Obesity affects pulmonary function in Japanese adult patients with asthma, but not those without asthma

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Abstract

Background

Obesity is a factor associated with the severity of asthma, which is characterized by airway obstruction. Pulmonary function testing is one of the important examinations for evaluating airway obstruction. However, the impact of obesity on pulmonary function in patients with asthma is not fully understood.

Methods

A total of 193 patients with asthma and 2,159 patients without asthma who visited Saga University Hospital were investigated retrospectively. Obesity was defined as a body mass index (BMI) greater than 25 kg/m^2. Pulmonary functions including forced vital capacity (FVC) and forced expiratory volume in 1 second (FEV_1) were compared between patients with asthma and those without asthma, focusing especially on obesity.

Results

%FVC, FEV_1, and $\%FEV_1$, but not FVC were significantly lower in patients with asthma than in those without asthma ($p < 0.01$, < 0.01, < 0.01, 0.25, respectively). In patients with asthma, FVC, %FVC, FEV_1, and $\%FEV_1$ were significantly lower in patients with obesity than in those without obesity (all $p < 0.01$). In addition, BMI was negatively correlated with FEV_1 ($r = -0.21$, $p = 0.003$) and FVC ($r = -0.15$, $p = 0.04$). On multivariate analysis in patients with asthma, FVC (β [95% confidence interval] 0.11 [0.01–0.02], $p = 0.02$) and FEV_1 (0.13 [0.05–0.21], $p < 0.01$) were still significant between patients with obesity versus patients without obesity. However, those obesity-associated differences were not observed in patients without asthma.

Conclusion

Obesity reduces pulmonary function including FVC and FEV_1 in patients with asthma but not in those without asthma.

Introduction

Asthma is a common respiratory disease the pathophysiology of which involves airway inflammation and airway hyperresponsivity, [1–3] which induce respiratory symptoms such as shortness of breath, coughing, and wheezing due to narrowing of the airway [4, 5]. To evaluate the disease control level precisely and objectively, pulmonary function testing has been widely recognized as a useful tool [6].

Pulmonary function testing is one of the important examinations for patients with asthma, and forced expiratory volume in 1 second (FEV_1) in particular is a parameter that reflects the situation of disease control related to airway obstruction [7, 8]. For example, corticosteroid therapy, which is a pivotal treatment for asthma, increases FEV_1 dramatically [9], and, in contrast, severe asthma patients showed decreased FEV_1 even with intense treatment [10, 11]. In addition, a previous report showed that a decreased FEV_1 is associated with poor outcomes in patients with asthma [12]. These data indicate that exploration of factors related to reduced FEV_1 is important for the management of asthma in daily clinical care.

Obesity and asthma are closely related, and obesity contributes to clinical outcomes of patients with asthma [13]. For example, obesity increases the incidence of asthma beyond age and race differences [14]. Obesity also affects the severity of asthma, and for severe asthma patients in the United States, the prevalence of obesity was 57.3% in adults, which is substantially higher than that in the general United States population [15]. In addition, the annual rate of exacerbation, which is one of the characteristics of asthma severity, is higher in asthma patients with obesity than in those without obesity in Japan [16, 17]. As for the mechanisms, it is considered that obesity increases systemic and airway inflammation and induces resistance to corticosteroid therapy, which augments asthma pathophysiology [18–21]. However, the impact of obesity on pulmonary function in patients with asthma is not fully understood.

In the present cross-sectional study, the effects of obesity, defined as a body mass index (BMI) greater than 25 kg/m^2, on pulmonary function in patents with asthma and in those without asthma were examined. It was found that forced vital capacity (FVC) and FEV_1 were significantly lower in patients with asthma than in those without asthma even after adjustment for confounding factors on multivariate analysis. Additionally, FVC and FEV_1 were significantly lower in obese patients with asthma than in non-obese patients with asthma, and these differences were not seen in obese patients without asthma and non-obese patients without asthma.

Results

Characteristics of patients with and without asthma

First, 193 patients with asthma and 2,159 patients without asthma were analyzed (Figs. 1, 2). The BMI was not different between the groups. Patients with asthma were significantly younger than those without asthma ($p < 0.01$). There were more females and fewer with a smoking history in patients with asthma than in those without asthma (both $p < 0.01$). For comorbidities, the rates of hypertension, diabetes mellitus, and cardiovascular diseases were significantly lower in patients with asthma than in those without asthma (all $p < 0.01$), and the rate of hyperlipidemia was not different between the groups. On pulmonary function testing, FVC was not different, but %FVC was significantly lower in patients with asthma than in those without asthma ($p < 0.01$). FEV_1 and $\%FEV_1$ were significantly lower in patients with asthma than in those without asthma (both $p < 0.01$) (Table 1). On multivariate analysis adjusted by BMI, age, sex, and smoking history with respect to known cofounding factors for asthma incidence,

pathophysiology, and decreased pulmonary function, FVC (β [95% confidence interval] 0.08 [0.04–0.12], p < 0.01) and FEV_1 (0.18 [0.15–0.22], p < 0.01) were significantly different between the patients with asthma versus those without asthma (Table 2).

Table 1
Characteristics of patients with and without asthma

	Patients without asthma	Patients with asthma	p value
n	2159	193	
Body mass index (kg/m^2)	23.0 ± 0.1	23.0 ± 0.3	0.96
Age	62.9 ± 0.4	53.2 ± 1.4	< 0.01
Sex (M/F)	1128/1031	69/124	< 0.01
Smoking history (pack-year)	14.5 ± 0.5	8.9 ± 1.3	< 0.01
Comorbidities			
Hypertension	895 (41.5%)	51 (26.4%)	< 0.01
Diabetes mellitus	406 (18.8%)	21 (10.9%)	< 0.01
Hyperlipidemia	446 (20.7%)	29 (15.0%)	0.05
Cardiovascular diseases	347 (16.1%)	16 (8.3%)	< 0.01
Pulmonary function testing			
FVC (L)	3.06 ± 0.02	2.98 ± 0.07	0.25
%FVC (%)	96.5 ± 0.3	93.5 ± 1.2	< 0.01
FEV_1 (L)	2.41 ± 0.02	2.19 ± 0.06	< 0.01
$\%FEV_1$ (%)	95.9 ± 0.3	83.4 ± 1.5	< 0.01
Abbreviations: FVC: forced vital capacity, FEV_1: forced expiratory volume in 1 second			

Table 2
Multivariate analysis of FVC and FEV_1 in patients with asthma versus patients without asthma

Patients with asthma versus patients without asthma	Multivariate analysis		
	β	95% CI	p value
FVC (L)	0.08	0.04–0.12	< 0.01
FEV_1 (L)	0.18	0.15–0.22	< 0.01

Abbreviations: FVC: forced vital capacity, FEV_1: forced expiratory volume in 1 second, CI: confidence interval

Impact Of Obesity In Patients With And Without Asthma

Of the patients without asthma, 1,570 were non-obese (average BMI 21.1 kg/m^2), and 589 were obese (average BMI 28.2 kg/m^2). Age, sex, and smoking history were not different. The rates of hypertension, diabetes mellitus, hyperlipidemia, and cardiovascular diseases were significantly higher in obese patients without asthma than in non-obese patients without asthma ($p < 0.01$. < 0.01, < 0.01, = 0.02, respectively). Parameters of pulmonary function testing including FVC, %FVC, FEV_1, and %FEV_1 were not different even after adjustment for confounding factors including age, sex, and smoking history on multivariate analysis (Tables 3, 4). Of the patients with asthma, 134 were non-obese (average BMI 20.9 kg/m^2), and 59 were obese (average BMI 27.7 kg/m^2). Obese patients with asthma were significantly older than non-obese patients with asthma ($p < 0.01$), but sex and smoking history were not different between the 2 groups. The rates of hypertension, diabetes mellitus, and hyperlipidemia, but not of cardiovascular diseases, were significantly higher in obese patients with asthma than in non-obese patients with asthma ($p < 0.01$, < 0.01. < 0.01, 0.09, respectively). Parameters of pulmonary function testing including FVC, %FVC, FEV_1, and %FEV_1 were significantly lower in obese patients with asthma than in non-obese patients with asthma (all $p < 0.01$) (Table 3). In addition, BMI was negatively correlated with FEV_1 ($r = -0.21$, $p = 0.003$) and FVC ($r = -0.15$, $p = 0.04$), even though the correlation coefficients were low (Fig. 3a, 3b). On multivariate analysis with adjustment for age, sex, and smoking history, FVC (0.11 [0.01–0.21], $p = 0.02$) and FEV_1 (0.13 [0.05–0.21], $p < 0.01$) were still significant between patients with obesity versus patients without obesity (Table 4).

Table 3
Characteristics of patients with and without asthma associated with obesity

	Non-obese without asthma	Obese without asthma	p value	Non-obese with asthma	Obese with asthma	p value
n	1570	589		134	59	
Body mass index (kg/m^2)	21.1 ± 0.1	28.2 ± 0.1		20.9 ± 0.2	27.7 ± 0.3	
Age	63.1 ± 0.4	62.3 ± 0.6	0.35	50.2 ± 1.8	60.1 ± 2.1	< 0.01
Sex (M/F)	802/768	326/263	0.34	48/86	21/38	0.98
Smoking history (pack-year)	14.6 ± 0.6	13.6 ± 0.9	0.34	8.5 ± 1.7	9.6 ± 2.3	0.71
Comorbidities						
Hypertension	599 (38.2%)	296 (50.3%)	< 0.01	22 (16.4%)	29 (49.2%)	< 0.01
Diabetes mellitus	270 (17.2%)	136 (23.1%)	< 0.01	9 (6.7%)	12 (20.3%)	< 0.01
Hyperlipidemia	298 (19.0%)	148 (25.1%)	< 0.01	14 (10.4%)	15 (25.4%)	< 0.01
Cardiovascular diseases	234 (14.9%)	113 (19.2%)	0.02	8 (6.0%)	8 (13.6%)	0.09
Pulmonary function test						
FVC (L)	3.04 ± 0.02	3.1 ± 0.04	0.14	3.12 ± 0.08	2.67 ± 0.11	< 0.01
%FVC (%)	96.6 ± 0.4	96.2 ± 0.6	0.6	95.9 ± 1.4	87.9 ± 2.2	< 0.01
FEV_1 (L)	2.4 ± 0.02	2.44 ± 0.03	0.24	2.35 ± 0.07	1.82 ± 0.09	< 0.01
$\%FEV_1$ (%)	96.0 ± 0.4	95.6 ± 0.6	0.57	87.1 ± 1.7	75.1 ± 2.7	< 0.01

Abbreviations: FVC: forced vital capacity, FEV_1: forced expiratory volume in 1 second, CI: confidence interval

Table 4
Multivariate analysis of FVC and FEV_1 in obese patients without asthma versus non-obese patients without asthma and obese patients with asthma versus non-obese patients with asthma

Patients without asthma				**Patients with asthma**			
obese versus non-obese	Multivariate analysis			obese versus non-obese	Multivariate analysis		
	β	95% CI	p value		β	95% CI	p value
FVC (L)	0.00	-0.03–0.03	1.0	FVC (L)	0.11	0.01–0.21	0.02
FEV_1 (L)	0.01	-0.01–0.03	0.5	FEV_1 (L)	0.13	0.05–0.21	< 0.01

Abbreviations: FVC: forced vital capacity, FEV_1: forced expiratory volume in 1 second, CI: confidence interval

Allergic Comorbidities, Therapies, And Laboratory Data In Patients With Asthma Focusing On Obesity

Allergic rhinitis was less common in obese patients with asthma than in non-obese patients with asthma ($p = 0.02$), but atopic dermatitis and sinusitis were not different between the groups. Food allergy and drug allergy were more frequently seen in obese patients with asthma than in non-obese patients with asthma (both $p = 0.01$). In terms of therapies for the treatment of asthma, low-dose ICS was used significantly less often ($p = 0.03$), and high-dose ICS tended to be used more often ($p = 0.05$) in obese patients with asthma than in non-obese patients with asthma. Other therapies, long-acting β2 adrenergic agonists (LABAs), long-acting muscarinic antagonists (LAMAs), leukotriene receptor antagonists (LTRAs), and daily use of oral corticosteroid (OCS), were not different between the groups, but molecular targeting drugs were more often used in obese patients with asthma than in non-obese patients with asthma ($p < 0.01$), even though the absolute numbers were quite low. On laboratory testing, white blood cell counts, percentages of eosinophils, and eosinophil counts were not different between obese patients with asthma and non-obese patients with asthma (Table 5).

Table 5
Allergic comorbidities, therapies, and laboratory data in non-obese patients with asthma and obese patients with asthma

	Non-obese with asthma	Obese with asthma	p value
n	134	59	
Allergic comorbidities			
Allergic rhinitis	31/131 (23.7%)	6/59 (10.2%)	0.02
Atopic dermatitis	9/132 (6.8%)	3/59 (5.1%)	0.64
Sinusitis	14/133 (10.5%)	3/59 (5.1%)	0.2
Food allergy	7/130 (5.4%)	10/58 (17.2%)	0.01
Drug allergy	16/130 (12.3%)	16/58 (27.6%)	0.01
Asthma therapies			
Low dose of ICS	25 (18.7%)	4 (7.0%)	0.03
Moderate dose of ICS	73 (54.5%)	27 (45.8%)	0.34
High dose of ICS	17 (12.7%)	14 (23.7%)	0.05
LABA	95 (70.9%)	43 (72.9%)	0.78
LAMA	13 (9.7%)	6 (10.2%)	0.92
LTRA	37 (27.6%)	21 (35.6%)	0.27
Daily use of OCS	16 (11.9%)	7 (11.9%)	0.72
Molecular targeting drugs	1 (0.8%)	5 (8.5%)	< 0.01
Laboratory data			
White blood cell (/ml)	6989.1 ± 256.4	7743.9 ± 318.3	0.09
Eosinophil (%)	6.3 ± 0.7	4.9 ± 0.7	0.21
Eosinophil count (/ml)	525.6 ± 111.0	396.5 ± 64.5	0.45
Abbreviations: ICS: inhaled corticosteroid, LABA: long-acting β2 adrenergic agonist, LAMA: long-acting muscarinic antagonist, LTRA: leukotriene receptor antagonist, OCS: oral corticosteroid			

Impact Of Sex Differences On Obesity For Patients With And Without Asthma

Given the sex difference in obesity-induced asthma severity augmentation [13, 16, 22], the impact of obesity on parameters of pulmonary function testing was examined by sex in patients with and without asthma.

In patients of both sexes without asthma, FVC, %FVC, FEV_1, and $\%FEV_1$ were not different between non-obese patients and obese patients. In patients with asthma, in males, FVC was significantly lower in obese patients than in non-obese patients ($p = 0.04$), but %FVC was not different. FEV_1 and $\%FEV_1$ were significantly lower in obese patients than in non-obese patients ($p = 0.01$, 0.02 respectively). In females, FVC, %FVC, FEV_1, and $\%FEV_1$ were significantly lower in obese patients than in non-obese patients (all $p < 0.01$) (Table S1). On multivariate analysis, FEV_1, but not FEV, were still significant after adjustment for confounding factors including age and smoking history in both sexes (male: 0.18 [0.01–0.35], $p = 0.04$, female: 0.09 [0.01–0.17], $p = 0.03$) (Table S2).

Discussion

In the present single-center, cross-sectional study, the impact of obesity on pulmonary function was examined in Japanese adult patients with asthma. Notably, all of the patients followed by pulmonary physicians with the disease name of asthma covered by medical insurance in our institute from 2005 to 2019 were included, and 193 patients definitely diagnosed as having asthma by a pulmonary physician were analyzed; this approach likely led to decreased rates of misdiagnosis and selection bias. In addition, the data of pulmonary function testing for patients without asthma were used for the comparison, which facilitated the evaluation of the effect of obesity in patients with asthma along with those without asthma. The present results showed that pulmonary functions were lower in patients with asthma than in those without asthma. Furthermore, FEV_1 and FVC was negatively correlated with BMI in patients with asthma even though the correlation coefficients were relatively low, and obese patients with asthma showed significantly lower pulmonary functions than non-obese patients with asthma. Importantly, those differences were not seen between obese patients without asthma and non-obese patients without asthma.

Pulmonary function testing is an important examination for patients with asthma [6] because decreased pulmonary function, including FEV_1 and FVC, is associated with the severity of asthma. For example, decreased FEV_1 is a major characteristic of severe asthma, along with increased symptoms and exacerbations [23, 24]. Reduced FVC is also correlated with uncontrolled asthma defined by emergency department visits [25] and is significantly lower in asthma patients with severe airflow obstruction than in those with moderate airflow obstruction based on their baseline FEV_1 [26]. These data and the present results show that decreased pulmonary function is an important parameter reflecting asthma severity.

There is increasing evidence that obesity is related to the severity of asthma. In Japan, To et al examined the impact of obesity defined by a BMI greater than 25 kg/m^2 in 492 patients with severe asthma, and they found that obesity was associated with severe acute exacerbations in females [16]. We also reported that the annual exacerbation rate was significantly higher in overweight patients than in non-overweight patients, although there was no significant difference in pulmonary function given the small sample size [17]. In a cohort of 28,016 patients with asthma in the USA, seasonal exacerbations were significantly increased in overweight patients defined as those whose BMI was 25–29.9 kg/m^2 and obese patients,

defined as those whose BMI was greater than 30 kg/m^2, than in those with normal BMIs [27]. Importantly, in patients with asthma, exacerbations contribute to excess lung function decline [28, 29]. Therefore, the present results indicating that obesity is related to lower pulmonary function in patients with asthma is consistent with those findings.

The present results showed that obese patients with asthma had significantly lower pulmonary functions than non-obese patients with asthma (Table 3). A population-based cohort study in the Netherlands of the epidemiology of obesity showed that in 472 patients with asthma, obesity (BMI > 30 kg/m^2) was associated with lower FEV_1 and FVC than non-obesity [30]. Another longitudinal study also showed that obesity was significantly associated with decreased lung function (FVC), and the risk was higher in patients with asthma than in those without asthma,[31] which supported the present results. As the mechanisms, decreased responses to corticosteroid therapy, which is one of the factors related to the severity of asthma with obesity [21, 32], might be involved. Indeed, the present study showed that obese patients with asthma were more often treated by high-dose ICS than non-obese asthma patients, even though their pulmonary functions were low (Table 5). In addition, the previous Japanese study mentioned above showed that obesity does not affect pulmonary functions when focusing on severe asthma patients treated by high-dose ICS compared to those without obesity, in contrast to the present study which included mild to severe patients with asthma. These data remind us of the possibility that obese patients with asthma might generally include more patients with resistance to corticosteroid therapy and, consequently, have reduced pulmonary function compared to non-obese patients with asthma.

Pulmonary function, especially FEV_1, was significantly lower in obese patients with asthma than in non-obese patients with asthma, and those differences were not seen between obese patients without asthma and non-obese patients without asthma (Tables 3, 4). Furthermore, the obesity-induced decline of FEV_1 was greater than that of FVC on correlation analysis and multivariate analysis (Fig. 3a, 3b, Table 4). As for the mechanisms, augmented airway and systemic inflammation induced by obesity should be considered [17, 19, 20, 33], but airway dysanapsis, a physiological incongruence between the growth of lung parenchyma and the caliber of the airway, might be involved in obese patients with asthma [34]. Several studies reported that airway dysanapsis, explained by high FVC, normal FEV_1, and low FEV_1/FVC, was more frequently seen in overweight/obese asthmatic children than in those of normal weight [35, 36]. Because obesity is associated with the incidence of asthma itself [37], the result that obesity decreased FEV_1 featured by airway dysanapsis, considered a diagnostic factor of asthma, might be consistent.

Interestingly, the present results showed that FVC was also lower in obese patients with asthma than in non-obese patients with asthma, and these differences were not seen between obese patients without asthma and non-obese patients without asthma (Table 3). These results cannot be explained by the obesity-induced physical effects caused by mobility regulation of the diaphragm and thorax by increased fat[37, 38]. While we do not yet know the reason, obesity appears to have a specific impact on FVC along with FEV_1 in patients with asthma, but not those without asthma. Sex differences are normally involved

in the pathophysiology of asthma with obesity, and studies in Japan of asthma incidence and exacerbation related to obesity showed more impact in female than male patients [16, 39]. In the present study, there were no sex differences in pulmonary function associated with obesity (Table S1, S2)

The present study has several limitations. First, the background characteristics were different between patients with asthma and those without asthma. This might have affected the impact of obesity on the results of pulmonary function testing in patients with and without asthma. We also exploratory performed propensity score matched (PSM) method to mitigate the risk of confounding due to differences between patients without and those with asthma and it was used to balance the groups with respect to known confounders including BMI, age, sex, and smoking history. The results of the PSM group comparison were also reproductively confirmed that pulmonary functions were lower in patients with asthma than those without asthma. And, FEV_1 and FVC were significantly lower in obese patients with asthma than in non-obese patients with asthma. Those obesity-associated differences were not observed in patients without asthma (data not shown), which we believe reduced the biases, were confirmatory. Second, patients with chronic obstructive pulmonary disease may not have been completely excluded from the patients with asthma and patients without asthma, which would have affected the results of pulmonary function testing. Third, it is unclear that the observed obesity-induced decreased pulmonary function in asthma contributes to poor outcomes, especially mortality. This was not assessed, and recently, overweight was found to be associated with improvement of long-term survival in a Japanese cohort [40]. Finally, the present study involved a small number of patients at a single hospital with limited ethnic diversity. To confirm the validity of the present results, multicenter, prospective studies designed with appropriate controls and larger numbers of patients should be performed.

Conclusion

The present cross-sectional study showed that parameters of pulmonary function testing including FEV_1 and FVC were significantly lower in obese patients with asthma than in non-obese patients with asthma. These differences were not observed between obese patients without asthma and non-obese patients without asthma. High-dose inhaled corticosteroid therapy was more common in obese patients with asthma than in non-obese patients, which might indicate obesity-related corticosteroid resistance as the mechanism.

Methods

Patients and diagnosis

To identify patients with asthma who underwent pulmonary function testing, 716 adult patients (age >18 years) who were followed by pulmonary physicians at Saga University Hospital with the International Classification of Diseases, 10^{th} Revision, Clinical Modification [ICD-10-CM] code of asthma (J459) from January 2005 to March 2019 were individually reviewed by a pulmonary physician. A total of 523 patients were excluded because of lack of medical information, other pulmonary diseases including

COPD, chronic cough, bronchitis, and 193 patients with asthma diagnosed by pulmonary physicians with reference to the Global Initiative for Asthma (GINA) guidelines [41] were identified (Figure 1). On the other hand, to identify patients without asthma who underwent pulmonary function testing, 2,710 adult patients (age >18 years) who underwent pulmonary function testing at Saga University Hospital for preoperative examination in 2019 were individually reviewed by a pulmonary physician. A total of 551 patients were excluded because of lack of medical information or complications of asthma and other diseases that contribute to decreased pulmonary function, such as chronic obstructive pulmonary disease (COPD), interstitial pneumonia, pleural effusion, and neuromuscular diseases in the medical record. Patients with a history of lung resection were also excluded, and 2,159 patients were identified as patients without asthma (Figure 2). An obese patient was defined as one whose BMI was greater than 25 kg/m^2, referring to criteria in Japan [42].

Ethics approval and consent to participate

The present study was approved by the ethics committees of Saga University Hospital (approval number: 2021-11-R-01) and was performed in accordance with the 1964 Declaration of Helsinki. Informed consent of the participants was obtained in the form of opt-out on the website of Saga University Hospital, and those who declined participation were excluded.

Data collection

The information for smoking history, comorbidities, asthma-related therapy, and laboratory data were collected from patients' medical records at the nearest time point when pulmonary function testing was performed. The data for BMI and age were obtained at the time when pulmonary function testing was performed. Comorbidities and allergic comorbidities were diagnosed by the physicians. Cardiovascular disease included coronary artery disease, valvular disease, cardiac arrhythmias such as atrial fibrillation, and chronic heart failure diagnosed by echocardiography. Treatments for asthma were also selected at the physicians' discretion, and doses of inhaled corticosteroid (ICS) were divided into 3 levels, low, moderate, and high, referring to the GINA guidelines [41]. In patients with asthma, the spirometry parameters were obtained in a stable condition, and the %FVC and $\%FEV_1$ predicted were calculated with the LMS method referring to the recommendations of the Japanese Respiratory Society.[43]

Statistical analysis

Quantitative data are expressed as means ± standard error (SE). The clinical data were analyzed by Student's *t*-test for continuous variables or the chi-squared test for categorical variables. For correlation analysis, Pearson's correlation coefficient was calculated to determine whether it was zero. Multivariate analysis with linear regression analysis for continuous variables and logistic regression analysis for categorical variables was performed, and the regression coefficient (β) were calculated. Comparing patients with and without asthma, FVC and FEV_1 were individually adjusted by confounding factors including BMI, age, sex, and smoking history. Comparing non-obese patients with asthma and obese

patients with asthma, and non-obese patients without asthma and obese patients without asthma, FVC and FEV_1 were individually adjusted by confounding factors including age, sex, and smoking history. Significance was considered if a p value was less than 0.05. Statistical analysis was performed with JMP Pro version 14.2.0 software (SAS Institute Inc., Cary, NC, USA).

Declarations

Data availability

The datasets used and analyzed during the current study are available from the corresponding author on reasonable request.

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Not applicable

Author Contributions

HT, KT and YK conceived and designed the project. HT, YK and HS analyzed clinical data. RT advised for statistical analysis. HT, KT and NA prepared the manuscript with input from all other authors. HS, SK and NA performed final check of manuscript. All authors have read and approved the manuscript.

Competing interests

The authors declare that they have no competing interests.

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Figures

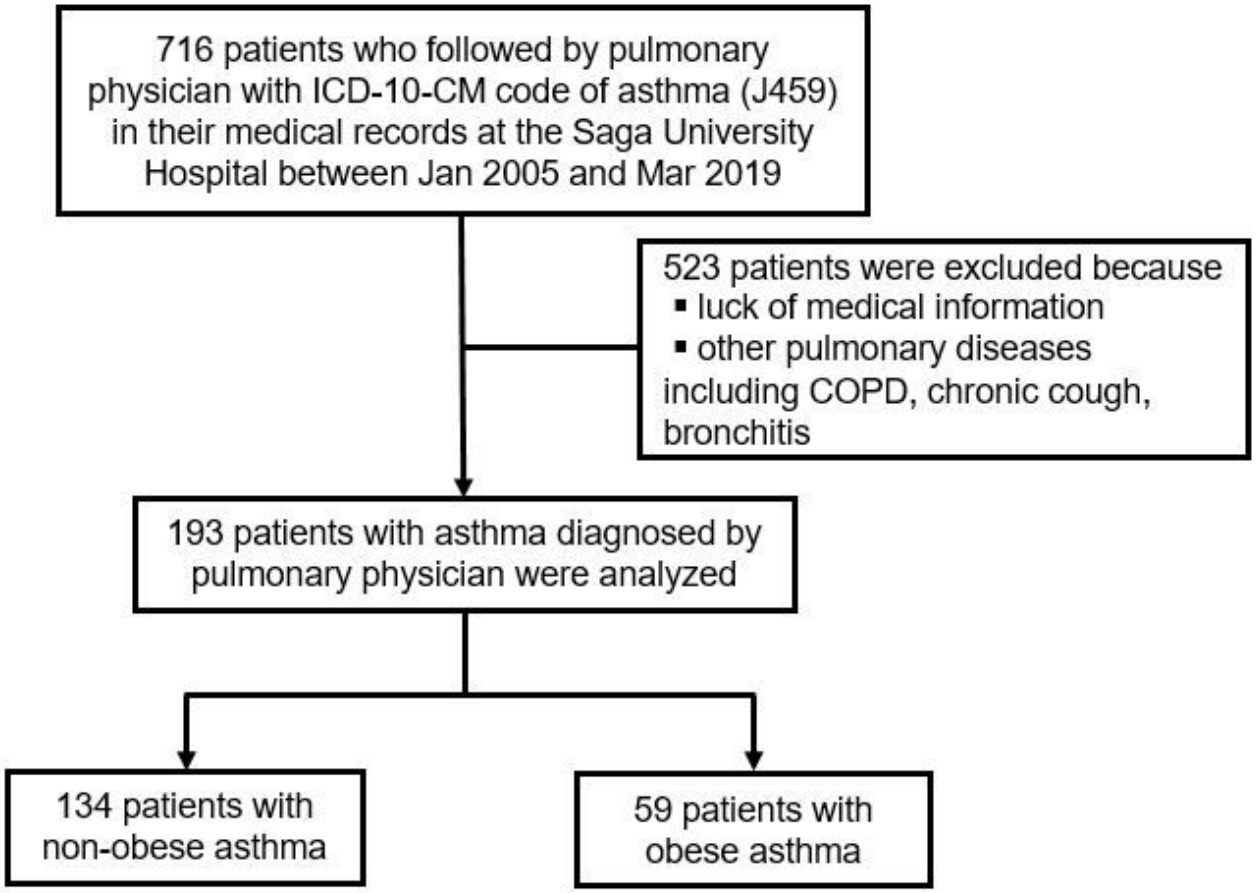

Figure 1

Study design. Patients with asthma

A total of 193 patients with asthma diagnosed by a pulmonary physician were identified, and 134 non-obese patients with asthma and 59 obese patients with asthma were analyzed.

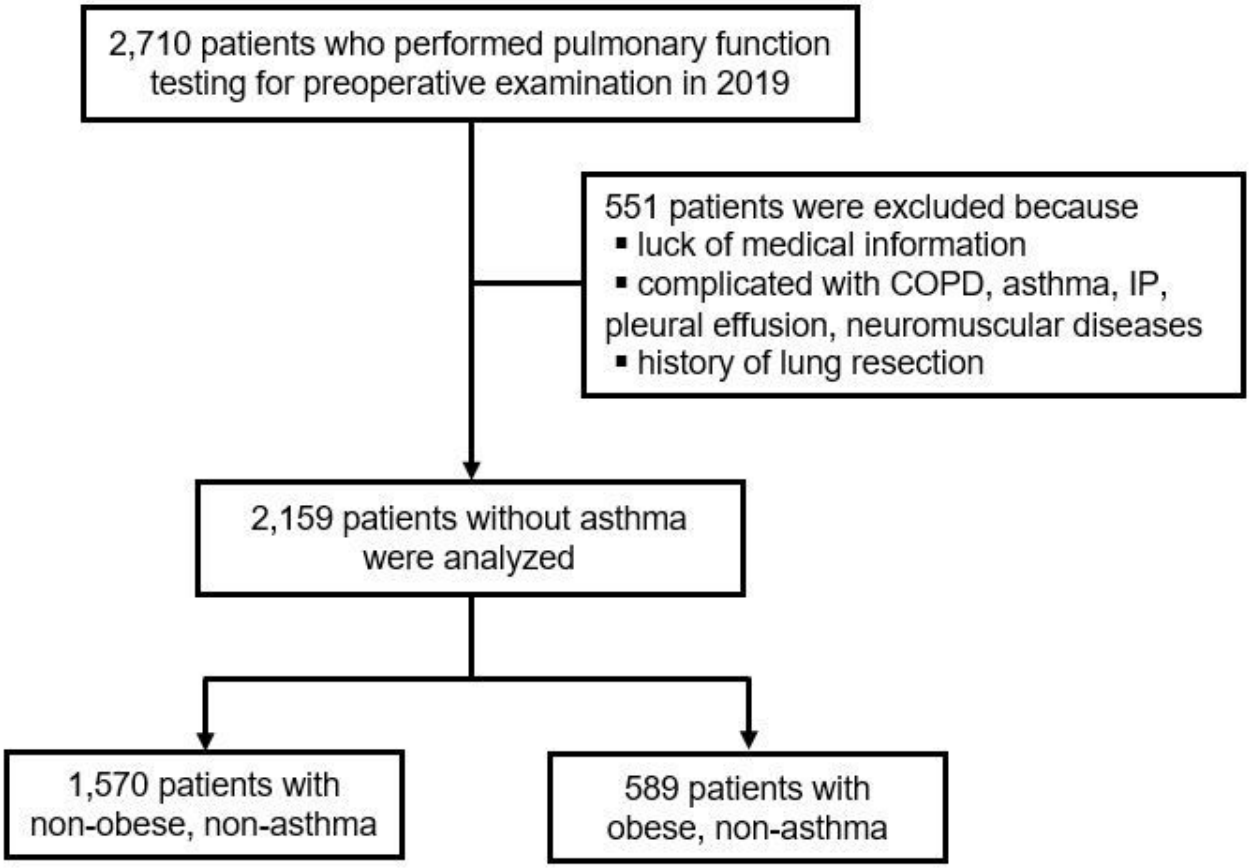

Figure 2

Study design. Patients without asthma

A total of 2,159 patients without asthma were extracted, and 1,570 non-obese patients without asthma and 589 obese patients without asthma were analyzed.

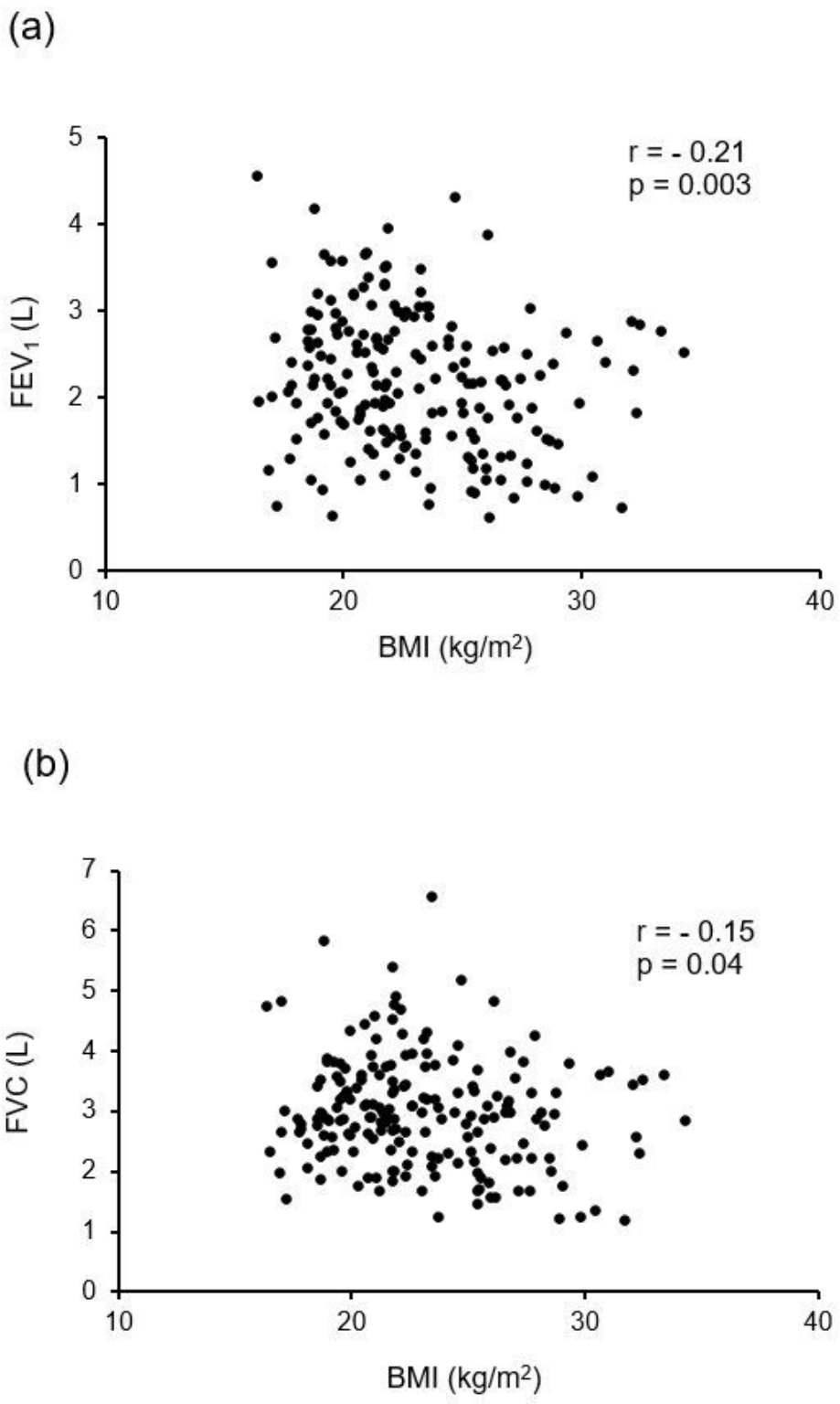

Figure 3

Correlation between forced expiratory volume in 1 second, forced vital capacity and the body mass index in patients with asthma

(a) The forced expiratory volume in 1 second is significantly negatively correlated with the body mass index (r = - 0.21, p = 0.003).

(b) The forced viral capacity is correlated with the body mass index even though the correlation coefficient is low (r = - 0.15, p = 0.04).

Supplementary Files

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