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A comparative study of clinical characteristics and quality of life between obese and non-obese asthmatics

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ABSTRACT

Background: Asthma is a chronic inflammatory disorder arising from heterogenic gene-environment interactions. Obesity is one of the risk factors for asthma. Evidence shows that several inflammatory markers are active in obese and overweight patients. There is growing evidence that obesity can affect the course and control of asthma. **Aims and Objectives:** The study was done to compare clinical characteristics, spirometry variables, and quality of life among obese and non-obese asthmatics. **Materials and Methods:** The study was a cross-sectional comparative study conducted in a tertiary care hospital using a semi-structured pre-tested questionnaire. Detailed history and examination, quality of life using AQLQ score, and spirometry severity were assessed. **Results:** A total of 80 patients, 40 obese asthmatics and 40 non-obese asthmatics were included in the study. The study found that most subjects belonged to the age group of 31–40 years and body mass index of 30.89 ± 1.94 among the obese group and 23.19 ± 3.77 in non-obese, respectively. The majority had symptoms of breathlessness and wheezing in both groups. The mean forced expiratory volume in 1 s was similar in both groups. However, the mean forced vital capacity was lower in the obese group. The asthma control test (ACT) score was poorer in obese group as compared to non-obese group (7 in obese vs. 15 in non-obese). The dosage of inhaled steroids was higher in the obese group. **Conclusion:** The symptomatology was similar among the two groups. Restrictive abnormalities, higher asthma symptoms with lower ACT scores, and higher dosages of medications were seen in obese asthmatics as compared to non-obese patients.

Key words: Asthma; Airway control; Airway remodelling; Obesity

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INTRODUCTION

Asthma is a chronic inflammatory disorder arising from heterogenic gene-environment interactions. It features variable airway obstruction and bronchial hyperresponsiveness. Asthma is characterized by recurrent attacks of breathlessness and wheezing, which vary in severity and frequency from person to person.[1]

Asthma can have multiple triggers which include allergenic agents (mites, dust, molds, dander, pollens, etc.), and non-allergenic agents (viral infections, exercise, cold air, etc.).[2] In some cases, other factors such as rhinosinusitis, occupational exposures, obesity, comorbidities such as gastroesophageal reflux, and drugs can also worsen asthma.[3]

Obesity is one of the risk factors for asthma. Obesity is also a state of inflammation. Evidence shows that several inflammatory mediators are active in obese and overweight subjects. Similarly, evidence also shows that asthma is associated with active inflammation.[4] Many studies have reported that obesity-related asthma is more often non-atopic, whereas few have suggested it worsens atopy.[5]

The relationships, interactions, and associations between obesity and asthma are complex. Although the direct

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relationships between asthma and obesity remain controversial, there is growing and significant evidence that the two conditions have multiple areas of interplay, particularly in the inflammatory microenvironment.[6]

Asthma causes symptoms such as wheezing, shortness of breath, chest tightness, and cough that vary over time in their occurrences, frequency, and intensity. These symptoms are associated with variable airflow due to bronchoconstriction (airway narrowing), airway wall thickening, and increased mucus. Some variation in airflow can also occur in people without asthma, but it is greater in untreated.[7]

Keeping this in mind, the study was undertaken to compare and contrast the clinical characteristics, lung function abnormalities, quality of life, and medication usage among obese and non-obese asthmatics.

Aims and objectives

- To assess clinical characteristics among obese and non-obese asthmatics
- To assess lung function abnormality and spirometry pattern among obese and non-obese asthmatics
- To assess the inhaled corticosteroid usage among obese and non-obese asthmatics

MATERIALS AND METHODS

This study was conducted in a tertiary care hospital using a semi-structured pre-tested questionnaire. It is a cross-sectional comparative study, among 80 patients with 40 obese and 40 non-obese asthmatics. The data were collected from patients attending the outpatient department and inpatients in the hospital fulfilling the inclusion–exclusion criteria. The study was conducted over a period of 2 years.

Inclusion criteria included subjects who were ≥18 years old, patients with diagnoses of asthma based on clinical history, and or spirometry results. Ethical committee clearance was obtained (SIEC/SIMS and RC/35(69)/2021) and only those willing to participate were included in the study. Exclusion criteria included those with obstruction or diseases of the upper airways, other airway diseases such as bronchiectasis/chronic obstructive pulmonary disease, and other pulmonary diseases such as pulmonary tuberculosis/interstitial lung diseases/obstructive sleep apnea. Those with other comorbidities such as a cardiac disease/stroke were also excluded from the study.

A detailed history regarding the onset and progression of the disease, symptomatology, and examination findings were noted. Spirometry assessment was done as per standard protocol. Height, weight, and body mass index (BMI) were noted and patients were categorized into obese and non-obese asthmatics based on BMI. Patients with BMI of more than 30 kg/m^2 were taken as obese and those lesser than 25 kg/m^2 were included in non-obese. The severity of asthma symptoms was assessed using an asthma control test (ACT) score with 5 being the lowest score and 25 being the highest score. Medication usage for asthma was noted. Data were analyzed using SPSS 21 software. The Chi-square test was applied to check for significance value, and $P<0.05$ was taken as significant.

RESULTS

The subjects were divided into two groups of obese and non-obese with BMI mean and standard deviation for the obese group being 30.89±1.94, and for non-obese 23.19±3.77, respectively. The study consisted of subjects with a mean age of 40.58±10.1 in the obese group and 42.5±9.41 non-obese group. The age group shows no significant differences (P=0.38). The study found that the majority of subjects belonged to the age group of 31–40 years. However, there was a significant difference in the distribution of patients after 40 years of age. It was found that 30.5% in the obese group were 41–50 years of age group, whereas 38.8% were in 51–60 years in the non-obese group (P=28.0, Chi-square – <0.00).

In the study, majority of the participants were males in non-obese, whereas in obese, the gender distribution was equal.

In this study, predominant symptoms were breathlessness (100% in the obese and 83% in the non-obese), and wheezing (100% in the obese group, and 83% in the non-obese group). Other symptoms of cough, expectoration, and chest tightness are shown in Table 1.

The spirometry analysis among the subjects showed that mean forced expiratory volume in 1 s (FEV1) was similar in both groups. However, mean forced vital capacity (FVC) was lower in the obese group (2.03 vs. 2.36) as compared to non-obese which was statistically significant (P=0.022) (Table 2). In the study, the predominant pattern was a mixed pattern (obstruction+restriction) 28 (70%) in the obese group, and a normal pattern 20 (50%) in the non-obese group.

Table 1: Distribution of subjects based on symptoms

Symptoms	Obese n=40	(%)	Non-obese n=40	(%)	P-value
Cough	30	75	33	83	0.384
Expectoration	20	50	32	81	0.006
Breathless ness	40	100	33	83	0.011
Wheezing	40	100	33	83	0.011
Chest tightness	33	83	32	81	0.759

Table 2: Distribution of subjects based on spirometry analysis

Parameter	Group	Mean	Standard deviation	P-value
FEV1 (pre)	Obese group	1.7983	0.52235	0.014
	Non-obese group	1.7969	0.77952	
V1 predicted	Obese group	2.6875	0.67769	0.93
	Non-obese group	2.4619	0.56217	
FVC (pre)	Obese group	2.0300	0.54645	0.022
	Non-obese group	2.3694	0.90810	
FEV1/FVC	Obese group	75.908	4.2553	0.013
	Non-obese group	74.736	9.7347	
FEF 25–75% (pre)	Obese group	1.7125	0.84230	0.70
	Non-obese group	1.6567	0.91700	

FVC: Forced vital capacity, FEV1: Forced expiratory volume in 1 s, FEF: forced mid-expiratory flow

Table 3: Distribution of patients based on spirometry pattern

Pattern	Obese n (%)	Non-obese n (%)	
Mixed	28 (70)	9 (22)	Chi-square – 36.57 P≤0.001
Obstruction	4 (11)	11 (27)	
Restriction	4 (11)	0 (0)	
Small airway obstruction	4 (11)	0 (0)	
Normal	0 (0)	20 (50)	

Table 4: Dosage of steroids among obese and non-obese patients

Drug history	Obese n (%)	Non-obese n (%)	
High dose ICS	16 (39)	4 (11)	Chi-square – 7.589 P=0.022
Low dose ICS	14 (36)	19 (47)	
Medium dose ICS	10 (25)	17 (42)	

ICS: Inhaled corticosteroid

The next common pattern in each group was obstruction (obese=4/11%); (non-obese=11/27%) (Table 3).

The ACT score was poorer among obese asthmatics (score of 7) as compared to the score among non-obese asthmatics (score of 15). A statistically significant (P=0.022) difference was seen in the dosing of inhaled steroids among the two groups. High-dose inhaled steroids were commonly prescribed in obese patients (39% in obese vs. 11% in non-obese) whereas the dose of steroids prescribed was low in the non-obese group (47% in non-obese patients vs. 36% in the obese group) (Table 4).

DISCUSSION

In this study, mean age of the obese group was 40.58±10.1 and the non-obese group was 42.5±9.4. Most subjects belonged to the age group of 31–40 years. In a similar study done by Rajasekhar which included 100 patients, the mean age of patients in the obese group was 40.27±7.583 and in the non-obese group 38.74±7.583 years and most of the subjects belonged to group 20–59 years.[8] The mean age of asthmatics between the two groups in our study was not statistically significant. However, it was lower than the age in which studies have shown the highest prevalence that is between 45 and 60 years.[9] Asthma can affect people in any stage of life from infancy to old age.[9] The clinical symptoms through younger (20–40 years), middle (41–65 years), and older adults (more than 66 years) can vary due to differences in pathophysiology.[9] Clinical differences are present in those who develop childhood or adolescent asthma as compared to young adulthood asthma (long-standing asthma) or those who develop asthma after 40 years of late-onset asthma (LOA). Those with LOA are less likely to have atopy, more frequently to have severe symptoms, and less responsive to standard therapy as compared to the other two groups.[10]

With regards to symptoms, the predominant symptom was breathlessness and wheezing in the obese and the non-obese, followed by chest tightness and cough with expectoration among all patients. Among the obese group, breathlessness was more prevalent as compared to the non-obese group, whereas cough with expectoration was more common in non-obese asthmatics. A study by Kasteleyn et al., which included 472 patients found that predominant symptoms were breathlessness 114 (46%) and wheezing 88 (36%) in the obese group, and breathlessness 78 (35%) and wheezing 72 (32%) in non-obese.[11] In another study done by Beuther and Sutherland, breathlessness was present in all patients in the obese group and 9% in the non-obese group.[12] The next common symptom in their study was cough with expectoration. Obesity alters the mechanics of the lungs and chest wall due to fat deposits. There is also an associated increase in inflammatory cytokines and immune cells. These factors lead to increased breathlessness, wheezing, and orthopnea among obese patients.[5]

Studies have shown that restrictive lung function defect is common among obese patients, especially among males due to android fat deposition. As mentioned previously fat deposits in the chest wall limit lung expansion and

diaphragmatic contractility which results in a lower FVC.[13] Huang et al. found that BMI was the best predictor of poor lung function among their study patients. They found a lower FVC and an accelerated FVC decline in obese asthmatics.[14] Our study also had similar findings. Lung function abnormalities were more common among obese asthmatics than non-asthmatics with restriction and mixed patterns being common among them.

Given all the pathogenetic mechanisms mentioned earlier that include alteration in lung mechanics, and inflammatory cytokine changes obese asthmatics tend to have more severe disease, more frequent exacerbations, and reduced response to medications. This eventually leads to poorer quality of life among obese asthmatics as compared to non-obese asthmatics.[5] A study done by Maalej et al. found that obese asthmatics had 6 times more frequent emergency visits, 5 times more frequent hospitalizations, increased missed work days, and poorer control of asthma and quality of life.[15] Our study also found lower ACT scores among obese asthmatics as compared to non-obese asthmatics, indicating more severe symptoms at presentation.

Inhaled steroids are the mainstay in achieving control in patients with asthma. Studies have shown a greater prevalence of higher doses of inhaled steroids in obese asthmatics as compared to non-obese asthmatics. This is due to the higher severity of disease in this group of patients.[16] Camargo et al. demonstrated that the time from treatment onset to peak improvement in FEV1 was longer in obese patients as compared to non-obese implying a slow response to therapy.[17] Another meta-analysis showed that obese asthmatics were more likely to use rescue short-acting beta-2 agonists, and maintenance oral steroids as compared to non-obese asthmatics.[18] Our study too had increased prescription of high-dose inhaled corticosteroids in obese patients as compared to non-obese patients. This could be attributed to more subjective feelings of breathlessness, poor quality of life, and more prevalent spirometry abnormalities among these patients in the study.

Limitations of the study

The study has few limitations. Small sample size and contributions of comorbidities like gastroesophageal reflux or sleep apnea towards poor control was not assessed. Additionally, follow up of patients to see if reduction in BMI improved the asthma control would help understand the influence of obesity on asthma better.

CONCLUSION

Obese asthmatics have complex pathogenetic mechanisms in play that make their asthma more severe as compared to non-obese asthmatics. These changes lead to more symptoms, frequent exacerbations, higher medication requirements, and poorer quality of life. Lung function abnormalities also show varied changes primarily in the form of lower FVC, restriction, or mixed obstruction with restriction which is not frequently seen in non-obese asthmatics. Although symptomatology was the same our study also found higher symptom severity, poorer lung function, and higher medication usage among the obese group as compared to the non-obese group.

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SR and **RB**- Idea of the study, literature review, data compilation, and article preparation; **RH** and **VN**- Data collection; **RBK**- Data analysis with statistical application of results.

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