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# RATIONAL PHARMACOTHERAPY IN THE TREATMENT OF ISCHEMIC HEART DISEASE

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*Ischemic heart disease, angina pectoris, chronic heart failure, pharmacotherapy, beta-blockers, statins, antithrombotic agents, ACE inhibitors, diuretics, atherosclerosis, rational treatment, polypharmacy.*

**ABSTRACT**

*This scientific article provides an in-depth analysis of the theoretical and practical aspects of rational pharmacotherapy in the treatment of ischemic heart disease (IHD). Ischemic heart diseases mainly include angina pectoris, myocardial infarction, post-infarction cardiosclerosis, chronic heart failure, acute heart failure, cardiac arrhythmias, and coronary insufficiency. The most common forms are angina pectoris and myocardial infarction. Chronic heart failure (CHF) is considered a major complication of the disease.The study examines the clinical efficacy, safety, and principles of using antianginal, antithrombotic, diuretic drugs, cardiac glycosides, ACE inhibitors, lipid-lowering agents, and medications affecting the neurohormonal system. In addition, issues such as individualized treatment approaches, limitation of polypharmacy, and pharmacoeconomic efficiency are discussed. The results obtained demonstrate that rational pharmacotherapy plays a crucial role in improving the prognosis of patients with ischemic heart disease.*

# РАЦИОНАЛЬНАЯ ФАРМАКОТЕРАПИЯ ПРИ ЛЕЧЕНИИ ИШЕМИЧЕСКОЙ БОЛЕЗНИ СЕРДЦА

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**ABSTRACT**

*В данной научной статье подробно анализируются теоретические и практические аспекты рациональной фармакотерапии при лечении ишемической болезни сердца (ИБС). К основным формам ишемической болезни сердца относятся: стенокардия, инфаркт миокарда, постинфарктный кардиосклероз, хроническая сердечная недостаточность, острая сердечная недостаточность, сердечные аритмии, коронарная*

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**KEYWORDS**

*Ишемическая болезнь сердца, стенокардия, хроническая сердечная недостаточность, фармакотерапия, бета-блокаторы, статины, антитромботические средства, ингибиторы АПФ, диуретики, атеросклероз, рациональное лечение, полипрагмазия.*

*недостаточность. Наиболее распространёнными являются стенокардия и инфаркт миокарда. В качестве осложнения заболевания часто развивается хроническая сердечная недостаточность (ХСН). В исследовании рассмотрены клиническая эффективность, безопасность и принципы применения антиангинальных, антитромботических, диуретических средств, сердечных гликозидов, ингибиторов АПФ, гиполипидемических препаратов, а также лекарственных средств, влияющих на нейрогормональную систему. Кроме того, освещены вопросы индивидуального подхода к лечению, ограничения полипрагмазии и фармакоэкономической эффективности. Полученные результаты показывают, что рациональная фармакотерапия играет важную роль в улучшении прогноза пациентов с ишемической болезнью сердца.*

# YURAK ISHEMIK KASALLIKLARINI DAVOLASHDA RATSIONAL FARMAKOTERAPIYA

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*Yurak ishemik kasalligi, stenokardiya, surunkali yurak yetishmovchiligi, farmakoterapiya, beta-blokatorlar, statinlar, antitrombotik vositalar, ACE ingibitorlari, diuretiklar, ateroskleroz, ratsional davolash, polifarmasiya.*

**ABSTRACT**

*Mazkur ilmiy maqolada yurak ishemik kasalliklarini (YIK) davolashda ratsional farmakoterapiyaning nazariy va amaliy jihatlari chuqur tahlil qilinadi. Asosan, yurak ishemik kasalliklariga: stenokardiya, miokard infarkt, infarktdan keyingi kardioskleroz, surunkali yurak yetishmovchiligi, o'tkir yurak yetishmovchiligi, yurak aritmiyasi, toj tomir yetishmovchiligi kabi kasalliklar kiradi. Eng keng tarqalgani yurak stenokardiyasi va miokard infarkt. Kasallik asorati sifatida esa surunkali yurak yetishmovchiligi (SYY). Tadqiqotda antianginal, antitrombotik, diuretik, yurak glikazidlari, APF ingiitorlari, gipolipidemik va neyrogormonal tizimga ta'sir qiluvchi dori vositalarining klinik samaradorligi, xavfsizligi hamda qo'llash prinsiplari ko'rib chiqilgan. Shuningdek, individual yondashuv, polifarmasiyani cheklash va farmakoiqtisodiy samaradorlik masalalari yoritilgan. Olingan natijalar ratsional farmakoterapiya YIK bilan og'rigan bemorlarning prognozini yaxshilashda muhim ahamiyat kasb etishini ko'rsatadi.*

**Introduction.** Ischemic heart disease is one of the most pressing problems of modern medicine and occurs as a result of insufficient oxygen supply to the heart muscle. This pathology is most commonly associated with atherosclerotic narrowing of the coronary arteries and clinically manifests as angina pectoris, myocardial infarction, and heart failure. According to the World Health Organization, cardiovascular diseases remain the leading cause of death worldwide. Therefore, effective treatment and prevention of ischemic heart disease are among the top priorities of contemporary medicine. Pharmacotherapy is the main component in the treatment of ischemic heart disease (IHD). However, incorrect selection of medications, their excessive use, or failure to consider individual patient characteristics can reduce the effectiveness of treatment. From this perspective, the concept of rational pharmacotherapy is of particular importance. For example, the impact of diuretics on morbidity and mortality in patients with chronic heart failure (CHF) has not been sufficiently studied in long-term trials. At the same time, the use of diuretics helps eliminate symptoms associated with fluid retention, such as peripheral edema, shortness of breath, and pulmonary congestion. This justifies their use in patients with CHF regardless of the level of left ventricular ejection fraction (LVEF). Diuretics are also recommended to relieve symptoms of heart failure and to improve physical activity in patients with signs of fluid accumulation. Fluid retention and the development of edema syndrome are typical and well-known manifestations of CHF, usually beginning from functional class II. Therefore, dehydration therapy is considered one of the most important components in the successful management of patients with CHF. However, it should be noted that complex neurohormonal mechanisms are involved in the development of edema syndrome, and inappropriate dehydration may lead only to adverse effects and result in “rebound fluid retention”. The condition commonly referred to as edema and dyspnea is characterized by the accumulation of fluid in the extracellular space [1].

**Materials and Methods.** This study has a systematic analysis design and employed the following methods: a systematic review of scientific literature, analysis of clinical trial results, as well as comparative and generalization methods. The sources included recommendations from international cardiology societies such as the European Society of Cardiology (ESC) and the American Heart Association (AHA); randomized clinical trials; meta-analyses; and review articles. The evaluation criteria included clinical efficacy, safety profile, patients’ quality of life, and pharmacoeconomic effectiveness. In addition, as noted above, lipid-lowering drugs have long been widely used in the treatment of ischemic heart disease (IHD). This is because one of the main etiological factors of IHD is atherosclerotic damage to the vascular walls. The process of atherosclerosis contributes not only to IHD but also to the development of pathologies in other organs. The risk of developing cardiovascular complications was

calculated using the SCORE scale. The diagnosis of chronic obstructive pulmonary disease (COPD) was established in accordance with the criteria of the Global Initiative for Chronic Obstructive Lung Disease (GOLD, 2017, 2020). The criteria for dyslipidemia were defined according to the 2019 European clinical guidelines [4].

Laboratory investigations included the assessment of the lipid profile (total cholesterol (TC), low-density lipoproteins (LDL), high-density lipoproteins (HDL), triglycerides (TG)), as well as the determination of primary (diene conjugates (DC)), secondary (triene conjugates (TC)), and end products (Schiff bases (SB)) of lipid peroxidation using the method of I.A. Volchegorsky (1989). Furthermore, the intensity of free radical oxidation in blood serum was evaluated using the method of induced biochemiluminescence (S, Imax) according to E.I. Kuzmina, A.S. Nelyubina, and M.K. Shchennikova (1983). To assess the tolerability of the prescribed therapy, creatinine, bilirubin, alanine aminotransferase (ALT), and aspartate aminotransferase (AST) levels were dynamically monitored.

**Analysis and Discussion.** The main factor in the development of ischemic heart disease (IHD) is atherosclerotic damage to the coronary arteries. As a result of plaque formation, the vascular lumen narrows, leading to myocardial ischemia. Therefore, based on the etiological and pathogenetic mechanisms of the disease, pharmacotherapy aims to achieve the following key goals: reduction of ischemia, prevention of thrombosis, slowing the progression of atherosclerosis, and reduction of complications. Antianginal pharmacotherapy. Antianginal drugs form the basis of symptomatic treatment. Beta-blockers reduce heart rate and decrease myocardial oxygen demand. Their effectiveness in reducing the risk of myocardial infarction and mortality has been well established. Nitrates dilate venous vessels and reduce venous return to the heart. Rapid-acting forms are used to relieve angina attacks. Calcium channel blockers dilate arterial vessels and improve coronary blood flow. They are particularly effective in vasospastic angina. Regarding the use of diuretics, there have been very few large placebo-controlled trials (with the exception of the AMCRA study), and therefore most recommendations are based on expert opinion. Formally, this corresponds to level C evidence. However, considering the extensive clinical experience with diuretics, there is no doubt about their appropriateness in all patients with chronic heart failure (CHF) who present with fluid overload. The main principles of dehydration therapy, including the use of diuretics, are as follows [2]: diuretics are used in patients with CHF to eliminate edema syndrome and reduce clinical symptoms. When used correctly, these drugs can decrease the number of hospitalizations, thereby achieving two of the six main goals in CHF management. Most diuretics (with the exception of torasemide) do not slow the progression of CHF or improve patient prognosis If administered in inappropriate doses (for example, in high “shock” doses every 3–4–5–7 days), they may negatively affect

patients' quality of life. Torasemide is considered one of the most effective and safe loop diuretics with an optimal pharmacokinetic profile. The initial dose of the drug is 2.5–5 mg, which can be increased up to 100–200 mg per day depending on clinical necessity. Torasemide is a typical loop diuretic that blocks the reabsorption of sodium and water in the ascending limb of the loop of Henle. In terms of pharmacokinetics, it is superior to furosemide (with a duration of action of up to 18 hours), has better and more predictable absorption (about 90% versus 50%), is independent of food intake and its bioavailability is almost twice as high as that of furosemide [3].

Antithrombotic therapy reduces the risk of acute coronary events by decreasing platelet aggregation. For example, acetylsalicylic acid (aspirin) is the main drug, along with P2Y12 receptor blockers such as clopidogrel. Dual antithrombotic therapy is recommended for high-risk patients. Lipid-lowering therapy. Statins are the cornerstone of IHD treatment. Their main effects include reducing LDL cholesterol, stabilizing atherosclerotic plaques, and decreasing inflammation. Intensive statin therapy is particularly beneficial in high-risk patients. After 4 weeks of treatment with pitavastatin at a starting dose of 4 mg, a significant improvement in all lipid profile parameters was observed: total cholesterol decreased by 26%, LDL by 33%, triglycerides by 19%, while HDL increased by 18% compared to baseline levels. Initially, the primary (DC, TC) and final (Schiff bases) products of lipid peroxidation were elevated above normal levels. Analysis using the nonparametric Kruskal–Wallis test showed that the accumulation of lipid peroxidation products and the level of ET-1 increased in parallel with the severity of broncho-obstructive disorders in COPD and cardiovascular risk. It should be noted that among endothelial dysfunction indicators, only ET-1 demonstrated a significant association with the degree of broncho-obstruction (Kruskal–Wallis criterion = 14.7; $p < 0.001$) and cardiovascular risk (Kruskal–Wallis criterion = 34.76; $p < 0.001$). After 4 weeks of treatment with pitavastatin, improvements were observed in endothelial dysfunction markers and lipid peroxidation processes. Statistically significant changes were noted for DC, TC, Schiff bases, S, and Imax. The increase in the Schiff bases/(DC + TC) ratio after treatment was associated with a decrease in primary products (DC) and accumulation of final products. The indicators of the antioxidant defense system (Imax) also normalized. Initially, during the ESPV test, patients showed a decrease in the diameter and blood flow velocity of the brachial artery, with ESPV at 4.9% [5]. After 4 weeks of treatment, a statistically significant increase in ESPV to 6.6% was observed [2]. ACE inhibitors lower blood pressure, improve endothelial function, and slow cardiac remodeling. Angiotensin receptor blockers (ARBs) are used as an alternative to ACE inhibitors.

Modern approaches. In recent years, new drugs have also been widely introduced into clinical practice: ivabradine, which selectively reduces heart rate, and ranolazine, which acts through metabolic mechanisms.

Principles of rational pharmacotherapy. Rational treatment includes the following key principles: individualization (considering patient age, and comorbidities), evidence-based approach, use of the minimum effective dose, consideration of drug interactions, limitation of polypharmacy. Consequences of irrational pharmacotherapy may include increased adverse effects, reduced treatment efficacy, and higher economic burden.

**Results.** The analysis showed the following: combination pharmacotherapy significantly reduces angina symptoms, statin use lowers cardiovascular events by 30–50%, antithrombotic therapy sharply decreases the risk of thrombosis, and ACE inhibitors improve long-term prognosis. Rational approach: Optimizing therapy improves patient adherence, reduces adverse effects, and optimizes healthcare costs. Among diuretics, torasemide stands out as the first diuretic that not only alleviates symptoms in CHF patients but also influences disease progression and mitigates pathological processes within the myocardium. Recent Russian studies have confirmed torasemide's ability to affect left ventricular (LV) remodeling and normalize the ratio of collagen synthesis/degradation markers. Additionally, torasemide use helps overcome major limitations of conventional diuretic therapy: it enhances diuretic efficacy while mitigating side effects such as electrolyte imbalances and RAAS activation. Its long-lasting, stable diuretic effect (14–18 hours for torasemide vs. 4–5 hours for furosemide) allows greater patient mobility, significantly improving adherence to therapy [4]. Another important pharmacological direction is the suppression of maladaptive renin–angiotensin–aldosterone system (RAAS) activation, which remains excessively active in CHF even with normal or increased blood volume. Chronic angiotensin II exposure promotes myocyte hypertrophy, fibrosis, endothelial dysfunction, and apoptotic signaling. ACE inhibitors and ARBs help mitigate these processes by reducing excessive load, stimulating natriuresis, and improving coronary perfusion. Furthermore, combining neprilysin inhibition with an angiotensin receptor blocker provides a more effective therapeutic platform. ARNIs (angiotensin receptor–neprilysin inhibitors) increase endogenous natriuretic peptides while suppressing RAAS, resulting in dual hemodynamic and metabolic benefits. These include improved cardiac output, enhanced diuresis independent of renal function, reduced ventricular wall stress, and accelerated reverse remodeling. Transitioning patients with reduced left ventricular ejection fraction from ACE inhibitors to ARNIs has become a critical step in achieving long-term stability [2].

**Conclusion.** Rational pharmacotherapy plays a critical role in the treatment of ischemic heart disease (IHD), as it helps control disease progression and reduce complications. A comprehensive clinical-pharmacological approach also considers the important interaction between the heart and kidneys, since impaired renal function complicates drug dosing, increases the risk of adverse effects, and limits

therapeutic options. Adjusting drug doses based on estimated glomerular filtration rate (GFR), avoiding nephrotoxic combinations, and monitoring electrolyte balance are key components of long-term safe management. Close monitoring is especially important when using MRAs, diuretics, or ARNIs, as hyperkalemia and worsening renal function can quickly destabilize patients.

Incorporating biomarkers such as NT-proBNP, soluble ST2 (sST2), and high-sensitivity troponins into pharmacological decision-making improves prognostic accuracy and helps individualize therapy [4]. The following conclusions were drawn: a comprehensive and individualized approach is the most effective; statins, antithrombotics, and diuretics should form the backbone of therapy; modern agents expand treatment possibilities, and rational pharmacotherapy improves healthcare system efficiency.

In the future, treatment strategies based on pharmacogenetics and personalized medicine are expected to advance further.

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