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Research article

Spirometry profiles of overweight and obese individuals with unexplained dyspnea in Saudi Arabia

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ABSTRACT

ARTICLE INFO

Kevwords: Background: Obesity is a common cause of dyspnea. However, the impacts of obesity on Obesity spirometry parameters, particularly among individuals with unexplained dyspnea, have not been Respiratory symptoms well investigated. Lung function Objectives: This study aimed to explore the prevalence of overweight and different classes of Overweight obesity and their effect on spirometry parameters among subjects with unexplained dyspnea in Spirometry Saudi Arabia. Dyspnea Methods: We conducted a retrospective electronic medical record review of individuals with Breathlessness Unexplained dyspnea unexplained dyspnea who visited our pulmonary clinic between January 2016 and December 2022 and assessed the association of body mass index (BMI) with spirometry parameters. After we classified the subjects based on their BMI values, we determined the impacts of increased BMI on spirometry parameters. *Results*: The sample included a total of 978 subjects with unexplained dyspnea. The prevalence of overweight and obesity among our study sample was high (33 % and 47 %, respectively). All spirometry parameters: forced vital capacity (FVC) L, forced expiratory volume in 1 s (FEV1) L, forced expiratory flow at 25-75 % (FEF 25-75 %) L/sec and peak expiratory flow (PEF) L/sec were significantly lower in obese individuals with dyspnea compared to normal weight subjects. In addition, our findings showed a negative correlation between BMI and FVC, FEV1, FEF 25-75 %, and PEF. Conclusion: The high prevalence of obesity and overweight and the impairment of lung function because of high body weight among subjects with dyspnea point to the need for routine assessment and the evaluation of nutritional status in primary health care facilities for early intervention.

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1. Introduction

Dyspnea, also known as breathlessness or shortness of breath, is a subjective experience of difficulty breathing [1]. Dyspnea is a common debilitating symptom in adults and can cause physical and psychological distress [2], affecting functional capacity and health-related quality of life. The prevalence of dyspnea in the general population varies from 10 % to 25 % [3,4], which increases among individuals with chronic health conditions [1]. Although the exact mechanisms underlying sensations of dyspnea are not fully understood, it can result from a wide spectrum of underlying diseases, including respiratory and cardiovascular conditions [5,6]. Other conditions that may contribute to dyspnea include obesity [7].

Obesity is one of the most common metabolic diseases in the world, characterised by abnormal or excessive accumulation of fat. The prevalence of obesity has been increasing for several decades, ranging from 12.9 % to 41.9 % among general populations [8–10], and it is estimated that 48.9 % and 24.2 % of the population in the United States will be obese and severely obese, respectively, by 2030 [11]. In patients with asthma, we have recently reported that the overweight and obesity prevalence together is 75 % [12]. These observations suggest that routine assessment of body weight in the primary care clinic is needed as a strategy for obesity prevention to reduce risk-associated obesity. Currently, different methods used to determine abnormal body weight including but not limited to body mass index (BMI), waist circumference and hydrostatic weighing. Among these methods, BMI, calculated by dividing weight by the square of height, is commonly used to determine overweight and obesity as a BMI of between 25 and 29.9 kg/m² and a BMI of 30 kg/m² and greater, respectively [13,14]. The WHO and CDC subdivide obesity into three categories: mild obesity (30–34.9 kg/m²), moderate obesity (35–39.9 kg/m²), and morbid obesity (40 kg/m² or above).

Obesity is a known risk factor for several non-communicable diseases (e.g., respiratory disease) [15] and can impair respiratory function and ultimately worsen health-related quality of life. Although overweight and obesity prevalence and their effect on lung function in people with breathlessness have not been well explored, we have previously shown that obesity is prevalent and can reduce lung function among patients with asthma and that increased body weight is associated with reduced spirometry parameters: forced vital capacity (FVC), forced expiratory volume in 1 s (FEV₁), forced expiratory flow at 25–75 % (FEF $_{25-75}$ %), and peak expiratory flow (PEF). These observations are supported by the fact that reduced lung volumes have been reported in healthy obese Black Americans who did not have evidence of respiratory diseases [16] and suggest a key role of obesity in the impairment of lung function.

Obesity is highly prevalent among general populations and is strongly associated with respiratory symptoms. Although the severity of dyspnea appears to increase with increasing airflow limitation, it is nevertheless unclear whether obesity is associated with impairment in airflow limitation in individuals with respiratory symptoms who showed no evidence of chronic respiratory or cardiovascular conditions. Therefore, our study aimed to determine the prevalence of overweight and all classes of obesity and examine the effects of overweight and obesity on spirometry parameters (FVC, FEV₁, FEF_{25 %-75 %} and PEF) among individuals with unexplained dyspnea in Saudi Arabia.

2. Materials and methods

2.1. Study design and setting

This is a retrospective study of spirometry profiles of overweight and obese individuals with unexplained dyspnea. The study was conducted at King Abdulaziz University Hospital, a large teaching hospital located in Jeddah, Saudi Arabia.

2.2. Study population

The medical records of individuals without known chronic respiratory or cardiovascular conditions who presented with respiratory symptoms, particularly dyspnea, in our pulmonary clinic during their scheduled visit. We identified a total of 1550 subjects who visited the clinic between the 1st of January 2016 and the 31st of December 2022. We collected demographic data (e.g., BMI, age, and sex), spirometry results (FVC, FEV₁, FEF_{25 %-75 %} and PEF) and medical conditions. A multidisciplinary team for subjects with respiratory symptoms often performed and reviewed spirometry tests and other related investigations to eliminate the presence of potential medical conditions. Thus, the final analysis of the current study only included patients who had shortness of breath without any other known medical conditions (e.g., asthma) either before or after the pulmonary function test. In addition, only subjects who were aged 18 years or older, had a BMI $\geq 18.5 \text{ kg/m}^2$ and presented acceptable and reproducible spirometry results were included in the analysis. Anonymity and confidentiality were maintained. Patient identifiers (e.g., dates of birth, addresses and names) were not collected. We obtained ethical approval before the start of the study from the Unit of Biomedical Ethics Research Committee at the Faculty of Medicine at King Abdulaziz University, Saudi Arabia (No 448-22). Given that this is a retrospective study (a study of medical records), the ethics committee waived the need for patient consent to participate or have the data included in this manuscript be published.

2.3. Spirometry parameters

In the current study, we only included spirometry results that were carried out in agreement with the most recent international guidelines [17]. An experienced pulmonary function technician conducted the tests in a pulmonary clinic using the Vmax® Encore pulmonary function testing system (SensorMedics Inc., Anaheim, California, United States of America). Although a respiratory

consultant evaluated and validated the pulmonary function tests regularly in the clinic, we manually reviewed all the spirometry tests used in the current study (A.A.A and R.A.S). Spirometry tests without acceptable and reproducible results were excluded. Only the most recent spirometry parameters (FVC, FEV₁, PEF and FEF_{25-75 %}) results were collected and reported in the present study if the patient had undergone several spirometry tests between the 1st of January 2016 and the 31st of December 2022. To derive z-scores for each subject, we applied Global Lung Function Initiative (GLI) 2012 reference values using the "other/mixed" classification [18].

2.4. BMI

We calculated the BMI of individuals included in the present study from the measured heights and weights using a medical scale. The measurements of heights and weights were routinely performed before the spirometry tests were conducted and while the subjects were standing in an upright position, barefoot and wearing light clothing. In the current study, we defined healthy weight, overweight and all classes of obesity using BMI following WHO and CDC recommendations. Individuals with a BMI of between 18.5 and 24.9 kg/m² and between 25 and 29.9 kg/m² were identified as having a healthy weight and being overweight, respectively. Subjects with a BMI of 30 kg/m² or above were classified as obese. Furthermore, those with obesity were subdivided into three different classes: mild obesity (30–34.9 kg/m²), moderate obesity (35–39.9 kg/m²), and morbid obesity (40 kg/m² or above).

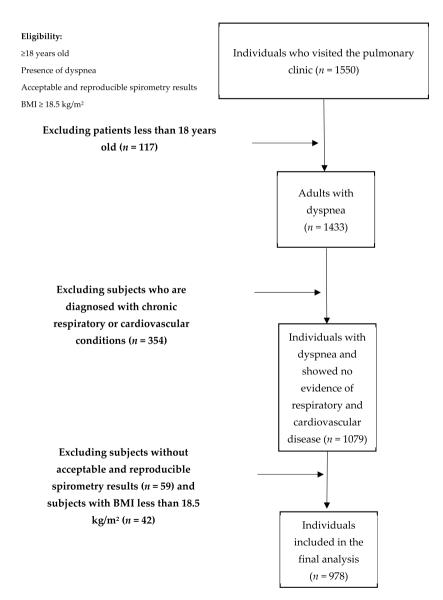


Fig. 1. Flow chart of the study.

2.5. Statistical analysis

Data were analysed using GraphPad Prism (version 9). The categorical variables were presented in percentages and frequencies, while arithmetic means \pm standard error of the mean (SEM) were used for continuous variables. A one-way analysis of variance (ANOVA) followed by a *t*-test was performed to determine whether there are differences between healthy weight and specific groups of obesity or overweight. Pearson's correlation coefficient and linear regression models were performed to assess the correlation of BMI with spirometry parameters and linear regression models to estimate the independent relationship between BMI and spirometry parameters, respectively. *p* < 0.05 was regarded as statistically significant.

3. Results

3.1. Subject characteristics

A total of 1550 individuals were identified. We excluded patients who were aged less than 18 years old (n = 117), patients who were diagnosed with chronic respiratory or cardiovascular conditions (n = 354), those who did not have acceptable and reproducible lung function results (n = 59) and individuals with a BMI less than 18.5 kg/m² (n = 42). As a result, 978 subjects met the inclusion criteria of the current study and, thus, were included in the analysis (Fig. 1).

The study population was divided into five groups based on their BMI as follows: normal weight (n = 201; 20 %), overweight (n = 321; 33 %), mild obesity (n = 214; 22 %), moderate obesity (n = 129; 14 %) and morbid obesity (n = 113; 11 %) (Table 1). We found that the mean age of subjects with unexplained dyspnea included in the analysis was 52 years. The proportion of females (55 %) was greater than males and the mean BMI was greater in females than in males (31.4 vs 29.8; p < 0.01).

3.2. Correlations between BMI and spirometry parameters

The association between BMI and spirometry measures was assessed using Pearson's correlation tests. As shown in Fig. 2A–D, our results revealed that BMI was negatively associated with FVC (r = -0.20, p < 0.0001) and PEF (r = -0.13, p < 0.01). Similarly, we also found a negative association between BMI and FEF $_{25-75\%}$ (r = -0.14, p < 0.001) as well as FEV₁ (r = -0.20, p < 0.0001) (Fig. 2A–D).

3.3. The effect of increased BMI on spirometry measures

Next, subjects were subdivided into five groups based on their BMI to explore whether spirometry parameters were affected by increased BMI (overweight and various levels of obesity) among those with unexplained dyspnea. Based on weight class, a one-way ANOVA test showed a significant difference among means of the tested spirometry parameters: FVC L (p < 0.0001), FEV₁ L (p < 0.0001), FEF _{25–75 %} L/sec (p < 0.001), and PEF L/sec (p < 0.001) (Fig. 3A–D). We next were interested in finding out whether the means of each spirometry parameter differed among subjects in different weight categories. Unpaired t-tests showed that the mean FVC did not differ significantly in overweight individuals compared with those of normal body weight (2.98 ± 0.07 L and 2.85 ± 0.06 L, respectively) ($p \ge 0.05$) (Fig. 3A). Furthermore, the mean FVC was reduced in subjects with mild (2.73 ± 0.07 L), moderate (2.57 ± 0.09 L) and severe (2.25 ± 0.07 L) obesity compared with normal body weight subjects (p < 0.05, p < 0.001, and p < 0.0001, respectively). A significant reduction of mean FVC was observed in subjects who were moderately and severely obese compared with overweight subjects (p < 0.05 and p < 0.0001, respectively) (Fig. 3A).

In contrast to the findings regarding FVC, the mean FEV₁ was significantly reduced in overweight subjects (2.26 ± 0.05 L/s, p < 0.05) compared with normal body weight (2.42 ± 0.06 L) (Fig. 3B). Similarly, our results showed a significant decrease in the mean FEV₁ in subjects with mild, moderate, and morbid obesity (2.18 ± 0.06 L, p < 0.01, 2.07 ± 0.07 L, p < 0.001, and 1.82 ± 0.07 L p < 0.0001, respectively) compared with normal body weight. As shown in Fig. 3B, the mean FEV₁ of individuals with moderate and morbid (but not mild) obesity was reduced (p < 0.05 and p < 0.0001, respectively) compared with those who were overweight.

The mean FEF $_{25-75\%}$ of subjects with normal body weight was 2.42 ± 0.06 L. Similar to the FEV₁ findings, a reduction of the mean FEF $_{25-75\%}$ was observed in the overweight subjects (2.40 ± 0.06 L/s, p < 0.05) as well as those with all the different levels of obesity: mild (2.34 ± 0.08 L, p < 0.05), moderate (2.24 ± 0.09 L, p < 0.01) and severe (2.04 ± 0.10 L, p < 0.001) as compared with normal

Table 1	
Subjects'	characteristics ($n = 978$).

Variable	Normal weight ($n = 201$)	Overweight ($n = 321$)	Mild Obesity ($n = 214$)	Moderate Obesity ($n = 129$)	Morbid Obesity ($n = 113$)
Age (years)	50 ± 1.2	51 ± 0.93	53 ± 0.96	54 ± 1.3	52 ± 1.4
Female, <i>n</i> (%)	116 (58 %)	138 (43 %)	127 (59 %)	83 (64 %)	76 (67 %)
Height (cm)	161 ± 0.006	163 ± 0.005	161 ± 0.007	159 ± 0.009	158 ± 0.01
Weight (kg)	58 ± 0.5	73 ± 0.49	84 ± 0.78	95 ± 1.2	113 ± 1.8
BMI (kg/m ²)	22 ± 0.12	27 ± 0.07	32 ± 0.09	37 ± 0.12	45 ± 0.50

Data are expressed as mean \pm SEM or numbers (%). Normal weight, overweight, and obesity are classified based on BMI according to the classification of the WHO: normal weight 18.5–24.9 kg/m², overweight 25–29.9 kg/m², mild obesity 30–34.9 kg/m², moderate obesity 35–39.9 kg/m², and morbid obesity 40 kg/m² and greater.

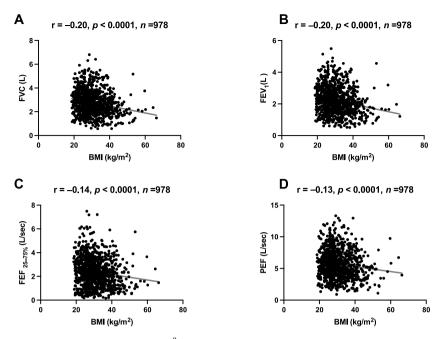


Fig. 2. The correlation between body mass index (BMI) kg/m^2 with forced vital capacity (FVC) L (A), forced expiratory volume in 1 s (FEV₁) L (B), forced expiratory flow at 25–75 % (FEF _{25–75 %}) L/sec (C), and peak expiratory flow (PEF) L/sec (D) among individuals with unexplained dyspnea.

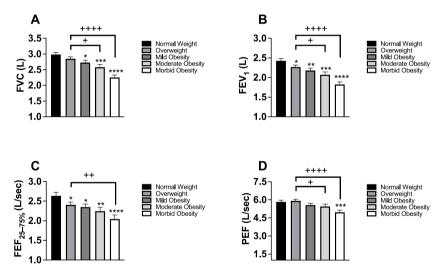


Fig. 3. Impact of overweight and obesity on all spirometry parameters among individuals with unexplained dyspnea. Subjects were subdivided into five different groups based on their body mass index (BMI): normal weight (BMI 18.5–24.9 kg/m², n = 201), overweight (BMI 25–29.9 kg/m², n = 321), mild obesity (BMI 30–34.9 kg/m², n = 214), moderate obesity (BMI 35–39.9 kg/m², n = 129) and morbid obesity (BMI 40 kg/m² or greater, n = 113). Being overweight and all distinct levels of obesity impact forced vital capacity (FVC) L (A), forced expiratory volume in 1 s (FEV₁) L, (B), forced expiratory flow at 25–75 % (FEF _{25–75 %}) L/sec (C), and peak expiratory flow (PEF) L/sec (D). Each data point represents mean \pm SEM. *p < 0.05, **p < 0.01, ****p < 0.001, ****p < 0.0001 compared with normal weight; +p < 0.05, ++p < 0.01, ++++p < 0.0001 compared with overweight.

body weight (Fig. 3C). Although there was no significant difference among the mean FEF $_{25-75}$ % between subjects with mild and moderate obesity and those overweight, the mean FEF $_{25-75}$ % of morbidly obese subjects was reduced compared with overweight subjects (p < 0.01) (Fig. 3C). Furthermore, it was interesting to find that overweight (5.92 ± 0.12 L/s) and mild and moderate obesity (5.55 ± 0.15 L/s and 5.44 ± 0.20 L/s, respectively) did not affect the mean PEF in subjects with dyspnea compared with normal body weight (5.82 ± 0.14 L/s) (Fig. 3D). In addition, the mean PEF was only reduced in subjects with morbid obesity compared with normal body weight subjects (p < 0.001) (Fig. 3D). Compared with overweight subjects, the mean PEF was also reduced in those with moderate (p < 0.05) and severe (but not mild) obesity (p < 0.0001) (Fig. 3D).

The z-score comparisons were also conducted to further confirm the apparent differences in main spirometry parameters. The mean FEV₁, FVC, FEV₁/FVC ratio, and FEF _{25–75} % z-scores for adults with healthy weight were -0.68 ± 0.14 , -0.79 ± 0.13 , 0.03 ± 1.12 , and -0.28 ± 0.10 , respectively (Table 2). We then compared these z-scores in people with normal weight with z-scores of spirometry parameters in people with overweight and various levels of obesity. Like the comparison on the absolute values, z-scores of spirometry parameters worsened in subjects with overweight or obesity compared with normal weight, except FEV₁/FVC ratio where no significant differences were seen between normal weight and overweight/obesity (Table 2). Further comparisons of z-score and % of predicted values can be found in the supplementary material (Tables S1–S8).

To assess the independent association between BMI and spirometry parameters, a multiple linear regression model was performed. As shown in Table 3, BMI was associated with FVC (L), FEV1 (L), FEF $_{25-75}$ %, and PEF (β : 0.03; 95 % CI: 0.04 to -0.02; p < 0.001), (β : 0.2; 95 % CI: 0.31 to -0.016; p < 0.001), (β : 0.02; 95 % CI: 0.03 to -0.01; p < 0.001) and (β : 0.04; 95 % CI: 0.06 to -0.01; p < 0.01), respectively. The associations remained significant even after adjusting for age, gender, and smoking.

4. Discussion

To the best of our knowledge, the present study was the first to explore the prevalence of overweight and all obesity classes and assess the effect of increased body weight on lung function in breathless subjects who showed no evidence of chronic respiratory or cardiovascular conditions in Saudi Arabia. Our main findings showed that the combined prevalence of overweight and obesity in our study population was 80 %. More importantly, we found that all spirometry parameters were negatively associated with BMI and were significantly reduced in subjects with obesity, particularly in those who are morbidly obese, compared with healthy weight and/or overweight. These findings strongly suggest that obesity, particularly morbid obesity, can lead to impairment in lung function and may also provide potential explanations for the presence of dyspnea in individuals with obesity. In addition, our observations point to the need for routine assessment and the evaluation of nutritional status in primary healthcare facilities for early intervention to reduce the severity of lung function impairment.

Obesity is a major public health concern and its prevalence in high-income countries has tripled since 1975 [13]. The prevalence of self-reported increased body weight (obesity) in the general population of Saudi Arabia [8], the United States [9], and Europe [10] is 24.7 %, 41.9 % and 12.9 %, respectively. Recently, obesity has been shown to be an independent risk factor for dyspnea [6]. Although the prevalence of overweight and obesity has not been well assessed in individuals with respiratory symptoms, we have demonstrated, for the first time, that overweight and obesity are highly prevalent among people with dyspnea without evidence of chronic respiratory or cardiovascular conditions in Saudi Arabia (33 % and 47 %, respectively). This observation is consistent with our previous findings showing overweight (31 %) and obesity (46 %) to be prevalent in Saudi subjects with asthma [12]. Elsewhere, it has been reported that the prevalence of overweight and obesity is 36.7 % and 23.8 %, respectively, in Australian community-dwelling adults with dyspnea, including healthy adults and patients with pulmonary conditions [19]. The disparity in the obesity proportion between the two studies could be attributed to the differences in the target study populations and their geographical locations. Another factor that may contribute to the discrepancy is that the calculated BMI in their study was based on self-reported heights and weights [19], whereas in the current study, these were measured using a medical scale in the clinic under the supervision of medical staff. The fact that the current study found that overweight and obesity to be highly prevalent among individuals with dyspnea (80 %) compared with the general population with and without respiratory symptoms (60 %) [19] suggests an association between increased body weight and

Table 2	
Spirometry measures of the included subjects ($n = 978$)).

Variable	Normal weight ($n = 201$)	Overweight ($n = 321$)	Mild Obesity ($n = 214$)	Moderate Obesity ($n = 129$)	Morbid Obesity ($n = 113$)
FEV1					
z-score	-0.68 ± 0.14	$-1.35 \pm 0.07^{*}$	$-1.23 \pm 0.09^{*}$	$-1.33 \pm 0.14^{*}$	$-1.94\pm0.11^{*+\&\#}$
% predicted	90.42 ± 2.09	$79.76 \pm 1.12^{*}$	$81.63 \pm 1.45^{*}$	$80.25 \pm 1.69^{*}$	$71.66 \pm 1.70^{*}$
FVC					
z-score	-0.79 ± 0.13	$-1.34 \pm 0.08^{*}$	$-1.29 \pm 0.10^{*}$	$-1.41 \pm 0.18^{*}$	$-2.11\pm0.12^{*^{+\&\#}}$
% predicted	90.70 ± 1.83	$81.87 \pm 1.16^{*}$	$82.81 \pm 1.39^{*}$	$80.91 \pm 1.58^{*}$	$72.53 \pm 1.65^{*^{+\&\#}}$
FEV ₁ /FVC ratio)				
z-score	0.03 ± 1.12	-0.18 ± 0.07	-0.10 ± 0.09	-0.08 ± 0.19	-0.07 ± 0.10
%	81.27 ± 0.82	79.78 ± 0.52	80.06 ± 0.61	80.33 ± 0.78	80.65 ± 0.73
FEF25-75 %					
z-score	-0.28 ± 0.10	-0.57 ± 0.06	-0.42 ± 0.08	-0.46 ± 0.15	$-0.77\pm0.11^*$
% predicted	99.62 ± 3.70	$86.29 \pm 2.11^*$	91.12 ± 2.73	$82.09 \pm 3.55^{*}$	$79.37\pm3.13^{\ast}$

Data are expressed as mean \pm SEM. In the groups' comparisons, the significance level for adjusted p value was set at 0.05.

Normal weight, overweight, and obesity are classified based on body mass index (BMI): normal weight 18.5–24.9 kg/m², overweight 25–29.9 kg/m², mild obesity 30–34.9 kg/m², moderate obesity 35–39.9 kg/m², and morbid obesity 40 kg/m² and greater. FEV₁: forced expiratory volume in 1 s. FVC: Forced vital capacity. FEF 25–75 %: forced expiratory flow at 25–75 %.

*Significantly different from normal weight.

+Significantly different from overweight.

&Significantly different from mild obesity.

#Significantly different from moderate obesity.

Table 3

Linear regression	on analysis of	f the associations	of BMI with	spirometry r	parameters.

Independent variable	BMI					
	β (95 % CI)	P-value	^a Adjusted β (95 % CI)	P-value		
FVC L	-0.03 (-0.04 to -0.02)	< 0.001	-0.016 (-0.026 to 0.007)	< 0.001		
FEV _{1 L}	-0.2 (-0.31 to -0.016)	< 0.001	-0.014 (-0.02 to -0.006)	< 0.001		
FEF 25-75 % 1/sec	-0.02 (-0.03 to -0.01)	< 0.001	-0.01 (-0.02 to -0.001)	0.033		
PEF L/sec	-0.04 (-0.06 to -0.02)	< 0.01	-0.011 (-0.03 to -0.01)	0.045		

BMI: body mass index. FVC: Forced vital capacity. FEV1: forced expiratory volume in 1 s. FEF 25–75 %: forced expiratory flow at 25–75 %. PEF: peak expiratory flow.

^a Adjusted for age, gender, and smoking.

respiratory symptoms (e.g., shortness of breath). In addition, our observations support the proposition that obesity can cause shortness of breath even in the absence of pulmonary or cardiac conditions.

It has been reported that there is an association between obesity and dyspnea among the general population [20] and that obesity is an independent risk factor for dyspnea [6]. Although the direct association between increased body weight and airflow limitation in those with dyspnea has not been well studied, our findings showed that BMI is negatively associated with spirometry parameters. In this case, when the BMI (kg/m²) increased by one unit, the FVC (L), FEV₁ (L), PEF (L/sec) and FEF_{25-75 %} (L/sec) decreased by 0.03, 0.2, 0.02 and 0.04 units, respectively, which suggests that increased body weight is associated with a reduction in lung function. These observations are similar to our previous findings from a study of patients with asthma that demonstrated reductions of the FVC, FEV₁, PEF and FEF_{25-75 %} by 0.03, 0.02, 0.03 and 0.03 units, respectively, as the BMI score increased by one unit [12]. These observations, together with the combined high prevalence of overweight and obesity (80 %) in our study population, also suggest that increased body weight may lead to dyspnea by reducing lung function. This is supported by the fact that obesity is a known independent risk factor for dyspnea [6] and that airflow limitation has been reported to contribute significantly to dyspnea [21]. However, the presence of chronic dyspnea induced by obesity cannot be excluded.

Although obesity is known to have detrimental effects on the respiratory system, to our knowledge, no prior studies have evaluated the direct effect of high body weight (overweight and obesity) on spirometry parameters in breathless people without evidence of chronic respiratory or cardiovascular conditions. Our observations in the current study demonstrate a progressive reduction in spirometry parameters as body weight increases and are strengthened by the previous findings of our groups and others. For instance, we have recently shown that spirometry parameters are reduced in patients with asthma [12]. In addition, reduced lung volumes have been reported in morbidly obese women [22] and in healthy obese Black Americans who did not have evidence of respiratory diseases [16]. Although the exact mechanism leading to reduced lung function in obese individuals remains unclear, the observed reduction in spirometry parameters in our study population is likely due to the mechanical effects of increased body weight on the pulmonary system, which lead to a narrowing and closure of the airway [23]. The production of inflammatory mediators due to obesity may lead to airway inflammation, which causes remodelling of the airway and a reduction of lung function. This is supported by a pre-clinical study demonstrating increased pro-inflammatory mediators (e.g., interleukin-6 and neutrophils) in the bronchoalveolar lavage fluid of obese or fat mice compared to non-obese controls [24]. Taken together, our findings suggest that obesity may play a key role in dyspnea, likely by reducing lung function. Given that weight reduction has been shown to improve lung function [25], our findings also highlight the importance of weight loss in improving respiratory symptoms by preventing further reduction in lung volumes and spirometry parameters.

4.1. Strengths

The current study has several strengths. First, although previous studies have examined changes in lung function among different obesity categories, these studies have not limited their participant population to those with dyspnea. Our study is the first to explore the spirometry profiles of obese and overweight individuals who have symptoms of breathlessness but have not been diagnosed with respiratory diseases, e.g., asthma and chronic obstructive pulmonary disease (COPD). Second, some previous studies used self-reported heights and weights to characterise overweight and obesity based on BMI values. In the current study, a trained nurse or respiratory therapist measured the heights and weights of the subjects using a medical scale. This took place during the patient's visit to the studied pulmonary clinic and before the spirometry tests were performed. Third, the included spirometry results were conducted following international guidelines (the American Thoracic Society/European Respiratory Society guidelines). In addition, although the tests were performed by a trained respiratory therapist and the results were reviewed by respiratory physicians to determine a diagnosis, two respiratory therapists manually reviewed the collected spirometry results. Tests without acceptable and reproducible results were excluded.

4.2. Limitations

This study has some limitations. First, it is known that obesity is a common cause of dyspnea even in the absence of respiratory disease. Given the current study design, it was difficult to determine the severity of dyspnea and its association with the degree of airflow limitation in our study population. Second, reduced functional residual capacity and expiratory reserve volume have been

reported in healthy Black subjects with obesity [16], suggesting that obesity can also reduce lung volumes. However, whether reductions in lung volumes are associated with increased body weight in those with dyspnea remains unknown. In the current study, we were unable to assess the impact of overweight and obesity on lung volumes as tests are not frequently carried out in our pulmonary clinic for individuals experiencing respiratory symptoms. Third, although patients with dyspnea and a diagnosis of respiratory disease (e.g., COPD and asthma) were excluded from the final analysis, we cannot exclude the possibility that the reduction in spirometry parameters came as a result of unknown conditions other than obesity. Fourth, the results are not representative of the whole population with unexplained dyspnea, given that data in the current study were collected from one pulmonary clinic. Further prospective multicentre studies are needed to further confirm these findings. Finally, we were not able to adjust for some factors (e.g., income, education, air pollution and profession) related to pulmonary function, due to insufficient information in our dataset. However, important factors such as age, gender, and smoking, which are known to greatly contribute to lung function, have been accounted for.

5. Conclusion

Our findings demonstrate that the prevalence of overweight and obesity is high and that obesity can reduce spirometry parameters in Saudi subjects with dyspnea. These findings point toward the importance of including routine screening and assessment of obesity as part of primary care for people with chronic dyspnea and also suggest that patient-specific treatment approaches (e.g., lifestyle intervention) should be considered to reduce body weight to prevent further reduction in lung function, thereby improving respiratory symptoms and quality of life in people with dyspnea.

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Institutional review board statement

The study was approved by the Unit of Biomedical Ethics Research Committee at the Faculty of Medicine at King Abdulaziz University, Saudi Arabia (No 448-22).

Informed consent statement

Not applicable.

Data availability statement

All data generated and analysed during this study are available from the corresponding author upon reasonable request.

CRediT authorship contribution statement

Abdullah A. Alqarni: Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Software, Resources, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. Abdulelah M. Aldhahir: Writing – review & editing, Writing – original draft, Methodology, Formal analysis, Data curation, Conceptualization. Jaber S. Alqahtani: Writing – review & editing, Writing – original draft, Formal analysis, Data curation, Conceptualization. Rayan A. Siraj: Writing – review & editing, Writing – original draft, Software, Formal analysis, Data curation. Joud H. Aldhahri: Writing – review & editing, Writing – original draft, Data curation, Conceptualization. Shahad A. Madkhli: Writing – review & editing, Writing – original draft, Methodology, Data curation. Wanas M. Fares: Writing – review & editing, Writing – original draft, Formal analysis, Data curation. Abeer A. Alqurayqiri: Writing – review & editing, Writing – original draft, Formal analysis, Data curation. Hassan Alwafi: Writing – review & editing, Writing – original draft, Supervision, Project administration, Methodology, Funding acquisition, Formal analysis, Data curation, Conceptualization, Abdallah Y. Naser, Writing – review & editing, Writing – original draft, Funding acquisition, Formal analysis, Data curation, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.heliyon.2024.e24935.

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