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The Level of Leptin in the Blood of Patients with Chronic Obstructive Pulmonary Disease and its Correlations with the Levels of Clinical, Functional and Anamnestic Indicators

Leptin, a hormone produced by white adipose tissue and recognised as an inflammatory biomarker, has an undefined role in the progression of chronic obstructive pulmonary disease (COPD).

Objective — to evaluate the leptin level and its correlations with the levels of clinical, functional and anamnestic indicators in COPD patients.

Materials and methods. This study employed a case-control design involving 42 patients experiencing acute exacerbation of COPD and 20 control subjects. The patients were examined according to the standard protocol. Additionally, the examination included ultrasonic scanning of the abdominal cavity, evaluation of the serum leptin level, scoring of the anxiety and depression and quality of life.

Results and discussion. It was revealed that hyperleptinemia in the patients with acute exacerbation of COPD occurred significantly more frequently than in the healthy persons ((73.8 ± 6.8) *vs* (40.0 ± 11.0) %; $p < 0.05$). Besides, the COPD patients manifested increased leptin levels much more frequently than normal levels ((73.8 ± 6.8) *vs* (26.2 ± 6.8) %; $p < 0.01$). Hyperleptinemia was found to co-occur with severe COPD cases belonging to E clinical group ((51.6 ± 9.0) *vs* (18.2 ± 11.6) % in group with normal leptin level; $p < 0.05$). Kendall correlation analysis established that elevated circulating leptin levels were associated with an increased body mass index ($\tau = 0.3$; $p = 0.02$), female gender ($\tau = 0.3$; $p = 0.01$), non-smoking history ($\tau = -0.2$; $p = 0.03$), stronger manifestation of such symptoms as shortness of breath ($\tau = 0.3$; $p=0.01$), disturbed home ($\tau = 0.3$; $p = 0.002$) and out-of-home activities ($\tau = 0.3$; $p = 0.02$) and lack of energy ($\tau = 0.3$; $p = 0.01$). It also correlated with increased severity of pulmonary insufficiency ($\tau = 0.3$; $p = 0.003$) and decreased forced vital capacity ($\tau = -0.2$; $p = 0.04$), elevated systolic blood pressure ($\tau = 0.2$; $p = 0.02$), elevated cholesterol ($\tau = 0.3$; $p = 0.01$) and β-lipoproteins levels ($\tau = 0.2$; $p = 0.04$), pronounced depression ($\tau = 0.3$; $p = 0.002$) and lowered quality of life ($\tau = 0.3$; $p = 0.01$).

Conclusions. COPD patients with elevated leptin levels manifest not only with an increased body weight, but also with arterial hypertension, disturbed lipid metabolism, more pronounced pulmonary insufficiency, depressive disorders and lowered life quality with more severe subjective respiratory symptoms.

Keywords

Chronic obstructive pulmonary disease, leptin, hyperleptinemia.

Chronic obstructive pulmonary disease (COPD) is one of the most common chronic pulmonary diseases on the global scale, associated with a large personal and social burden. Despite the numerous studies and availability of new medicines, COPD along with bronchial asthma continue to be the most frequent chronic respiratory diseases globally (4.1 % and 3.7 %, respectively) and the main reasons for mortality caused by respiratory diseases [5]. Contemporary COPD treatment strategies aim at

reducing the occurrence of acute exacerbation and its prevention. However, the scientists focus on developing new, personalized approaches to the treatment with a view of modifying the disease course. Creation of new therapeutic approaches aims to distinguish certain populations of patients based on their phenotype. Promising in this respect is the analysis of indicators of inflammatory biomarkers. One of them is leptin, a hormone mostly synthesized by the white adipose tissue with a number of pleiotropic functions. It participates in the regulation of cellular homeostasis and metabolism, has an effect on immune and inflammatory responses: it is involved in the stimulation of neutrophil chemotaxis and macrophage phagocytosis and production of proinflammatory interleukins-6, 12 and tumour necrosis factor-alpha [9].

In addition to adipocytes, leptin is also expressed by bronchial epithelial cells, type 2 alveolar pneumocytes and lung macrophages, which suggests its potential effect on the respiratory system [3, 11]. It is known that COPD acute exacerbation features elevated leptin levels [13], but the effect of leptin on the COPD course has been insufficiently studied. The limited contemporary researches point to the correlation of COPD with severity of bronchial obstruction as well as to the fact that leptin can be considered a potential biomarker for evaluating the severity and prognosis in COPD patients [12, 14].

Objective — to evaluate the leptin level and its correlations with the levels of clinical, functional and anamnestic indicators in COPD patients.

Materials and methods

The survey involved 42 patients with acute exacerbation of COPD, comprising 20 females and 22 males with a median age of 60.0 [52.0; 68.0] years. The control group consisted of 20 healthy individuals of similar age and gender distribution.

The study included patients with a confirmed diagnosis of COPD, established based on clinical symptoms (chronic cough, dyspnea, sputum production) and spirometric findings showing a postbronchodilator forced expiratory volume in 1 second (FEV_1)/forced vital capacity (FVC) ratio < 0.7. The median duration of the disease among the participants was 5.0 [2.0; 10.0] years. Among the patients, (57.1 ± 7.6) % were current or former smokers. At the time of examination, (57.1 ± 7.6) % of patients were classified in clinical groups A or B of COPD, while (42.9 ± 7.6) % were in clinical group E.

The diagnosis of acute exacerbation of COPD was made by eliciting a history of acute symptomatic worsening from the stable state and beyond normal day-to-day variations and necessitating a change in regular medication. According to the criteria of GOLD, there were 7 cases of GOLD Grade 1, 17 cases of Grade 2, 14 cases of Grade 3 and 4 cases of Grade 4. The FEV_1 of the examined patients was 54.0 [40.0; 73.0] %, which corresponds to moderate airflow limitation severity.

The patients were examined according to the protocol (COPD Assessment Test; the body mass index (BMI) assessment using Quetelet formula (the medium BMI was 27.6 [23.0; 33.7] kg/m^2 for the COPD patients and 25.1 [24.5; 29.1] kg/m^2 for the healthy persons; $p > 0.05$; the medium BMI for the male patients was 25.5 [22.6; 31.7] kg/m^2 and for the female patients — 29.5 [24.9; 36.2] kg/m^2; $p > 0.05$); assessment of the main indicators of pulmonary function testing using computer spirometry). Additionally, the examination included ultrasonic scanning of the abdominal cavity, evaluation of the serum leptin level using enzyme-linked immunosorbent assay with DRG Leptin ELISA (Germany) (normal reference range is 3.6—11.1 ng/ml for females and 1.1—5.6 ng/ml for males), calculation of the ratio of the leptin level to BMI (L/BMI) [8], scoring of the anxiety and depression using the Hospital Anxiety and Depression Scale (HADS) and quality of life using the St. George Respiratory Questionnaire (SGRQ). In the latter, four subscales were applied: symptoms, activity, impacts, scoring (from 0 to 100 %, the latter indicating the worst result). The digital data was processed in Statistica for Windows 10.0 (Statsoft, USA); the results were presented as a median [lower; upper quartile] and correlations were assessed using Kendall's τ coefficient. The significance was assumed to be $p < 0.05$.

Results

In the COPD patients, the circulating leptin level was somewhat higher than in the control (16.3 [7.2; 39.9] *vs* 12.0 [5.6; 21.2] ng/ml; $p > 0.05$). At the same time, elevated leptin levels in the COPD patients were observed 2.7 times more frequently than normal levels ((73.8 ± 6.8) *vs* (26.2 ± 6.8) %; $p < 0.01$) and 1.8 times more frequently than in the control ((73.8 ± 6.8) *vs* (40.0 ± 11.0) %; $p < 0.05$) (Fig. 1).

As it was expected, the leptin level depended on the body weight. For instance, in the COPD patients with a normal weight (n = 16; BMI 22.7 [21.0; 23.4] kg/m^2), the leptin level was 4.1 [1.3; 17.0] ng/ml; in the overweight patients (n = 9; BMI 27.6 [27.0; 28.3] kg/m^2) it was significantly higher (16.3 [14.0; 34.5] ng/ml; $p = 0.02$), whereas in the obese patients (n = 17; BMI 34.4 [31.7; 39,8] kg/m^2) it was maximal and significantly higher than in normal weight patients (27.1 [18.1; 60.1] ng/ml; $p = 0.03$) (Fig. 2). Additionally, hyperleptinemia much more frequently occurred in the overweight and obese patients than in those with a normal body weight

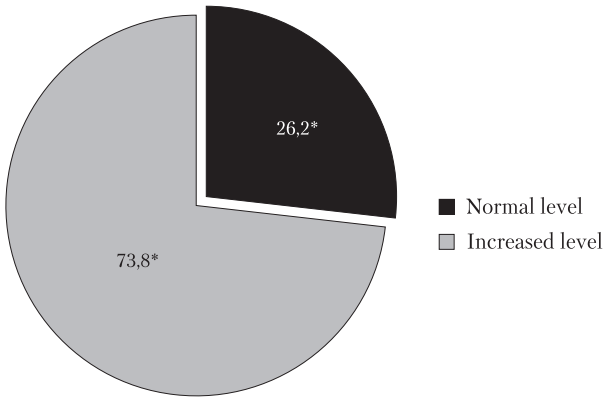

Fig. 1. **The occurrence of normal and elevated leptin levels in patients with chronic obstructive pulmonary disease (%)**

* The difference between the indicators is significant (p < 0.05).

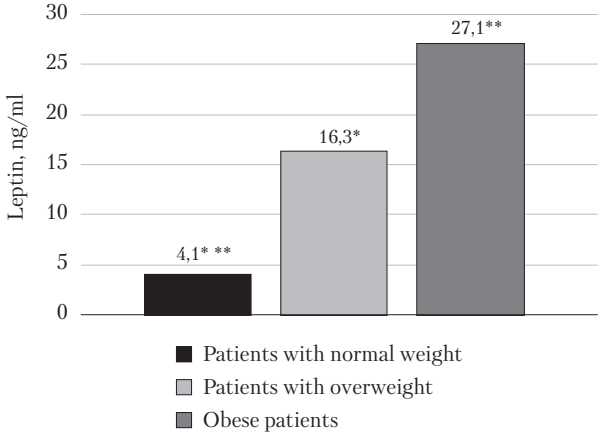

Fig. 2. **Leptin levels in chronic obstructive pulmonary disease patients *vs* body weight**

* The difference between the leptin levels in patients with normal weight and overweight is significant (p < 0.05); ** the difference between the leptin levels in patients with normal weight and obesity is significant (p < 0.05).

((100 ± 0) % and (88.2 ± 7.8) *vs* (37.5 ± 12.1) %; p < 0.01 for both).

The circulating leptin level also correlated with gender. In male COPD patients, it was significantly lower (13.4 [2.6; 25.1] *vs* 28.7 [12.4; 60.1] ng/ml; p = 0.04; Fig. 3). A similar tendency was observed in the control group (2.6 [1.2; 8.0] ng/ml in men *vs* 16.7 [8.0; 22.1] ng/ml in women; p = 0.04).

The secretory activity of adipocytes, assessed by the index L/BMI in COPD patients, also depended on body weight. In obese cases, it was maximal, amounting to 0.7 [0.4; 1.4] arbitrary unit (a.u.); somewhat lower in overweight patients (0.6 [0.5; 1.2] a.u.) and significantly lower in those with normal body weight (0.2 [0.1; 0.8] a.u.; p_{1-3} = 0.03) (Fig. 4). Adipocyte secretory activity was also influenced by gender: in females, it was higher (0.9 [0.4; 1.7] *vs* 0.4 [0.1; 0.7] a.u., p = 0.07).

Kendall's correlation analysis established positive correlations of leptin level with BMI (τ = 0.3; p = 0.02), gender (higher in females; τ = 0.3; p = 0.01), smoking history (lower in smokers; τ = –0.2; p = 0.03), a number of COPD Assessment Test indicators (stronger manifestation of shortness of breath, disturbed home and out-of-home activities, lack of energy and scoring: τ_{1-5} = 0.3; $p_{1,4}$ = 0.01; p_2 = 0.002; p_3 = 0.02; p_5 = 0.03), severity of depression (τ = 0.3; p = 0.002), quality of life indicators (symptoms: τ = 0.2; p = 0.03; activity: τ = 0.3; p = 0.02; impacts and scoring: $\tau_{1,2}$ = 0.3; $p_{1,2}$ = 0.01) (Table). Therefore, elevated circulating leptin levels were associated with the development of depression and lower quality of life with increasing anxiety caused by respiratory symptoms, limited physical activity, psychological and social problems stemming from the disease.

A clear correlation was established between the leptin level and systolic blood pressure (BP) in the COPD patients (τ = 0.2; p = 0.02), which implies

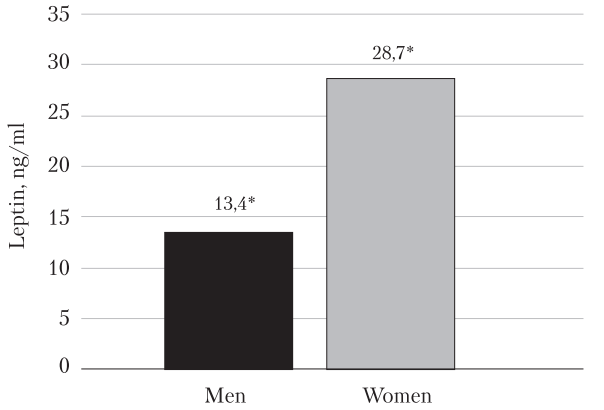

Fig. 3. **Gender differences in the leptin levels in patients with chronic obstructive pulmonary disease**

* The difference between the indicators is significant (p < 0.05).

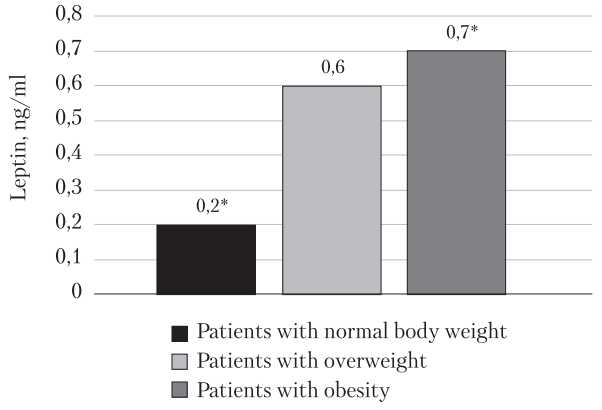

Fig. 4. **Secretory activity of adipocytes in patients with chronic obstructive pulmonary disease vs body weight**

* The difference between the indicators is significant (p < 0.05).

Table. **Significant correlations between the leptin level and level of different clinical indicators**

Indicator	τ	p-value
BMI	0.3	0.02
Female gender	0.3	0.01
Non-smoking history	–0.2	0.03
COPD Assessment Test indicators (total)	0.3	0.03
Severity of depression (according to HADS)	0.3	0.002
Quality of life (according to SGRQ)	0.3	0.01
Systolic blood pressure	0.2	0.02
Degree of pulmonary insufficiency	0.3	0.003
Forced vital capacity (FVC)	–0.2	0.04
Blood cholesterol level	0.3	0.01
Blood β-lipoproteins levels	0.2	0.04
Ultrasonographic right liver lobe size	0.4	0.01
Ultrasonographic gall bladder length	0.4	0.03
Ultrasonographic right kidney width	0.4	0.02

the risk of development of arterial hypertension in case of hyperleptinemia. At the same time, the direct correlations between the leptin level and diastolic and pulse BP were established in the male patients only ($\tau_1 = 0.5$; $p_1 = 0.001$; $\tau_2 = 0.4$; $p_2 = 0.003$).

Also, the leptin level was found to directly correlate with the degree of pulmonary insufficiency (PI) ($\tau = 0.3$; $p = 0.003$): it was the lowest in degree I PI patients (1.7 [0.9; 14.9] ng/ml), and as PI progressed, it increased (II degree PI: 16.1 [7.2; 43.6] ng/ml; $p_{I–II} = 0.02$; III degree PI: 24.7 [12.9; 60.4] ng/ml; $p_{I–III} = 0.006$). Besides, the leptin level was directly proportional to the forced vital capacity (FVC) ($\tau = -0.2$; $p = 0.04$).

In terms of lab test indicators, there were established direct correlations of the leptin level with the absolute neutrophil count in women ($\tau = 0.3$; $p < 0.05$), cholesterol ($\tau = 0.3$; $p = 0.01$) and β-lipoproteins levels ($\tau = 0.2$; $p = 0.04$) regardless of the gender. The leptin level was also associated with the structural dimensions in ultrasonic imaging: with the right liver lobe size ($\tau = 0.4$; $p = 0.01$), gall bladder length ($\tau = 0.4$; $p = 0.03$), right kidney width ($\tau = 0.4$; $p = 0.02$) and left liver lobe in women ($\tau = 0.6$; $p = 0.03$).

Then the patients were split into two groups. The patients with a normal leptin level (n = 11; BMI 23.0 [20.5; 24.1] kg/m^2) went into group 1 and those with an elevated leptin level (n = 31; BMI 28.7 [25.7; 34.4] kg/m^2) made group 2. The groups were similar in terms of gender complement and age (58.0 [44.0; 72.0] *vs* 61.0 [54.0; 67.0] years; $p > 0.05$). It was revealed that severe COPD occurred much more frequently in group 2 patients (clinical group E: (51.6 ± 9.0) *vs* (18.2 ± 11.6) %; $p < 0.05$), whereas group 1 patients had mild COPD (clinical groups A-B: (81.8 ± 11.6) *vs* (48.4 ± 9.0) %; $p < 0.05$).

For the normal leptin levels, its concentration directly correlated with the gender, being higher in women ($\tau = 0.5$; $p = 0.03$), systolic and pulse BP ($\tau = 0.5$ for both; $p = 0.02$), WBC and RBC counts in the urine sediment ($\tau_1 = 0.5$; $p_1 = 0.04$; $\tau_2 = 0.6$; $p_2 = 0.01$), right kidney width ($\tau = 0.7$; $p = 0.03$). Inversed correlation was observed with smoking (lower in smokers: $\tau = -0.5$; $p = 0.03$), haemoglobin and RBC levels ($\tau_{1,2} = -0.6$; $p_1 = 0.005$; $p_2 = 0.01$).

In group 2 patients, the leptin level was in direct correlation with the gender, being higher in women ($\tau = 0.4$; $p = 0.003$), total cholesterol level ($\tau = 0.3$; $p = 0.04$) and gall bladder length ($\tau = 0.5$; $p = 0.01$), and in inverse correlation with the monocytes count ($\tau = -0.3$; $p = 0.04$). Further elevation of the leptin level will associate with disturbed lipid metabolism and risk of infectious complications.

Discussion

Our study revealed that hyperleptinemia was much more frequent in patients experiencing acute exacerbation of COPD compared to healthy individuals. Similar findings, with subsequent reductions in leptin levels during remission periods, were reported by A. Vassiliou et al. [13]. Furthermore, research has shown that during COPD acute exacerbation, leptin levels not only increase in the patient's blood but also in their sputum [7]. This suggests that leptin plays a role not only in systemic inflammation but also in local inflammation associated with COPD, potentially serving as one of the biomarkers for its acute exacerbation.

We established that the leptin level inversely correlated with smoking history. Similar outcomes were obtained in the study by Y.A. Zhelyazkova et al., showing that it was COPD patients with no smoking history in whom the highest leptin

levels were found [15]. Earlier, the inverse correlation between the leptin level and nicotine addiction established using Fagerström test was described by M. Suhaimi et al. [10]. A suggestion can be made that these results stem from the effect of nicotine on major metabolism — higher energy expenditure results in a reduced body weight and, respectively, lower leptin levels. However, the role of excessive secretion of catecholamines because of smoking, which can have an effect on expression and secretion of leptin, should not be ruled out.

We detected a direct correlation between the leptin level and the severity of depression by HADS. Taking into consideration that leptin has anxiolytic and anti-depressant effects, this result can be put down to the fact that patients with hyperleptinemia caused by excessive body weight or acute exacerbation of COPD with inflammation syndrome activation, can develop leptin resistance, this leading to the loss of these effects, which causes depression and anxiety disorders [1]. Therefore, overweight and obesity due to leptin resistance can cause development of depression. On the other hand, depressive conditions can manifest themselves as eating disorders and cause body weight gain.

In our study, elevated circulating leptin levels in the COPD patients were associated with the disturbed pulmonary function. This may stem from the fact that in case of excessive weight or obesity, which are accompanied by hyperleptinemia, the inspiratory volume as well as the expiratory reserve volume decrease, which occurs due to the fat deposits in the chest and abdomen areas. Besides, the recent researches show that leptin participates in the regulation of synthesis and secretion of MUC5AC protein, which is the main component of the respiratory tract mucus [2]. In addition, leptin can promote not only the hyper secretion of mucus at COPD but also stimulate the production of a number of anti-inflammatory mediators, which with time leads to the thickening of the walls of the respiratory tract and its remodelling [4].

The relation between the enlarged liver and increased leptin level that we established is rather unclear. On the one hand, leptin suppresses lipogenesis and stimulates lipolysis, thereby making an antisteatotic effect on the liver; on the other hand, its pro-inflammatory and pro-fibrotic actions can cause the development of inflammation and fibrosis in the hepatic tissues, which indirectly corroborates the fact that leptin is recognized as a risk factor of hepatocellular carcinoma [6].

Conclusions

COPD patients with elevated leptin levels manifest not only with an increased body weight, but also with arterial hypertension, disturbed lipid metabolism, more pronounced pulmonary insufficiency, depressive disorders and lowered life quality with more severe subjective respiratory symptoms.

No conflict of interest.
Participation of authors: research concept and design — L.I. Pylypiv, O.M. Radchenko; collection of research materials, analysis of the research materials, statistical analysis, text writing — L.I. Pylypiv; supervision of the research — O.M. Radchenko, O.Y. Komarytsia; critical analysis of manuscript draft version — O.M. Radchenko, O.Y. Komarytsia, Z.I. Piskur; editing — O.M. Radchenko, L.I. Pylypiv; technical preparation of manuscript — L.I. Pylypiv, Z.I. Piskur.

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Рівень лептину в крові хворих на хронічне обструктивне захворювання легень та його кореляції з рівнями клініко-функціональних і анамнестичних показників

Лептин — гормон білої жирової тканини і біомаркер запалення, роль якого в перебігу хронічного обструктивного захворювання легень (ХОЗЛ) остаточно не встановлена.

Мета роботи — визначити рівень лептину та його кореляції з клініко-функціональними й анамнестичними показниками в пацієнтів із ХОЗЛ.

Матеріали та методи. За стандартним протоколом обстежено 42 пацієнтів із загостренням ХОЗЛ і 20 здорових осіб як контрольну групу. Проведено ультразвукове сканування органів черевної порожнини, оцінку рівня лептину в сироватці крові, тривоги і депресії та якості життя.

Результати та обговорення. Установлено, що гіперлептинемія в пацієнтів із загостренням ХОЗЛ траплялася значно частіше, ніж у здорових осіб ((73,8 ± 6,8) і (40,0 ± 11,0) %; $p < 0,05$). У пацієнтів із ХОЗЛ підвищений рівень лептину зафіксовано частіше, ніж нормальні показники ((73,8 ± 6,8) і (26,2 ± 6,8) %; $p < 0,01$). Гіперлептинемія значно частіше супроводжувалася тяжким перебігом ХОЗЛ із встановленням клінічної групи Е порівняно з нормолептинемією ((51,6 ± 9,0) і (18,2 ± 11,6) %; $p < 0,05$). Кореляційний аналіз Кендалла виявив, що підвищення рівня лептину в крові асоціювалося зі зростанням індексу маси тіла ($\tau = 0,3$; $p = 0,02$), жіночою статтю ($\tau = 0,3$; $p = 0,01$), відсутністю куріння в анамнезі ($\tau = -0,2$; $p = 0,03$), збільшенням виразності низки симптомів (задишки ($\tau = 0,3$; $p = 0,01$), порушення діяльності вдома ($\tau = 0,3$; $p = 0,002$) та поза домом ($\tau = 0,3$; $p = 0,02$), нестачею енергії ($\tau = 0,3$; $p = 0,01$)), зростанням тяжкості легеневої недостатності ($\tau = 0,3$; $p = 0,003$), зниженням форсованої життєвої ємності легень ($\tau = -0,2$; $p = 0,04$), зростанням систолічного артеріального тиску ($\tau = 0,2$; $p = 0,02$), вмісту холестерину ($\tau = 0,3$; $p = 0,01$) і β-ліпопротеїнів ($\tau = 0,2$; $p = 0,04$), виразності депресії ($\tau = 0,3$; $p = 0,002$) та погіршенням якості життя ($\tau = 0,3$; $p = 0,01$).

Висновки. Пацієнти з ХОЗЛ і підвищеним рівнем лептину характеризуються не лише збільшеною масою тіла, а й артеріальною гіпертензією, порушенням ліпідного обміну, виразнішою легеневою недостатністю, депресивними розладами, зниженням якості життя та виразнішими суб'єктивними респіраторними симптомами.

Ключові слова: хронічне обструктивне захворювання легень, лептин, гіперлептинемія.

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