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MARKERS OF SYSTEMIC INFLAMMATION IN COPD PATIENTS WITH OBESITY

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Chronic obstructive pulmonary disease is one of the most common respiratory diseases in which comorbid pathology is detected in almost 90% of patients. Particular attention in the clinic of internal medicine deserves a coexisting with alimentary obesity due to the following risk factors: population aging, hypodynamia, over-nutrition, which contributes to the progression of comorbid pathology and the development of complications.

The chronic inflammatory process is the main link in the pathogenesis of chronic obstructive pulmonary disease, which causes structural changes of the respiratory tract and lungs (bronchial obstruction and loss elasticity of the lung parenchyma) against the background of systemic inflammation, especially in overweight patients.

So, *the purpose of the work* was to analyze literature data on the common markers of systemic inflammation in chronic obstructive pulmonary disease patients with obesity.

High sensitivity C-reactive protein is an acute-phase protein synthesized mainly by hepatocytes in response to the bronchopulmonary tissue damage by the inflammatory process. The research has demonstrated the relationship between the high sensitivity C-reactive protein and functional ventilatory capacity, clinical chronic obstructive pulmonary disease severity.

Adipose tissue is considered as an endocrine organ and a source of biologically active substances: adipokines, bioactive peptides, free fatty acids, monocytic chemotactic factor-1 (monocyte chemotactic protein-1), pro-inflammatory cytokines, involved in systemic inflammation in many pathological conditions, including respiratory diseases. There are many works focused on the study of leptin as a pleiotropic hormone determining the state of immune homeostasis and angiogenesis. However, a remarkable discovery was the determination of the leptin role in the respiratory tract. Leptin is a stimulator of ventilation, a factor determining the processes of maturation and development of the lungs, as well as respiratory diseases, including obstructive sleep apnea syndrome, bronchial asthma, chronic obstructive pulmonary disease and lung cancer. It should be noted that leptin is involved in airway inflammation in chronic obstructive pulmonary disease, possibly due to the regulation of infiltration and apoptosis of immune cells in the submucosal basis of the bronchi.

Conclusion. Thus, obesity has a significant effect on the intensity of systemic inflammation in patients with chronic obstructive pulmonary disease. High sensitivity C-reactive protein and leptin are the common biomarkers of systemic inflammation that can be used to assess the severity of chronic obstructive pulmonary disease and obesity.

Keywords: chronic obstructive pulmonary disease, obesity, high sensitivity C-reactive protein, leptin.

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Introduction. The chronic inflammatory process is the main link in the pathogenesis of chronic obstructive pulmonary disease (COPD). It causes structural changes in the respiratory tract and lungs (bronchial obstruction and loss elasticity of the lung parenchyma) against the background of systemic inflammation, especially in overweight patients, given the significant contribution of fat cells to the inflammatory process [1, 2, 3].

So, **the purpose of the work** was to analyze literature data on the common markers of systemic inflammation in COPD patients with obesity.

It is common knowledge that the main mechanisms of inflammation in COPD are an increasing number of alveolar macrophages, neutrophils, T-lymphocytes (mainly T-helper cells (Th1 and Th17). These mechanisms, together with epithelial, endothelial cells, and fibroblasts, express the synthesis of

pro-inflammatory mediators such as cytokines, chemokines, growth factors, and lipid mediators [4]. It is considered that T-lymphocytes play an essential role in the pathogenesis of COPD. However, the mechanisms of this inflammatory process remain understudied. One of the researches found that the blood of COPD patients demonstrated a higher expression of CC chemokine 5 receptors (CCR5) on CD8 + T cells in bronchoalveolar lavage and a higher percentage of CXCR3 + CD8 + T lymphocyte cells, as compared to apparently healthy individuals. Moreover, the results obtained were dependent on the gender of the patients. The Th1 / Tc1 immune response was associated with macrophages and neutrophil density in the bronchi, and the reaction of Th2 / Tc2 was associated with the severity of emphysema [5].

Two large-scale COPD Gene and ECLIPSE studies (total of 6299 participants) are devoted to the relationship between changes in circulating immunocompetent cell populations and COPD severity and progression. The results of these studies suggested a positive correlation between FEV_1 and lymphocyte counts against the feedback with myeloid cell counts (neutrophils, monocytes). The number of lymphocytes, monocytes, and eosinophils determined the three-year change in spirometric parameters of airflow limitation. In COPD patients, a decreased number of CD4 + memory cells and naive B cells was observed, suppressing the maturation of lymphocytes against the background of enlargement of myeloid cells. This fact indicates an essential place of immune mechanisms in the pathogenesis of COPD progression [6].

Most studied mediators of chronic inflammation in COPD patients are inflammatory interleukins (IL-1, IL-2, IL-6, IL-8, IL-9, IL-12, IL-18), tumor necrosis factor-alpha (TNF- α), high sensitivity C-reactive protein (hsCRP), matrix metalloproteases. They also include specific markers, namely desmosine isomers, leukotriene-B4, neutrophil elastase and surfactant protein D, natriuretic peptide (NT-proBNP), and T-troponin [7, 8, 9, 10].

The hsCRP is a recognized biomarker of systemic inflammation, synthesized mainly by hepatocytes in response to the bronchopulmonary tissue damage by the inflammatory process [11]. The hsCRP binds phagocyte receptors and plays a role in cell apoptosis, correlating with other inflammatory mediators, particularly with IL-6, IL-1 [12].

It is essential to determine the dependence of the hsCRP concentration and the number of COPD exacerbations, which determine the prognosis of the disease. The obtained data are contradictory, as evidenced by the results of research, which did not show a significant correlation between the hsCRP concentration and the number of exacerbations. Other studies found a significant correlation between the hsCRP concentration and the number of exacerbations, identifying hsCRP as a marker for the development of COPD exacerbations [13, 14].

In the study, which involved 116 patients in the stable phase of COPD and 35 practically healthy individuals, the researchers found that serum hsCRP concentration was significantly higher in COPD patients than in practically healthy individuals (4.48 ± 0.83 vs. 1. 1 ± 0.27 mg / l; p <0,05). Noteworthy the conclusion that serum hsCRP concentration > 3 mg / l has a negative effect on COPD prognosis compared to hsCRP concentration ≤ 3 mg / l (odds ratio (OR) = 2.71; 95% confidence interval (CI) = 1.05–6.99; p <0.05). The results of four studies (1750 patients with COPD), which provided statistically similar results (OR = 1.54; 95% CI = 1.14–2.07; p <0.01), confirmed this statement. These studies emphasized the importance of identifying the hsCRP biomarker in patients with stable COPD [15].

A systematic review and meta-analysis, conducted to clarify the relationship between hsCRP concentration and COPD mortality, included 15 sources on COPD mortality. In the general analysis, increase the concentration of hsCRP was significantly associated with the mortality rate (OR = 1.53, 95% CI = 1.32–1.77; p <0.001). However, it is necessary to observe that this relationship was established at maximal indices of hsCRP and primarily in the Asian population of COPD patients [16].

Considering that systemic inflammation is the primary pathogenetic mechanism of COPD with obesity, the question remains about the role of adipose tissue in the progression of the inflammatory process, the intensity of which depends on the patient's body mass index (BMI). According to a randomized ECLIPSE trial, systemic inflammation activity was significantly higher in patients with BMI 29.4 kg / m^2 compared to the group of patients where BMI was within normal limits. The scholars also found that 30% of COPD patients had no manifestations of systemic inflammation both in the initial stage of the disease and after one year of observation against the background of availability bronchial obstructive syndrome. The results of this study also demonstrated a correlation between inflammatory marker concentration and hypodynamia, especially in COPD patients with obesity, which contributes to the growth of pro-inflammatory markers [17, 18].

The results of the research by R. Agarwal et al., 2013 [19] proved that BMI had an inverse correlation with serum hsCRP concentration. According to another study, COPD patients with obesity had an increased serum hsCRP concentration compared to COPD patients with normal BMI [20]. The study by

M. Breyer et al., 2009, confirmed that in COPD patients with BMI ≥30 kg / m^2 serum hsCRP concentrations was by 3.3 times higher (95% CI, 1.5–7.0; p = 0.002) than in patients with BMI 21-24.9 kg / m^2. However, in COPD patients with low BMI (<21 kg / m^2), hsCRP concentration was by 2 times higher (OR = 0.5; 95% CI = 0.3-0.9; p = 0.022) compared with control groups (BMI within normal limits) [21].

Today, adipose tissue is considered an endocrine organ and a source of biologically active substances. These substances include adipokines, bioactive peptides, free fatty acids, monocytic chemotactic factor-1 (monocyte chemotactic protein -1 (MCP-1)), and proinflammatory cytokines (IL-1β, TNF- α, hsCRP, IL-6). They are the modulators of cardiovascular function, insulin sensitivity, inflammation, and function of adipose tissue against the background of inhibition of production the anti-inflammatory mediators [22, 23]. Adipokines can also be synthesized by inflammatory cells, participating in systemic low-intensity inflammation in many pathological conditions, including respiratory diseases (COPD, bronchial asthma, interstitial lung diseases) [24]. Thus, adipose tissue plays a vital role in the processes of systemic inflammation and the formation of carbohydrate metabolism disorders in COPD patients. It acts as an independent factor in the potentiation of the bronchopulmonary inflammatory process.

Leptin is involved in many vital functions of the body. These functions include energy metabolism, reproductive function, and angiogenesis. It also participates in the expression of cytokines synthesized by immunocompetent cells and regulates the processes of cell apoptosis, involved in the regulation of T-cell proliferation. Furthermore, leptin can activate macrophages and promote vascular proliferation. The regulation of leptin synthesis occurs through the hypothalamus-pituitary-adrenal axis and depends on the body fat mass. Leptin activates the synthesis of proinflammatory cytokines such as IL-2, interferon-γ produced by Th1, and inhibits the synthesis of IL-4 synthesized by Th2 [25, 26, 27, 28].

An important discovery was the determination of the leptin role in the respiratory tract. Leptin is a stimulator of ventilation, a factor determining the processes of maturation and development of the lungs, as well as respiratory diseases, including obstructive sleep apnea syndrome, bronchial asthma, COPD, and lung cancer [29, 30].

Scientific evidence suggests that leptin is involved in airway inflammation in COPD, possibly due to the regulation of infiltration and apoptosis of immune cells in the submucosal basis of the bronchi. Increased leptin concentration in the proximal airways correlates with the expression of activated T lymphocytes (mainly CD8 +) and the absence of apoptotic T cells. Leptin concentration in the induced sputum positively correlated with inflammatory markers. However, the question remains debatable on the existence of a paracrine cross-section between resident lung epithelial cells and immune cells in response to harmful particles, requiring further scientific studies on the role of leptin in the pathogenesis of COPD [27, 29, 31, 30, 32, 33].

Leptin concentration in COPD patients depends on BMI. The studies confirmed this fact by demonstrating a close relationship between serum leptin concentration and TNF-ά content and soluble TNF receptor (sTNF-R55 and -R75) and a direct correlation with BMI and body fat percentage (% of fat) in patients with COPD [34, 35, 36].

Another study found a link between genetic variants in the leptin-R gene and decreased lung function in COPD patients showed a significant association (p <0.05). Haplotype analyzes confirmed some of these associations, identified by distinct markers, suggesting that genetic variants in the leptin-R gene are significantly associated with decreased lung function in the COPD smoker population. The results confirmed the genetic nature of the leptin effect on the severity of ventilatory capacity in COPD patients. They may identify leptin-R as a new gene candidate for COPD [37].

The purpose of one study was to evaluate the relationship between leptin levels in COPD patients and changes in respiratory function depending on his concentration. Analysis of the results of this study revealed an increase in leptin concentration in COPD patients with overweight and obesity compared to patients with normal body weight (p <0.01). The research found the most pronounced obstructive and restrictive changes in patients with hyperleptinemia, and its level is closely related to the degree of bronchial obstruction [38].

Conclusion. Thus, in COPD patients, obesity has a significant effect on the intensity of systemic inflammation. The hsCRP and leptin are markers of this inflammation and, therefore, can be used to assess the severity of COPD and obesity.

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МАРКЕРИ СИСТЕМНОГО ЗАПАЛЕННЯ У ХВОРИХ НА ХОЗЛ У ПОЄДНАННІ З ОЖИРІННЯМ

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Резюме. Одним з найбільш розповсюджених захворювань органів дихання залишається хронічне обструктивне захворювання легень при якому коморбідну патологію виявляють майже у 90 % хворих. Особливої уваги у клініці внутрішньої медицини заслуговує поєднання з аліментарним ожирінням за рахунок наступних факторів ризику: постаріння населення, гіподинамія, надмірне харчування, що сприяє прогресуванню коморбідної патології та розвитку ускладнень.

Хронічний запальний процес є головною ланкою патогенезу хронічного обструктивного захворювання легень, що викликає структурні зміни дихальних шляхів і легень (бронхіальна обструкція та втрата еластичності паренхіми легень) на тлі системного запалення, особливо у пацієнтів з надмірною вагою. Тому метою роботи було провести аналіз літературних даних про найбільш поширені маркери системного запалення у хворих на хронічне обструктивне захворювання легень із ожирінням.

Високочутливий С-реактивний протеїн являє собою гострофазний білок, що синтезується гепатоцитами у відповідь на ушкодження бронхолегеневої тканини внаслідок запального процесу. Доведений тісний зворотний зв'язок між показниками високочутливого С-реактивного протеїну та функціональними показниками вентиляційної здатності легень, клінічними показниками тяжкості хронічного обструктивного захворювання легень.

Жирова тканина розглядається як ендокринний орган і джерело біологічно активних речовин – адипокінів, біоактивних пептидів, вільних жирних кислот, моноцитарного хемотаксичного фактору-1 (моноцитарний хемотаксичний білок-1-MCP-1), прозапальних цитокінів, що приймають участь в системному запаленні низької інтенсивності при багатьох патологічних станах, в тому числі при захворюваннях органів дихання. Вивченню лептину як плейотропного гормону, який визначає стан імунного гомеостазу та ангіогенезу присвячена велика кількість робіт. Однак важливим відкриттям стало визначення ролі лептину в ураженні органів дихання, враховуючи що лептин є стимулятором вентиляції, фактором визначаючим процеси дозрівання та розвитку легень, а також респіраторних захворювань, включаючи обструктивний синдром нічного апное, бронхіальну астму, хронічне обструктивне захворювання легень та рак легень.

Отже, високочутливий С-реактивний протеїн та лептин є загальними біомаркерами системного запалення низької інтенсивності, котрі можна використовувати для оцінки ступеня тяжкості хронічного обструктивного захворювання легень та ожиріння.

Ключові слова: хронічне обструктивне захворювання легень, ожиріння, високочутливий С-реактивний протеїн, лептин.

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МАРКЕРЫ СИСТЕМНОГО ВОСПАЛЕНИЯ У БОЛЬНЫХ ХОБЛ В СОЧЕТАНИИ С ОЖИРЕНИЕМ

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Резюме. Одним из самых распространенных заболеваний органов дыхания остается хроническая обструктивная болезнь легких, при которой коморбидную патологию выявляют почти у 90% больных. Особое внимание в клинике внутренней медицины заслуживает сочетание с алиментарным ожирением за счет следующих факторов риска: старение населения, гиподинамия, избыточное питание, что способствует прогрессированию коморбидной патологии и развитию осложнений.

Хронический воспалительный процесс является главным звеном патогенеза хронической обструктивной болезни легких, вызывает структурные изменения дыхательных путей и легких (бронхиальная обструкция и потеря эластичности паренхимы легких) на фоне системного воспаления, особенно у пациентов с избыточным весом. Поэтому целью работы было провести анализ литературных данных о наиболее распространенных маркерах системного воспаления у больных хронической обструктивной болезнью легких с ожирением.

Высокочувствительный С-реактивный протеин представляет собой острофазный белок, синтезируемый гепатоцитами в ответ на повреждение бронхолегочной ткани вследствие воспалительного процесса. Доказана тесная обратная связь между показателями высокочувствительного С-реактивного протеина и функциональными показателями вентиляционной способности легких, клиническими показателями тяжести хронической обструктивной болезни легких.

Жировая ткань рассматривается как эндокринный орган и источник биологически активных веществ – адипокинов, биоактивных пептидов, свободных жирных кислот, моноцитарного хемотаксического фактора-1 (моноцитарный хемотаксический белок-1-MCP-1), провоспалительных цитокинов, участвующих в системном воспалении низкой интенсивности при многих патологических состояниях, в том числе при заболеваниях органов дыхания. Изучению лептина как плейотропного гормона, который определяет состояние иммунного гомеостаза и ангиогенеза посвящено большое количество работ. Однако важным открытием стало определение роли лептина в поражении органов дыхания, учитывая, что лептин является стимулятором вентиляции, фактором, определяющим процессы созревания и развития легких, а также респираторных заболеваний, включая обструктивный синдром ночного апноэ, бронхиальную астму, хроническая обструктивная болезнь легких и рак легких.

Таким образом, высокочувствительный С-реактивный протеин и лептин являются общими биомаркерами системного воспаления низкой интенсивности, которые можно использовать для оценки степени тяжести хронической обструктивной болезни легких и ожирения.

Ключевые слова: хроническая обструктивная болезнь легких, ожирение, высокочувствительный С-реактивный протеин, лептин.

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