ustnogo sustava i zhevatel'nyh myshc [Etiology and pathogenesis of diseases of the temporomandibular joint and masticatory muscles]. Vestnik Novgorodskogo gosudarstvennogo universiteta im. Yaroslava Mudrogo [Bulletin of Yaroslav Mudry Novgorod State University. Temporomandibular joint and masticatory muscles. 2020; 4 (120): 50-59 (In Russ.).]

29. Fazylova M.B., Chapin D.L. Rol' sanitarno-prosvetitel'skoj raboty s naseleniem v snizhenii rasprostranennosti i intensivnosti stomatologicheskih zabolevanij [The role of sanitary-educational work with the population in reducing the prevalence and intensity of stomatologic diseases]. Molodye uchenye – gordost' strany [Young scientists – the pride of the country. 2023. P. 142-145 (In Russ.).]

30. Tsakoev A.G. Periodontit: osobennosti, simptomy, lechenie [Periodontitis: peculiarities, symptoms, treatment]. Nauchnyj Lider [Scientific Leader. 2022; 25: 45-46 (In Russ.).]

31. Agbulut N., Delilbasi C. The anatomy of the temporomandibular joint. Handbook of Oral and Maxillofacial Surgery and Implantology. Cham: Springer International Publishing, 2024.P.1-12.

32. Akbarjon S.K. Adentia types and methods of their study. Ethiopian International Journal of Multidisciplinary Research.2024; 11(03): 100-102.

33. Anvarjonovich A.M. History and treatment plan for patients with complete edentia. Basics of prevention. Ethiopian International Journal of Multidisciplinary Research.2023; 10 (11): 276-280.

34. Gromova S.N., Pyshkina O.A., Agalakova T.B., et al. Conservative treatment of pulpitis. Vyatsk Medical Bulletin.2021; 1(69): 47-52 (In Russ.).]

35. Donnermeyer D, Dammaschke T, Lipski M, et al. Effectiveness of diagnosing pulpitis: a systematic review. International Endodontic Journal.2023; 56: 296-325.

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COMBINATION OF CHRONIC KIDNEY DISEASE WITH CHRONIC NON-COMMUNICABLE DISEASES

The scientific review examines chronic kidney disease (CKD) in combination with chronic non-communicable diseases. Chronic kidney disease is a serious and growing public health problem worldwide, characterized by a gradual and irreversible decline in kidney function and is one of the leading causes of death worldwide. The priority task of healthcare is the prevention of chronic non-communicable diseases, among which the most significant include cardiovascular diseases, bronchopulmonary diseases, diabetes mellitus, and oncological diseases. The association of CKD with chronic non-communicable diseases, in particular with cardiovascular diseases, makes this pathology especially dangerous, which leads to a deterioration in the quality of life of patients and an increase in mortality. The treatment of patients with comorbid pathology requires a comprehensive and interdisciplinary approach.

Keywords: chronic non-communicable diseases, chronic kidney disease, diabetes mellitus, arterial hypertension, oncological diseases, coronary heart disease, bronchial asthma, chronic obstructive pulmonary disease.

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Introduction. CNID is a long-term ongoing disease caused by prolonged exposure to various causes: environmental factors and genetic characteristics combined with an unhealthy lifestyle (smoking, alcohol abuse, unhealthy diet, low physical activity). The World Health Organization (WHO) estimates that 41 million people die from these diseases every year, accounting for 71% of all deaths; more than 15 million of them are people aged 30 to 69 years. CNID is the main cause of early death and disability of the population [14].

WHO identifies the following categories of NCDs with corresponding mortality rates:

- cardiovascular diseases (CVD): most often – arterial hypertension (AH), coronary heart disease (CHD) and CKD) claiming 17 million lives;
- oncological diseases lead to the death of about 9.3 million people annually;
- diseases of the respiratory system (chronic obstructive pulmonary disease (COPD) and bronchial asthma (BA)) cause 4.1 million deaths per year;
- diabetes mellitus (DM) is the cause of death of 1.5 million people.

Special attention should be paid to CKD, which is a persistent organ lesion for three or more months due to the action of various etiological factors, the anatomical basis of which is the process of replacing normal anatomical structures with fibrosis, leading to its dysfunction by CKD [20]. The causes of CKD include genetic factors and the consequences of concomitant diseases such as hypertension, diabetes, abdominal obesity and lipid metabolism disorders (dyslipidemia). Today, hypertension consistently holds a leading position among all the causes contributing to the progression of CKD, affecting about 10-15% of the world's population [28].

Arterial hypertension and CKD. The basis for the development of hypertension in patients with CKD is the activation of the sympathetic nervous system against the background of deterioration of kidney function. This, in turn, contributes to an even greater increase in blood pressure (BP) in people with CKD. In the process of decreasing glomerular filtra-

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tion rate (GFR), the renin-angiotensin-aldosterone system (RAAS) is activated, resulting in accumulation of Na+ and fluid in the body [2].

The most significant way to detect CKD in patients with hypertension at the outpatient level is to determine the level of creatinine in the blood with the calculation of GFR, which is carried out in conjunction with the determination of daily protein loss. Analysis of both indicators of CKD is especially important, since a decrease in eGFR and an increase in urinary albumin levels, independently of each other and other cardiovascular risk factors, are associated with an increased risk of general and cardiovascular mortality, which increases over time with a decrease in eGFR and an increase in albuminuria [11]. This focuses on the need for timely detection and monitoring of the development of CKD in patients with hypertension.

High blood pressure significantly worsens the prognosis of the disease for patients with pre-existing CKD, as the risk of CKD developing into end-stage renal failure (ESRD) increases. According to the MRFIT (Multiple Risk Factorl ntervention Trial) study, data on 332,544 men over 16 years were collected. After analyzing all the results, it was concluded that people with high and normal blood pressure, unlike patients with optimal blood pressure (less than 120/80 mmHg), are 1.9 times more likely to develop ESRD [5]. The data obtained allow us to conclude that the development of CKD contributes to the aggravation of the course of pre-existing hypertension and is one of the main reasons for the ineffectiveness of antihypertensive therapy.

One of the key aspects contributing to slowing the development of CKD and significantly reducing the risk of cardiovascular diseases is monitoring blood pressure while meeting targets. In addition to blood pressure monitoring, lifestyle modification is necessary to slow the progression of CKD: to give up bad habits, regularly monitor blood glucose levels, avoid taking medications with nephrotoxic effects, and simultaneously carry out effective treatment of the underlying disease, which led to the development of CKD [21].

When hypertension and CKD are combined, antihypertensive therapy should be carefully selected in the patient. It is extremely important to start treatment as early as possible to prevent cardiovascular complications and the progression of CKD. It is recommended to use renin-angiotensin-angiotensin system blockers (RAAS blockers) as the main drugs for the treatment of hypertension combined with CKD, and, if necessary, give preference to HMG-CoA reductase inhibitors, type 2 sodium-glucose cotransporter inhibitors, and glucagon-like peptide 1 agonists [11]. It is important to understand that patients with CKD require special attention and an individual approach. It is necessary to select drugs individually for each patient, taking into account his characteristics and needs [21, 28].

Coronary heart disease and CKD. Coronary artery disease (CHD) is a disease that is caused by insufficient blood flow in the coronary arteries [17]. Modern scientific evidence indicates a close relationship between coronary heart disease and CKD, and coronary heart disease is not only a concomitant disease, but can also provoke the development of kidney complications. The most significant complication of coronary heart disease is the development of chronic heart failure (CHF) [24]. Current epidemiological data confirm a stable relationship between CHF and CKD: CHF is a risk of developing CKD and vice versa [22, 24, 31]. This is the so-called cardiovascular continuum [9].

Among the main mechanisms leading to the formation of CKD on the background of coronary heart disease, one can single out:

1. hemodynamic disorders (due to a decrease in the left ventricular ejection fraction, perfusion decreases, proinflammatory cytokines are activated and, as a result, proliferative processes, in particular fibrosis) [21, 24];

2. Microvascular changes (impaired autoregulation of renal blood flow caused by systemic inflammation in atherosclerosis, worsens the course of ischemic nephropathy) [25];

3. neurohumoral changes (in CHF, a decrease in the shock volume of blood discharged by the left ventricle leads to activation of neurohumoral mechanisms, which is manifested by excessive production of vasoconstrictors (norepinephrine, angiotensin, endothelin, vasopressin and a decrease in secretion or reaction to internal vasodilating factors. Such disorders provoke deterioration of blood supply to the kidneys, progression of oxygen starvation of tissues and structural organ damage) [18].

Dyslipidemia is an important pathogenetic mechanism in coronary heart disease. Endothelial dysfunction, which is the first stage in the development of atherosclerosis, leads to the development of CKD. Vascular cell adhesion molecule 1 (VCAM-1) is a marker of endothelial dysfunction. Its level can be used to judge the onset of inflammatory and atherosclerotic changes in the renal vessels [12]. In 1963, lipoprotein (a) was discovered, which is now used as a marker of increased risk of CVD. It should be noted that this indicator increases at the initial stages of CKD development, however, its study in the blood is currently not widespread [8].

Homocysteine is also an indicator associated with both coronary heart disease and CKD. In the early stages of CKD, homocysteine levels are moderately elevated and continue to rise with the progression of a decrease in GFR [17]. An indicator of CKD in the early stages is also myeloperoxidase, which is one of the main causes of oxidative stress leading to calcification and atherosclerosis of the renal vessels [19].

Thus, coronary artery disease is a significant risk factor for the development and progression of CKD. Understanding the above mechanisms allows us to develop strategies for the early diagnosis and prevention of CKD in patients with coronary heart disease.

Chronic obstructive pulmonary disease, bronchial asthma and CKD. The conducted clinical and epidemiological studies indicate that for many years CKD has been in the shadow of chronic kidney disease, including COPD and asthma. Hypoxia, which develops against the background of chronic bronchopulmonary diseases, can lead to kidney damage. Characteristic kidney cells have a particularly high oxygen demand (especially the cells lining the proximal sinuous tubules), and even subclinical hypoxia can lead to apoptosis of these cells and tissue fibrosis. Hypercapnia in patients with COPD can cause renal afferent arteriolar vasoconstriction, decreased renal blood flow, and activation of the adrenergic pathway. It is obvious from the definition of COPD that harmful gases play a vital role in the pathogenesis of the disease. Nicotine activates the sympathetic autonomic system, causing an increase in blood pressure. This can worsen any nephropathy that the patient already has. The most significant risk factors that worsen kidney function in patients with COPD include lead and hydrocarbons from smoke, systemic inflammation, hypoxemia and hypercapnia, and sympathetic activation [6, 35].

In 2020, a study was conducted with the participation of the Amur State Medical Academy aimed at a comprehensive assessment of the functional state of the kidneys in patients aged 45 to 60 years with documented COPD. The study did not include patients with extremely severe

COPD; patients with hypertension, coronary heart disease, acute cerebrovascular accident, kidney and urinary tract pathology, tuberculosis, and oncological diseases of any localization. The results of the study: in patients with frequent exacerbations of COPD and severe symptoms, the urea level was higher and the GFR value was lower than in the group with mild clinical manifestations and low frequency of exacerbations. Albuminuria (albumin/creatinine ratio in a single portion of urine) of more than 30 mg/g was observed mainly in people with frequent exacerbations of COPD. Thus, the data obtained allow us to consider COPD as a risk factor for the development of impaired renal function. Patients with frequent exacerbations of COPD and severe clinical symptoms require increased monitoring by specialists [4].

The problem of the risk of developing CKD in asthma is also being considered. A number of studies have indicated the relationship between the presence of asthma in a patient and the risk of developing CKD, and risk factors such as gender, age, obesity, diabetes, hypertension, and smoking do not significantly affect this relationship [10]. Huang H.L. et al. In 2014, it was shown that the relative risk (RR) of developing CKD in patients with asthma remains at a high level. However, it is worth noting that in patients who take glucocorticosteroids, the risk of developing CKD (HR=0.56; 95% CI: 0.62-0.61; p <0.001) is more than 2 times lower than in patients with asthma who do not take glucocorticosteroids (HR=1.40; 95% CI: 1.33–1.48; p=0.040). In addition to glucocorticosteroids, expectorant and cholinolytic drugs, leukotriene receptor antagonists, bronchodilators, and muscle relaxants reduce the risk of developing CKD. A 6-year follow-up of patients with ASTHMA to assess the risk of developing CKD showed that in patients with controlled asthma or asthma in remission, proteinuria and a decrease in GFR were observed less frequently than in patients diagnosed with ASTHMA for more than 20 years, uncontrolled or persistent [29].

Oncological diseases and CKD. According to WHO, in 2022, about 20 million new cases of malignant neoplasms (ZNO) and about 9.7 million deaths from them were registered worldwide. Oncological diseases are the second leading cause of death after diseases of the cardiovascular system, not only in the Russian Federation, but throughout the world. CKD and ZNO are quite closely related: Just as cancer can cause direct or indirect damage to the renal tissue, leading to decreased kidney function, CKD is one of the risk factors for kidney cancer [37]. One of the reasons for the development of CKD in CHF is the nephrotoxicity of antitumor drugs [23]. This is largely due to the fact that most drugs are excreted by the kidneys. The components of the drug itself and its mechanism of action can also influence the development of CKD. For example:

- cisplatin contains the heavy metal platinum, which is toxic to the body, and the antitumor effect of the drug is due to the selective suppression of DNA synthesis, the products of its metabolism can damage mitochondria and block the cell cycle [13];

- isophosphamide enhances the expression of cytochrome p450, which metabolizes it into toxic chloroacetaldehyde, which causes acute and chronic damage to the renal tubules [13];

- methotrexate is safe in low doses [34], but in high (1 g/m2) doses, uric acid crystals gradually precipitate under the influence of the acidic environment of urine, which can lead to acute renal failure or CKD [13].

The nephrotoxic effects of cytostatic therapy are dose–dependent (the rate and degree of their realization depend on the dose of the drug) and are most often realized at the level of the proximal tubules, less often the distal tubules and glomeruli [13, 23].

Another reason for the development of CKD on the background of ZNO is paraneoplastic nephropathies. Paraneoplastic nephropathy is damage to the filtering apparatus of the kidneys caused by immunological and metabolic disorders in oncological diseases. In case of HYPERTENSION, excessive production of growth factors, proinflammatory cytokines, and lymphokines causes cell dysfunction and malfunction of the glomerular filter, can lead to the formation of intracapillary blood clots, infiltration of kidney tissue by macrophages and monocytes [37].

The other side of the issue is the development of cancer on the background of CKD. Numerous studies show that patients with CKD have a higher risk of developing cancer than patients without CKD. One of the reasons for this is that due to impaired kidney function, nitrogenous substances and carcinogenic compounds accumulate in the body (for example, 2–amino-6-methyldipyridoimidazole), DNA repair decreases, and oxidative stress constantly occurs [30].

Diabetes mellitus and CKD. Diabetes mellitus (DM) is a group of chronic metabolic diseases characterized by hyperglycemia and/or impaired insulin metabolism. Currently, this chronic non-communicable disease is characterized by a wide prevalence. Constantly elevated glucose levels in diabetes are accompanied by damage to target organs: kidneys, nerves, eyes, and the cardiovascular system [1]. The variety of renal pathology in DM (diabetic glomerulosclerosis proper, chronic glomerulonephritis, urinary tract infection, medicinal nephritis, tubulointerstitial fibrosis, atherosclerotic renal artery stenosis, etc.), having different mechanisms of development, progression dynamics, and treatment methods, is a particular problem for DM patients, since their frequent combination is mutually aggravating [16]. Diabetes is the leading cause of CKD in the world [32]. In patients with impaired carbohydrate metabolism, diabetic nephropathy occupies one of the leading positions among the causes of death, following diseases of the cardiovascular system and hypertension [7].

Along with hereditary predisposition, hyperglycemia is the main mechanism that damages the kidney structure. An increase in glucose levels contributes to the activation of the RAAS, as well as an increase in cell resistance to insulin and, as a result, endothelial dysfunction. These effects are enhanced in the presence of modifiable risk factors.: increased blood pressure, obesity and smoking, which determines the formation of glomerular hyperfiltration and hypertension. As the process progresses, the mechanism of automatic regulation of the tone of the delivering arterioles is disrupted, which is why systemic blood pressure directly affects intraclubular pressure and hypertension becomes constant.

Albuminuria is one of the most important mechanisms of progression of glomerular lesions. At this stage, proteinuria and hypertension develop, which stimulate damage to kidney structures, which leads to a decrease in their functions, up to terminal CKD [3].

It is also known that under the influence of hyperglycemia, end products of glycation (AGE) are formed in the patient's body. The formation of a small amount of AGE is part of the metabolism in a healthy person, but a large amount of glycotoxins produced can have negative consequences [15]. High levels of glycation toxins activate processes (profibrotic and inflammatory changes) that trigger the progression of diabetic nephropathy. One of the mechanisms of action of toxins is the transformation of protein complexes of the extracellular matrix. Glycation of collagen leads to a decrease in its properties, including solubility and

inversion of the charge of monomers, changing the configuration of the basement membrane of nephrons, which destroys the architecture. Glycation of another protein, laminin, also leads to a decrease in its binding to the components of the basement membrane. Glycotoxins affect other matrix proteins, among other things, which worsens the degradation by metalloproteinases, contributing to an increase in the thickness of the basement membrane and the width of the mesangium. These changes lead to a violation of the function of the filtration apparatus of the kidneys, therefore, albuminuria increases and progressive deterioration of kidney function occurs [26].

The diagnosis of diabetic nephropathy is based on the determination of albumin levels in urine and/ or a decrease in GFR. It is typical to have a history of diabetes mellitus, as well as the presence of other complications (for example, retinopathy) [33]. At the same time, CKD in a patient with type 2 diabetes may be the result of diabetes, it may worsen, or it may not be related to diabetes at all [1]. Thus, diabetic nephropathy can be diagnosed mainly on the basis of clinical and laboratory data (general clinical urine tests, eGFR, general blood test, blood creatinine level, determination of glycated hemoglobin, lipid spectrum, blood pressure measurement, ultrasound examination of the kidneys) [27]. It is important to note that kidney biopsy in diabetic nephropathy is performed relatively rarely, only when differential diagnosis with other glomerular lesions, such as glomerulonephritis and amyloidosis, is required [3].

The combination of diabetes and CKD requires an integrated approach to therapy, which boils down to observing the basics of a healthy lifestyle, with a high body mass index (BMI over 26 kg/m2), body weight control and rejection of bad habits are necessary. The main goal of drug treatment is to preserve kidney function and achieve intermediate targets for blood glycemia, lipid spectrum, and blood pressure [1]. In the latest clinical recommendations of KDIGO-2024, it was noted that sodium-glucose cotransporter-2 (SGLT2) inhibitors reduce the risk of renal failure in patients with diabetes mellitus. Thus, both RAAS blockers and SGLT2 inhibitors belong to therapies with proven efficacy that slow the progression of CKD in patients with and without diabetes [36]. In patients with CKD without diabetes, the presence of even mild albuminuria means a potential benefit from the use of RAAS blockers, and in the case of severe albuminuria, significant benefits are expected from the use of SGLT2 inhibitors. It is important that the earlier treatment is started, the higher the chance of slowing down the development of the disease and complications. Prevention of diabetic nephropathy is carried out by monitoring blood pressure, sugar and cholesterol levels and achieving targets. In case of diabetes, it is necessary to screen biochemical parameters every 3 months, in addition, it is necessary to monitor blood pressure levels on a systematic basis, as well as to evaluate GFR in patients with more than 5 years of diabetes experience [26].

Conclusion. The focus of domestic healthcare on the early diagnosis of chronic non-communicable diseases with the aim of timely effective therapy to prevent premature mortality and disability is fixed in the orders on medical examination and dispensary supervision of the adult population of the Russian Federation. Among the most significant chronic non-communicable diseases cardiovascular, chronic bronchopulmonary diseases, diabetes mellitus and malignant neoplasms are considered. The progression of CKD against the background of ineffective treatment of CKD mutually aggravates the prognosis in this category of patients. Monitoring of the functional state of the kidneys by the level of eGFR at the outpatient stage serves as an indicator of the effectiveness of chronic non-communicable diseases therapy. Therefore, an indication of the presence and stage of CKD in the clinical diagnosis is mandatory.

The authors declare no conflict of interest in the submitted article.

References

1. Algoritmy specializirovannoj medicinskoj pomoshchi bol'nym saharnym diabetom [Standards of Specialized Diabetes Care]. Edited by Dedov I.I., Shestakova M.V., Mayorov A.Yu. 11th Edition. Diabetes mellitus. 2023; 26 (2S): 1-157. (In Russ.).] DOI: 10.14341/DM13042

2. Chazova I.E., Kislyak O.A., Podzolkov V.I., et al. Arterial'naya gipertenziya i hronicheskaya bolezn' pochek: konsensus po vedeniyu pacientov [Arterial hypertension and chronic kidney disease: consensus on patient management]. Sistemnye Gipertenzii [Systemic Hypertension. 2023; 20 (1): 5-19. (In Russ).] DOI: 10.38109/2075-082X-2023-1-5-19

3. Bagriy A.E., Homenko M.V., Shverova O.I. Diabeticheskaya nefropatiya: voprosy epidemiologii, terminologii, patogeneza, klinicheskoj kartiny i lechebnoj taktiki [Diabetic nephropathy: issues of epidemiology, terminology, pathogenesis, clinical picture and treatment tactics]. Krymskij Terapevticheskij Zhurnal [Crimean Therapeutic Journal. 2022; 1: 15-20. (In Russ.).]

4. Bakina AA, Pavlenko VI, Naryshkina SV. Kompleksnaya ocenka funkcional'nogo sostoyaniya pochek u bol'nyh hronicheskoj obstruktivnoj bolezn'yu legkih [Comprehensive assessment of the functional state of the kidneys in patients with chronic obstructive pulmonary disease]. Sibirskoe medicinskoe obozrenie [Siberian Medical Review. 2020; 2: 45-51 (In Russ.).] DOI: 10.20333/2500136-2020-2-45-51

5. Zhaybergenova Zh.B., Abdirashitova G.S., Ametova A.S. Hronicheskaya pochechnaya nedostatochnost' kak oslozhnenie gipertonicheskoj bolezni [Chronic renal failure as a complication of hypertension]. Ekonomika i Cocium [Economy and Society. 2021; 1 (1): 80. (In Russ.).] Accessed March 4, 2025. https://sciup.org/hronicheskaja-pochechnaja-nedostatochnost-kak-oslozhnenie-gipertonicheskoj-140260254

6. Murkamilov I.T., Sabirov I.S., Fomin V.V., et al. Kliniko - patogeneticheskie aspekty formirovaniya hronicheskoj bolezni pochek pri hronicheskoj obstruktivnoj bolezni legkih [Clinical and pathogenetic aspects of the formation of chronic kidney disease in chronic obstructive pulmonary disease]. The Scientific Heritage [The Scientific Heritage]. 2020; 55 (2): 18-26. (In Russ.).] URL: https://cyberleninka.ru/article/n/kliniko-patogeneticheskie-aspekty-formirovaniya-hronicheskoy-bolezni-pochek-pri-hronicheskoy-obstruktivnoy-bolezni-legkih (date of access: 10.03.2025).

7. Dedov I.I., Shestakova M.V., Mayorov A.Yu., et al. Klinicheskie rekomendacii Rossijskoj associacii endokrinologov. Saharnyj diabet 2 tipa u vzroslyh [Clinical recommendations of the Russian Association of Endocrinologists. Type 2 diabetes mellitus in adults]. Rossijskaya associaciya endokrinologov [Russian Association of Endocrinologists. 2022. (In Russ.).]

8. Belida A.I., Tikhomirova A.D., Kipina E.G., et al. Kompleksnyj podhod k diagnostike ishemicheskoj bolezni serdca v molodom vozraste [An integrated approach to the diagnosis of ischemic heart disease in young people]. Vestnik Nacional'nogo mediko-hirurgicheskogo centra im. N.I. Pirogova [Bulletin of N.I. Pirogov. National Medical and Surgical Center. 2024; 19 (1): 93-98. (In Russ.).] DOI 10.25881/20728255-2024-19-1-93.

9. Kuzmin O.B. Hronicheskaya bolezn' pochek i sostoyanie serdechno-sosudistoj sistemy [Chronic kidney disease and cardiovascular system]. Nefrologiya [Nephrology. 2007; 11 (1): 28-37 (In Russ.).] DOI 10.24884/1561-6274-2007-11-1-28-37

10. Mineev V.N., Vasilieva T.S., Deev D.M. Sushchestvuet li risk razvitiya hronicheskoj bolezni pochek u pacientov s bronhial'noj astmoj? [Is there a risk of developing chronic kidney disease in patients with bronchial asthma?]. Nefrologiya [Nephrology. 2021; 4: 40-47. (In Russ.).] DOI:10.24884/1561-6274-2017-21-4-40-47

11. Medvedeva E.A., Usova E.I., Solovyova A.E., et al. Pacienty s arterial'noj gipertenziej i hronicheskoj bolezn'yu pochek kak prioritetnaya gruppa dlya programm pervichnoj profilaktiki [Patients with arterial hypertension and chronic kidney disease as a priority group for primary prevention programs]. Rossijskij Kardiologicheskij Zhurnal [Russian Journal of Cardiology. (In Russ.).] 2024; 29 (3):5812. DOI: 10.15829/1560-4071-2024-5812.

12. Samakaev A.S., Glova S.E., Zimoveiskaya E.E., et al. Pacienty s ishemicheskoj bolezn'yu serdca i hronicheskoj bolezn'yu pochek - vozmozhnost' primeneniya molekuly kletochnoj adgezii kak prediktora progressirovaniya serdechno-sosudistyh zabolevanij [Patients with coronary heart disease and chronic kidney disease - the possibility of using a cell adhesion molecule as a predictor of the progression of cardiovascular diseases]. Yuzhno-Rossijskij zhurnal terapevticheskoj praktiki [South Russian Journal of Therapeutic Practice. 2023; 4 (2): 56-63 (In Russ.).] DOI: 10.21886/2712-8156 2023-4-2-56-63

13. Gromova E.G., Biryukova L.S., Dzhumabaeva B.T., et al. Prakticheskie rekomendacii po korrekcii nefrotoksichnosti protivoopuholevyh preparatov [Practical recommendations for the correction of nephrotoxicity of antitumor drugs]. Zlokachestvennye Opuholi [Malignant Tumors. 2021; 11 (3s2-2): 132–144 (In Russ.).] DOI:10.18027/2224-5057-2021-11-3s2-46

14. Drapkina O.M., Koncevaya A.V., Kalinina A.M., et al. Profilaktika hronicheskih neinfekcionnyh zabolevanij v Rossijskoj Federacii. Nacional'noe rukovodstvo 2022 [Prevention of chronic non-communicable diseases in the Russian Federation]. Kardiovaskulyarnaya terapiya i profilaktika [Cardiovascular therapy and prevention. 2022; 21 (4):3235 (In Russ.).] DOI:10.15829/1728-8800-2022-3235.

15. Gavrilova A.O., Severina A.S., Shamkhalova M.Sh., et al. Rol' konechnyh produktov glikirovaniya v patogeneze diabeticheskoj nefropatii [The role of advanced glycation end products in the pathogenesis of diabetic nephropathy]. Saharnyj Diabet [Diabetes Mellitus. 2021; 24 (5): 461-469. (In Russ.).] DOI: 10.14341/DM12784

16. Saharnyj diabet s porazheniem pochek. Klinicheskie rekomendacii RF 2013-2017 (Rossiya) [Diabetes mellitus with kidney damage. Clinical guidelines of the Russian Federation 2013-2017 (Russia)]. Rossijskaya associaciya endokrinologov. Utverzhdeny Minzdravom RF 2016 [Russian Association of Endocrinologists. Approved by the Ministry of Health of the Russian Federation 2016 (In Russ.).] The link is active on 04/09/2025. https://diseases.medelement.com/disease/saharnyj-diabet-s-porazheniem-pochek-kr-rf-2016/16692

17. Barbarash O.L., Karpov Yu.A., Panov A.V., et al. Stabil'naya ishemicheskaya bolezn' serdca. Klinicheskie rekomendacii 2024 [Stable ischemic heart disease. Clinical guidelines 2024]. Rossijskij Kardiologicheskij Zhurnal [Russian Cardiology Journal. 2024; 29 (9): 166-229. (In Russ.).] DOI 10.15829/1560-4071-2024-6110

18. Khalilova F.A. Komorbidnoe izmeneniya pochek i serdca u bol'nyh hronicheskoj serdechnoj nedostatochnost'yu [Comorbid changes in the kidneys and heart in patients with chronic heart failure]. Ta\'lim va Rivojlanish Tahlili Onlayn Ilmiy Jurnali. 2023; 3 (5): 524-529. (In Russ.).]

19. Khvatova, Yu.G., Chernyshev A.V. Faktory riska i profilaktika ishemicheskoj bolezni serdca [Risk factors and prevention of ischemic heart disease]. Tambovskij Medicinskij Zhurnal [Tambov Medical Journal. 2024; 6. (2): 92-107. (In Russ.).] DOI 10.20310/2782-5019-2024-6-2-92-107.

20. Hronicheskaya bolezn' pochek (HBP). Klinicheskie rekomendacii 2024 [Chronic kidney disease (CKD). Clinical Guidelines 2024]. Nacional'naya Associaciya nefrologov [National Association of Nephrologists. 2024-2025-2026 (11/19/2024). Approved by the Ministry of Health of the Russian Federation. 2024; 242 p. (In Russ.).]

21. Ebzeeva E.Yu., De V.A., Ni L.I. [et al.] Khronicheskaya bolezn' pochek i arterial'naya gipertenziya: kak razorvat' porochnyj krug? [Chronic Kidney Disease and Arterial Hypertension: How to Break the Vicious Cycle?] Russkij Medicinskij Zhurnal [Russian Medical Journal. 2022; 5: 30-34.(In Russ.).]

22. Mezhonov E.M., Reytblat O.M., Yu. A. Vyalkina [et al.] Hronicheskaya bolezn' pochek i hronicheskaya serdechnaya nedostatochnost': vliyanie na prognoz i vybor patogeneticheskoj terapii [Chronic kidney disease and chronic heart failure: impact on prognosis and choice of pathogenetic therapy]. Terapevticheskij Arhiv [Therapeutic Archives. 2024; 7: 666–674 (In Russ.).] DOI: 10.26442/00403660.2024.07.202781 (In Russian)

23. Murkamilov I.T., Sabirov I.S., Fomin V.V., et al. Khronicheskaya bolezn' pochek i zlokachestvennye novoobrazovaniya: sovremennoe sostoyanie problem [Chronic kidney disease and malignant neoplasms: current state of the problem]. Arhiv" Vnutrennej Mediciny [Archives of Internal Medicine. 2022; 12 (2): 104–112 (In Russ.).] DOI:10.20514/2226-6704-2021-12-2-104-112

24. Galyavich A.S., Tereshchenko S.N., Uskach T.M., et al. Khronicheskaya serdechnaya nedostatochnost'. Klinicheskie rekomendacii 2024 [Chronic heart failure. Clinical guidelines 2024]. Rossijskij Kardiologicheskij Zhurnal [Russian Cardiology Journal. 2024; 29. (11): 251-349. (In Russ.).] DOI 10.15829/1560-4071-2024-6162.

25. Kilessa V.V., Zhukova N.V., Itskova E.A., et al. Hronicheskaya serdechnaya nedostatochnost' pri ishemicheskoj bolezni serdca – sistemnaya mikrocirkulyatornaya patologiya [Chronic heart failure in ischemic heart disease is a systemic microcirculatory pathology]. Krymskij terapevticheskij zhurnal [Crimean Therapeutic Journal. 2024; 3: 51-58. (In Russ.).]

26. Khudoerova D.R. Prognoz i profilaktika diabeticheskoj nefropatii [Prognosis and prevention of diabetic nephropathy]. Nauchno-issledovatel'skij zhurnal po travmatologii i invalidnosti [Research Journal of Trauma and Disability Studies. 2023; 2 (4): 75–84. (In Russ.).] Accessed March 4, 2025. https://journals.academiczone.net/index.php/rjtds/article/view/693

27. Shestakova M.V. Saharnyj diabet i hronicheskaya bolezn' pochek: vozmozhnosti prognozirovaniya, rannej diagnostiki i nefroprotekcii v XXI veke [Diabetes mellitus and chronic kidney disease: possibilities of prognosis, early diagnosis and nephroprotection in the 21st century]. Terapevticheskij arhiv [Therapeutic Archive. 2016; 6: 84-88 (In Russ.).] DOI: 10.17116/terarkh201688684-88

28. Evans M, Lewis R.D., Morgan A.R., et al. A Narrative Review of Chronic Kidney Disease in Clinical Practice: Current Challenges and Future Perspectives. Adv Ther. 2022; 39 (1): 33-43. DOI: 10.1007/s12325-021-01927-z.

29. Huang H.L., Ho S.Y., Li C.H., et al. Bronchial asthma is associated with increased risk of chronic kidney disease. BMC Pulm Med. 2014; 14-80. DOI:10.1186/1471-2466-14-80

30. Hu M, Wang Q, Liu B, et al. Chronic Kidney Disease and Cancer: Inter-Relationships and Mechanisms. Frontiers in Cell and Developmental Biology. 2022; 10: 868715. DOI: 10.3389/fcell.2022.868715

31. Szlagor M, Dybiec J, Młynarska E, et al. Chronic Kidney Disease as a Comorbidity in Heart Failure. International Journal of Molecular Sciences. 2023; 24: 3:2988. DOI: 10.3390/ijms24032988.

32. Jha V, Garcia-Garcia G, Iseki K, et al. Chronic kidney disease: global dimension and perspectives. Lancet. 2013; 382 (9888): 260-272. DOI:10.1016/S0140-6736(13)60687-X

33. Solomon S.D., Chew E, Duh EJ, et al. Diabetic Retinopathy: A Position Statement by the American Diabetes. Diabetes Care Association. 2017; 40 (3): 412–418. DOI: 10.2337/dc16-2641

34. Sparks J.A., Vanni K.M.M., Sparks M.A., et al. Effect of Low-Dose Methotrexate on eGFR and Kidney Adverse Events: A Randomized Clinical Trial. Journal of the American Society of Nephrology. 2021; 32 (12): 3197-3207. DOI: 10.1681/ASN.2021050598.

35. Maduros N., Jarvis S, Saleem A, et al. Is There an Association Between Chronic Obstructive Pulmonary Disease and Chronic Renal Failure? Cureus: Journal of Medical Science. 2022; 14 (6): e26149. DOI:10.7759/cureus.26149

36. KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. Kidney International. 2024; 105 (Suppl 4S): 117–S314. DOI: 10.1016/j.kint.2023.10.018. The link is active on 04/09/2025. https://congress-med.ru/assets/files/Полезная%20информация/2024/25.08.2024/2024-kdigo-rekomendaczii-po-xbp.pdf

37. Lees J.S., Elyan B.M.P., Herrmann S.M., et al. The 'other' big complication: how chronic kidney disease impacts on cancer risks and outcomes. Nephrol Dial Transplant. 38 (5): 1071–1079. DOI: 10.1093/ndt/gfac011