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YURAKNING KORONAR QON-TOMIRLAR KASALLIGI BILAN OG'RIGAN BEMORLARDA BUYRAK DISFUNKSIYASINI ZAMONAVIY TASHXISLASH TAMOYILLARI

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✓ ***Rezyume***

Buyrak disfunksiyasi muammosi va uning yurak qon-tomir kasalligi bilan og'rigan bemorlarning turli guruhlariga ta'siri keng qo'llanilmoqda. Buyrak disfunksiyasi bir qator yurak-qon tomir xavf omillari bilan bog'liq. Buyrak shikastlanishini aniqlash uchun yangi biomarkerlardan, xususan, sistatin C dan foydalanish buyrak yetishmovchiligi xavfini erta bashorat qilishga yordam beradi. Sistatin C nafaqat buyrak shikastlanishining dastlabki shakllarini aniqlashda, balki buyrak o'rnini bosuvchi terapiya ehtiyojlarini va shifoxonalarda reanimatsiya bo'limidagi bemorlarda o'lim xavfini baholashda yordam beradigan ideal biomarker sifatida ko'plab xususiyatlarni ochib beradi.

Kalit so'zlar: buyrak disfunktsiyasi, yurak-qon tomir kasalligi, sistatin S.

СОВРЕМЕННАЯ ДИАГНОСТИКА ПОЧЕЧНОЙ ДИСФУНКЦИИ У ПАЦИЕНТОВ С ИШЕМИЧЕСКОЙ БОЛЕЗНЬЮ СЕРДЦА

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✓ ***Резюме***

Проблема дисфункции почек и ее влияние на исходы в различных группах кардиологических больных продолжает широко использоваться. Дисфункция почек связана с рядом традиционных факторов риска сердечно-сосудистых заболеваний. Использование новых биомаркеров, в частности цистатина С, для выявления повреждения почек может способствовать улучшению раннего прогнозирования риска развития почечной недостаточности (РФ). Цистатин С удовлетворяет многим характеристикам в качестве идеального биомаркера, который может помочь не только в выявлении ранних форм повреждения почек, но и в оценке риска ФР, потребности в заместительной почечной терапии и риска смерти у пациентов отделений интенсивной терапии в кардиологических клиниках. Поражение почек при многих заболеваниях, в том числе и тех, которые изначально не рассматриваются как почечные, обуславливает необходимость разработки единых подходов к ведению пациентов с выявленной хронической ФР, особенно к ранней профилактике и лечению ее осложнений, таких как анемия, фосфорно-кальциевые метаболические нарушения, которые существенно ухудшают прогноз других заболеваний.

Ключевые слова: почечная дисфункция, ишемическая болезнь сердца, цистатин С.

THE CURRENT DIAGNOSIS OF KIDNEY DYSFUNCTION IN PATIENTS WITH CORONARY HEART DISEASE

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✓ ***Resume***

The problem of kidney dysfunction and its impact on outcomes in different groups of cardiac patients continue to being widely used. Kidney dysfunction is associated with a number of traditional cardiovascular risk factors. The use of new biomarkers, cystatin C in particular, to identify kidney injury can contribute to the improvement of early prediction of a risk for renal failure (RF). Cystatin C satisfies many characteristics as an ideal biomarker that can assist in not only detecting the early forms of kidney injury, but also in assessing the risk of RF, the needs for renal replacement therapy, and the risk of death in intensive care unit patients in cardiac clinics. Kidney involvement in many diseases, including those that are not initially regarded as renal, necessitates the elaboration of uniform approaches to managing patients with identified chronic RF, especially to the early prevention and treatment of its complications, such as anemia, phosphorus-calcium metabolic disorders, which substantially worsen the prognosis of other diseases.

Key words: renal dysfunction, coronary heart disease, cystatin C.

Relevance

Since the mid-20th century, cardiovascular diseases (CVD) have been the leading cause of death and disability in the industrialized world, leading to a steady increase in healthcare costs. At the same time, experts from the World Health Organization predict a further increase in cardiovascular morbidity and mortality in both economically developed and developing countries due to population aging and lifestyle factors [1]. Epidemiologists pay special attention to the early detection and correction of risk factors (RF) that determine the destabilization of the course of diseases associated with atherosclerosis, including coronary heart disease (CHD). Numerous studies have proven the role of renal dysfunction (RD) as an independent predictor of cardiovascular morbidity and mortality. It has been shown that a moderate decrease in renal function increases the risk of developing arterial hypertension (AH), CHD, heart failure (HF) and death from CVD [1,2]. Chronic kidney disease (CKD) is defined as kidney damage or decreased kidney function for 3 months or more, i.e. the presence of chronic DP. The disease is classified into 5 stages, which differ in patient management tactics, the risk of developing end-stage renal failure (ESRF) and cardiovascular complications (CVC). Anamnestic indication of the presence of morphological kidney pathology is not necessary for verification of CKD. It has been established that a patient with CKD is 20 times more likely to die from heart disease than from end-stage kidney disease [3]. At the same time, in patients with end-stage CKD, CVD is the cause of 43-50% of all deaths [4], which determines the relevance of identifying DP in patients with CVD and the need for their aggressive prevention in patients with CKD. The problem of DP and its impact on outcomes in various groups of cardiac patients continues to be widely discussed. The increase in the number of patients with coronary heart disease with DP can be explained by both an increase in the proportion of elderly patients (given the known association of decreased renal function with age [5] and the influence of increasingly common background and concomitant pathology (type 2 diabetes mellitus - DM-2, hypertension, chronic heart failure - CHF). It is known that mortality due to acute DP in the general population is 41%; among patients with myocardial infarction (MI) with ST segment elevation - 55%, among patients with MI without ST segment elevation - 22% [6]. According to F. Masoudi et al. [7], in the USA, among hospital patients, normal renal function was determined in only 16%, mild decrease in renal function (glomerular filtration rate - SFR 60-89 ml/min/1.73 m2) - in 43%, moderate (SFR 30-59 ml/min/1.73 m2) - in 32% and severe (SFR 30 ml/min/1.73 m2) - in 9%. Mortality in hospitals in patients with concomitant CKD is 21% compared to 6-8% in the general population of patients with MI [8]. The presence of DP is associated with a number of traditional risk factors for the development of CVD. In our previously published studies, we have shown that the prevalence of DP among patients with ST-segment elevation MI is 37%, it is more often detected in older patients, women, as well as in patients with a complicated cardiovascular history and more severe manifestations of acute HF according to Killip. In addition, patients with DP are less likely to undergo percutaneous interventions, which is an additional factor worsening their prognosis. Decreased renal function is associated with such background (for coronary heart disease) diseases as hypertension, diabetes mellitus (DM), CHF and increases the likelihood of death during the hospital period [9]. Thus, among patients with CHF, kidney pathology occurs in 44% [10]. The problem of acute kidney injury (AKI) is also relevant when performing therapeutic and diagnostic radiological procedures with contrast. The prevalence of nephropathy induced by a contrast agent (contrast-induced - CIN) in the general population does not exceed 2% [11]. In high-risk groups (elderly patients, those with previous kidney disease, diabetes, heart failure, arterial hypotension, anemia, dehydration, and those using nonsteroidal anti-inflammatory drugs), the prevalence of CIN reaches 20–30% [11]. There is evidence that taking rosuvastatin

at a dose of 10 mg/day 2 days before angiographic examination and for 3 days after it significantly reduces the risk of developing contrast-induced AKI [12].

It is also important to remember about assessing renal function when prescribing any long-term drug therapy, which is especially important for drugs in the groups of anticoagulants, hypoglycemic drugs (HDA) and statins. The problem of undiagnosed CKD is especially relevant among patients with diabetes. According to registry studies, almost 40% of patients with type 2 diabetes are characterized by the presence of CKD of various stages, which, however, is not taken into account when prescribing oral HDA. This fact is the cause of therapy-induced transient episodes of hypoglycemia, which naturally worsens the prognosis of the underlying disease [13]. At the same time, most antidiabetic drugs have limitations, and in some cases, contraindications when prescribed to patients with DP. The daily dose of HDA should be adjusted depending on the SCF, this is especially true for metformin, since it is contraindicated in moderate and severe DP [14]. New antidiabetic drugs are better tolerated by patients with severe renal impairment, although clinical experience remains limited for many of them [15]. For patients with a cardiological profile, the problem of dosing and indirect anticoagulants is relevant. In a multicenter prospective study EPICA, which included 4093 patients over 80 years old who took vitamin K antagonists for atrial fibrillation (AF), the prevalence of CKD, determined by SCF, was assessed. The risk of bleeding was determined using the Cockcroft-Gault and MDRD (Modification of Diet in Renal Disease) formulas. It was found that the risk of bleeding is higher in patients taking vitamin K antagonists with moderate and severe atrial fibrillation, regardless of the formula for determining SCF. These data confirm the need for more frequent monitoring of blood coagulation parameters in elderly patients [15]. The ROCKET-AF study assessed the efficacy and safety of rivaroxaban, a new factor X inhibitor. The study included 14,264 patients with non-valvular AF and additional stroke risk factors compared with standard warfarin therapy under the control of an international normalized ratio of 2 to 3 [15]. Rivaroxaban is known to be primarily metabolized by the liver, but about 1/3 of it is metabolized by the kidneys [13]. Patients with an SCF of 30 ml/min were excluded from the study, while the daily dose of rivaroxaban was reduced from 20 to 15 mg in patients with an SCF of 30 to 49 ml/min. It has been established that new anticoagulants (X-factor inhibitors), such as rivaroxaban, dabigatran and apixaban, are not associated with a higher risk of bleeding in patients with non-valvular AF and concomitant CKD [12]. Thus, DP is associated with a number of traditional risk factors for the development of CVC, while it has not only an indirect but also an independent effect on the occurrence of adverse outcomes in patients with coronary heart disease; its identification is an important condition for safe treatment. Currently, the diagnosis of AKI or chronic kidney injury is based on the detection of changes in the volume of hourly diuresis, the concentration of creatinine in the blood and SCF. It is known that an increase in the level of creatinine in the blood serum can depend not only on kidney function, but also on many other factors not associated with the excretory system [12], in particular, on the pathology of the musculoskeletal system and liver. The main limitation of kidney injury diagnostics using creatinine level assessment is its late increase: it has been determined that in many cases it occurs only 24-48 hours after kidney injury. The explanation may be that the kidneys have a significant functional reserve, so the creatinine concentration does not change until 60% of the renal parenchyma is morphologically lost. Renal function itself is traditionally assessed using the formulas for calculating the SCF: Cockcroft-Gault and MDRD. The MDRD formula was developed on the basis of a study involving 1,628 people, of whom 1,070 were randomly selected and 500 people formed the control group. The aim of this study was to determine the most accurate formula for calculating the SCF. Calculations were made using stepwise regression analysis. The results of the study showed that the MDRD formula calculates the SCF more accurately and, accordingly, has a higher sensitivity compared to the calculation of creatinine clearance in determining the stage of DP [11]. The advantages of the MDRD formula are that it is derived from the determination of the renal clearance of 125I-iothalamate in a large group of patients, both Caucasian and Negroid, with a wide range of kidney diseases. The formula allows one to estimate the SCF standardized by body surface area. There are two versions of the MDRD formula: full and abbreviated. To calculate the SCF using the full (original) formula, a number of biochemical parameters are required along with serum creatinine. To use the abbreviated MDRD formula, only demographic data (gender, age, race) and serum creatinine level are required. The formula is designed for men, and a correction factor is proposed for women. In the MDRD study, which evaluated the Cockcroft-Gault formula in one laboratory, it overestimated the SCF by 23%. In addition, the Cockcroft-Gault formula overestimates the CrCr at a SCF level of 60 ml/min. Thus, the assessment of DP, especially at normal or borderline creatinine levels, is more accurately carried out using the MDRD formula and, in general, the presented formulas reflect different processes. However, MDRD is not ideal either, since errors may be detected in patients with initial manifestations of DP. It is clear that for prospective and early diagnostics of AKI at the earliest possible stage according to the RIFLE criteria [13] adopted in leading clinics, more sensitive markers than an increase in

the level of creatinine in the blood are needed. The limitations of the use of SCF are reduced to the listed reasons, since the main criterion for calculating the SCF is the same level of creatinine in the blood. Thus, in recent years, there has been great interest in the search for new, highly sensitive biological markers that allow one to determine renal dysfunction at the initial stages and to assess the individual risk and prognosis of DP development in a patient, as well as to monitor the effectiveness of treatment of the underlying disease in relation to potential nephrotoxicity. It is assumed that the "ideal" biomarker should be produced by damaged kidney cells, its level should increase immediately after even a small volume of damage to the renal tissue, when it is still potentially reversible for the organ. In addition, the level of such a biomarker should be proportional to the degree of damage for potential riskometry and monitoring of treatment effectiveness. It should decrease soon after improvement of kidney function. Such a marker should be collected for laboratory testing in a non-invasive way, provide the possibility of simple and rapid measurement, reflect the pathophysiology of the disease and have high prognostic significance [14]. According to many authors, the most promising biomarkers for early diagnostics of kidney damage are those not associated with impaired renal filtration function, but reflecting morphological damage to the renal parenchyma, proliferation, differentiation and apoptosis of cells, immune response disorders and production of cytokines and chemokines [15]. Based on the results of experimental studies, a number of biomarkers have been proposed for early diagnostics of DP, including interleukin-18 (IL-18), kidney injury molecule type 1 (KIM-1), neutrophil gelatinase-associated lipocalin (NGAL), and cystatin C [12]. IL-18 is a pleiotropic proinflammatory cytokine that plays an important role in the inflammatory cascade [7]. Thus, the results of a number of studies have shown a slight increase in its content in kidney tissue under conditions of experimentally induced damage and its key role in the induction of ischemic kidney damage [8,9]. In isolated damage to the proximal tubules of the nephron in mice, an increase in the content of IL-18 in the hypoxic zone and an increase in its level in the urine in the ischemic variant of AKI were detected [10]. This prompted a number of clinical studies aimed at proving the possible role of IL-18 as a biomarker of AKI. There is evidence that the expression of IL-8 may be associated with the progression and damage of the atherosclerotic plaque [13]. In the study by C. Parickh et al. [12], it was demonstrated that elevated concentrations of IL-18 are an independent risk factor for the development of CVD. Another biological marker that can potentially be considered as an early indicator of AKI is KIM-1, which is determined in urine. This is a transmembrane protein with immunoglobulin and mucin domains, first described in 1998 [7]. In intact renal tissue, it is contained in small amounts, but after renal ischemia, its level in regenerating proximal tubules increases significantly. As shown in experimental models, an increase in the KIM-1 level is associated with ischemic kidney injury and is not always accompanied by an increase in blood urea nitrogen and creatinine [14]. Numerous studies show that KIM-1 is a highly sensitive and specific marker of kidney injury and has a high prognostic value in predicting the risk of developing acute renal failure [15] in patients with CVD. NGAL is a protein with a molecular weight of 25 kDa, initially detected in neutrophils and somewhat later in small amounts in the epithelium of renal tubules. In healthy humans, NGAL is not detected in the blood or is present in small concentrations in various tissues or organs with activated epithelial cells [15]. In ischemic kidney injury, its expression in tubular epithelial cells increases many times, its concentration in the blood and excretion in the urine increase, outpacing the increase in creatinine concentration by 24-48 hours [7].

In a study devoted to the prognostic role of NGAL in urine in patients after open heart and vascular surgery under conditions of prolonged artificial circulation, it was found that the NGAL level in urine increased 2-6 times already 2 hours after surgery, while creatinine and urea concentrations remained within the permissible values. 24 hours after surgery, the NGAL level in urine returned to baseline. An increase in the concentration of creatinine in the blood and urea was noted only on the 2nd day [8]. Thus, lipocalin has proven its effectiveness in early prediction of the risk of AKI, especially in patients with coronary artery disease, after planned coronary artery bypass grafting (CABG) [9]. Another actively discussed biological marker of early kidney injury, cystatin C, is a non-glycosylated protein with a molecular weight of 13.4 kDa and an isoelectric point at pH 9.3. Belongs to the family of cysteine proteinase inhibitors, is identical to post-γ-globulin and has two disulfide bonds located in the C-terminal region of the molecule. This protein is encoded by the CS73 gene, localized on the short arm of chromosome 20 [4]. As early as 1979, it was suggested that serum cystatin C could serve as a marker reflecting SCF [5]. In patients on hemodialysis, its level was 13 times higher than that in healthy individuals [40]. Early studies (1984-1985) showed that serum cystatin C was indeed a marker of SCF and was not inferior to creatinine in early studies [9]. In addition, it has been shown that the serum cystatin C level is a more reliable marker of SCF than other low-molecular proteins (β2-microglobulin, retinol-binding protein, and complement factor D) [4]. Currently, these facts are explained by the fact that cystatin C is characterized by a constant rate of production by almost all nucleated cells of the body, free glomerular filtration, is completely metabolized in the kidneys and is not subject to

tubular secretion, which makes it an almost ideal marker for determining SCF [2]. It is generally accepted that the serum cystatin C level does not depend on race, gender, muscle mass, and its concentration in plasma is relatively stable [3]. According to some data, with increasing age, the cystatin C level slightly increases, which is associated with a natural decline in kidney function with aging [2]. However, there is currently evidence that the formation of cystatin C is not constant. The main factors influencing the serum cystatin C level are considered to be height and body weight, smoking, alcohol consumption, C-reactive protein (CRP) level, as well as systemic inflammatory diseases such as rheumatoid arthritis and steroid therapy [4]. Some studies have noted a relationship between increased serum cystatin C levels and obesity [8], which raised the question of the mechanisms of cystatin C hyperproduction by adipocytes and the limitation of the use of cystatin C for assessing GFR in patients with an increased body mass index (BMI) [7]. Another extrarenal factor influencing the cystatin C level is thyroid function: in hyperthyroidism, its level increases, while in hypothyroidism, it decreases [9]. The advantage of cystatin C over serum creatinine has been proven in patients with diabetic nephropathy, when the GFR remains normal or increased. It has been established that there is a pronounced disproportion between the SCF calculated using the MDRD formula based on the concentration of creatinine in the blood serum and cystatin C: cystatin C turned out to be a more accurate marker of the progression of renal dysfunction than creatinine, with a SCF > 60 ml / min / 1.73 m2, which can be used for early prediction of renal dysfunction in diabetes mellitus [6]. It has also been shown that cystatin C is superior to creatinine in terms of prognostic significance in relation to assessing the SCF and predicting the risk of death and developing CVE [7]. Numerous studies demonstrate a strong relationship between the level of cystatin C and the risk of developing CVE. Elevated cystatin C levels are associated with both progression of renal impairment and increased risk of all-cause mortality and development of CV events such as MI, ischemic stroke, HF, as well as peripheral arterial disease, severity of atherosclerotic process, and metabolic syndrome [8]. Some authors suggest that elevated cystatin C levels may be associated with high CV mortality regardless of renal impairment [9]. Elevated cystatin C levels in patients with MI are associated with increased mortality. In addition, it was found that in patients with multiple atherosclerotic lesions of the coronary arteries according to coronary angiography, the concentration of cystatin C is higher than in patients with lesions of only one coronary artery [10]. According to data published in the Annals of Internal Medicine, cystatin C predicts the risk of developing HF more accurately than serum creatinine, a more commonly used parameter of renal function. Elevated cystatin C levels were associated with an increased risk of developing HF even after adjustment for confounders, while creatinine concentration was not significantly associated with the risk of developing HF [12]. In a recent study evaluating the prognostic role of cystatin C in relation to the development of hospital complications in patients undergoing CABG, no statistically significant differences were found in serum creatinine concentrations and SCF either before or after surgery between patients in the low, intermediate, and high risk groups according to the EuroSCORE scale. At the same time, with an increase in risk according to the EuroSCORE scale, the concentration of cystatin C in the blood serum increases. There were no significant differences in serum creatinine concentrations and SCF in patients with favorable and unfavorable outcomes either before or after CABG. At the same time, the concentration of cystatin C in patients with an unfavorable outcome was significantly higher than in patients with a favorable outcome one day before CABG and on the 7th day after it [14]. In a study by Russian authors, a prognostic model for the risk of complicated postpericardiotomy syndrome was developed in patients who underwent heart valve replacement. A high risk of developing postpericardiotomy syndrome is established based on a serum cystatin C level of more than 5.45 mg/l [15]. In a study by A.P. Rebrov et al. [11], a relationship was determined between the level of cystatin C and target organ damage in hypertension, including hypertensive nephropathy. The study group included 94 patients (men and women) diagnosed with hypertension aged 30-65 years. It was found that the median serum cystatin C level in individuals with hypertension was 1029.4 ng/ml (772; 1216), minimum — 350 ng/ml, maximum — 2371.95 ng/ml. Relationships were found between the cystatin C level and age over 50 years, obesity, stage and risk of hypertension, heart failure. In men under 50 years, a more rapid and statistically significant increase in the cystatin C level was determined than in women. In addition, in women, an increase in serum cystatin C was detected with hypertension duration of more than 10 years. Relationships have been established between the level of cystatin C and SCF, left ventricular myocardial mass index, the presence of atherosclerotic plaques in the carotid arteries and an increase in the thickness of the intima-media complex, and the size of the heart cavities [8]. According to S. Zhao et al. [9], cystatin C is a marker that reflects not only AKI, but also oxidative status. In addition, it was found that the combination of elevated concentrations of cystatin C, total bilirubin and CRP in patients undergoing percutaneous coronary intervention are independent risk factors for in-stent restenosis. Recent studies have shown that DP is associated with microbleeds in the brain. It has been proven that elevated serum cystatin C concentrations are associated with the presence of microbleeds in the

brain in patients with acute stroke, regardless of other risk factors, such as gender, age and risk factors for the development of CVE [10].

Conclusion

In addition, older age, BMI, and CRP levels are independently associated with cystatin C levels. All of the above determines serious limitations of the most common methods for determining renal failure using the traditional indicator of kidney damage — creatinine. Currently, many biomarkers are known that reflect kidney damage at the earliest stages, many of which have sensitivity and specificity, as well as reproducibility, that are quite acceptable for clinical use. In addition, the special value of a number of markers is determined by their versatility — the possibility of using them not only for diagnosing renal failure, but also for predicting cardiovascular events. Perhaps the most promising in this regard is cystatin C, since its elevated concentrations can be associated with the risk of developing not only nephropathy, but also fatal cardiovascular events. The use of "new" biomarkers, in particular cystatin C, to detect kidney damage can help improve early prediction of the risk of developing renal failure. In our opinion, cystatin C meets many characteristics of an "ideal" biomarker, which can be used not only to detect early forms of kidney damage, but also to assess the risk of developing renal failure, the need for renal replacement therapy, and the risk of death in patients in intensive care units of cardiology clinics. Kidney involvement in many diseases, including those not initially considered renal, makes it necessary to develop unified approaches to the management of patients with identified chronic renal failure, especially in terms of early prevention and treatment of its complications: anemia, phosphorus-calcium metabolism disorders, which significantly worsen the prognosis of other diseases.

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