UDC: 616.248-008.4:616-056.52

Lung ventilation impairment in asthma patients with obesity

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A – research concept and design; B – collection and/or assembly of data; C – data analysis and interpretation; D – writing the article; E – critical revision of the article; F – final approval of the article

BA is characterized by more severe course, worse response to standard therapy in obese patients, therefore asthma with obesity is considered not just as comorbidity but as a distinct BA phenotype.

Aim. To study the lung ventilation impairment in asthma patients with obesity using capnometry. The tasks are to study parameters of lung ventilation in asthma patients and healthy persons and to compare these parameters in asthma patients depending on the presence of obesity.

Materials and methods. The study involved 60 participants (29 men and 31 women) aged 24 to 74 years, the mean age was 52.3 ± 2.7 years, including 30 patients with asthma and 30 healthy subjects. For all participants, body plethysmography and capnometry were performed.

Results. The respiratory minute volume and alveolar ventilation volume were higher in asthma patients with obesity, and the volume of dead space ventilation and its fraction in the tidal volume were not substantially different among the groups of patients with BA. Specifically in relation to the gas exchange parameters, it was found that in obese asthmatics, the end-tidal carbon dioxide pressure was significantly increased – 4.8 ± 0.1 kPa compared with non-obese patients 4.3 ± 0.1 kPa, $P < 0.05$. The end-tidal oxygen concentration was significantly decreased – 15.3 ± 0.2 % compared to normal weight patients 16.0 ± 0.2 %, $P < 0.05$.

Conclusions. Asthma patients with obesity have characteristics that include reduced total lung capacity, inspiratory capacity and forced vital capacity with a higher degree of airways obstruction. These changes lead to lung ventilation impairment with a tendency to carbon dioxide retention, which may be an unfavorable factor for the process of gas exchange with increased oxygen demands for the entire organism in obesity.

Key words: asthma, obesity, capnometry.

Zaporozhye medical journal 2019; 21 (6), 733–738

DOI: 10.14739/2310-1210.2019.6.186486

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Порушення легеневої вентиляції у хворих на бронхіальну астму з ожирінням

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Для бронхіальної астми (БА), що розвивається на тлі ожиріння, притаманні тяжчий перебіг, зниження відповіді на стандартну терапію. Таке поєднання розглядають як окремий фенотип захворювання.

Мета роботи – вивчити порушення легеневої вентиляції у хворих на астму з ожирінням за допомогою методики капнометрії. Завдання дослідження полягали у вивченні параметрів легеневої вентиляції у хворих на астму та здорових осіб і порівнянні параметрів легеневої вентиляції у хворих на астму залежно від наявності ожиріння.

Матеріали та методи. У дослідженні взяли участь 60 осіб (29 чоловіків і 31 жінка) віком від 24 до 74 років, серед них – 30 хворих на БА і 30 здорових осіб, яким виконали бодіплетизмографію та капнометрію.

Результати. У хворих на астму з ожирінням визначають більші об'єм хвилинної вентиляції та об'єм альвеолярної вентиляції порівняно з хворими на БА без ожиріння. При ожирінні вірогідно збільшується парціальний тиск вуглекислого газу в кінці видиху (4,8 ± 0,1 кПа) порівняно з пацієнтами без ожиріння (4,3 ± 0,1 кПа), $p < 0,05$. Встановили вірогідне зменшення фракційної концентрації кисню в кінці видиху до 15,3 ± 0,2 % при ожирінні порівняно з 16,0 ± 0,2 % у хворих на астму з нормальною масою тіла, $p < 0,05$.

Висновки. Для хворих на бронхіальну астму з ожирінням притаманні зниження загальної ємності легень, ємності вдиху та життєвої ємності легень, а також значно виражений ступінь обструкції дихальних шляхів. Ці зміни призводять до порушення легеневої вентиляції з тенденцією до затримки вуглекислоти в організмі, що служить несприятливим тлом функціонування газообміну в умовах підвищеної загальної потреби організму в кисні при ожирінні.

Ключові слова: бронхіальна астма, ожиріння, капнометрія.

Запорізький медичний журнал. – 2019. – Т. 21, № 6(117). – С. 733–738

Нарушения легочной вентиляции у больных бронхиальной астмой с ожирением

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Бронхиальная астма (БА) у больных с ожирением характеризуется более тяжелым течением, сниженным ответом на стандартную терапию и рассматривается как отдельный фенотип заболевания.

Цель работы – изучить нарушения вентиляции легких у больных астмой с ожирением с использованием методики капнометрии. Задачи исследования – изучить параметры вентиляции легких у пациентов с астмой и здоровых людей и сравнить параметры вентиляции легких у пациентов с астмой в зависимости от наличия ожирения.

Ключевые слова: бронхиальная астма, ожирение, капнометрия.

Запорожский медицинский журнал. – 2019. – Т. 21, № 6(117). – С. 733–738

Материалы и методы. В исследовании приняли участие 60 участников (29 мужчин и 31 женщина) в возрасте от 24 до 74 лет, среди них 30 больных БА и 30 здоровых лиц, которым проведено бодиплетизмографию и капнометрию.

Результаты. У больных астмой с ожирением установлены более высокие минутный объем дыхания и объем альвеолярной вентиляции по сравнению с больными БА без ожирения. При ожирении достоверно увеличивается парциальное давление углекислого газа в конце выдоха (4,8 ± 0,1 кПа) по сравнению с пациентами без ожирения (4,3 ± 0,1 кПа), $p < 0,05$. Отмечено достоверное уменьшение фракционной концентрации кислорода в конце выдоха до 15,3 ± 0,2 % при ожирении по сравнению с 16,0 ± 0,2 % у больных астмой с нормальной массой тела, $p < 0,05$.

Выводы. Для больных БА с ожирением характерно снижение общей емкости легких, емкости вдоха и жизненной емкости легких, а также более выраженная степень обструкции дыхательных путей. Данные изменения приводят к нарушению легочной вентиляции с тенденцией к задержке углекислоты в организме, что служит неблагоприятным фоном функционирования газообмена при повышенной общей потребности организма в кислороде при ожирении.

The prevalence of both obesity and bronchial asthma (BA) is increasing now as well as their comorbidity [1]. The problem of asthma and obesity has been studied since 1999, when it was found that overweight people have a higher risk of asthma development. After 10 years, the results of more than 50 studies worldwide have shown a higher incidence of asthma in obese individuals. Later, a causal relationship between weight gain and asthma development was proved [2].

In GINA (Global Initiative for Asthma) guidelines, obesity is included in the list of the risk factors and clinical manifestations of asthma. Weight reduction in obese and overweight patients with asthma is recommended in order to improve lung function, symptoms and health status. BA is characterized by more severe course, worse response to standard therapy in obese patients, therefore asthma with obesity is considered not just as a comorbidity but as a distinct BA phenotype [3].

Obesity is a state of the systemic inflammatory response, and fatty tissue is an active source of proinflammatory mediators (adipokines) secretion such as C-reactive protein, tumor necrosis factor α, interleukin-6, -4, -5 and -13, plasminogen activator inhibitor, eotaxin, vascular endothelial growth factor, monocyte chemoattractant protein-1. Obesity is accompanied by oxidative stress, as evidenced by the increase in isoprostane-8 and other markers in both plasma and bronchiolar alveolar lavage fluid with increasing body mass index [4,5].

Obesity has a significant pathophysiological impact on the respiratory system function including ventilation, respiratory mechanics, respiratory muscle strength and endurance, gas exchange, control of respiration, decreasing exercise capacity and resulting in respiratory failure [6].

Repeated episodes of airway obstruction in asthma also lead to gas exchange disturbances especially due to ventilation-perfusion mismatch. The presence of significant gas exchange abnormalities along with almost normal clinical and spirometric parameters in patients is the peculiarity of asthma [7].

We study the possibilities of capnometry in evaluating pulmonary ventilation in patients with asthma. Capnometry is a measurement and a digital representation of the concentration or partial pressure of carbon dioxide in the air that inhales or exhales the patient during the respiratory cycle. Due to the integration of capnometers and pneumotachometers in one device and the unique capnometry capability to calculate CO_2-free air during exhalation as a dead space volume and its fraction in the tidal volume, it is possible to evaluate the effectiveness of alveolar ventilation [7].

Aim

To study the lung ventilation impairment in asthma patients with obesity using capnometry. The tasks of the work are:

– to study parameters of lung ventilation in asthma patients and healthy persons;

– to compare the lung ventilation parameters in asthma patients depending on the presence of obesity.

Materials and methods

The study involved 60 participants (29 men and 31 women) aged 24 to 74 years, the mean age was 52.3 ± 2.7 years including 30 patients with asthma and 30 healthy subjects.

The inclusion criteria for asthma patients – women and men from 18 years old, bronchial reversibility – percentage change in baseline forced expiratory volume in the first second (FEV1) of >12 % (or ≥200 ml) after administering short-acting β_2-agonists. Only asthma patients who had no signs of disease exacerbation at the time of the examination participated in the study. The inclusion criteria for healthy individuals – women and men from 18 years old with normal respiratory function without bronchial obstructive pathology at the time of the examination. In addition, for all participants – study protocol awareness and signed informed consent to participate in the study, the ability to understand and perform maneuvers of the diagnostic procedures.

The exclusion criteria were having other than asthma respiratory diseases, severe uncontrolled progressive chronic diseases that can affect the examination results, mental disorders.

The diagnosis of asthma was determined by the Order of Ministry of Health of Ukraine No 868 from 08.10.2013 “About the approval and implementation of medical-technological documents on the standardization of medical care for bronchial asthma” [8].

Body mass index (BMI) was calculated as body weight in kilograms divided by height in meters squared. Obesity was defined in subjects with BMI 30.0 kg/m^2 or higher.

The BA group consisted of 30 patients (11 men and 19 women) with the mean age of 57.3 ± 2.3 years and the mean FEV_1 72.3 ± 1.8 % with both normal weight and obesity.

The group of healthy individuals consisted of 30 subjects (18 men and 12 women) with the mean age of 48.7 ± 3.7 years and the mean FEV_1 100.2 ± 1.5 % with normal weight.

Pulmonary function tests and capnometry were performed for all subjects.

Pulmonary function tests (body plethysmography and spirometry) were performed on the "Master Screen PFT" apparatus of the "Cardinal Health" company (Germany) according to the manufacturer's methodology. The following parameters were studied:

– total resistance (Rtot),
– total lung capacity (TLC),
– residual volume (RV),
– RV/TLC ratio,
– intrathoracic gas volume (ITGV),
– inspiratory capacity (IC),
– forced expiratory volume in the first second (FEV_1),
– forced vital capacity (FVC),
– FEV_1/FVC ratio,
– maximal expiratory flow at 75 %, 50 % and 25 % of remaining FVC ($MEF_{25\,\%}$, $MEF_{50\,\%}$, $MEF_{75\,\%}$).

Capnometry was performed on a set for cardiorespiratory system examination "Oxycon Pro", "Cardinal Health" (Germany), the following parameters were studied:

– volume of dead space (the volume of gases in non-perfused alveoli), ml (VD, ml),
– dead space to the tidal volume ratio % (VD%VT),
– minute ventilation (V'E, l/min),
– alveolar ventilation (VA, l/min),
– end-tidal CO_2 pressure, ($PETCO_2$, kPa),
– end-tidal O_2 fraction ($FETO_2$, %).

The data collection and mathematical processing was carried out by licensed software products included in the Microsoft Office Professional 2007 package, Russian Academic OPEN 1 License No Level 43437596. Statistical analysis was performed using MS Excel with descriptive statistics (mean (M), the mean error (m)) and nonparametric statistics (Mann–Whitney test for two independent samples) and the level of significance (P) was determined. Statistically significant differences were estimated at p-values of less than 0.05.

The study was agreed with the Ethics Committee of the NIPhP NAMS, all participants were acquainted with the study protocol and gave their written informed consent.

Results

Data on age, body weight and BMI of study participants are shown in *Table 1*.

Anthropometric data, except for BMI, of the asthma patients did not differ from healthy subjects as healthy subjects were not obese. Accordingly, there were statistically significant differences only in body weight and BMI between asthma groups due to having obesity. 11 patients from 30 asthmatics were obese.

Based on the pulmonary function test results it was found that unlike healthy individuals, in asthma patients, all the parameters of body plethysmography and spirometry were abnormal, except for the TLC and IC, with the statistically significant difference (*Table 2*).

In determining the effect of obesity on the pulmonary function test in patients with asthma, we obtained evidence showing that lung volumes were reduced in patients with obesity. In particular, TLC, IC and FVC were reduced. At the same time, the severity of bronchial obstruction was more pronounced in obese patients compared with those without obesity.

Capnometry was performed for all 60 participants in the study. To solve the first task of our work, we analyzed the capnometry results in patients with asthma and healthy individuals. It was found that the overall pulmonary ventilation parameters in patients with asthma were slightly

Table 1. Anthropometric data of the subjects (M ± m)

Indicators, units	Asthma patients, (n = 30)	Asthma patients with obesity, (n = 11)	Asthma patients without obesity, (n = 19)	Healthy subjects, (n = 30)
Age, years	57.3 ± 2.3	60.3 ± 2.6	55.5 ± 3.4	48.7 ± 3.7
Body weight, kg	79.6 ± 3.5	99.0 ± 4.2	68.3 ± 2.5#	78.5 ± 2.7
BMI, kg/m^2	28.7 ± 1.1	34.5 ± 1.4	25.4 ± 0.7#	24.2 ± 0.7*

*: statistically significant difference between asthma patients and healthy subjects, P < 0.05; #: statistically significant difference between asthma patients with and without obesity, P < 0.05.

Table 2. Pulmonary function test results (M ± m)

Indicators, units	Asthma patients, (n = 30)	Asthma patients with obesity, (n = 11)	Asthma patients without obesity, (n = 19)	Healthy subjects, (n = 30)
Rtot, %	181.1 ± 13.4	180.38 ± 27.4	181.5 ± 15.6	91.2 ± 5.2*
TLC, %	103.4 ± 2.8	96.3 ± 3.9	107.7 ± 3.7#	101.7 ± 2.8
RV, %	128.7 ± 6.4	121.9 ± 13.4	132.8 ± 6.8	103.0 ± 5.1*
RV/TLC, %	44.7 ± 1.4	44.8 ± 3.2	44.6 ± 1.3	32.5 ± 1.7*
ITGV, %	106.7 ± 4.1	99.1 ± 8.1	111.3 ± 4.5	93.2 ± 4.4*
IC, %	107.8 ± 4.2	101.1 ± 3.3	111.6 ± 3.8#	113.4 ± 3.5
FEV_1, %	72.3 ± 1.8	67.8 ± 2.1	74.9 ± 2.4#	100.2 ± 1.5*
FVC, %	93.9 ± 2.1	87.1 ± 2.3	97.9 ± 2.7#	102.4 ± 1.9*
FEV_1/FVC, %	64.4 ± 1.6	65.1 ± 2.4	64.0 ± 2.2	81.6 ± 1.2*
MEF_{75}, %	52.3 ± 3.5	52.1 ± 7.1	52.4 ± 4.1	92.0 ± 3.2*
MEF_{50}, %	31.8 ± 2.1	30.3 ± 3.3	32.7 ± 2.8	88.7 ± 3.6*
MEF_{25}, %	22.5 ± 1.9	23.9 ± 3.3	21.6 ± 2.3	68.4 ± 5.9*

*: statistically significant difference between asthma patients and healthy subjects, P < 0.01; #: statistically significant difference between asthma patients with and without obesity, P < 0.05.

Table 3. Capnometry results (M ± m)

Indicators, units	Asthma patients, (n = 30)	Asthma patients with obesity, (n = 11)	Asthma patients without obesity, (n = 19)	Healthy subjects, (n = 30)
V'E, L/min	10.7 ± 0.4	11.5 ± 0.8	10.3 ± 0.4	10.6 ± 0.3
VD, ml	189 ± 8	210.9 ± 15.0	199.1 ± 11.9	181 ± 8
VD%VT	27.4 ± 1.3	27.4 ± 1.8	29.3 ± 1.6	27.1 ± 1.2
VA, L/min	7.8 ± 0.6	8.2 ± 0.5	7.4 ± 0.4	7.9 ± 0.2
$PETCO_2$, kPa	4.5 ± 0.1*	4.8 ± 0.1	4.3 ± 0.1#	4.6 ± 0.1
$FETO_2$, %	15.7 ± 0.1	15.3 ± 0.2	16.0 ± 0.2#	15.5 ± 0.1

*: statistically significant difference between asthma patients and healthy subjects, P < 0.01;#: statistically significant difference between asthma patients with and without obesity, P < 0.05.

different from those in healthy individuals. Thus, the average value of V¢E was 10.6 ± 0.3 l/min in healthy individuals and 10.7 ± 0.4 l/min in asthma patients (*Table 3*). As for the VD, it was, on average, higher in patients with asthma – 189 ± 8 ml compared with healthy individuals – 181 ± 8 ml, but without statistically significant difference between groups. VD%VT was also higher in asthma patients 27.4 ± 1.3 % than in healthy persons 27.1 ± 1.2 %, but without statistical significance.

In resolving the second task of our work, it was found that the indicators of V'E and VA were higher in patients with obesity, while VD and VD%VT were not substantially different among the groups of patients with BA (table 3). Specifically in relation to the gas exchange parameters, it was found that in obese asthmatics, the indicators of $PETCO_2$ were significantly increased – 4.8 ± 0.1 kPa compared with non-obese patients 4.3 ± 0.1 kPa, p <0,05, but the indicators of $FETO_2$ were significantly decreased – 15.3 ± 0.2 % compared to normal weight patients 16.0 ± 0.2 %, P < 0.05.

Discussion

The obtained results of the pulmonary function tests correspond to the generally accepted data that in obesity, the TLC decreases due to an increased pressure in the abdominal cavity impeding the normal diaphragm excursion. Another TLC reduction mechanism is an excessive fat deposition in the thoracic cavity, which reduces the space for complete lung expansion during inhalation. The same mechanism is suggested for the decrease in IC [9]. In our study, a more pronounced bronchial obstruction was in obese asthmatics with FEV_1 67.8 ± 2.1 % compared to those without obesity 74.9 ± 2.4 %, P < 0.05 that can be explained by a higher degree of bronchial hyperreactivity in BA-obesity phenotype [10]. The literature indicates that even obese non-asthmatics may have a significant airway obstruction due to small airway collapse when lung volume is reduced [11].

A similar mechanism underlies a higher $PETCO_2$ in obesity. An accumulation of carbon dioxide in the alveolar space is associated with inadequate ventilation through mechanisms of lung volumes reduction, inhibition of neural respiratory drive, respiratory muscle weakness and ventilation-perfusion mismatch [12]. Given the fact that carbon dioxide easily dissolves in blood and penetrates biological membranes, the value of $PETCO_2$ in the ideal pulmonary diffusing capacity should correspond to the partial pressure of carbon dioxide in arterial blood [13]. Although the level of $PETCO_2$ in the examined patients 4.8 ± 0.1 kPa did not exceed the normal values for this indicator (4.0–5.7 kPa) [14], but it was significantly higher than that in patients without obesity – 4.3 ± 0.1 kPa, P < 0.05, indicating the compromised processes of pulmonary ventilation with a tendency to accumulate carbon dioxide in these patients.

Regarding the $FETO_2$, it can be calculated from inspired atmospheric oxygen concentration of 20.85 % and expired oxygen concentration of 16.0 % by a non-obese asthma patient, so oxygen absorbance from the atmosphere is 4.85 %. Similarly, we assume that if expired oxygen concentration is 15.3 % in obese asthmatics, that is oxygen absorbance is 5.55 %, which is 14 % higher compared to patients without obesity.

According to the literature data, body weight gain causes an increased total oxygen demand. The research results show that oxygen consumption in obese patients at rest is 25 % higher than in patients with normal body weight. Metabolism in overweight individuals requires higher lung ventilation both for oxygen supply to tissues and carbon dioxide elimination [11]. In our study, oxygen consumption in obese patients was not as high as in the sources mentioned. We explain this difference by the fact that in our case, asthma patients with bronchial obstruction were studied rather than obese non-asthmatics.

Despite the lack of pharmacological strategies for a specific treatment of obese asthmatics, weight loss strategies have been thoroughly investigated and shown to be effective in improving respiratory health in these patients [15]. Thus, obesity is a current problem of clinical practice which significantly disrupts overall health of patients and complicates the course of BA, in particular [16].

Conclusions

1. Asthma patients with obesity have characteristics that include reduced total lung capacity, inspiratory capacity and forced vital capacity with a higher degree of airways obstruction.

2. These changes lead to lung ventilation impairment with a tendency to carbon dioxide retention, which may be an unfavorable factor for the process of gas exchange with increased oxygen demands for the entire organism in obesity.

Prospects for further research. Capnometry diagnostics criteria development for non-invasive hypercapnia detection in obese asthma patients for initial assessment prior to therapy and control evaluation of treatment efficacy.

Funding
The study was conducted in SI "National Institute of Phthisiology and Pulmonology named after F. H. Yanovsky NAMS of Ukraine" (NIPhP NAMS) as the part of the scientific research work "To develop effective medical treatment for patients with obstructive pulmonary disease" (state registration number 0113U000264).

Conflicts of interest: authors have no conflict of interest to declare.
Конфлікт інтересів: відсутній.

Надійшла до редакції / Received: 14.08.2019
Після доопрацювання / Revised: 05.09.2019
Прийнято до друку / Accepted: 16.09.2019

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