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ТИББИЁТДА ЯНГИ КУН НОВЫЙ ДЕНЬ В МЕДИЦИНЕ NEW DAY IN MEDICINE

*Илмий-рефератив, маънавий-маърифий журнал
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THE ROLE OF BRONCHIAL ASTHMA IN THE DEVELOPMENT OF NEPHROPATHIES

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✓ Resume

Chronic kidney disease (CKD) is one of the most common chronic non-communicable diseases due to its prevalence among all segments of the population, a sharp decrease in the quality of life of patients and a high mortality rate. The role of bronchial asthma in the development of BCS has not been sufficiently studied. This article presents an analysis of articles devoted to studying the role of bronchial asthma in the development of chronic kidney diseases.

Key words: chronic kidney disease, bronchial asthma, leptin, cytokines, apoptosis, oxidative stress.

РОЛЬ БРОНХИАЛЬНОЙ АСТМЫ В РАЗВИТИИ НЕФРОПАТИЙ

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✓ Резюме

Хроническая болезнь почек (ХБП) является одним из наиболее распространенных хронических неинфекционных заболеваний в связи с ее распространенностью среди всех слоев населения, резким снижением качества жизни больных и высоким уровнем смертности. Роль бронхиальной астмы в развитии СБК недостаточно изучена. В данной статье представлен анализ статей, посвященных изучению роли бронхиальной астмы в развитии хронических заболеваний почек.

Ключевые слова: хронические болезни почек, бронхиальная астма, лептин, цитокины, апоптоз, оксидативный стресс.

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✓ Rezyume

Surunkali buyrak kasalligi (SBK) – surunkali noinfeksion kasalliklar orasida aholining barcha qatlamlari orasida keng tarqalganligi, bemorlar hayot sifatining keskin pasayishi, o'lim holatining yuqoriligi va katta iqtisodiy xarajatlar talab qiladigan o'rin bosuvchi terapiya (gemodializ) va buyrak transplantatsiyasi muolajalariga muhtoj bo'lib qolishlariga olib kelishi bilan muhim o'rin egallagan. SBK rivojlanishida bronxial astmaning roli yetarli darajada o'rganilmagan. Ushbu maqolada Bronxial astmaning surunkali buyrak kasalliklari rivojlanishidagi o'rnini o'rganishga bag'ishlangan maqolalar tahlili keltirilgan.

Kalit so'zlar: surunkali buyrak kasalliklari, bronxial astma, leptin, sitokinlar, apoptoz, oksidlovchi stress.

Relevance

Scientific research has shown that there is a commonality in the molecular (biomarkers of kidney injury and inflammation) pathogenic mechanisms between bronchial asthma (BA) and chronic kidney disease (CKD) [1]. In 1953, academician M.V. Chernorutskiy wrote: "The principle of a single pathology plays a significant organizational and guiding role in the process of medical reasoning when diagnosing. According to this principle, in every case of illness, the physician, above all, should base their understanding on the assumption that all painful signs observed in the body stem from one cause, one disease" [2]. Another quote from a modern article dedicated to the issue of comorbidity [3] states: "Everything in the body is interconnected. No function, organ, or system works in isolation. Their continuous joint activity maintains homeostasis, ensures the harmony of ongoing processes, and protects the body. However, in real life, this mechanism, which is ideal from nature's point of view, encounters numerous pathological agents every second. Under their influence, individual components cease to function, leading to the development of disease. As the same authors emphasize, "Nothing happens without consequence. Despite timely elimination of a deficiency, the disruption of even a seemingly minor joint can lead to changes in many processes, mechanisms, and functions. This contributes to the emergence of new diseases, whose debut may take years."

For any practicing physician, these quotes are familiar and understandable, and undoubtedly, their significance is taken into account in daily practice. It is precisely from these positions that the issue of comorbidity between BA and CKD can be considered. It is known that bronchial asthma affects the course of chronic diseases of internal organs such as ischemic heart disease [4], diabetes [5], and others. On the other hand, CKD can also have a significant systemic effect. It is well known that CKD often leads to the development of arterial hypertension and dyslipidemia [6,20], which exacerbate kidney failure and contribute to the development of atherosclerosis, creating conditions for vascular damage.

It is known that the interaction between bronchial asthma and chronic kidney disease has been scarcely studied. The purpose of this article is to review several studies aimed at identifying the risk of CKD development in patients with BA.

One such study was conducted by H.L. Huang and colleagues in 2014 [7]. As part of a large-scale study, data from 141,064 patients were analyzed – all of whom were free from CKD at the start of the study. Of these, 35,086 had bronchial asthma, and 105,258 did not. The average age of the patients was 47.7 years, and most of the patients lived in industrial cities. The proportional hazard model (Cox regression) was used to calculate the risk of CKD development over a three-year follow-up period.

In the group of patients with bronchial asthma, CKD developed in 2,196 patients (6.26%), while in the control group, it developed in 4,120 patients (3.91%). The application of the proportional hazard model showed that patients with asthma were more likely to develop chronic kidney disease (hazard ratio [HR]: 1.56; 95% CI: 1.48-1.64; $p < 0.001$).

At the same time, many other factors are known to influence the risk of CKD development. Authors [7,16,17,18] have noted gender, age, monthly income, urbanization level, geographic region, diabetes, hypertension, hyperlipidemia, and the use of glucocorticoids as relative risk factors for CKD development in asthma patients, with a relative risk of 1.40 (95% CI: 1.33-1.48; $p = 0.040$). However, in patients who received glucocorticoid treatment, the risk of CKD development was significantly lower (HR: 0.56; 95% CI: 0.62-0.61; $p < 0.001$). Additionally, it was hypothesized that expectorants, bronchodilators, muscle relaxants, and leukotriene receptor antagonists may reduce the risk of CKD development [7].

Indirect data indicating the risk of CKD development in asthma patients were assessed through studies on pathogenetic factors influencing the development of CKD, as well as the expression of biomarkers of acute and chronic kidney injury in asthma. These findings are discussed from the perspective of the problem at hand.

Authors like Mineev V.N., Zelenkova Z.A., and Sadovnikova O.M. studied glomerular filtration rate (GFR) in 103 patients with different types of asthma. For calculations, the CKD-EPI formula, which is currently recommended as the most appropriate screening method for assessing GFR in clinical and outpatient practice, was used. The CKD-EPI is considered a universal and accurate method for assessing CKD at any stage. The results of glomerular filtration assessment using the Rehberg test and CKD-EPI formula (eGFR) showed reliable correlation ($R_s = 0.634$, $p = 0.004$, $n = 19$).

It was shown that in patients with non-allergic asthma and aspirin-induced asthma (AspBA), the glomerular filtration rate (hGFR) was significantly lower than in allergic asthma patients [9]. In patients

with systemic (oral) glucocorticoid treatment for asthma, hGFR was also reduced, but the differences were not statistically significant. Additionally, it was found that in allergic BA, hGFR values were within normal limits. In the AspBA group [9], and also in the randomly selected group by age, hGFR was found to be the lowest, indicating a pathogenic imbalance in the metabolism of prostaglandins and leukotrienes in kidney autoregulation in this form of BA.

To date, direct evidence of the involvement of leukotrienes (LTB₄) in the development of CKD has been established [10]. According to some sources [7,8,9], the use of leukotriene receptor antagonists in the treatment of asthma reduces the risk of CKD development. Interestingly, in two patients treated with montelukast, the hGFR was within normal limits, measuring 96 and 97 ml/min/1.73 m² [9].

Several authors [11] analyzed the effect of anti-inflammatory cytokines on glomerular filtration rate in patients with bronchial asthma. When analyzing the factors in allergic asthma, IL-6 was found to play a crucial role in the development of both BA symptoms (bronchial obstruction) and CKD (hGFR), as well as in the disruption of the anti-inflammatory/inflammatory balance in the system [11]. In non-allergic asthma, particularly, TNF- α expression was found to reflect the severity of the disease and hGFR values. The more severe the bronchial asthma, the greater the reduction in hGFR [11,13,14,15].

When studying the expression of major adipokines and the relationship between glomerular filtration rate in different variants of the disease [12], a correlation was found between the expression of major adipokines (leptin, adiponectin, resistin) and glomerular filtration rate, as well as the involvement of the STAT-3 transcription factor in the effects of anti-inflammatory adipokines.

It is known that the kidneys are the primary organ involved in the endogenous leptin degradation process. Leptin can also contribute to the development of morphological changes, particularly leading to glomerulosclerosis in obese patients. Leptin triggers renal fibrogenesis primarily by activating the expression of growth factor-b (TGF-b) and its receptors in mesangial cells and endothelial cell membranes.

Additionally, the hormone resistin, produced excessively in obesity, may disrupt endothelial function. In obese patients, both hyperleptinemia and increased plasma resistin levels are associated with elevated TNF- α and IL-6 soluble receptors in the blood plasma.

An analysis was conducted to evaluate the role of peripheral blood lymphocyte apoptosis in the development of CKD in patients with asthma. The analysis included information on known risk factors such as cell apoptosis readiness, late-stage apoptosis, gender, smoking, combination of bronchial asthma and heart pathology, age, and the composition of inflammatory effector cells – particularly eosinophil count in peripheral blood [19]. The study showed that lymphocyte apoptosis could play a role in the reduction of glomerular filtration rate in asthma patients and contribute to the development of CKD [19].

Conclusion

A review of several published studies allows us to conclude that the link between bronchial asthma and chronic kidney disease is not coincidental. This link may primarily be based on mechanisms involving common biological processes such as inflammation, oxidative stress, apoptosis, and fibrosis.

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