Deniz and Kuranoğlu
BMC Pulmonary Medicine (2024) 24:586
https://doi.org/10.1186/s12890-024-03408-9

BMC Pulmonary Medicine

RESEARCH

Open Access

Comparison of impulse oscillometry measurements according to body mass index in patients with asthma

Sami Deniz[1,2*] and Nil Kuranoğlu[1]

Abstract

Background Obesity can lead to increased airway resistance, especially in the peripheral airways. Impulse oscillometry (IOS) may detect small airway changes in asthma. In this study, we aimed to investigate the effect of obesity on the small airways of the lungs in asthmatic patients by IOS measurements.

Methods One hundred newly diagnosed asthmatic patients (28 male) were divided into three groups (BMI (< 25.0 kg/m^2: Group-1; 25.0 to 29.9 kg/m^2: Group-2; ≥30 kg/m^2: Group-3). Demographic data, comorbidities, Beck anxiety and depression questionnaires, PFTs, and IOS measurements of the patients were recorded.

Results There was no detected significant difference concerning age, gender, smoking history, pack year, comorbidities (except hypertension and anxiety), asthma cardinal symptoms (except dyspnea), and pulmonary function tests among groups ($p > 0.05$). There were significant differences regarding R5, R5%, R20%, X5, X20, Fres, and R5-R20 among the three groups. Group 1 significantly differed about R5-R20, X5, AX, and Fres compared to groups 2 and 3 in pairwise comparisons. Based on these results, a difference was detected between BMI < 25 and ≥ 25. There was only one significant variable (R20%) between BMI < 30 and ≥ 30. When performing a correlation test between IOS parameters and BMI, it was observed to be significantly correlated ($p < 0.05$) except X5% and X20% ($p > 0.05$).

Conclusion Obesity and being overweight affected peripheral airways and reactance; however, obesity also affects central airways, based on our results. In addition, it was thought that IOS may detect earlier than PFTs on small airway changes.

Keywords Asthma, Small airways, Impulse oscillometry, Obesity

Background

Asthma is a common disease characterized by airway inflammation presenting clinically variable airway narrowing, airway hyperresponsiveness, and, in some cases, loss of FEV_1. Although asthma pathophysiology has not yet been fully understood, inflammation, structural abnormalities, injury, and repair processes have been shown in small airways despite previously being regarded as in large airways [1, 2]. The diameter of small airways is less than 2 mm, and the contribution of total resistance is less than 20%; for that reason, early diagnosis of small airway diseases (SADs) is challenging to detect utilizing conventional pulmonary function tests [3]. However, multiple-breath nitrogen washout, respiratory oscillometry, and ventilation imagining pave the way for diagnosing SADs. No matter what it is, conventional spirometry will still be used, and it is considered that three peripheral

*Correspondence:
Sami Deniz
sami_deniz@yahoo.com
[1] Dr Suat Seren Chest Diseases and Thoracic Surgery Research and Training Hospital, İzmir, Turkey
[2] The Department of Chest Diseases, University of Health Sciences Turkey Medical Faculty, İzmir Faculty of Medicine, İzmir, Turkey

airway measurements will be complementary to draw the whole picture [2].

Impulse oscillometry (IOS), a non-invasive and established technique, provides some data about respiratory mechanical features. R5 (total airway resistance), R20 (resistance of the central airway), and R5-R20 reflect changes in the obstruction of peripheral airways. However, it remains unclear whether R5–R20 reflects small airway caliber [4]. A study showed that R5, R5–R20, area of reactance (AX), and resonant frequency (Fres) significantly increased in patients with SAD ($n=42$) compared to those without SAD [5].

Obesity includes mechanical and inflammatory lung and airway effects [6]. It significantly affects both distal and central airway function [7]. Because obesity-related asthma may be a different kind of inflammation (such as TNF-α, IL-1β, and IL-6 and pro-inflammatory cytokines are increased, anti-inflammatory adiponectin is reduced) and contributory factors, asthma control is difficult in these patients [1]. Obesity can lead to increased airway resistance, especially in the peripheral airways, leading to difficulty breathing and reduced FRC and ERV, which indicate peripheral airway dysfunction [8]. Small airway abnormalities present in healthy obese individuals and body weight may negatively affect distal airway function. Therefore, IOS may be an early functional marker of asthma, especially in obese individuals reporting symptoms despite having normal airflow [9].

We hypothesized that obesity affects small airways independently. In this study, we aimed to investigate the effect of obesity on the small airways of the lungs in asthmatic patients by IOS measurements.

Methods

The study was designed as a prospective cross-sectional study, which involved observing a defined population simultaneously. The study was clearly explained to all patients, and an informed consent form was obtained from them. This study complies with the Declaration of Helsinki and was performed according to ethics committee approval. Ethical approval was obtained from the University of Health Sciences Turkey Medical Faculty, Dr Suat Seren Chest Diseases, and Thoracic Surgery Research and Training Hospital.

Patients

The eligible patients aged > 18 years visiting the outpatient clinic with newly diagnosed asthma who had symptoms and confirmation with a 12% and 200 mL change in FEV_1 after a short-acting bronchodilator and/or positive bronchial provocation test (BPT) (PC20 less than 16 mg/mL) were included. Seventeen patients used ICS in a very short time previously (n:6,6,5 patients, separately, for each group); however, they did not use it in the past six months. After the definitive diagnosis of asthma, demographic data (age, gender, BMI, smoking history, treatment), comorbidities, pulmonary function tests, and questionnaire scores were recorded. They were divided into three groups according to BMI, which measures body fat based on height and weight. The groups were Group 1, with a BMI of less than 25.0 kg/m^2; Group 2, with a BMI between 25.0 and 29.9 kg/m^2; and Group 3, with a BMI of 30 kg/m^2 or higher. The study did not include a control group consisting of obese patients without a concurrent diagnosis of asthma. Pregnants, current ICS use, immunosuppressive therapy for any reason, patients with other lung disorders (COPD, interstitial lung diseases, emphysema, active/sequel tuberculosis, post-COVID), smoking history > 10 PY, surgery or myocardial infarction in the past three months, insufficient cognitive function were excluded.

Procedures

Pulmonary Function Tests: For the diagnosis, all patients performed pulmonary function testing as a post-bronchodilator test (Zan 100 Bodyplethysmographie, Germany). A 200 ml and 12% change in FEV_1 and clinical agreement were considered asthma. A bronchial provocation test with methacholine was performed in eligible patients, and a 20% decrease in FEV_1 (PC20) was considered a positive bronchial provocation test (Zan 300 Bodyplethysmographie, Germany). A positive BPT was considered asthma if clinical features agreed with asthma [10]. PFTs were performed according to Quanjer 2012 GLI norm.

Impulse Oscillometry: It was measured all variables (R5, R20, R5-R20, X5, X20, Fres, and AX) with Vyntus IOS (CareFusion, Germany 234 GmbH), a non-invasive and established technique that provides data about respiratory mechanical features. IOS measurement was performed at least one day after PFT. Patients were asked to breathe at average tidal volume for 30–60 s with their noses clipped and cheeks supported by their hands. The mean values of three technically acceptable measurements compatible with ERS guideline [4] were used in the analysis.

Beck Anxiety and Depression Inventory: The Beck Depression and Anxiety Inventory consists of 21 questions separately. Each question contains four options. According to total scores for depression, 11–16: Mild mood disturbance, 17–20: Borderline clinical depression, 21–30: Moderate depression, 31–40: Severe depression, over 40: Extreme depression. According to total scores for anxiety, a score of 0–21: low anxiety, a score of 22–35: moderate anxiety, and a score of 36 and above: potentially concerning levels of anxiety.

Statistical Analysis: Analyses were performed using the statistical software SPPS Version 28 (IBM Corp., Armonk, NY, USA). Categorical variables were presented as percentages and numbers. The Chi-Square test was used to compare categorical variables. If numerical variables fit the normal distribution, a t-test was used for two-group comparisons. The one-way ANOVA test was used for three-group comparisons, and these variables were presented as mean ± SD. If the variables did not fit a normal distribution, the Mann-Whitney U test was used for two-group comparisons. The Kruskal-Wallis test was used for three-group comparisons, and these variables were presented as median (IQR25,75). Adjusted *p*-value was used in post hoc analysis. In addition, the correlation test was performed between BMI and IOS parameters. All variables were analyzed as two-tailed, and $p<0.05$ was considered significant.

Results

The patients were categorized into three groups according to BMI (kg/m^2) (<25: 31 patients; ı 25–29.9: 38 patients; >30: 31 patients). There were no significant differences concerning age, gender, smoking history, and pack year among groups ($p>0.05$) (Table 1). In addition, there was no detected significance in terms of comorbid conditions such as depression, gastroesophageal reflux, allergic rhinitis, diabetes mellitus, coronary artery disease, or additional comorbidities (the three patients had thalassemia, mitral valve prolapse, and epilepsy, separately for each group and some other comorbidities such as migraine, varicose veins, benign prostatic hypertrophy). We determined a significant difference regarding anxiety (8, 12, 15 of median value; respectively; for groups) ($p=0.035$) and hypertension (7%, 21%, 35% of percentage; respectively; for groups) ($p=0.019$) (there was a difference between groups 1 and 3 in both variables, respectively; $p=0.030$, 0.013) (Table 2). Of all asthma cardinal symptoms, they were similar between groups except for dyspnea ($p=0.027$, the adjusted difference between groups 1 and 2 ($p=0.029$)) (Table 1). Pulmonary function tests were similar in both pre and post-bronchodilator and changes after bronchodilator (ml and %) between the groups ($p>0.05$) (Table 3). Nine patients were treated in step 4; the remaining were treated in steps 3 and 2. When compared to groups in this respect, there was no significant difference between the groups ($p=0.200$).

There were statistical differences between groups regarding IOS measurements except for R20, X5%, and X20%. The distribution of groups were 0.29 ± 0.15, 0.34 ± 0.21,0.44 ± 0.21 kPa/(L/s) for R5 ($p=0.007$, difference between group 1 and 3); 103 (79;120), 110 (92;120), 131 (105;155) for R5% ($p=0.005$, the difference between group 1 and 3); 94 (79;99), 87.5 (77;105), 100 (90;127) for R20% ($p=0.015$, difference between group 2 and 3); −0.04 (−0.11;0), −0.07 (−0.12;−0.05) −0.10 kPa/(L/s) (−0.13;−0.06) for X5 ($p=0.010$, difference between group 1,2 and 1,3); 0.16 (0.05;0.49), 0.59 (0.34;1.09), 0.75 (0.34;1.24) kPa/L for AX ($p<0.001$, difference between group 1,2 and 1,3); 11.8 ± 6.1, 17.4 ± 5.6, 17.9 ± 7.5 Hz for F_{res} ($p<0.001$, difference between group 1,2 and 1,3) and 19.0 (2.6;432), 44.4 (28.3;63.3), 40.5 (24.8;55.7) kPa/(L/s) for R5-R20 ($p<0.003$, difference between group 1,2 and 1,3) (*p* values presented or all variables were adjusted). There were no differences between groups about R20, X5%, and X20%. Except for those, R20% differed between

Table 1 Demographic data of the patients

Variables		Group 1	Group 2	Group 3	*P* value
Age		34 (27,53)	43 (28,57)	45 (38,61)	0.112
Gender n (%)	Male	10 (32%)	11 (29%)	7 (23%)	0.688
	Female	21 (68%)	27 (71%)	24 (77%)	
Smoking n (%)	Never	16 (52%)	18 (47%)	18 (58%)	na
	Active	12 (39%)	15 (39%)	7 (23%)	
	Exsmoker	3 (10%)	5 (13%)	6 (19%)	
Smoking (PY)		8 (5,9)	7 (5,10)	5 (3;8)	0.394
Body mass index		22.7(22.5,24)	27 (25.7,28.5)	33.3 (31.6,37.1)	<0.001
Previous ICS use n (%)		6 (19%)	6 (16%)	5 (16%)	0.846
Cough n (%)		25 (81%)	32 (84%)	25 (81%)	0.904
Wheezing n (%)		24 (77%)	30 (79%)	26 (84%)	0.800
Chest tightness n (%)		18 (58%)	18 (47%)	21 (68%)	0.233
Dyspnea n (%)		27 (87%)	23 (61%)	25 (81%)	**0.027**
Eosinophil count		330 (170,450)	200 (150,300)	200 (90,300)	0.161

The categoric variables were presented as n (%), and the numeric variables were presented as median (IQR 25,75) in this table

Table 2 Comorbid conditions of the patients

Variables	Group 1	Group 2	Group 3	*p*-value	Adjusted *p*-value		
					1 vs 2	1 vs 3	2 vs 3
Anxiety	8 (2,18)	12 (6,20)	15 (7,26)	**0.035**	0.336	**0.030**	0.799
Depression	7 (3,10)	8 (4,11)	9 (5,14)	0.189	-	-	-
Gastroesophageal reflux n (%)	15 (48%)	17 (45%)	17 (55%)	0.703	-	-	-
Allergic rhinitis n (%)	23 (74%)	19 (50%)	15 (48%)	0.066	-	-	-
Hypertension n (%)	2 (7%)	8 (21%)	11 (35%)	**0.019**	0.167	**0.013**	0.287
Diabetes mellitus n (%)	2 (7%)	2 (5%)	6 (19%)	na	-	-	-
Coronary artery disease n (%)	0 (0%)	0 (0%)	1 (3%)	na	-	-	-
Epilepsy n (%)	0 (0%)	1 (3%)	0 (0%)	na	-	-	-
Thalessemia n (%)	0 (0%)	0 (0%)	1 (3%)	na	-	-	-
Heart valve disease n (%)	1 (3%)	1 (3%)	0 (0%)	na	-	-	-
Heart failure n (%)	3 (10%)	4 (10%)	4 (12%)	0.780	-	-	-
Additional comorbidities n (%)	4 (13%)	5 (13%)	5 (16%)	0.271			

The categoric variables were presented as n (%), and the numeric variables were presented as median (IQR 25,75) in this table

Table 3 Pulmonary function tests of the patients

Variables	Group 1	Group 2	Group 3	*p*-value
FEV_1, %	75 (68,82)	75 (61,87)	72 (66,85)	0.957
FEV_1, ml	2342 ±693	2271 ±733	2032 ±553	0.165
FVC, %	86 (73,93)	86 (78,93)	81 (75,93)	0.510
FVC, ml	3084 ±904	2998 ±1039	2656 ±761	0.156
FEV_1/FVC	76 ±7	74 ±9	76 ±7	0.344
Post FEV_1, %	86 ±11	86 ±15	91 ±17	0.240
Post FEV_1, ml	2639 ±578	2642 ±787	2439 ±758	0.445
Post FVC, %	90 ±13	91 ±13	95 ±15	0.449
Post FVC, ml	3282 ±653	3214 ±1094	3016 ±950	0.519
Post FEV_1/FVC	81 ±8	79 ±8	81 ±6	0.608
FEF 25–75, %	47 (38,62)	43 (31,62)	45 (38,57)	0.513
FEF 25–75, ml	1855 (1280,2610)	1635 (1230,2340)	1710 (1310,2030)	0.664
Post FEF 25–75, %	69 ±19	68 ±26	73 ±28	0.690
Post FEF 25–75, ml	2600 ±918	2591 ±1058	2528 ±1022	0.954
FEV_1, % (Change)	16 (11,21)	14 (12;19)	17 (12,22)	0.553
FEV_1, ml (Change)	345 (250,485)	305 (220,410)	310 (260,360)	0.761

If the numeric variables fitted the normal distribution, they were presented as mean±SD; if not, they were presented as median (IQR 25,75) in this Table. Post: Post-bronchodilator

BMI < 30 and ≥ 30. The major deviations were observed between BMI < 25 and ≥ 25 for the other IOS parameters (Table 4).

When the correlation test between IOS parameters and BMI was performed, it was determined as $p = 0.003$ and $p < 0.0001$ for R5 and Fres (Pearson correlation coefficient; 0.252 and 0.295, respectively). The other IOS parameters were also significant except for two (for R5%, R20, R20%, R5-R20, X5, X5%, X20, X20%, and AX; p-value; <0001, 0.007, 0.007, 0.007, 0.020, 0.403, 0.002, 0.148, < 0.0001; respectively) (Kendall's tau; 0.247, 0.188, 0.184, 0.183, −0.161, 0.057, −0.222, −0.099, 0.293; respectively). In addition, there was no significant difference between groups for FEF%25–75 in both ml and % (0.156, 0.834) (Fig. 1).

Discussion

In this study, it was determined that there were significant differences concerning R5, R5%, R20%, X5, X20, Fres, and R5-R20 among the three groups. In comparing

Table 4 Impulse oscillometry measurements of the patients

Variables	Group 1	Group 2	Group 3	*P* value	Adjusted *p* value		
					1 vs 2	1 vs 3	2 vs 3
R5	0.29 ±0.15	0.34 ±0.21	0.44 ±0.21	**0.007**	0.707	**0.006**	0.104
R5%	103 (79,120)	110 (92,120)	131 (105,155)	**0.005**	0.689	**0.004**	0.089
R20	0.24 (0.21,0.31)	0.25 (0.21,0.32)	0.29 (0.23,0.35)	0.077	-	-	-
R20%	94 (79,99)	87 (77,105)	100 (90,127)	**0.015**	1.000	0.070	**0.019**
R5-R20	19.03 (2.6,43.2)	44.3 (28.3,63.3)	40.5 (24.8,55.7)	**0.003**	**0.004**	**0.015**	1.000
X5	−0.04 (−0.11,0)	−0.07 (−0.12,−0.05)	−0.10 (−0.13,−0.06)	**0.010**	**0.041**	**0.015**	1.000
X5%	23 (−162,234)	107 (−223,302)	128 (−73,228)	0.755	-	-	-
X20	0.04 (0.03,0.07)	0.03 (0,0.05)	0.03 (−0.01,0.06)	**0.035**	0.057	0.092	1.000
X20%	69 (37,98)	49 (10,84)	52 (−16,109)	0.328	-	-	-
AX	0.16 (0.05,0.49)	0.59 (0.34,1.09)	0.75 (0.34,1.24)	**<0.001**	**0.001**	**<0.001**	1.000
Fres	11.8 ±6.1	17.4 ±5.6	17.9 ±7.5	**<0.001**	**0.001**	**0.001**	1.000

If the numeric variables fitted the normal distribution, they were presented as mean±SD; if not, they were presented as median (IQR 25,75). R5, X5, R20, X20, R5-R20: hPa/L/s, AX: kPa/L, Fres: Hz

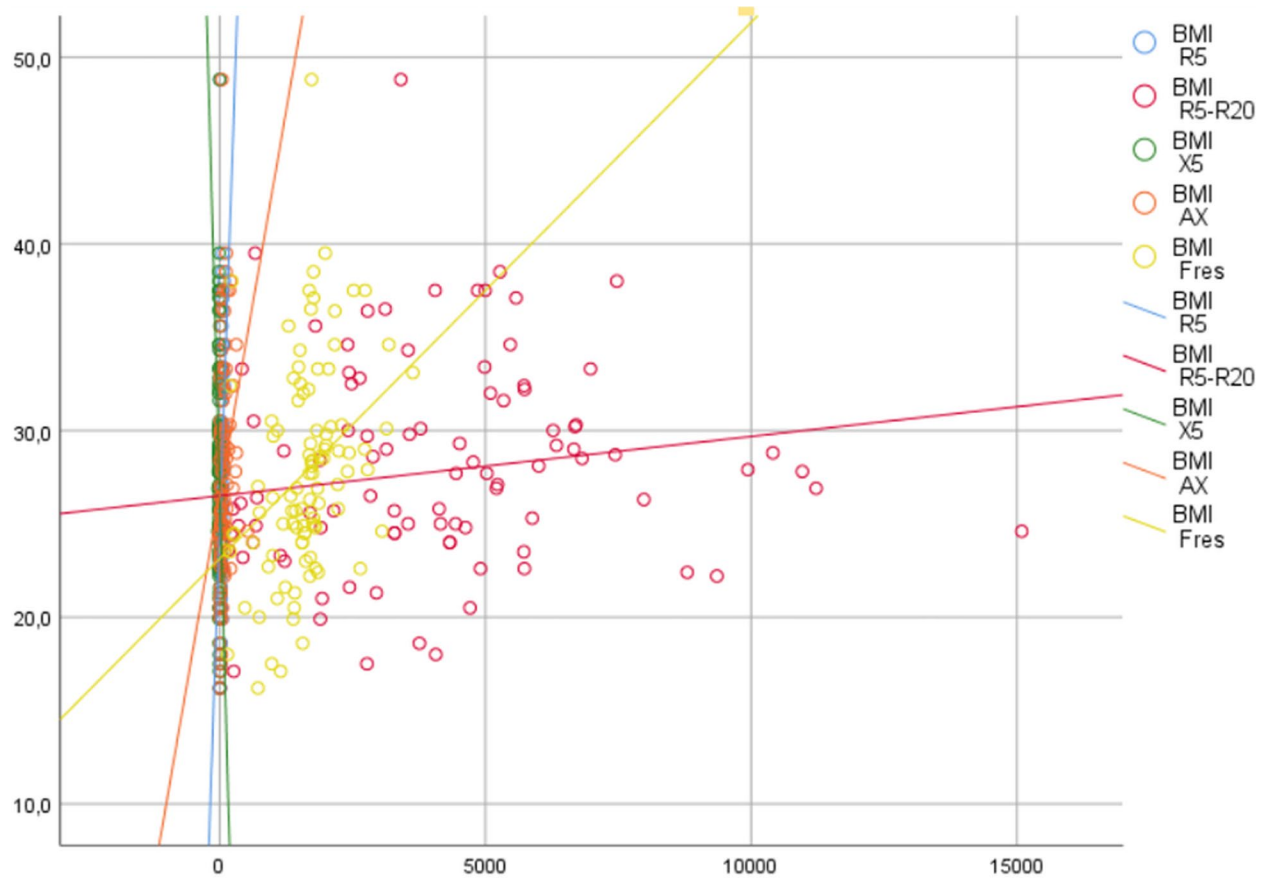

Fig. 1 The correlation test between IOS parameters and BMI

the two groups, group 1 significantly differed about R5-R20, X5, AX, and Fres compared to groups 2 and 3. Based on these results, a difference was detected between BMI < 25 and ≥ 25 kg/m². Only one significant variable (R20%) between BMI < 30 and ≥ 30. Obesity and being overweight affected peripheral airways and reactance;

on the other hand, obesity also affected central airways, based on our results. Remarkable differences were observed in terms of AX and Fres rather than R5-R20.

As for PFT, even though it is not specific to SAD, a study showed FEF25-75% as an indicator of SAD, and it was suggested that FEF25-75% may be more sensitive than FEV_1 for small and middle airway obstruction [11]. R5 and Fres are increased in peripheral airway obstruction for patients with asthma. R5-R20 is higher because R5 increases more than R20. X5 is more negative due to hyperinflation and decreased elasticity. AX increase is observed due to more negative X5 values and increased Fres [12]. In our study, PFTs of our patients did not differ among the three groups, including FEF25%−75%. Increased R5 and R5-R20 and normal R20 are shown in peripheral airway obstruction. Considering our results, the IOS results of the patients showed that obese patients had more peripheral airway obstruction than lean asthmatic patients, even though spirometry results of the patients were distributed homogenous.

There are several studies in this field on asthmatic children. A study on school-age children showed high R5, R5-20, and FEF25-75% at baseline measurement in obese asthmatics compared to lean asthmatics and obese children [13]. In a study by Assumpção et al., patients were divided into three groups (81 children, 30 in control, 21 in the overweight, and 30 in the obesity group). There was a difference between the control group and the obesity group for impedance, R5, Fres, and AX [14]. Another study detected that the median age of 75 participants was seven years old, and R5, Z5, and AX in obesity with asthma were statistically significantly higher than in asthma only. X5's median was higher in obesity than in asthma and obesity with asthma group. In a comparison of asthma patients and obese patients with asthma, the authors found a significant difference regarding R5-R20 in obese patients with asthma compared to patients with asthma alone [15]. Even though the populations of the studies were different, our results were similar to those of the above studies. However, FEF25-75% was not different among groups in our study; in addition to R5, Fres, and AX, R5-R20 was also significantly different in our groups. Our results were highly similar to those of Sangsawang et al. 's study. In addition to the above variables, X5 was also a significant difference in our study. In comparison to all variables, overweight and obese asthmatic patients, some variables that demonstrated peripheral airway obstruction were higher than lean asthmatic patients in our study.

In a study that was performed on 188 patients, they were categorized into four groups according to BMI (kg/m^2) (< 25 (control group), 25–30, > 30, and > 40). Demographic data and PFTs of the patients were homogeneous distribution among groups. The authors detected that obesity (BMI 30–39.9) and morbid obesity (BMI ≥ 40) were linked to a significantly worse R5-R20, X5, AX, and Fres compared with normal weight [16]. Similarly, demographic data and PFTs were similar in our study; besides R5-R20, X5, AX, and Fres, we determined that R20% was a significant difference. Conversely, a significant difference was not detected about R20 ($p = 0.077$). Although R20 increases in central airway obstruction, it also increases in peripheral airway obstruction; however, it does not increase so much as R5. In a study comparing morbid obesity and normal weight in terms of IOS measurements, R5, R20, R5-20, Z5, X5, and Fres were found to be increased [17]. Although our study was not included in morbid obese groups and was included in asthmatic patients, similar to the study, R20% was increased in our patients. Considering that obesity affects both peripheral and large airway functions [8], increasing R20 is not surprising; interestingly enough, the significant difference was between overweight and obese patients; there was no difference between lean and overweight or obese asthmatic patients.

In a cross-sectional study, it was examined whether obesity affects lung mechanics. In this context, 45 patients (22 females) were included in the study to examine BMI and other measurements. There were no differences between BMI < 25 and > 25 regarding IOS parameters and PFTs in males and females [18]. Compared to our results, although there was no significant difference between groups concerning PFTs, similar to the above study, there were notable deviations in IOS parameters between BMI < 25 and > 25 kg/m^2. The study consisted of a relatively small sample size compared to our study. Asthma might increase airway resistance and reactance in obese patients based on the above study.

The ATLANTIS study found that R5-20, AX, and X5 are all strongly related to small airway disease and have a high prevalence in asthma compared to other physiological measures thought to reflect the small airways [19]. The resonant frequency(Fres), the zero crossing of the reactance spectrum, and the reactance area (AX) are used as indirect markers of the lung periphery, especially for pre-post examinations and provocation tests [4]. In our study, despite indirect markers for peripheral airways, they were strongly significant between BMI > 25 and < 25.

It was detected between groups 1 and 3 regarding hypertension (7,21,35%, respectively) and anxiety (8,12,15 of median value, for groups, respectively). It is well-established that obesity is a significant risk factor for several diseases, such as anxiety [20]. Similarly, the association between obesity and hypertension has been known clearly for a long time [21]. In our study, the more the weight was increased, the more the diagnosis of hypertension was increased. However, there was only one patient diagnosed with CAD in group 3.

Limitations

The study did not include a control group consisting of obese patients without a concurrent diagnosis of asthma. Some comorbid conditions, such as hypercholesterolemia, were based on patients' self-reporting. On the other hand, there have been few studies in the literature evaluating IOS parameters according to BMI.

Conclusion

The small airways might affect other factors, such as the severity of asthma, genetic features, socioeconomic situation, and obesity. According to our results, obesity and being overweight were detected to negatively affect the peripheral airways in adult asthma patients. Compared with lean asthmatic patients, significant changes related to R5-R20%, R5, Fres, and AX demonstrating small airway disease were worse in overweight and obese asthmatic patients. However, there were no differences concerning PFTs between groups. Therefore, it was thought that IOS may detect small airway changes earlier than PFTs, especially in obese individuals.

Acknowledgements
Not applicable.

Clinical trial number
Not applicable.

Authors' contributions
S.D. conceptualized the study. N.K. and S.D. curated the data. S.D. contributed to the methodology and drafted the original manuscript. All authors participated in reviewing and editing the manuscript. S.D. supervised the study. The authors read and approved the final manuscript.

Funding
No funding.

Data availability
The datasets used and/or analyzed during the current study are available from the corresponding author (Sami Deniz, e-mail: sami_deniz@yahoo.com) upon reasonable request.

Declarations

Ethics approval and consent to participate
The study was approved by the Institutional Review Board for Human Studies and Ethics Committee (2023/65–70) University of Health Sciences Turkey Medical Faculty, Dr Suat Seren Chest Diseases and Thoracic Surgery Research and Education Hospital, Ethics Committee) and was conducted using the principles of the Declaration of Helsinki.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

Received: 13 August 2024 Accepted: 21 November 2024
Published online: 27 November 2024

References

1. Global Initiative for Asthma (GINA). Global strategy for asthma management and prevention. http://www.ginasthma.org. Accessed 2023.
2. Siora A, Vontetsianos A, Chynkiamis N, et al. Small airways in asthma: from inflammation and pathophysiology to treatment response. Respir Med. 2024;222:107532.
3. Kelly MM et al. Pathology of Asthma. In: Burks AW, O'Hehir RE, Broide DH, editors. Middleton Allergy Principles and Practice 9th Edition Chapter:57, Amsterdam, 2019, Academic, p: 957–969.
4. Thamrin C, Dellacà RL, Hall GL, et al. Technical standards for respiratory oscillometry: test loads for calibration and verification. Eur Respir J. 2020;56(4):2003369.
5. Li LY, Yan TS, Yang J, et al. Impulse oscillometry detects small airway dysfunction in subjects with chronic respiratory symptoms and preserves pulmonary function. Respir Res. 2021;22(1):68.
6. Tay TR, Hew M. Comorbid treatable traits in difficult asthma: current evidence and clinical evaluation. Allergy. 2018;73:1369–82.
7. Perossi L, Holtz M, Santos DOD, et al. Increased airway resistance can be related to decreased functional capacity in obese women. PLoS ONE. 2022;17:e0267546.
8. Pepys J, Lombardi C, Comberiati P, et al. Small airway dysfunction and obesity in asthmatic patients: a dangerous liaison? Explore Asthma Allergy. 2023;1:73–88.
9. van de Kant KDG, Paredi P, Meah S et al. The effect of body weight on distal airway function and airway inflammation. Obes Res Clin Pract 201610:564–73.
10. Stanojevic S, Kaminsky DA, Miller MR, et al. ERS/ATS technical standard on interpretive strategies for routine lung function tests. Eur Respir J. 2022;60(1):2101499.
11. Arshad SH, Hodgekiss C, Holloway JW, et al. Association of Asthma and smoking with lung function impairment in adolescence and early adulthood: the isle of wight birth cohort study. Eur Respir J. 2020;55:1900477.
12. Comberiati P, Landi M, Cottini M, Berti A. Impulse oscillometry for the evaluation and management of pediatric asthma. Explor Asthma Allergy. 2023;1:219–29.
13. Uysal P, Anik A, Anik A. School-age obese asthmatic children have distinct lung function measures from lean asthmatics and obese children. J Asthma. 2022;59(8):1548–59.
14. Assumpção MS, Ribeiro JD, Wamosy RMG, et al. Impulse oscillometry and obesity in children. J Pediatr (Rio J). 2018;94(4):419–24.
15. Sangsawang T, Daengsuwan T. Comparative impulse oscillometry parameters among childhood asthma, obesity with and without Asthma. J Allergy Clin Immunol. 2020;145(2):AB119.
16. Chan R, Lipworth B. Clinical impact of obesity on oscillometry lung mechanics in adults with asthma. AnnAllergy Asthma Immunol. 2023;131(3):338–42.
17. Venâncio R, Karsten M, Gonçalves R, et al. Morbidly obese patients have altered respiratory mechanics when assessed by the impulse Oscillometry System. Eur Respir J. 2017;50:PA2473. https://doi.org/10.1183/1393003.congress-2017.PA2473.
18. Shanmugasundaram K, Bade G, Sampath M, Talwar A. Effect of obesity on Airway mechanics. Indian J Endocrinol Metab. 2023;27(2):161–6.
19. Postma DS, Brightling C, Baldi S, et al. Exploring the relevance and extent of small airways dysfunction in asthma (ATLANTIS): baseline data from a prospective cohort study. Lancet Respir Med. 2019;7(5):402–16.
20. Gariepy G, Nitka D, Schmitz N. The Association between obesity and anxiety disorders in the Population: a systematic review and Meta-analysis. Int J Obes. 2010;34:407–19.
21. Shah NP, Lu R, Haddad F, Project Baseline Health Study Group. Relationship between body mass index and cardiometabolic health in a multiethnic population: A project baseline health study. Am J Prev Cardiol. 2024;18:100646.

Publisher's Note