RESEARCH ARTICLE

Effect of Obesity and Hypertension on Pulmonary Functions

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ABSTRACT

Background: Obesity is a serious nutritional problem in the world and a source of multiple co-morbid conditions in metabolic and cardiopulmonary disorders. It offers mechanical obstruction to the movements of the body and the respiratory system. Further, it is hypothesized that the lung functions can be reduced to varying extents in obese hypertensive and obese normotensive subjects compared to normal subjects in the same age group.

Aims & Objective: The present work is undertaken to study the effect of obesity and hypertension on the pulmonary functions.

Materials and Methods: By using a computerized spirometer, the pulmonary functions FVC, FEV_1 and PEF were evaluated in 20 obese normotensive subjects (BMI of 33.04 ± 2.42) and 20 obese hypertensive subjects (BMI of 32.89 ± 2.58) & 20 normal (BMI of 22.39 ± 1.65) male subjects in the age group of 40 to 60 years. Spirometry was performed by the subject in sitting posture. Trails were given for the subject to familiarize with the equipment, then they were asked to perform 3 trails and best of the three readings were computed for analysis.

Results: The percentage of predicted value of FVC, FEV_1 and PEF in normal subjects were 94.82 ± 13.07; 96.62 ± 15.19; 91.49 ± 23.21 and the corresponding values for obese normotensive subjects were 84.45 ± 15.75; 89.35 ± 16.48; 81.14 ± 20.54 and in obese hypertensive subjects were 76.60 ± 13.38; 78.65 ± 17.62; 71.09 ± 19.29 respectively.

Conclusion: There was a significant decline in FVC, FEV_1 and PEF in obese normotensive & obese hypertensive subjects when compared to normal individuals. Further, the extent of decline was greater in obese hypertensive when compared to obese normotensive subjects. It can be concluded that obesity and hypertension will have an adverse impact on pulmonary functions.

Key Words: Pulmonary Function; Obesity; Hypertension

INTRODUCTION

Obesity is a major preventable cause of death. Prevalence of obesity is increasing in both adults and children. It is viewed as a serious public health problem.[1] Obesity was perceived as a symbol of prosperity during some period of history & even now in some parts of the world. Obesity has stigmatized the western world and a phenomenal increase in its incidence in developing countries.[2]

Obesity can lead to multiple co-morbid conditions. No direct correlation is seen between excessive energy or lipid intake and damage to trachea, bronchi or alveoli.[3] Subjects with excessive body weight tend to have reduced cardiopulmonary fitness and are chronically hypo ventilated. This is indicated by a reduction of aerobic capacity and exercise tolerance. These limitations are due to heavy thoracic wall, increased abdominal fat and diminished compliance of the lungs. Obesity and hypertension are common manifestations with the advancement of age. These are due to an interplay of variety of endocrine, genetic and metabolic mechanisms. Lung functions and respiratory muscle strength are closely associated with body weight and lean body mass. Initially, a higher BMI in young individuals may result in better pulmonary function. However, due to greater fat deposition, increased BMI is associated with compromised pulmonary function.[4]

There is substantial increase in morbidity and mortality in obese hypertensive in comparison to obese normotensive & healthy individuals. Earlier studies have given conflicting results about the influence of obesity and hypertension on pulmonary functions.[5-7] In the present study, efforts are made to compare pulmonary functions in obese normotensive, obese hypertensive subjects with non-obese South Indians.

MATERIALS AND METHODS

The study was carried out in MS Ramaiah teaching hospital. Twenty male normotensives & twenty male hypertensive subjects with a BMI of ≥ 30 kg/m^2 & BP ≥ 140/90 mmHg were recruited as subjects. They were in the age group of 40 to 60 years. Twenty age and anthropometrically matched males attending the OPD for master health checkup, satisfying the inclusion and exclusion criteria were recruited as controls. Subjects with coronary artery disease, respiratory diseases, skeletal abnormalities and smokers were excluded from the study. Ethical clearance was obtained from the institutional ethical review board. The subjects participated in this study by giving written informed consent. The medical and dietary history, use of drugs, personal habits of the participating subjects was recorded by using a standardized questionnaire. Later, they were subjected to general physical examination. BMI, waist circumference and the blood pressure was measured. Height was measured in meters using a stadiometer and weight was measured in kilograms using sensitive balance. BMI was calculated by using the formula weight in Kg / (Height in meter)^2. Waist circumference was measured at a level midway between the lowest rib and the iliac crest, and the hip circumference at the level of the greater trochanter and Waist hip ratio (WHR) was calculated. Blood pressure was recorded in supine position using mercury sphygmomano-meter and readings were expressed as mmHg.

Spirometry was performed under standard conditions of ambient temperature and pressure in sitting position. Tests were carried out between 9-30 am and 12-30 pm in order to avoid circadian influences. The subjects were instructed to rest for 15 minutes prior to the test. The area in which the tests were carried out was quiet; temperature and humidity were maintained at constant levels. After instruction and practice attempts, final measurements were done by using a spirometer (Model RESMED'S Spirobank G, MIR SRL, ROMA, ITALY) with nose closed using a nose clip. Each subject performed 3 trials (with at least two reproducible and acceptable maneuvers) according to the American Thoracic Society recommendations.[8] The values were considered reproducible when

the difference between two highest recorded values was not more than 5%.[9] The best value of FVC, FEV_1, FEV_1% and PEF were considered for evaluation and comparison.

Statistical Analysis

The actual and the predicted values of pulmonary function & the percentage of the predicted value were expressed as Mean ± SD. The percentage change of the predicted values in normal, obese & obese hypertensive subjects were compared by ANOVA. Post hoc analysis was done to assess the significant difference between groups. P value less than 0.05 was considered significant.

RESULTS

Age, blood pressure & the anthropometric parameters are shown in Table 1. There was a significant difference in BMI, hip & waist circumference, waist hip ratio between the controls & obese subjects. There was a significant difference in systolic & diastolic BP between obese normotensive & obese hypertensive subjects.

Pulmonary functions like FVC, FEV_1, FEV_1% & PEF are indicated in Table 2. The predicated value for the same age group, anthropometric measurements and ethnicity were obtained. The two values were used to get percentage of predicted value. The differences in percentage of predicated values were used for comparison. There was a significant reduction in FVC, FEV_1 & PEF in obese normotensive & obese hypertensive subjects when compared to their normal counter parts.

Table-1: Comparison of Anthropometric Data between Controls, Obese Normotensives & Obese Hypertensive Subjects

Study Characteristics	Controls	Obese Normotensive	Obese Hypertensive
Age in years	47.20 ± 6.70	49.50 ± 7.35	49.48 ± 6.43
BMI (kg/m^2)	22.36 ± 1.54	33.19 ± 2.59*	32.89 ± 2.58*
Waist (cm)	81.85 ± 3.49	115.20 ± 5.28*	113.09 ± 5.34*
HIP (cm)	92.12 ± 3.91	112.95 ± 4.67*	109.62 ± 7.19*
W/H ratio	0.88 ± 0.01	1.02 ± 0.03*	1.03 ± 0.03*
SBP (in mmHg)	123.90 ± 9.14	127.85 ± 9.28	158.76 ± 10.4*#
DBP (in mmHg)	78.90 ± 6.66	82.60 ± 4.01	95.90 ± 4.92*#

*# P<0.05; * Comparison between controls, obese normotensive and obese hypertensive; # Comparison between obese normotensive and obese hypertensive

Table-2: Comparison of Change in Percentage of Pulmonary Functions between Controls, Obese Normotensive & Obese Hypertensive Subjects

PFT	Controls	Obese Normotensive	Obese Hypertensive
FVC (L)	94.82 ± 13.07	84.45 ± 15.75*	76.60 ± 13.38*
FEV_1 (L)	95.62 ± 15.19	89.35 ± 16.48*	78.65 ± 17.62*
FEV_1%	101.48 ± 7.36	101.47 ± 20.50	102.10 ± 12.76
PEF (L/sec)	91.49 ± 23.21	81.14 ± 20.54*	71.09 ± 19.29*

* P value <0.05, (Comparison between control & obese normotensive & obese hypertensive subjects)

DISCUSSION

In present study, pulmonary functions were estimated in normal, obese normotensive & obese hypertensive subjects in the comparable age groups. The comparison of the percentage predicted values indicate that there was a significant reduction in these parameters in obese normotensive & obese hypertensive when compared to controls. Our observations are consistent with the earlier reports.[6,7] These reports have established a negative correlation between fat mass, central distribution of fat with lung functions. Our observations are suggestive of obstructive and restrictive type of airway dysfunction in obese normotensive & obese hypertensive subjects. King G.G et al. have reported a reduction in lung volume and size of airway with an increase in BMI.[10]

In obese individuals, mechanical obstruction due to accumulation of abdominal fat hinders the decent of diaphragm. The problem is compounded by a reduction in the compliance of the chest wall, the work of breathing and an elastic recoil of lungs.[11] The reduction in the chest wall compliance, reduction in the pulmonary function is attributable to the deposition of fat in the abdominal cavity and thoracic cage. The reduction in elasticity of the lung is an important factor causing the reduction in the size of the airways.

Obesity produces an increase in total blood volume and cardiac output because of high metabolic activity of excessive fat.[12] Pressure and volume overloads are present in obese hypertensive subjects leading to mixed eccentric and concentric form of left ventricular hypertrophy (LVH) and left ventricular diastolic

dysfunction.[12,13] This causes pulmonary hypertension and congestive heart failure, which are major pathophysiological sequelae for increased pulmonary airway resistance leading to low lung volumes.[14]

The earlier studies do not provide sufficient information about the alteration in lung functions (FVC, FEV_1, FEV_1% and PEF) in middle-aged obese hypertensive and normotensive subject compared to non-obese individuals of comparable age group. Further it is possible that the lung functions can be reduced to varying extents in obese hypertensive and obese normotensive subjects compared to normal subjects. In our study there was a significant decline in the pulmonary functions in obese and obese hypertensive subjects when compared to normal subjects. The extent of reduction was greater in obese hypertensive as compared to obese normotensive subjects. However the difference was not statistically significant. The declining trends in lung function in obese hypertensive subjects suggest that hypertension can adversely affect the lung function in addition to obesity. A study with a larger sample size can conclusively establish the role of hypertension in altering the lung functions.

CONCLUSION

There was a significant decline in FVC, FEV1 and PEF in obese normotensive & obese hypertensive subjects when compared to normal individuals. Further, the extent of decline was greater in obese hypertensive when compared to obese normotensive subjects. It can be concluded that obesity and hypertension will have an adverse impact on pulmonary functions.

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