporate body composition assessments, such as BIA or DEXA, to better distinguish fat and lean mass. Similarly, the use of wearable devices or accelerometers may allow for objective monitoring of physical activity and its relationship to asthma outcomes.

5. Conclusion

This study demonstrates that even in patients with mild asthma, functional capacity is significantly reduced compared to healthy individuals, and the presence of obesity further exacerbates this impairment. Given the increasing prevalence of obesity worldwide, these findings underscore the importance of early functional assessment and the integration of structured exercise and weight management into asthma rehabilitation. Although current guidelines emphasize the role of exercise in asthma care, our results highlight the need for a more comprehensive approach that addresses both respiratory limitations and obesity-related physical constraints. We believe this study raises clinical awareness about the dual burden of asthma and obesity and supports the development of tailored rehabilitation strategies. Further research is warranted to explore how varying degrees of obesity influence respiratory health and asthma control, ideally through studies with larger and more diverse populations to enhance the generalizability of findings and deepen our understanding of this complex interaction.

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