Impact of morbid obesity on pulmonary function

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Abstract

Introduction: While studies have examined the effects of mild-moderate obesity on pulmonary function, there have been no large studies that have specifically looked at the impact of morbid obesity, especially with regard to the influence of gender and age. Some studies have demonstrated an increased incidence of asthma among obese patients, suggesting that this may be due to a decrease in airway caliber, although there is little evidence to support this. The aim of this study was to examine the impact of morbid obesity on pulmonary function with a particular emphasis on the influence of gender and age. By including a large number of patients in the study, the goal was to also get some insight into the association between obesity and asthma.

Material and methods: Pulmonary function test results were collected on 433 patients with a body mass index BMI ≥40 kg/m^2 who were being evaluated for bariatric surgery between January 2001 and August 2006.

Results: BMI had a significant impact on the forced vital capacity (FVC), forced expiratory volume in the first second (FEV_1), expiratory reserve volume (ERV), residual volume (RV), total lung capacity (TLC) and partial pressure of oxygen in arterial blood (PaO_2). In addition, there were significant differences in pulmonary function based on the gender and age of the patient. FVC, FEV_1, FEV_1/FVC, ERV, TLC and PaO_2 were all significantly reduced in males compared to females. Similarly, FVC was significantly reduced among subjects >40 years when compared to those ≤40 years.

Conclusions: Our study demonstrated the effects of morbid obesity on pulmonary function. It also established the greater impact of obesity in men, which along with the absence of an obstructive impairment in almost all our patients suggests that asthma maybe being over-diagnosed in obese patients, especially women.

Key words: morbid obesity, pulmonary function, asthma.

Introduction

About 25% of the adult U.S. population is obese while at least 5% is morbidly obese. Over the past several decades, this has been an increasing trend with some projections estimating that 20% of the population will be morbidly obese by the year 2010 [1]. This growing problem will have an enormous impact on healthcare costs in the future. Annual medical spending due to obesity already accounted for more than 9% of the total annual U.S. medical expenditures in 1998 [2].

each unit increase in BMI [3-9]. The effect of obesity on diffusing capacity of the lung for Carbon Monoxide (DLCO) appears to be somewhat more unpredictable with some studies demonstrating an increased DLCO in obese patients, possibly related to the increased pulmonary blood volume seen in such patients [3, 10, 11].

The most common complaint of obese patients is dyspnea. Obesity can lead to dyspnea by a number of different mechanisms – a reduction in respiratory system compliance, lung volumes and peripheral airway diameter and an increase in pulmonary blood volume, ventilation-perfusion mismatch and airway hyperresponsiveness [4-9, 12-14]. Respiratory system compliance is reduced mainly by the increased pulmonary blood volume, fatty infiltration of the chest wall and compression of the thoracic cage by the excess soft tissue. Thus, men who tend to have a central (upper body) pattern of obesity can be expected to have lower lung volumes and poorer pulmonary compliance compared to women who tend to have a peripheral (lower body) pattern of obesity [15-18]. However, this has not been conclusively demonstrated in the literature. For instance, a recent study showed no significant differences between men and women for the effects of BMI on TLC, FVC, RV, ERV or DLCO [3].

Body fat distribution in obese patients varies with age. Middle-aged men often undergo an increase in abdominal girth with relatively little change in body mass. As a result, obesity can be expected to have a greater impact on pulmonary function in older patients [19].

Although the association between obesity and asthma remains controversial, wheezing is sometimes seen in obese patients and asthma is often diagnosed [20]. This maybe due to decrease in airway caliber secondary to the low lung volumes rather than to increased airway hyperresponsiveness [20, 21]. Obese women are more than twice as likely as men with equivalent BMIs to be diagnosed with asthma [22]. While this may be related to the effects of sex hormones, differences in the way obesity impacts men and women cannot be excluded. For instance, King et al. have suggested that central

raises the possibility that obese patients are being over-diagnosed with asthma, possibly based more on their subjective symptoms rather on objective data.

Most previous studies have examined the effects of mild and moderate obesity on pulmonary function. In addition to examining the effects of morbid obesity on pulmonary function, we also looked at the impact of age and gender on pulmonary function in the morbidly obese patient. We thereby hoped to provide some insight into the association between obesity and dyspnea as well as possible explanations for the increased incidence of asthma diagnoses among obese patients, especially women.

Material and methods

This was a retrospective study of all patients who were being evaluated for bariatric surgery at Westchester Medical Center between January 2001 and August 2006. All such patients were required to undergo a pulmonary function test (PFT) as part of their pre-operative evaluation. The height and weight of each patient was recorded at the time of their PFT, from which their BMI was calculated. Spirometry was done with the patient in a sitting position using a wedge spirometer with an X-Y recorder. Lung volumes were calculated either by the Nitrogen washout method or by Plethysmography. An arterial blood gas was obtained and the DLCO was corrected for Hemoglobin. The studies were performed with the Medgraphics Elite series plethysmograph using the BreezeSuite software. Predicted values were based on the Crapo reference values. Institutional Review Board approval was obtained.

Only subjects with a BMI $\geq$40 kg/m^2 were included in the analysis. Subjects were not excluded based on their smoking history or a diagnosis of asthma. Use of controller or rescue medications did not lead to exclusion from the study although patients had to have been off bronchodilators for at least 4 hours prior to pulmonary function testing. All values recorded were those prior to the administration of a bronchodilator, if used during the test.

The data were analyzed using Student's t-tests or Wilcoxon rank-sum tests to compare the patient

Table I. Patient demographics and baseline characteristics of the 433 patients

Male	108
Female	325
Age ≤40 years	189 (45 males, 144 females)
Age >40 years	244 (63 males, 181 females)
Age (years)	43±11 (17-76)
Height (cm)	167±9 (147-198)
Weight (kg)	142.2±27.2 (90.3-254.5)
BMI (kg/m^2)	50.7±8 (40-90.5)

BMI – body mass index
Data are presented as mean ± SD (range) or No.

Table II. Gender differences in pulmonary function and gas-exchange of subjects with BMI ≥40 kg/m^2

	Male (n=108)	Female (n=325)	Value p
FVC (% predicted)	78.3±11.0	86.7±14.8	<0.0001
FEV_1 (% predicted)	77.6±12.1	86.1±15.2	<0.0001
FEV_1/FVC (%)	80.0±6.1	82.6±5.1	<0.0001
ERV (% predicted)	26.0±13.3	31.8±18.0	0.0005
RV (% predicted)	103.1±33.8	98.8±37.4	0.297
TLC (% predicted)	87.1±13.4	92.5±16.0	0.0007
DLCO (% predicted)	78.7±13.9	78.9±13.6	0.897
PaO_2 (mm Hg)	74.8±8.3	84.1±10.6	<0.0001
$PaCO_2$ (mm Hg)	40.5±3.8	38.3±3.4	<0.0001

FVC – forced vital capacity, FEV_1 – forced expiratory volume in the first second, ERV – expiratory reserve volume, RV – residual volume, TLC – total lung capacity, DLCO – diffusing capacity of the lung for carbon monoxide, PaO_2 – partial pressure of oxygen in arterial blood, $PaCO_2$ – partial pressure of carbon dioxide in arterial blood
Data are presented as mean ± SD

characteristics between two groups. Multiple linear regression analyses were conducted to investigate the effects of BMI, age and gender on pulmonary function. Significance was taken as p ≤0.05 for all tests.

Results

The records of a total of 472 morbidly obese patients who underwent PFTs as part of their evaluation for bariatric surgery were reviewed. Of these, 433 patients had a BMI ≥40 kg/m^2. Of the 433 patients (325 female and 108 male), 244 were older than 40 years while 189 were ≤40 years (Table I). The mean BMI was 50.2±7.5 kg/m^2 among the female subjects and 52.1±9.3 kg/m^2 among the male subjects (p=0.03). There was no difference between the BMIs of subjects >40 years and ≤40 years of age (50.7±7.8 vs. 50.7±8.3, p=0.97).

The mean FEV_1/FVC ratio was 80.0±6.1 among the male patients while it was 82.6±5.1 among the female patients. Only 9 patients (4 male, 5 female) had an FEV_1/FVC ratio <70%.

There were significant differences in pulmonary function based on the gender and the age of the patient. FVC, FEV_1, FEV_1/FVC, ERV, TLC and PaO_2 were all significantly reduced in males as compared to females (Table II). Similarly, FVC was significantly reduced among the subjects >40 years when compared to those ≤40 years (Table III). Although there was a significant reduction in the FEV_1/FVC and PaO_2 among subjects >40 years, these were not considered clinically significant since these values are absolute and not percent predicted values, and are expected to decline with increasing age.

Table IV shows the results of multiple linear regression analyses with each pulmonary function parameter as the outcome variable and BMI, gender and age as the predictor variables. BMI had a significant impact on FVC, FEV_1, ERV, RV, TLC and PaO_2 while male gender had an impact on FVC, FEV_1, FEV_1/FVC, ERV, TLC and PaO_2 and age >40 years had an impact on FVC, FEV_1 and PaO_2.

Discussion

To our knowledge, this is the largest known study of pulmonary function in the morbidly obese population. We confirmed the well-described effects of obesity on various aspects of pulmonary function, especially on FVC, FEV_1, TLC, ERV and PaO_2. While some studies have demonstrated an increased DLCO in obese patients, we saw a minimal decrease in the DLCO (corrected for hemoglobin) in our patients. This decrease is possibly secondary to the slight reduction in alveolar volume seen in these patients due to their obesity. Another interesting observation that has often been seen in the morbidly obese population is that the RV is increased [26, 27]. This has been thought to be due to the fact that in morbidly obese people, the chest wall pressure-volume curve below FRC becomes flatter limiting the action of action of expiratory muscles, thus increasing the RV. However, we saw no increase in the RV in our patients, similar to the findings of Zerah et al. [12].

In terms of the quantitative impact of obesity on the various aspects of pulmonary function, our study was similar to those previously described in the literature. Increasing BMI had no impact on the RV, a minimal impact on the TLC (0.3% decrease per unit increase in BMI), a slightly greater impact on the FVC and FEV_1 (0.6% decrease per unit increase in BMI) and the greatest impact on the ERV (2.7%

decrease per unit increase in BMI).

Studies have shown that males have a predominantly upper-body or central (abdominal) distribution of fat while females have a predominantly lower-body or peripheral (gluteal and femoral) distribution [17, 18]. The distribution of body fat has been shown to affect pulmonary function, with an abdominal fat pattern leading to a greater compression of the thoracic cage by the excess soft tissue [15, 16, 19, 28]. Our study confirmed these effects, with a significantly greater impact of the obesity on pulmonary function (FVC, FEV_1, FEV_1/FVC ratio, TLC, ERV and PaO_2) in the male subjects compared to the female subjects.

Obese patients tend to have an increase in abdominal girth with age. This effect was probably the reason for the small but significant decrease in the FVC and FEV_1 that we saw in our morbidly obese population older than 40 years.

Studies have shown that obese patients, especially women, are much more likely to be diagnosed with asthma by their physicians than non-obese patients [20, 25, 29]. A recent meta-analysis of seven studies demonstrated an increased incidence of asthma in obese individuals although all the studies used either self-reporting, physician diagnosis or use of asthma medications as the criteria for a diagnosis of asthma [30]. However, the majority of obese patients diagnosed with asthma have no objective evidence of airway hyper-responsiveness [20, 24]. Our study showed that the morbidly obese population did not have a baseline decrease in their FEV_1/FVC ratio or an increase in their RV. Within the obese population, male patients tended to have a slightly but significantly lower FEV_1/FVC ratio than the female patients, although both the mean values were within the normal range and only 9 patients (2%) had an FEV_1/FVC ratio <70%. Thus our study does not lend support to the thought that obesity leads to an increased incidence of asthma. In addition, if the obesity was contributing to a decreased baseline airway caliber leading to increased airway hyper-responsiveness, then based on our data, an obese male patient would be more likely to develop asthma than an obese female subject. This is contrary to published literature which has shown that women are more than twice as likely as men with equivalent BMIs to be diagnosed with asthma [22]. Therefore, while other mechanisms might be the cause of the increased association between obesity and asthma, our study suggests that there might be an over-diagnosis of this condition in the obese population.

There are some limitations to this retrospective study. A patient's smoking history and a diagnosis of asthma were not used to exclude patients from the analysis. However despite this, we did not see any evidence of obstructive airway disease in our patients, thus only further adding evidence to our belief that asthma is over-diagnosed in the obese population. Although, a normal FEV_1/FVC ratio does not exclude a diagnosis of asthma in these patients, one of the main theories behind the increased diagnosis of asthma in these patients has been a decreased baseline caliber of the airways. We saw

Table III. Age differences in pulmonary function and gas-exchange of subjects with BMI ≥40 kg/m²

	Age ≤40 years (n=189)	Age >40 years (n=244)	Value p
FVC (% predicted)	87.4±13.2	82.4±14.9	0.0003
FEV_1 (% predicted)	85.6±13.8	82.7±15.6	0.050
FEV_1/FVC (%)	82.6±5.6	81.4±5.3	0.023
ERV (% predicted)	31.4±17.2	29.5±17.0	0.244
RV (% predicted)	101.0±39.2	99.0±34.4	0.579
TLC (% predicted)	92.3±14.8	90.3±16.1	0.181
DLCO (% predicted)	79.6±11.8	78.2±14.8	0.307
PaO_2 (mm Hg)	84.9±11.2	79.5±10.0	<0.0001
$PaCO_2$ (mm Hg)	38.3±3.5	39.2±3.7	0.017

FVC – forced vital capacity, FEV_1 – forced expiratory volume in the first second, ERV – expiratory reserve volume, RV – residual volume, TLC – total lung capacity, DLCO – diffusing capacity of the lung for carbon monoxide, PaO_2 – partial pressure of oxygen in arterial blood, $PaCO_2$ – partial pressure of carbon dioxide in arterial blood
Data are presented as mean ± SD

Table IV. P-values from multiple linear regression analyses of pulmonary function and gas-exchange

	BMI	Male gender	Age >40 years
FVC	0.0001	<0.0001	0.0002
FEV_1	<0.0001	<0.0001	0.050
FEV_1/FVC	0.315	<0.0001	0.027
ERV	0.471	0.004	0.271
RV	0.030	0.195	0.560
TLC	0.0004	0.006	0.198
DLCO	0.243	0.822	0.348
PaO_2	0.123	<0.0001	<0.0001
$PaCO_2$	0.042	<0.0001	0.018

FVC – forced vital capacity, FEV_1 – forced expiratory volume in the first second, ERV – expiratory reserve volume, RV – residual volume, TLC – total lung capacity, DLCO – diffusing capacity of the lung for carbon monoxide, PaO_2 – partial pressure of oxygen in arterial blood, $PaCO_2$ – partial pressure of carbon dioxide in arterial blood

no evidence of this in our study. Another limitation was the fact that we did not have the measurements of patients' waist:hip ratios available and thus had to assume that our male subjects had a predominantly central pattern of obesity and our female subjects had a predominantly peripheral pattern of obesity. However, since our population of morbidly obese patients was not unique in any fashion and given the large number of patients in our study, we can probably accurately assume that the patterns of obesity in our patients were not significantly different from that described traditionally in the literature.

In conlusion our study provides some insights into the effects of morbid obesity on pulmonary function. It also potentially raises the possibility that obese patients, especially women, are being over-diagnosed with asthma. A prospective study with anthropometric analysis and bronchial provocation testing may help to resolve these issues.

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