Current Research in Diabetes & Obesity Journal
ISSN: 2476-1435

Juniper Publishers
key to the Researchers

Short Communication
Volume 17 Issue 1 - August 2023
DOI: 10.19080/CRDOJ.2023.17.555952

Curr Res Diabetes Obes J

The Relationship Between Body Fat Distribution on Pulmonary Function Test in Morbidly Obese Patients

Pérez-Cruz Elizabeth[1,2,3], Aldo Alfredo Pérez Manjarrez[4] and Ortiz-Gutiérrez Salvador[1,2]

[1]*Department Metabolic Unit and Nutritional Support. Hospital Juárez de México, México City*

[2]*Obesity Clinic. Hospital Juárez de México, México*

[3]*National Autonomous University of Mexico, México*

[4]*Department Internal Medicine. Hospital Juárez de México, México*

Submission: August 06, 2023; **Published:** August 18, 2023

***Corresponding author:** Elizabeth Pérez-Cruz, MD, Instituto Politécnico Nacional Núm. 5160, Col. Magdalena de las Salinas, C.P. 07760, Del Gustavo A. Madero, México City, México, Email: pece_liz@hotmail.com

Abstract

Introduction: Obesity can affect pulmonary function by different mechanisms. We aim was to evaluate if there are any differences in pulmonary volumes, measured through spirometry, and fat mass distribution by sex and its correlation with obesity.

Methods: A retrospective cohort that included 114 patients with obesity. Anthropometric measurement, fat mass, muscle mass through bioelectrical impedance analysis and spirometry data were measures.

Results: 82% were female, the mean age was 40.8±10 years old for all participants. 9.3 % of patients with obesity type III showed an FVC <80%; meanwhile, patients with obesity type II did not show alterations in pulmonary volumes (p=0.02). Differences by sex were not observed. A higher presence of altered spirometry was recorded among those with central obesity (elevated waist-to-hip ratio), 7.9% abnormal spirometry tests compared to 0.9% in patients with normal WHR (p=0.04).

Conclusion: The negative contribution of obesity on pulmonary volumes seems to be related to the presence of central distribution of body fat. No differences were found between sex

Keywords: Obesity; Fat mass; Spirometry; Respiratory function tests

Abbreviation: FEV: Forced Expiratory Volume; FVC: Forced Vital Capacity; BIA: Bioelectrical Impedance Analysis; WHR: Waist-to-Hip Ratio; BMI: Body Mass Index

Introduction

Obesity is a public health problem worldwide, and it could affect pulmonary function by different mechanisms [1]. Adipose tissue secretes various adipokines with inflammatory consequences; such effects have been related to changes in pulmonary function [2]. People living with obesity develop a reduction in thoracic distensibility due to the limited expansion of the thoracic cavity and diaphragm; in addition, has been described the presence of microatelectasis on lung bases resulting in altered oxygenation and respiratory distress [3].

Some studies showed that the higher obesity is, the more damaged breathing physiology and exhale volumes. Nevertheless, it has been observed that weight does not always explain the differences in pulmonary function. There is a lack of research describing changes in normal pulmonary function related to sex and body composition and distribution. Therefore, this study aimed to evaluate if there are any differences in pulmonary volumes, measured through spirometry, and fat mass distribution by sex and its correlation with obesity.

Methods

A retrospective cohort that included male and female patients between 18 and 55 years old. Patients were diagnosed with obesity based on body mass index (BMI): type II obesity = BMI >35 plus comorbidity, and type III obesity = BMI ≥40 with or without comorbidity. Those patients with a record of previous pulmonary disease, smoking index = >20 packages/year, or biomass exposure index = >200 hours/year were excluded, as well as patients with spirometry contraindications.

Anthropometric measurements

Weight (kg), height (m), waist circumference (cm), and hip circumference (cm) were measured following a standardized technic. BMI and waist-to-hip ratio (WHR) were computed. Bioelectrical impedance analysis (BIA) was used to determine fat mass (%) and muscle mass (Kg) with a TANITA® scale, model BC-418.

Spirometry data

Forced vital capacity (FVC) and forced expiratory volume in one second (FEV1) were used as measures of ventilatory function. Values of ≥80% were considered normal. The measurements were obtained by a baseline forced spirometry; patients were sitting. The spirometry was performed and assessed by an expert for meeting acceptability and repeatability criteria and following the standards of the American Thoracic Society. The spirometer was a Spiro senseâ of Burdick, calibrated after each test.

Statistical analysis

Continued variables were expressed as mean ± SD, and data comparison was performed by a t-student test for independent samples. Categorical variables were expressed as frequencies and percentages, and a chi-squared test was computed to observe differences. Pearson correlation coefficient (r) was used to study the degree of linear association between quantitative variables. Statistical significance was accepted with a p-value of <0.05. Data were analyzed using SPSS software (version 21.00, SPSS Inc). The study was approved by the institution's committees of ethics and research.

Results

The study included a total of 146 eligible participants. 114 cases were analyzed, and 82% were female. The mean age was 40.8±10 years old for all participants. Characteristics of participants are shown in table 1. In relationship with pulmonary volumes and obesity, the mean of FVC was 102±10.8% for patients with obesity type II; meanwhile, for patients with obesity type III FVC mean was 98.73±15.1% (p=0.06). FEV1 for patients with obesity type II and III was 100.4±10.6% and 96±15.6%, respectively (p=0.06). The mean of the FEV1/FVC ratio was 91±6.3% for patients with obesity type II and 91.73±7.2% for patients with obesity type III. Any pulmonary volume with a value of <80% was considered altered. 9.3 % of patients with obesity type III showed an FVC <80%; meanwhile, patients with obesity type II did not show alterations in pulmonary volumes (p=0.02). Differences by sex were not observed.

Table 1: Demographic characteristics and spirometry data (by sex).

Variable	Total (n=114)	Female (n=94)	Male (n=20)
Age (years)	40.8±10	39.9±10	43.5±11
Weight (Kg)	114±19	112±17	125.6±7
BMI (kg/m^2)	44.4±6	44.7±6	43.8±7
Waist circumference (cm)	119±18	116.7±18	130.6±14
Hip circumference (cm)	130.9±14	132.1±14	119.5±12.7
WHR	0.91±0.15	0.89±0.16	1.01±0.10
Fat mass (%)	37.6±7.2	38.5±7	29.4±6
Muscle mass (Kg)	55.8±11	57.3±8	51.6±9
FVC (predicted %)	99.9±15.1	101±14.2	97±14.5
FEV1 (predicted %)	97.3±15.5	96.3±15.8	97.2±14.7
FEV1/FVC (predicted %)	91.5±7	94±72	93±19

BMI: Body Mass Index; WHR: Waist-to-Hip Ratio; FVC: Forced Vital Capacity; FEV1: Forced Expiratory Volume in the First Second; FEV1/FVC, ratio of FEV1 to FVC.
Values are expressed as mean and standard deviation or frequencies and percentages.

The presence of central obesity was established using WHR. Values of WHR of >0.94 for men and >0.85 for women were considered central obesity. A higher presence of altered spirometry was recorded among those with central obesity, 7.9% (n=9) abnormal spirometry tests compared to 0.9% (n=1) in patients with normal WHR (p=0.04). Significative correlations between pulmonary volumes and anthropometric data are shown in table 2.

How to cite this article: Pérez-Cruz E, Aldo Alfredo Pérez M, Ortiz-Gutiérrez S. The Relationship Between Body Fat Distribution on Pulmonary Function Test in Morbidly Obese Patients. Curre Res Diabetes & Obes J 2023; 17(1): 555952.DOI:10.19080/CRDOJ.2023.17.555952

Table 2: Pulmonary volumes and anthropometric data correlation.

Variable	Correlation Coefficient (r)	p-Value
BMI (kg/m^2)	0.376	0.06
WHR	-0.222	0.04
Fat mass (%)	0.148	0.05
Muscle mass (Kg)	0.47	0.32

BMI: Body Mass Index; WHR: Waist-to-Hip Ratio

Discussion

Some studies have reported that obesity impacts pulmonary capacity. It has been found that BMI directly influences the lung's ability to expand and its elasticity [4,5]. In comparison, our study did not find any relationship between BMI and pulmonary capacity, but we observed a significant relationship with central obesity established by WHR. This brings the idea that central distribution of body fat might be more important than the relationship between total weight with height. White adipose tissue secretes a significant amount of adipokines, an important number of which have proinflammatory and proatherogenic activity. WHR could be helpful as an indirect biomarker of white adipose tissue mass and central obesity. WHR has been related to dysglycemia and several other metabolic abnormalities [6].

Although some authors such as Sutherland et al6 found no association between body fat distribution and trunk fat with respiratory volumes. Our findings are consistent with Collins et al. [7] who reported a decrease in lung volumes relation to body fat distribution in upper body, which interestingly, decreased in patients with higher degree of obesity. One hypothesis for our findings is that pulmonary tissue could be increased due to chronic inflammation of the perivascular adipose tissue. Thus, gas exchange is altered, increasing respiratory load and decreasing pulmonary reserve.

The limitations of our study are the higher proportion of females and the differences between obesity types. It is necessary to perform more studies with a more homogenous population.

Conclusion

The negative contribution of obesity on pulmonary volumes seems to be related to the presence of central distribution of body fat instead of total weight or BMI. No differences were found between sex.

Acknowledgments: The authors thank all participants in the study

Competing Interests: The authors declare no conflict of interest.

References

1. Kim SH (2015) Maturity-onset diabetes of the young What do clinicians need to know. Diabetes Metabolism Journal 39(6): 468-477.
2. Brodosi L, Baracco B, Mantovani V, Pironi L (2021) Neurod1 mutation in an Italian patient with maturity onset diabetes of the young 6: A case report. BMC Endocrine Disorders 21(1): 202.
3. Malecki MT, Jhala US, Antonellis A, Fields L, Doria A, et al. (1999) Mutations in NEUROD1 are associated with the development of type 2 diabetes mellitus. Nat Genet 23(3): 323-328.
4. Kristinsson SY, Thorolfsdottir ET, Talseth B, Steingrimsson E, Thorsson AV, et al. (2001) MODY in Iceland is associated with mutations in HNF-1alpha and a novel mutation in Neuro-D1. Diabetology 44(11): 2098-2103.
5. Naya F J, Huang HP, Qiu Y, Mutoh H, DeMayo F J, et al. (1997) Diabetes, defective pancreatic morphogenesis, and abnormal enteroendocrine differentiation in beta2/neurod-deficient mice. Development 11(18): 2323-2334.
6. Massari ME, Murre C (2000) Helix-loop-helix proteins regulators of transcription in eucaryotic organisms. Mol Cell Biol 20(2): 429-440.
7. Romer A I, Singer R A, Sui L, Egli D, Sussel L, et al. (2019) Murine perinatal β-cell proliferation. and the differentiation of human stem cell–derived insulin-expressing cells require neurod1.Diabetes 68(12): 2259-2271.
8. Richards S, Aziz N, Bale S, Bick D, Das S, et al. (2015) Standards and guidelines for the interpretation of sequence variants: A joint consensus recommendation of the American College of Medical Genetics and genomics and the Association for Molecular Pathology. Genetics in Medicine 17(5): 405-424.
9. Gonsorčíková L, Průhová Š, Cinek O, Ek J, Pelikánová T, et al. (2008) Autosomal inheritance of diabetes in two families characterized by obesity and a novel H241Q mutation inneurod1. Pediatric Diabetes 9(4 pt 2): 367-372.
10. Abreu G de, Tarantino R M, Cabello P H, Zembrzuski V M, da Fonseca, et al. (2019) The first case of neurod1-mody reported in Latin America. Mol Genet Genomic Med 7(12): 2323-2334.
11. Liu L, Furuta H, Minami A, Zheng T, Jia W, et al. (2007) A novel mutation ser159pro in the NEUROD1/beta2 gene contributes to the development of diabetes in a Chinese potential mody family. Molecular and Cellular Biochemistry 303(1-2): 115-120.
12. Chapla A, Mruthyunjaya M D, Asha H S, Varghese D, Varshney M, et al. (2014) Maturity onset diabetes of the young in India - a distinctive mutation pattern identified through targeted next-generation sequencing. Clinical Endocrinology 82(4): 533-542.

How to cite this article: Pérez-Cruz E, Aldo Alfredo Pérez M, Ortiz-Gutiérrez S. The Relationship Between Body Fat Distribution on Pulmonary Function Test in Morbidly Obese Patients. Curre Res Diabetes & Obes J 2023; 17(1): 555952.DOI: 10.19080/CRDOJ.2023.17.555952

13. Brodosi L, Baracco B, Mantovani V, Pironi L (2021) Neurod1 mutation in an Italian patient with maturity onset diabetes of the young 6: A case report. BMC Endocrine Disorders 21(1): 202.

14. Ming Qiang Z, Yang Li D, Ke H, Wei W, Jun Fen F, et al. (2019) Maturity onset diabetes of the young (MODY) in Chinese children Genes and clinical phenotypes. Journal of Pediatric Endocrinology and Metabolism 32(7): 759-765.

DOI: 10.19080/CRDOJ.2023.17.555952

How to cite this article: Pérez-Cruz E, Aldo Alfredo Pérez M, Ortiz-Gutiérrez S. The Relationship Between Body Fat Distribution on Pulmonary Function Test in Morbidly Obese Patients. Curre Res Diabetes & Obes J 2023; 17(1): 555952.DOI:10.19080/CRDOJ.2023.17.555952