Pulmonary Function in Morbid Obesity: Influence of Sex and Body Distribution

Alberto Herrejón, Ignacio Inchaurraga and Julio Palop

Department of Pneumology, Dr. Peset University Hospital, Valencia, Spain

Corresponding author: Herrejón A, Department of Pneumology, Dr Peset University Hospital, Avda Gaspar Aguilar, 9046017-Valencia, Spain, Tel: 34961622438; E-mail: herrejon_alb@gva.es

Received date: July 26, 2016; Accepted date: August 26, 2016; Published date: August 29, 2016

Abstract

Objective: To describe the respiratory functional alterations that occur in patients with morbid obesity (MO) and the possible influence of gender and distribution of body fat, measured as an index waist/hip ratio (wai/hip).

Method: Prospective study in 2 years of anthropometric and functional parameters breathing in MO, determining age, body mass index (BMI), waist, hip, wai/hip index, blood gas values, flow-volume curve, plethysmography, diffusion of CO and maximum inspiratory (MIP) and expiratory (PEM) pressures. The results between men and women and the relationship between respiratory parameters and wai/hip index are compared.

Results: We studied 171 patients, 80 men and 91 women, with 44 ± 12 years, weighing 130 ± 22 kg, with BMI of 48 ± 6 Kg/m² and wai/hip index of 0.989-0.097 ± 48. There is a decrease in lung function parameters in the MO, showing men MIP worse, but better MEP, than women. Hypoxemia is common (55%), related to an older and worse flows and static volumes. Hypercapnia is 15% more common in men, and is related to the decrease in expiratory flows and increased residual volume. Wai/hip index does not correlate with respiratory parameters in the MO.

Conclusions: Morbid obesity affects respiratory blood gas and has functional alterations. There are differences between men and women in the presence of hypercapnia and maximal respiratory pressures, without influencing the type of obesity measured with the wai/hip index.

Keywords: Morbid obesity; Lung function; Type of obesity

Introduction

MO is a common clinical situation defined by a BMI ≥ 40 kg/m². Two types of obesity are recognized, android or central and peripheral or ginoide obesity [1]. Central obesity is the one with rate of wai/hip ratio>1 in men and >0.9 in women [2]. It is associated with increased cardiovascular risk such as hypertension, diabetes or dyslipidemia and also increases the risk of two major respiratory problems: Obesity hypoventilation syndrome and sleep obstructive apneas syndrome [3]. Obesity, in terms of the distribution of body fat, has been linked to alterations in the pulmonary function [4,5].

Respiratory function of patients with MO is poorly studied, usually in limited samples. Two patterns of respiratory functional impairment in the OM are described. In the first pattern functional impairment, which is proportional to the degree of obesity is increasing diffusion of carbon monoxide (DLCO) and DLCO/alveolar ventilation (KCO) relationship, with reduced expiratory reserve volume (ERV). The second pattern, typical of most obese patients, is characterized by a decrease in vital capacity (VC), total lung capacity (TLC) and maximum ventilation voluntary [6]. However, it has not assessed the possible influence of sex and type of obesity on pulmonary function of patients with MO.

The aim of our work is the study of the parameters of conventional lung function in a large group of patients with OM and its relationship to the degree and type of obesity. The importance of the issue is due to the close relationship between lung function and morbid obesity, analyzed a large sample of subjects of both sexes, which condition their clinical situation and are likely to improve with weight reduction.

Material and Methods

Patients

We have studied prospectively patients with MO in a university hospital with an area of 321,361 inhabitants, for 5 years.

Method

We included consecutive patients with MO, excluding those with a history of respiratory disease known. We studied anthropometric and functional parameters.

Registered anthropometric parameters were: age (years), weight (kg), height (cm), BMI (kg/m²), waist circumference (wai) at the navel (cm) and hip circumference (hip) at the level of the greater trochanters (cm) and wai/hip ratio (wai/hip) index, both perimeters made in raising the standing position, if necessary, the abdominal fat mass.

The respiratory function parameters studied were: blood gas analysis, curve forced expiratory flows through flow/volume, static lung volumes with body plethysmography, CO diffusing capacity, maximum respiratory muscle pressures, inspiratory (MIP) and expiratory (PEM). Arterial blood gas analysis was performed in supine, determining pH, PO₂ (mmHg), PCO₂ (mmHg) and sat. O₂ (%) with the ABL 300 gas analyzer Radiometer Co. (Copenhagen), according to
Results

At the level of TLC in cm H\textsubscript{2}O, measuring total lung capacity (TLC) described by Morales [12].

...and hypercapnia if PCO\textsubscript{2} is greater than 45 mmHg. The flow/ volume curve was performed using a pneumotachograph system 2800 SensorMedics (SensorMedics Corporation, California 1984), following the European regulations [8] and registering forced vital capacity (FVC) (L), forced expiratory volume in one second (FEV\textsubscript{1}) (L/sec) and FEV\textsubscript{1}/FVC. Static volumes were determined by body plethysmography (Body Box 2800 SensorMedics), measuring residual volume (RV) (L) and functional residual capacity (FRC) (L). The diffusion of CO was obtained by the method of breathing unique with the team SensorMedics 2100, following the recommendations of the ATS [9], analyzing the DLCO (mmol/kPa.min) and KCO (mmol/kPa.min/L). Spirometric, plethysmographic and diffusion of CO values are expressed as percentages of the theoretical values, as CECA tables [10]. Maximal respiratory pressures were determined in mouth, nasal occlusion using a portable digital manometer (MicroMPM of SensorMedics, Brethoven Netherland) according to the method of Black and Hyatt [11]. PEM was measured at the level of TLC in cm H\textsubscript{2}O, hands on her cheeks to ignore the effect of the buccinator and PIM (expressed in absolute values) in cm H\textsubscript{2}O from RV, with a leak of a 1 mm diameter to avoid suction effect. To calculate the percentage of the theoretical reference used values described by Morales [12].

A descriptive study was conducted of the entire group and separated by sexes, including mean, standard deviation (SD), range and percentiles level 25, 50 and 75%, using the Statistical Package for Social Science (SPSS 9.0 for Windows). In comparisons of means he was used the Student t test, valuing equality of variances with the Levene test. For qualitative variables the Chi-square test was performed with Yates correction and Fisher’s exact test. Correlations were established by Pearson r. The level of statistical significance was of p<0.05.

Results

We studied 171 patients with MO, of which 80 (47%) were men and 91 (53%) women. They have a history of smoking 27% of them with predominance of males (42% of men versus 13% of women). Our patients have an average weight of 130 Kg (SD=22), a BMI of 48 kg/m\textsuperscript{2} (SD=6) and an average rate of wai/hip<1. The patient characteristics are expressed in Table 1.

More than half of patients have KCO, FRC, PIM and PEM below the limit of confidence of their theoretical values (Table 2), with no differences between the sexes, except for the maximum pressures. Men have less and less women PIM PEM (Table 3).

55% of patients, 57% of men and 54% of women (p<0.08), have hypoxemia in 10% being serious. Hypercapnia has 15% of cases: 18% of men and 13% of women (p<0.0001). The presence of hypercapnia and hypoxemia day in the OM is not related to weight or the type of obesity, but with older and decrease of forced ventilatory flows and static volumes (Table 4). However, this relationship is different depending on whether men or women (Table 5). There is a correlation between the degree and type of obesity and some parameters of lung function, which is small but statistically significant (Table 6).

| Table 1: Anthropometric parameters in the MO population subject to study. |
|--------------------|-----------------|-----------------|-----------------|
|                     | Mean (Men=80)   | Mean (Women=91) | P value (p<)    |
| Age (years)         | 42 ± 12         | 46 ± 12         | 0.03            |
| Weight (kilos)      | 137 ± 22        | 123 ± 20        | 0.0001          |
| Height (cm)         | 171 ± 30        | 158 ± 23        | 0.0001          |
| BMI (Kg/m\textsuperscript{2}) | 46 ± 6  | 48 ± 6          | 0.04            |
| Waist (cm)          | 140 ± 15        | 136 ± 15        | 0.1             |
| Hip (cm)            | 137 ± 14        | 142 ± 14        | 0.05             |
| Waist/hip           | 1.025 ± 0.087   | 0.954 ± 0.094   | 0.0001         |
|                    | BMI: Bosy mass index |

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Table 2: Blood gas and respiratory functional parameters in the MO.

|                          | Men            | Women           | P value (p<) |
|--------------------------|----------------|-----------------|--------------|
| FVC (%)                  | 95 ± 12        | 100 ± 17        | 0.06         |
| FEV1 (%)                 | 91 ± 14        | 96 ± 19         | 0.1          |
| FEV1/FVC                 | 80 ± 7         | 81 ± 7          | 0.3          |
| DLCO (%)                 | 86 ± 16        | 82 ± 15         | 0.2          |
| KCO (%)                  | 77 ± 13        | 76 ± 14         | 0.9          |
| TLC (%)                  | 98 ± 14        | 98 ± 13         | 0.9          |
| RV (%)                   | 88 ± 29        | 88 ± 22         | 0.9          |
| FRC (%)                  | 72 ± 20        | 71 ± 16         | 0.7          |
| MIP (%)                  | 59 ± 15        | 77 ± 24         | 0.0001       |
| MEP (%)                  | 68 ± 19        | 54 ± 14         | 0.001        |
| PO2 (mmHg)               | 78 ± 15        | 78 ± 14         | 0.7          |
| PCO2 (mmHg)              | 42 ± 5         | 42 ± 5          | 0.9          |
| Sat. O2 (%)              | 93 ± 4         | 93 ± 4          | 0.8          |

FVC: Forced Vital Capacity; FEV1: First Second Forced Expiratory Volume; DLCO: Diffusing Capacity for Carbon Monoxide; KCO: DLCO/Alveolar Ventilation; TLC: Total Lung Capacity; RV: Residual Volume; FRC: Functional Respiratory Capacity; Values expressed as mean ± standard deviation.

Table 3: Comparison of respiratory functional parameters among men and women with MO.

|                          | Age (years) | FVC (%) | FEV1 (%) | FEV1/FVC | RV (%) | FRC (%) |
|--------------------------|-------------|---------|----------|----------|--------|---------|
| Hypercapnia              | 47 ± 13     | 92 ± 16 | 86 ± 18  | 78 ± 7   | 98 ± 27| 76 ± 18 |
| Normocapnia              | 43 ± 12     | 95 ± 16 | 95 ± 16  | 81 ± 7   | 85 ± 26| 70 ± 18 |
| P value (p<)             | 0.2         | 0.04    | 0.01     | 0.04     | 0.04   | 0.1     |
| Severe hypoxemia         | 48 ± 11     | 94 ± 16 | 90 ± 17  | 79 ± 7   | 94 ± 24| 74 ± 18 |
| No Severe hypoxemia      | 39 ± 12     | 102 ± 12| 99 ± 15  | 82 ± 6   | 78 ± 28| 67 ± 18 |
| P value (p<)             | 0.0001      | 0.003   | 0.002    | 0.02     | 0.005  | 0.05    |

FVC: Forced Vital Capacity; FEV1: First Second Forced Expiratory Volume; RV: Residual Volume; FRC: Functional Respiratory Capacity; Values expressed as mean ± standard deviation.

Table 4: MO: comparisons between patients with severe hypoxemia and hypercapnia and normal.

|                  | Hypercapnia | Normocapnia | P value (p<) | Severe hypoxemia | No severe hypoxemia | P value (p<) |
|------------------|-------------|-------------|--------------|------------------|---------------------|--------------|
| Men              |             |             |              |                  |                     |              |

Citation: Herrejón A, Inchaurrega I, Palop J (2016) Pulmonary Function in Morbid Obesity: Influence of Sex and Body Distribution. J Obes Weight Loss Ther 6: 318. doi:10.4172/2165-7904.1000318
Pulmonary mechanical alterations caused by obesity produce an increase in alveolar-arterial oxygen difference that determines a lower PO2 [15]. Thus, there has been a high prevalence of hypoxemia in obesity has been linked to a decreased volume of closing and alteration in the distribution of ventilation [16]. In our study, 75% of the cases had a PO2 lower to 85 mmHg and 25% PO2 below to 70 mmHg, in adult patients without respiratory disease, with an average age of 44 years. The prevalence of hypoxemia is similar between men and women. Hypercapnia is observed more in men, perhaps due to the prevalence of obesity and lower central rate PEm, which could condition worse muscular efficiency, especially the diaphragm.

In obese patients it has not been stated clearly why some have hypercapnia and others not. Hypercapnia has been associated with decreased TLC, VC and FEV1, unrelated to age, BMI, or obesity [17]. Our patients with MO and hypercapnia have lower FVC, FEV1 and RV, with no differences in muscle pressures. There may be in thegenesis of hypercapnia mechanical alteration of the chest wall, with varying degrees of air trapping. There is no correlation between hypercapnia and BMI and waist circumference. The decrease in forced expiratory flows in men with hypercapnia does not occur in women.

Published values of DLCO in obesity are contradictory. While a decrease in DLCO study demonstrated in 20% of the obesity patients [18], another 10% increase in DLCO and 20-25% in the KCO [18] are observed. It has also described a significant increase in DLCO after a weight loss important [6]. Our patients with MO and hypercapnia have lower FVC, FEV1 and FEV1/FVC and higher RV, with no differences in muscle pressures. There may be in thegenesis of hypercapnia mechanical alteration of the chest wall, with varying degrees of air trapping. There is no correlation between hypercapnia and BMI and waist circumference. The decrease in forced expiratory flows in men with hypercapnia does not occur in women.

Table 5: Comparisons, depending on the presence of hypoxemia and hypercapnia, in men and women with MO.

|                     | PO2  | FVC  | Tiff | TLC  | MEP  |
|---------------------|------|------|------|------|------|
| **BMI (Kg/m²)**     |      |      |      |      |      |
| r                   | 0.143| -0.107| 0.169| -0.078| -0.244|
| p                   | 0.08 | 0.2  | 0.05 | 0.4  | 0.03 |
| **Waist (cm)**      |      |      |      |      |      |
| r                   | 0.195| -0.322| 0.108| -0.350| -0.058|
| p                   | 0.07 | 0.005| 0.3  | 0.006| 0.6  |
| **Hip (cm)**        |      |      |      |      |      |
| r                   | 0.229| -0.165| 0.087| -0.341| -0.188|
| p                   | 0.03 | 0.1  | 0.4  | 0.007| 0.1  |
| **waist/hip**       |      |      |      |      |      |
| r                   | -0.031| -0.215| 0.050| -0.081| 0.134|
| p                   | 0.7  | 0.06 | 0.6  | 0.5  | 0.3  |

BMI: Body Mass Index; FVC: Forced Vital Capacity; Tiff.: FEV1/FVC; TLC: Total Lung Capacity; MEP: Maximum Expiratory Pressure.

Table 6: Correlation between parameters determining the type of obesity and blood gas and respiratory functional parameters in MO.

**Discussion**

It is accepted that obese subjects have a restrictive ventilatory pattern type with decreased lung volumes and ventilation/perfusion ratio with lower closing volume in the pulmonary regions declines, which condition an increase in the difference alveolar-arterial of O2 [13].

In patients with OM it has also been reported that restrictive ventilatory pattern is attributed to increased abdominal volume, the displacement of the diaphragm, the greater weight of the chest wall and the deposit of fat in both the diaphragm and intercostal muscles. Thus, the MO causes a reduction in lung volumes, especially at the level of the ERV, which is reversible with ponderal loss [14]. This occurs in half of our patients who have less than 70% of its theoretical value FRC. ERV likely decrease as they maintain their values RV and TLC, but with great variability. The so-called dynamic volumes, obtained by spirometry, are in the range of normal for the entire group. Men with higher incidence of smoking, have lower FVC, which does not become significant.
index of 0.950, is also related to a decrease in dynamic volumes [5]. Similarly, our morbidly obese patients with increased waist have lower FVC and TLC. However, no correlation with cin/cad index [19].

The reduction in lung volumes obesity causes increased resistance and lower chest wall respiratory distensibility [20] without increased maximal respiratory pressures. Thus, although both measures maxim as FRC in patients with MO, respiratory muscle pressures are usually lower than in non-obese subjects, for both men and women, these differences are not significatives [21]. Our results, referring to a Mediterranean population, show greatly diminished percentage values of PIM and PEM. This decrease behaves differently according to sex. Thus, men have higher PEM than women, probably because increased inspiratory muscle strength and lower obesity but lower PIM that they, perhaps because diaphragmatic dysfunction, since the residual volume is similar in both groups. No correlation between respiratory pressures and type of obesity.

We conclude that in morbid obesity is a widespread decline in flows, volumes and respiratory maximum pressures, with frequent blood gas involvement occurs. There are differences between men and women for the presence of hypercapnia and respiratory maximum pressures. The type of obesity, measured as an waist/hip index, has little relevance in these alterations.

Acknowledgment

To Mr. Vicente Abella, Ms. Carmen Sempere and Ms. Pilar García for his estimable collaboration in the conduct of the respiratory function.

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