Editorial

Respiratory repercussions of obesity

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Obesity currently constitutes a serious public health challenge due to its increasing prevalence in the Brazilian population and its consequences as a causative factor of various systemic comorbidities. Studies have shown that obese patients present considerable functional-respiratory repercussions, which vary in intensity depending on the degree of obesity. Such repercussions include the following: decreased expiratory reserve volume (ERV); increased lower airway resistance; higher ratio between the residual volume and the total lung capacity (RV/TLC ratio); decreased pulmonary and thoracic compliance; decreased arterial oxygen pressure (PaO_2); increased arterial-alveolar oxygen difference; alveolar hypoventilation; and sleep disorders. In addition, the increase in the prevalence of dyspnea in obese patients is evident, and its intensity is directly proportional to the body mass index (BMI). The literature provides several explanations for the dyspnea seen in obese individuals. They can be subdivided as follows:

- Mechanical disorders, such as elevation of the diaphragm by the increased abdominal volume, increased closing volume, and decreased compliance of the chest cavity.
- Biochemical disorders, such as increased production of leptin, which is responsible for increasing lower airway resistance.
- Respiratory disorders, such as alterations in the ventilation/perfusion ratio and increased oxygen consumption.

However, we still do not have enough evidence to definitely determine the etiopathogenic factors of dyspnea in obese patients.

In this issue, the study carried out by Teixeira *et al.*,[1] of the department of Pulmonology of the University of São Paulo at Ribeirão Preto School of Medicine in Ribeirão Preto, Brazil, proposes to clarify the issues related to dyspnea in obese patients, based on functional and respiratory alterations. The authors studied 49 patients (41 women and 8 men) with class II or III obesity. The group was subdivided into patients who reported dyspnea (75.5%) and patients who reported no dyspnea (24.5%). The group that reported dyspnea presented lower ERV values, maximal expiratory pressure and arterial pH. The Mahler baseline dyspnea index (BDI) was, on average, lower in the group that reported dyspnea, indicating that this symptom was of greater intensity in these individuals. The average RV/TLC ratio for the group as a whole was above the upper limit of normality (considered 120% of the predicted value in the study), and there was a significant negative correlation between RV/TLC ratio and BDI. It was therefore suggested that the increased lower airway resistance, which resulted in the air trapping phenomenon, could be one of the factors responsible for dyspnea in obese patients. A positive correlation was observed between ERV and PaO_2. Therefore, the uncompensated elevation of the diaphragm and alterations in gas exchange can also be considered etiopathogenic factors for dyspnea. Although there was no control group, the inclusion of which would have allowed comparisons to be made between individuals with class I obesity and nonobese individuals, these findings are important for establishing the respiratory physiopathology in obesity.

In a study carried out in Brazil, spirometry, correlation with BMI, and the Borg dyspnea scale score were used to compare patients with class I or II obesity with individuals in a control group comprising nonobese individuals.[2] Decreased ERV and increased inspiratory capacity in obese patients were observed, showing clear compensatory phenomena, which is a mechanism that can be lost in individuals presenting a higher class of obesity. In addition, a negative correlation was observed between abdominal circumference and ERV, especially in men, whose breathing is predominantly abdominal. Other Brazilian studies should be conducted in order to determine whether dyspnea indexes and respiratory functional parameters correlate with markers of overweight status and obesity, such as abdominal circumference, cervical circumference, waist/hip ratio, and percentage of visceral/subcutaneous fat.

In the discussion section of their paper, Teixeira *et al.*[1] mention the high prevalence of asthma in obese patients. Among other related factors, the effect of leptin – a substance produced in the adipose tissue – has been emphasized in the literature because of its high concentration in the obese. Leptin seems to have immunomodulatory and inflammatory effects. Therefore, it can potentiate the structural changes in the bronchial tissue of asthmatic patients.

One study revealed that the controlled loss of weight in obese individuals with asthma was related to increased forced expiratory volume in one second and greater forced vital capacity.[3] Subsequently, another investigation involving 58 obese individuals showed that weight loss improved the functional parameters, albeit without reducing bronchial hyperreactivity.[4]

We concluded that obesity causes respiratory disorders, which result in dyspnea and correlate with the degree of obesity. Some authors argue that etiopathogenic factors (mechanic, biochemical, and immunological) are related.

Lines of research on this theme should be expanded in order to determine the mechanisms involved in the respiratory repercussions of obesity, including those in patients with respiratory disorders, such as asthma and chronic obstructive pulmonary disease.

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