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THE STATE OF CARBOHYDRATE METABOLISM AND RENAL FUNCTION IN PATIENTS WITH ACUTE CORONARY SYNDROME. LITERATURE REVIEW

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Abstract

Introduction. Cardiovascular diseases (CVD) are the most common cause of morbidity and mortality worldwide [41,84]. In most cases, kidney pathology also develops in parallel with the conditions described above, manifesting as albuminuria and a reduction in glomerular filtration rate (GFR), which can progress to end-stage renal failure and a fatal outcome [37].

Objective. To summarize the literature on the state of carbohydrate metabolism and renal function in patients with acute coronary syndrome (ACS).

Search strategy. An analysis of open-access literature sources from the scientific databases PubMed, Medline, Google Scholar, Cyberleninka for the past 7 years (2018-2025) was conducted. The following keywords were used for the search: carbohydrate metabolism, diabetes mellitus, prediabetes, acute coronary syndrome, renal function, cardiorenal syndrome, cardiorenometabolic syndrome. This literature review included full-text articles in Russian and English. A total of 206 papers were analyzed, of which 100 met the study objectives and inclusion criteria.

Results. This literature review presents data on the state of carbohydrate metabolism and renal function in patients with ACS.

Conclusion. Based on the literature review, it was found that carbohydrate metabolism disorders (CMD) are quite common in patients with ACS - data vary from 11 to 67%. ACS is often accompanied by renal dysfunction (RDI). The incidence of renal dysfunction among patients with ACS, according to literature data, is 10 - 61%. Simultaneous damage to the cardiovascular system and kidneys in combination with CMD is called cardio-renometabolic syndrome, the presence of which significantly aggravates the prognosis.

Key words: *carbohydrate metabolism, diabetes mellitus, prediabetes, acute coronary syndrome, renal function, cardiorenal syndrome, cardiorenometabolic syndrome*

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Резюме

СОСТОЯНИЕ МЕТАБОЛИЗМА УГЛЕВОДОВ И ФУНКЦИИ ПОЧЕК У ПАЦИЕНТОВ С ОСТРЫМ КОРОНАРНЫМ СИНДРОМОМ. ОБЗОР ЛИТЕРАТУРЫ

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Введение. Сердечно-сосудистые заболевания (ССЗ) являются наиболее распространенной причиной заболеваемости и смертности во всем мире [41,84]. В большинстве случаев параллельно с вышеописанными состояниями появляется и почечная патология в виде альбуминурии и снижения скорости клубочковой фильтрации (СКФ) вплоть до развития терминальной почечной недостаточности и летального исхода [37].

Цель. Обобщение литературных сведений о состоянии углеводного обмена и функции почек у пациентов с острым коронарным синдромом (ОКС).

Стратегия поиска. Проведен анализ литературных источников в открытом доступе из научных баз данных PubMed, Medline, Google Scholar, Cyberleninka за последние 7 лет (2018-2025 гг.). Для поиска использовались ключевые слова: углеводный обмен, сахарный диабет, предиабет, острый коронарный синдром, функция почек, кардиоренальный синдром, кардиоренометаболический синдром. В данный литературный обзор были включены полнотекстовые статьи на русском и английском языках. Всего проанализировано 206 работ, из них цели исследования и критериям включения соответствовала – 100.

Результаты. В данном литературном обзоре представлены данные о состоянии углеводного обмена и почечной функции у пациентов с ОКС.

Заключение. На основании проведенного обзора литературы установлено, что нарушения углеводного обмена (НУО) достаточно часто встречаются у пациентов с ОКС – данные варьируют от 11 до 67%. ОКС нередко сопровождается и нарушением функции почек (НФП). Частота встречаемости почечной дисфункции среди пациентов с ОКС согласно литературным данным составляет 10 - 61%. Одновременное поражение сердечно-сосудистой системы и почек в сочетании с НУО именуется термином кардио-рено-метаболический синдром, наличие которого значительно отягощает прогноз пациентов.

Ключевые слова: *углеводный обмен, сахарный диабет, предиабет, острый коронарный синдром, функция почек, кардиоренальный синдром, кардиоренометаболический синдром*

Для цитирования:

Казарян С.У., Базарбекова Р.Б., Досанова А.К., Касымалиева Р.А., Нурлыкаимова Ж.А., Абсатарова К.С., Якупова М.М. Состояние метаболизма углеводов и функции почек у пациентов с острым коронарным синдромом. Обзор литературы // Наука и Здравоохранение. 2025. Vol.27 (6), С.172-184. *doi 10.34689/SH.2025.27.6.019*

Түйіндеме

КӨМІРСУ АЛМАСУЫНЫҢ ЖАҒДАЙЫ ЖӘНЕ БҮЙРЕК ҚЫЗМЕТІ ЖЕДЕЛ КОРОНАРЛЫҚ СИНДРОМЫ БАР НАУҚАСТАРДА. ӘДЕБИЕТТІК ШОЛУ.

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Кіріспе. Жүрек-қан тамырлары аурулары (ЖҚА) әлем бойынша сырқаттанушылық пен өлім-жітімнің ең кең таралған себебі болып табылады [41,84]. Көп жағдайда жоғарыда сипатталған жағдайлармен қатар альбуминурия және шумақтық сүзілу жылдамдығының (ШСЖ) төмендеуі түріндегі бүйрек патологиясы да пайда болады, бұл терминалдық бүйрек жеткіліксіздігінің дамуына және өлімге дейін әкелуі мүмкін [37].

Мақсаты. Жедел коронарлық синдромы (ЖКС) бар науқастарда көмірсу алмасуының жағдайы мен бүйрек қызметі туралы әдеби деректерді талдап, жинақтау.

Іздеу стратегиясы. Соңғы 7 жыл ішінде (2018–2025 жж.) PubMed, Medline, Google Scholar, CyberLeninka ашық ғылыми дерекқорларындағы әдеби дереккөздерге талдау жүргізілді. Іздеу барысында келесі негізгі сөздер пайдаланылды: көмірсу алмасуы, қант диабеті, предиабет, жедел коронарлық синдром, бүйрек қызметі, кардиоренальды синдром, кардиоренометаболикалық синдром. Шолу аясында қазақ және ағылшын тілдеріндегі толық мәтінді мақалалар қарастырылды. Жалпы 206 жұмыс талданып, оның 100-і іріктеу критерийлеріне сәйкес келді.

Нәтижелер. Әдеби шолуда ЖКС бар науқастарда көмірсу алмасуы мен бүйрек қызметінің жағдайына қатысты мәліметтер ұсынылған.

Қорытынды. Әдеби деректерді талдау ЖКС бар науқастар арасында көмірсу алмасуының бұзылыстары (КАБ) жиі кездесетінін көрсетті, олардың таралу жиілігі 11%-дан 67%-ға дейін. Сонымен қатар, ЖКС жиі бүйрек қызметінің бұзылуымен (БҚБ) қатар жүреді, оның таралу жиілігі 10%-дан 61%-ға дейін жетеді. Жүрек-қантамыр жүйесі мен

бүйректің бір мезгілде зақымдалуы, көмірсу алмасуының бұзылыстарымен бірге кардиореометаболикалық синдром деп аталады. Бұл синдромның болуы науқастың болжамын айтарлықтай нашарлатады.

Түйінді сөздер. *Көмірсу алмасуы, қант диабеті, предиабет, жедел коронарлық синдром, бүйрек қызметі, кардиоренальды синдром, кардиореометаболикалық синдром.*

Дәйексөз үшін:

Казарян С.У., Базарбекова Р.Б., Досанова А.К., Касымалиева Р.А., Нурлыкаимова Ж.А., Абсатарова К.С., Якупова М.М. Көмірсу алмасуының жағдайы және бүйрек қызметі жедел коронарлық синдромы бар науқастарда. Әдебиеттік шолу // Ғылым және Денсаулық сақтау. 2025. Vol.27 (6), Б. 172-184. *doi 10.34689/SH.2025.27.6.019*

Introduction

Cardiovascular diseases (CVD) are the most common causes of morbidity and mortality worldwide [22]. Similarly, diabetes mellitus (DM) is also a major contributor to these adverse outcomes [73]. At the same time, global data on the prevalence of CVD among individuals with type 2 diabetes (T2D) remain insufficient. From this standpoint, the findings of the recent CAPTURE study are highly important: among 9,823 participants with T2D, more than one-third (n = 3,582; 36.5%) had confirmed CVD. Confirmed CVD was defined as the presence of any of the following conditions: coronary artery disease (CAD), cerebrovascular disease, heart failure, arrhythmias or cardiac conduction disorders, aortic disease, or peripheral or carotid artery disease [55]. The prevalence of CAD in patients with T2D reaches 21%. Noteworthy are also data demonstrating a high prevalence of prediabetes and T2D among individuals with CVD, particularly those with CAD [8]. The incidence of DM in the setting of acute coronary syndrome (ACS) ranges between 25–30% [13, 6]. It is also well known that many patients with CVD present with impaired renal function (IRF) [39].

Aim. To summarize the literature data on carbohydrate metabolism and renal function in patients with acute coronary syndrome.

Search Strategy

The literature search for the present review was conducted using major scientific databases, including PubMed, Medline, Google Scholar, and CyberLeninka. Publications from the last seven years (2018–2025) were considered. Selection was carried out among systematic reviews, meta-analyses, and original research articles published in both Russian and English. The search strategy was based on the use of the following key terms: «carbohydrate metabolism», «diabetes mellitus», «prediabetes», «acute coronary syndrome», «renal function», «cardiorenal syndrome» and «cardiorenometabolic syndrome».

The inclusion criteria were: articles published between 2018–2025, available in full-text format, written in either Russian or English, and classified as systematic reviews, meta-analyses, or original research studies. The exclusion criteria were: publications in other languages, materials presented in the form of theses or abstracts, as well as studies not relevant to the stated topic of the present review.

A total of 206 publications were identified through the search. The majority of the studies were retrieved from PubMed (96 publications) and CyberLeninka (54 publications). Google Scholar and Medline contained 30 and 26 publications respectively. At the stage of initial screening, 12 duplicate articles were excluded. Additionally, 58 studies published in foreign sources requiring paid access, as well as 36 publications unrelated to the research topic, were excluded. As a result of the selection, 100 publications that most comprehensively reflected current data on the studied issue were included in the analytical part of the review. The selection algorithm is presented in Fig. 1.

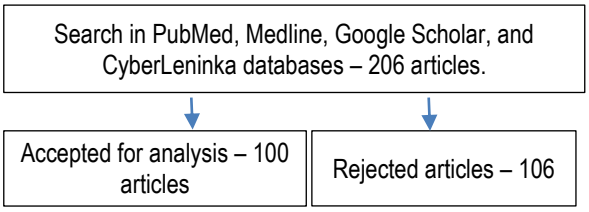

Figure 1. Flow diagram of materials extraction.

Definition and Classification of ACS

Acute coronary syndrome (ACS) is a modern synonym for the concept of acute coronary artery disease (CAD). CAD is a supra-nosological concept that may present in acute or chronic forms [9].

ACS includes the following conditions: myocardial infarction (MI), sudden coronary death, acute coronary thrombosis without MI development in the context of thrombolytic therapy (TLT) and/or percutaneous coronary intervention (PCI), and unstable angina (UA) [9].

According to the definition by Thygesen et al., ACS encompasses conditions characterized by acutely occurring clinical symptoms, 12-lead electrocardiogram (ECG) changes (with or without such changes), and marked elevation of cardiac troponin (cTn) concentrations (with or without such elevation) [87,40].

Patients with suspected ACS may be diagnosed with acute MI or unstable angina (UA). The diagnosis of MI is made according to the Fourth Universal Definition, which is based on the detection of elevated serum cTn levels in patients [87,3].

UA is an ACS manifesting with a characteristic clinical presentation (progression of exertional angina functional class, onset of rest angina, and a high likelihood of progression to MI) in the absence of biochemical markers of cardiomyocyte injury. The clinical and pathogenetic variants of UA are reflected in the Braunwald classification (Table 1) [27].

In patients presenting with sudden-onset acute chest pain or its equivalent and persistent ST-segment elevation or its equivalents, ST-segment elevation myocardial infarction (STEMI) is diagnosed. In most of these patients, myocardial necrosis and elevated cTn levels are observed.

In cases of anginal chest pain without persistent ST-segment elevation (or its equivalents) on ECG, a preliminary diagnosis of non–ST-segment elevation ACS (NSTE-ACS) is established. ECG changes may include the following: transient ST-segment elevation, persistent or transient ST-segment depression, T-wave abnormalities including hyperacute T-waves, T-wave inversion, biphasic or flattened T-waves, and T-wave pseudonormalization. In

some cases, the ECG may remain normal. In most such patients with characteristic rises and falls in cTn levels, non–ST-segment elevation myocardial infarction (NSTEMI) is diagnosed. In patients with normal cTn levels, unstable angina (UA) is diagnosed [21].

Table 1.

Classification of unstable angina by E. Braunwald.

Variants ⇩ ⇨ Causes Angina pectoris	A – presence of an external factor that increases ischemia Secondary UA (unstable angina)	B – no external factor causing angina Primary UA (unstable angina)	C – возникает в течение 2-х недель после инфаркта миокарда Постинфарктная НС
I – severe exertional angina (new-onset or progressive)	IA	IB	IC
II – angina within the past month, but not during the preceding 48 hours; subacute rest angina	IIA	IIB	IIC
III – rest angina during the preceding 48 hours; acute rest angina	IIIA	IIIB	IIIC

Importantly, at the Cardiology Congress held in London in 2024, it was emphasized that ACS may arise as a result of progression and destabilization of a condition referred to as chronic coronary syndrome (CCS). This term was first introduced in 2019 at the European Cardiology Congress in Paris, where it was adopted to replace the previous term «stable coronary (ischemic) disease». CCS represents a condition evolving from the formation of an atherosclerotic plaque to the development of functional coronary artery impairment. The mechanisms underlying CCS are associated with myocardial ischemia caused by diffuse atherosclerotic involvement rather than solely by flow-limiting stenoses [88,12].

Classification of Myocardial Infarction

The current MI classification distinguishes five types [25]:

Type 1. MI resulting from rupture or erosion of an atherosclerotic plaque (AP), leading to thrombus formation, significant reduction of blood flow distal to the affected AP, and development of myocardial necrosis. Less commonly, type 1 MI may be caused by the formation of an intraplaque hematoma with progressive enlargement and narrowing of the arterial lumen.

Type 2. Occurs due to ischemia either in the presence or absence of coronary atherosclerosis, and may result from embolism or spontaneous coronary artery dissection, hypoxia, arrhythmias, blood pressure fluctuations, and other causes.

Type 3. Type 3 MI corresponds to cases of acute MI confirmed at autopsy, as patients die before blood samples can be obtained to detect biochemical markers of myocardial necrosis. Supporting evidence for acute MI may include a history of ischemic symptoms and characteristic electrocardiographic changes.

Type 4a. MI associated with complications occurring during percutaneous coronary intervention (PCI) or within the first 48 hours following the procedure.

Type 4b. MI caused by coronary stent thrombosis confirmed by either coronary angiography (CAG) or autopsy. Depending on the time elapsed after stent implantation, stent thrombosis is classified as acute (0–24 h), subacute (>24 h–30 days), late (>30 days–1 year), and very late (>1 year).

Type 4c. MI due to restenosis in the artery corresponding to the necrosis zone, occurring in the absence of thrombosis and other lesions in the infarct-related artery.

Type 5. MI associated with coronary artery bypass grafting (CABG).

This MI classification takes into account several factors, including the underlying causes and mechanisms, updated ECG criteria, coronary imaging findings, and levels of high-sensitivity biomarkers.

In ESC member countries, cardiovascular diseases (CVD) account for approximately 2.2 million deaths among women and more than 1.9 million deaths among men. Among mortality causes attributed to CVD, coronary artery disease (CAD) accounts for 38% of deaths in women and 44% in men [86]. ACS is often the first clinical manifestation of CAD. According to expert data, in 2019, 5.8 million new cases of CAD were recorded across 57 ESC member countries [86].

Prevalence of Disorders of Carbohydrate Metabolism among Patients with ACS

A significant proportion of patients with cardiovascular diseases (CVD) have diabetes mellitus (DM). The risk of peripheral artery disease and the development of coronary artery disease (CAD) in DM is associated with a range of factors, primarily hyperglycemia, insulin resistance, and elevated levels of advanced glycation end-products (AGEs) [38,28,34,41,71,45,79,80].

It is well known that disorders of carbohydrate metabolism (DCM) are frequently detected in patients with ACS. According to registry data, DM in ACS patients is observed at a frequency of 22–65% [30,31,98,97].

Among patients with ST-segment elevation myocardial infarction (STEMI), one quarter had a previously documented diagnosis of DM, and more than 40% had prediabetes or newly diagnosed type 2 DM (T2DM) [56,82,37].

According to the results of a well-known epidemiological study, The Euro Heart Survey on Diabetes and the Heart (EHS), which included 2,107 ACS patients with no prior history of DCM, such disorders were identified in 58% of cases, with newly diagnosed T2DM in 22%. Even more

patients (36%) were found to have impaired glucose tolerance (IGT) [70].

In a study by *Rizaeva M.A.* [17], among 344 patients with MI aged 35 to 90 years, DCM were detected in 55% of cases.

Sheen M. et al. [76] found T2DM in 19.03% of 529 STEMI patients, of whom newly diagnosed T2DM accounted for 11.11%.

According to data from *Nikitina E.A.,* the prevalence of T2DM among patients with non–ST-segment elevation ACS (NSTE-ACS) was 12.17%, while in patients with ST-segment elevation ACS it was 13.89%.

The detection rate of DM among MI patients ranges from 14.3% to 40.9% [13].

According to *Shestakova M.V. et al.* [19], T2DM was identified in 8–14% of patients with CVD, and prediabetes in 14.6–36.4% of cases, which exceeds the corresponding population indicators reported in the NATION study (5.4% and 19.3%, respectively) [96].

In a study by *E.H. Ploumen et al.,* among 988 patients who underwent PCI, newly diagnosed T2DM was detected in 68 patients (6.8%), while 132 patients (13.4%) had prediabetes [66].

According to a meta-analysis conducted by *J. Yang et al.* in 2020, the prevalence of prediabetes among patients with ACS ranged from 26% to 38% [93].

In another study published by *L. Nguyen et al.* in 2020, approximately 34% of ACS patients had prediabetes [62].

According to a separate meta-analysis by N*. Laichuthai et al.*, the prevalence of prediabetes among patients with acute MI was 48.4% [50].

It is widely recognized that prediabetes is a major predictor of T2DM development. It has been demonstrated that T2DM develops in nearly all individuals with previously diagnosed prediabetes over the course of life, with one quarter of these patients progressing within the first 3–5 years. In addition to the high likelihood of T2DM development, individuals with prediabetes have an increased risk of both macrovascular and microvascular diabetes-related complications [44,59].

According to the literature, the course of ACS is significantly aggravated by the occurrence of another type of DCM known as «stress hyperglycemia». Stress hyperglycemia is a transient elevation of plasma glucose levels in critically ill patients, with or without pre-existing DM [29,26,35]. Critical states in this context include major surgical interventions, acute illnesses, life-threatening conditions requiring intensive care, and major trauma. According to the Consensus Statement of the American Association of Clinical Endocrinologists and the American Diabetes Association (ADA), stress hyperglycemia is defined as any elevation of blood glucose above 7.8 mmol/L in the absence of a history of diabetes [52].

The incidence of stress hyperglycemia in critically ill patients reaches 90%. In MI, its frequency, according to various authors, ranges from 20% to 80% [64,4]. According to another study, stress hyperglycemia during the acute phase of MI is observed in 44.4% of cases [14].

The pathogenesis of stress hyperglycemia is associated with stress-induced activation of the sympathetic nervous system, the release of insulin antagonists - particularly cortisol - and proinflammatory cytokines [96]. The relationship between stress hyperglycemia and elevated cortisol levels has been examined in detail in a study by *R.K. Pandey* [63].

In a well-known study [43], predictors of stress hyperglycemia in patients after cardiac surgery were analyzed. Among 3,658 medical records reviewed, 1,453 patients developed stress hyperglycemia postoperatively. The analysis demonstrated that the factors significantly associated with its development included female sex, older age, body mass index, elevated serum creatinine, left ventricular ejection fraction, history of cardiac surgery, and cardiogenic shock.

Confirmed biochemical predictors of stress hyperglycemia in patients with acute MI (AMI) include elevated glycated hemoglobin, hyperinsulinemia, and proinflammatory cytokines [72,20].

It is important to note that the adverse prognostic impact of disorders of carbohydrate metabolism in ACS patients has been well known for a long time. For example, according to a meta-analysis conducted in Sweden in 2007 that included 22 randomized clinical trials and 6,315 STEMI patients, mortality was higher in individuals with diabetes compared with those without [23].

Similar results were obtained by *Xu F. et al.* [92], who found that in-hospital MI mortality was significantly higher among patients with diabetes than among those without (16% vs. 6%, $P < 0.001$).

According to a meta-analysis by *M. Zeng et al.* [95], prediabetes also exerts a negative prognostic influence in MI patients. As in T2DM, mechanisms contributing to these adverse outcomes include insulin resistance, endothelial dysfunction, dyslipidemia, and oxidative stress. By promoting the formation of unstable atherosclerotic plaques, these mechanisms create a high risk of plaque rupture and erosion, and consequently acute coronary events, including MI. Glucotoxicity, leading to myocardial remodeling, is also considered relevant [95,51].

Renal Function in Patients with ACS

ACS is frequently accompanied by impaired renal function (IRF). The mechanism of kidney injury in MI is primarily associated with inadequate renal perfusion due to reduced cardiac output [99,100,18,5].

Another cause of IRF is exposure to radiographic contrast agents during coronary angiography (CAG), leading to contrast-induced nephropathy (CIN) [40,85,47]. CIN develops in approximately 3–19% of patients undergoing the procedure [34], and in about 1% of such cases, hemodialysis becomes necessary [74]. In patients with pre-existing chronic kidney disease (CKD) and additional risk factors, the likelihood of CIN development increases up to 50% [54].

Acute kidney injury (AKI) is a serious complication of MI [1]. This condition is observed in 10–60% of patients with ACS [16,22]. Renal impairment in ACS ranges from 4.4% to 13.2%, depending on the method used to calculate estimated glomerular filtration rate (eGFR) [89].

Roumeliotis S. et al. assessed the prevalence of unrecognized renal insufficiency in 12,830 ACS patients and found it to be 19.8% (defined as eGFR below 60 mL/min/1.73 m²) [70].

In a study by *Qi L. et al.,* the prevalence of IRF among ACS patients was 61% (1,146 out of 1,860). Specifically, mild IRF (eGFR 60–90 mL/min/1.73 m²) was present in 40.1% (746) of patients, while severe impairment was noted in 20% (400) [67].

In a joint Chinese-American research project, mild IRF was detected in 36.17% of cases, and significant impairment in 17.87% [91].

In another study involving a retrospective analysis of 314 ACS cases, mild renal impairment was more common in men (51.5%) than in women (42.7%). Moderate renal dysfunction was observed in 12.2% of men and 30.9% of women [18].

It has been demonstrated that IRF occurs more frequently in non–ST-segment elevation ACS (NSTE-ACS) cases (42.9%) compared to ST-segment elevation ACS (STE-ACS) (30.5%) [6,32]. Findings from various clinical studies indicate that 35-40% of ACS patients exhibit renal insufficiency of varying severity [92]. Other studies have also confirmed that ACS is often associated with reduced renal function [91,90,75].

In a meta-analysis conducted by *J.W. Pickering et al.* including 36 unique studies and 100,476 ACS patients, the prevalence of IRF reached 15.8%. The authors also reported that ACS complicated by AKI was associated with more than a threefold increase in early mortality and more than a twofold increase in long-term mortality [65].

In a study by *M. Zykov et al.,* aimed at establishing the clinical and prognostic significance of IRF in ACS patients, it was found that renal impairment assessed within the first 24 hours of ACS-regardless of initial treatment strategy-served as a predictor of cardiovascular complications during a three-year follow-up period [6].

According to another meta-analysis including 25 studies with a total of 254,408 adults with ACS, IRF was associated with an 86% increase in cardiovascular mortality risk, a 38% increase in major adverse cardiovascular events (MACE), and a 58% increase in heart failure risk [87].

Cardiorenal-Metabolic Continuum

The simultaneous involvement of the heart and kidneys against the background of metabolic disturbances is referred to as the cardiorenal-metabolic (cardiovascular) continuum (CRMC)**.** According to the definition by *Reznik E.V.,* «the cardiovascular continuum is a chain of interconnected changes in the cardiovascular system-from exposure to risk factors (arterial hypertension, diabetes mellitus, dyslipidemia, obesity, smoking, etc.) through the gradual development and progression of endothelial dysfunction, atherosclerosis, left ventricular (LV) hypertrophy, coronary artery disease (CAD), myocardial infarction (MI), to the development of heart failure (HF) and death» [15]. Regardless of the etiological factor, endothelial dysfunction represents the initial («triggering») link in the formation of this syndrome.

In the setting of dysglycemia, the major causal factor of endothelial dysfunction is hyperglycemia, which induces activation of the polyol pathway, heparanase, heparan sulfate degradation, and non-enzymatic protein glycation [15,58].

Subsequently, increased activity of the local renin-angiotensin system (RAS) plays a significant role, leading to the development of systemic and intraglomerular hypertension. The latter is observed in 40% of patients with type 2 diabetes (T2DM). There is evidence suggesting an association between the risk of renal damage in T2DM patients and RAS-related genes, as well as genes encoding endothelial factors (eNOS3) [7].

One of the variants within the CRMC framework is the **cardiorenal syndrome (CRS).**

The concept of «cardiorenal syndrome» (CRS) was formulated in 2008. This term encompasses the bidirectional interaction of pathological processes between the cardiovascular system and the kidneys: acute or chronic dysfunction of one organ leads to acute or chronic dysfunction of the other. In CRS, the primary disease may involve the kidneys, resulting in renal failure and subsequently cardiovascular complications and heart failure. Conversely, primary cardiac pathology may lead to HF, which in turn causes renal dysfunction and injury, leading up to end-stage renal disease (ESRD) [81] (Table 2).

Acute Cardiorenal Syndrome (CRS Type 1)

Acute CRS refers to sudden kidney injury occurring in the setting of rapidly developing cardiac pathology, such as myocardial infarction with cardiogenic shock, decompensation of chronic heart failure, coronary angiography (CAG), or cardiac surgery. Chronic kidney disease (CKD) is usually a predisposing factor [69,48]. This condition is associated with an increased risk of mortality [42,9,70]. A significantly adverse prognostic factor is reduced left ventricular ejection fraction (LVEF) [77].

The abrupt decline in renal function reflects the severity of the patient's condition, as it is accompanied by activation of inflammatory cascades and an increased risk of mortality [84].

Table 2.

Classification of CRS [68].

Type	Title	Clinical situations
1	Acute cardiorenal syndrome	AKI in the setting of ACS, acute heart failure, decompensated chronic heart failure, pulmonary embolism, after coronary angiography, and cardiac surgery
2	Chronic cardiorenal syndrome	Chronic kidney disease (CKD) in patients with chronic heart failure secondary to coronary artery disease, hypertension, cardiomyopathies, valvular heart disease, and other etiologies
3	Acute renocardiac syndrome	Hypertension, acute coronary syndrome, acute heart failure, and neurohormonal activation in the setting of acute kidney injury
4	Chronic renocardiac syndrome	Cardiovascular pathology (hypertension, left ventricular hypertrophy, cardiac structural calcifications, valvular heart disease, myocardial infarction) in chronic kidney disease
5	Secondary cardiorenal syndrome	systemic disease involving both cardiac and renal systems

Chronic Cardiorenal Syndrome (CRS Type 2)

The development of chronic cardiorenal syndrome type 2 is usually caused by the presence of pre-existing chronic cardiac pathology (for example, chronic heart failure) with prolonged reduction of renal perfusion. However, hemodynamic disturbances are not the only reason for the decline in glomerular filtration rate. Neurohormonal shifts are also considered important, including disturbances in the balance between the production of vasoconstrictors and vasodilators, and the use of diuretics, which may lead to hypovolemia [49,60]. The onset of chronic kidney disease in such patients serves as a predictor of the development of systolic and diastolic left ventricular dysfunction and cardiovascular death [24].

Acute Renocardiac Syndrome (CRS Type 3)

Acute renocardiac syndrome develops as a consequence of sudden renal dysfunction that leads to acute impairment of cardiac function, including heart failure, arrhythmia, and ischemia. Such a situation may be observed in acute kidney injury, acute glomerulonephritis or pyelonephritis, acute tubular necrosis, or acute obstruction of the urinary tract [94,57].

Chronic Renocardiac Syndrome (CRS Type 4)

In chronic renocardiac syndrome type 4, the primary condition is chronic renal damage, which causes impairment of cardiac function in the form of left ventricular hypertrophy, diastolic dysfunction, and other abnormalities [55].

Secondary Cardiorenal Syndrome (CRS Type 5)

Cardiorenal syndrome type 5 is associated with pre-existing acute or chronic diseases that simultaneously affect both the heart and the kidneys. Such conditions may include sepsis, systemic connective tissue diseases, and sarcoidosis.

Chronic heart failure significantly increases the risk of deterioration of kidney function and adverse renal outcomes, particularly when associated with a rapid decrease in estimated glomerular filtration rate (more than 5 milliliters per minute per 1.73 square meters per year). In turn, reduction of kidney function and the occurrence of microalbuminuria increase the risk of developing heart failure.

Thus, the cardiorenal syndrome is part of the cardiorenal-metabolic axis [78]. Cardiorenal syndrome may be diagnosed in 32-90.3% of patients with heart failure. Impairment of kidney function has an unfavorable prognostic significance, as it leads to increased mortality in patients with heart failure.

Conclusion

The literature data on the prevalence of disorders of carbohydrate metabolism and renal impairment in acute coronary syndrome are characterized by considerable variability. The prevalence of disorders of carbohydrate metabolism in acute coronary syndrome ranges from 11% to 67%. With regard to renal pathology, its frequency among patients with acute coronary syndrome, according to different authors, ranges from 10% to 60%.

Meanwhile, timely detection of impaired renal function, as well as disorders of carbohydrate metabolism, in the context of acute coronary syndrome is extremely important from the standpoint of the possible development of cardiorenal syndrome in this category of patients. Considering the mechanisms of the formation of cardiorenal syndrome, cardiorenal syndrome in acute coronary syndrome most likely corresponds to type 1 and, consequently, requires immediate intervention aimed at eliminating hemodynamic, metabolic, and other disturbances [16].

In particular, in cases where type 2 diabetes mellitus is diagnosed in the setting of acute coronary syndrome, it is reasonable to prescribe medications that modify the course of the disease, specifically sodium-glucose cotransporter 2 inhibitors and glucagon-like peptide-1 receptor agonists. These classes of medications were originally developed as "hypoglycemic agents." However, in numerous studies assessing cardiorenal safety in patients with type 2 diabetes mellitus, they have convincingly demonstrated beneficial effects on both cardiovascular and renal outcomes. This is very important, since patients with type 2 diabetes mellitus exhibit a high association with adverse development of both microvascular and macrovascular complications [10,11]. By suppressing the reabsorption of glucose and sodium in the proximal renal tubules, sodium-glucose cotransporter 2 inhibitors contribute to a reduction in preload and afterload, improvement of energy metabolism, and cardiac remodeling. Clinically, this manifests as a decrease in arterial blood pressure, improvement of cardiac contractility, and reduction of heart failure symptoms [11,46].

At the same time, due to a decrease in intraglomerular pressure, the workload on the kidneys is reduced, which is expressed as stabilization of estimated glomerular filtration rate, reduction of ischemic kidney injury, and ultimately-improvement of renal function [33,36].

Despite the absence of a pronounced effect of glucagon-like peptide-1 receptor agonists on renal hemodynamic parameters, this class of medications demonstrates nephroprotective capacity by reducing albumin excretion in patients with type 2 diabetes mellitus. Presumably, their nephroprotective effect is associated with decreased formation of proinflammatory cytokines and reduction of oxidative stress, which contributes to prevention of damage to structures of the glomerular filtration barrier [43,92].

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Литература:

1.*Арсеничева О.В.* Предикторы острого повреждения почек у больных инфарктом миокарда с подъёмом сегмента ST, осложнённым кардиогенным шоком, перенёсших чрескожное коронарное вмешательство. Медицинский вестник Юга России. 2022;13(3):118-126. DOI 10.21886/2219-8075-2022-13-3-118-126

2.*Барбараш О.Л., Воевода М.И., Галстян Г.Р., Шестакова М.В.* Предиабет как междисциплинарная

проблема: определение, риски, подходы к диагностике и профилактике сахарного диабета 2 типа и сердечно-сосудистых осложнений. *Российский кардиологический журнал*. 2019;(4):83-91. https://doi.org/10.15829/1560-4071-2019-4-83-91

3.*Бойцов С.А., Барбараш О.Л., Вайсман Д.Ш., Галявич А.С. и др.* Клиническая, морфологическая и статистическая классификация ишемической болезни сердца: консенсус Российского кардиологического общества, Российского общества патологоанатомов и специалистов по медицинской статистике. 2020. 24 с.

4.*Дедов И.И., Мельниченко Г.А.* Эндокринология. Национальное руководство / М. : ГЭОТАР-МЕДИЦИНА, 2024. – 433 с.

5.Демчук О.В., Сукманова И.А. Прогнозирование частоты развития хронической болезни почек у пациентов с инфарктом миокарда и острым повреждением почек. Российский кардиологический журнал. 2023;28(6):5247. doi:10.15829/1560-4071-2023-5247.

6.*Зыков М.В., Дьяченко Н.В., Велиева Р.М., Кашталап В.В., Барбараш О.Л.* Возможности совместного использования шкалы GRACE и различных индексов коморбидности для повышения эффективности оценки риска госпитальной летальности у больных с острым коронарным синдромом. Терапевтический архив. 2022;94(7):816–821. DOI: 10.26442/00403660.2022.07.201742

7.*Демчук О.В., Сукманова И.А.* Острое почечное повреждение у пациентов с инфарктом миокарда и чрескожными коронарными вмешательствами: лабораторные маркеры и влияние на частоту повторных сердечно-сосудистых событий. Комплексные проблемы сердечно-сосудистых заболеваний. 2022;11(4):25-34. https://doi.org/10.17802/2306-1278-2022-11-4-25-34

8.*Козиолова Н.А., Полянская Е.А., Миронова С.В.* Сахарный диабет 2 типа и микрососудистые осложнения у больных стабильной ишемической болезнью сердца: распространенность, прогноз и выбор антитромботической терапии. Южно-Российский журнал терапевтической практики. 2022;3(4):7-24. https://doi.org/10.21886/2712-8156-2022-3-4-7-24

9.*Компанец Н.В., Щукин Ю.В., Лимарева Л.В., Комарова М.В.* Прогностическое значение нейтрофильного желатиназа-ассоциированного липокалина и цистатина С у пациентов с хронической сердечной недостаточностью и перенесенным инфарктом миокарда. Российский кардиологический журнал. 2021;26(1):4126. doi:10.15829/1560-4071-2021-4126

10. *Леонова М.В.* Ингибиторы натрий-глюкозного котранспортера 2: механизмы кардиоренальной защиты. Consilium Medicum. 2024;26(4):225–231. DOI: 10.26442/20751753.2024.4.202763

11. *Мартынов С.А., Шамхалова М.Ш.* Роль ингибиторов натрий-глюкозного котранспортера 2 в замедлении прогрессирования почечной недостаточности у пациентов с сахарным диабетом 2 типа // Эффективная фармакотерапия. 2021. Т. 17. № 5. С. 16–23.

12. *Масленникова О.М., Ойноткинова О.Ш., Стародубова А.В., Мацкеплишвили С.Т.* Обзор рекомендаций Европейского общества кардиологов 2024 г. по ведению хронических коронарных синдромов: что нового. Эффективная фармакотерапия. 2024; 20 (31): 9–22.

13. *Никитина Е.А.* Клинико-функциональные и прогностические особенности реабилитации пациентов с острым коронарным синдромом и сахарным диабетом 2 типа : диссертация ... кандидата медицинских наук : 14.01.05 - Киров, 2019. - 102 с. : ил.

14. *Никулина Н.А., Акулова Е.А., Доценко Э.А., Кривелевич Н.Б., Сейфидинова С.Г., Ковалев А.А., Зинкевич Д.Д., Пономаренко Д.А., и др.* Транзиторная гипергликемия при острых формах ишемической болезни сердца. Проблемы здоровья и экологии. 2023;20(2):49–58. DOI: https://doi. org/10.51523/2708-6011.2023-20-2-07

15. *Попыхова Э.Б., Степанова Т.В., Лагутина Д.Д., Кириязи Т.С., Иванов А.Н.* Роль сахарного диабета в возникновении и развитии эндотелиальной дисфункции. Проблемы Эндокринологии. 2020;66(1):47-55.https://doi.org/10.14341/probl12212

16. *Резник Е.В., Никитин И.Г.* Кардиоренальный синдром у больных с сердечной недостаточностью как этап кардиоренального континуума (часть I): определение, классификация, патогенез, диагностика, эпидемиология (обзор литературы). Архивъ внутренней медицины. 2019; 9(1):5-22. DOI: 10.20514/2226-6704-2019-9-1-5-22

17. *Ризаева М.А.* Нарушения углеводного обмена при инфаркте миокарда и возможности гипогликемической терапии. Amaliy va tibbiyot fanlari ilmiy jurnali. vol. 3 No. 5 (2024). 153-155

18. *Сейтмаганбетова Н.А., Векленко Г.В., Хибина М.А., Жаубатырова А.А., Курмашева Г.С.* Состояние функции почек у пациентов с острым коронарным синдромом: ретроспективное исследование // West Kazakhstan Medical Journal. 2020. №1 (62). С. 60-67.

19. *Чазова И.Е., Шестакова М.В., Жернакова Ю.В.* Евразийские рекомендации по профилактике и лечению сердечно-сосудистых заболеваний у больных с диабетом и предиабетом (2021). Евразийский кардиологический журнал. 2021; (2):6-61, https://doi.org/10.38109/2225-1685-2021-2-6-61

20. *Чистова Н.П.* Роль полиморфизмов генов-кандидатов эндотелиальной дисфункции и метаболических нарушений в развитии сердечно-сосудистых заболеваний при воздействии производственных факторов. Мед. труда и пром. экол. 2022; 62(5): 331–336. https://elibrary.ru/jdniwu https://doi.org/10.31089/1026-9428-2022-62-5-331-336

21. *Acar B., Ozeke O., Karakurt M., Ozen Y., Ozbay M.B., Unal S. et al.* Association of Prediabetes with higher coronary atherosclerotic burden among patients with First Diagnosed Acute Coronary Syndrome. Angiology. 2019;70(2):174–80. doi: 10.1177/0003319718772420. [DOI] [PubMed] [Google Scholar]

22. *Albert C., Haase M., Albert A. et al.* Biomarker-Guided Risk Assessment for Acute Kidney Injury: Time for Clinical Implementation? Ann Lab Med. 2021; 41(1): 1–15. DOI: 10.3343/alm.2021.41.1.1.

23. *Alfaddagh A., Khraishah H., Romeo G.R.* Cardiovascular Outcomes Among Patients with Acute

Coronary Syndromes and Diabetes: Results from ACS QUIK Trial in India. Glob Heart. 2024 Apr 24;19(1). 37.doi: 10.5334/gh.1290. eCollection 2024.

24. *Bergman M., Abdul-Ghani M., DeFronzo R.* Review of methods for detecting glycemic disorders. Diabetes Res Clin Pract. 2020 Jul:165. 108233.doi: 10.1016/j.diabres.2020.108233. Epub 2020 Jun 1.

Biomolecules. 2023 Jan 27;13(2). 239. doi: 10.3390/biom13020239.

25. *Birnbaum Y., Fiol M., Nikus K.A* counterpoint paper: Comments on the electrocardiographic part of the 2018 Fourth Universal Definition of Myocardial Infarction. J Electrocardiol. 2020 May-Jun. 60:142-147. doi: 10.1016/j.jelectrocard.2020.04.012. Epub 2020 Apr 18.

26. *Brennan S., Esposito S., Abdelaziz M.* Selective protein kinase C inhibition switches time-dependent glucose cardiotoxicity to cardioprotection. Front Cardiovasc Med. 2022 Sep 7:9:997013.doi: 10.3389/fcvm.2022.997013. eCollection 2022.

27. *Budzianowski J., Faron W., Rzeźniczak J.* Predictors of Revascularization in Patients with Unstable Angina. J Clin Med. 2024 Feb 15;13(4). 1096.doi: 10.3390/jcm13041096.

28. *Cahn A., Wiviott S.D., Mosenzon O., Goodrich E.L., Murphy S.A., Yanuv I., Rozenberg A., Bhatt D.L., Leiter L.A., McGuire D.K. et al.* Association of baseline HbA1c with cardiovascular and renal outcomes: analyses from DECLARE-TIMI 58. Diabetes Care 2022. [DOI] [PubMed]

29. *Caturano A., Galiero R., Pafundi P.* Does a strict glycemic control during acute coronary syndrome play a cardioprotective effect? Pathophysiology and clinical evidence. Diabetes Res Clin Pract. 2021 Aug:178:108959.doi: 10.1016/j.diabres.2021.108959. Epub 2021 Jul 17.

30. *Chen S., Huang Z., Chen L., Zhao X., Kang Y. et al.* Does diabetes mellitus increase the short- and long-term mortality in patients with critical acute myocardial infarction? Results from American MIMIC-III and Chinese CIN cohorts. Front Endocrinol (Lausanne) 2021;12:797049. doi: 10.3389/fendo.2021.797049. [DOI] [PMC free article] [PubMed] [Google Scholar]

31. *Chen D., Zhang Y., Yidilisi A.* Combined risk estimates of diabetes and coronary angiography-derived index of microcirculatory resistance in patients with non-ST elevation myocardial infarction. Cardiovasc Diabetol. 2024 Aug 16;23(1):300. doi: 10.1186/s12933-024-02400-1.

32. *Chen W., Chen P., Ni Z.* Prognostic of different glomerular filtration rate formulas in patients receiving percutaneous coronary intervention: insights from a multicenter observational cohort. BMC Cardiovascular Disorders (2020) 20:341 https://doi.org/10.1186/s12872-020-01621-y

33. *Chiriacò M., Tricò D., Anna Solini.* Mechanisms of cardio-renal protection of sodium-glucose cotransporter-2 inhibitors. Curr Opin Pharmacol. 2022 Oct:66. 102272.doi: 10.1016/j.coph.2022.102272. Epub 2022 Aug 11.

34. *Cho E., Ko G.J.* The Pathophysiology and the Management of Radiocontrast-Induced Nephropathy. Diagnostics (Basel). 2022 Jan 12;12(1):180.doi: 10.3390/diagnostics12010180

35. *Christian Jung, Raphael Romano Bruno, Marwan Jumean.* Management of cardiogenic shock: state-of-the-art. Intensive Care Med. 2024 Nov;50(11):1814-1829. doi: 10.1007/s00134-024-07618-x. Epub 2024 Sep 10

36. *Dai Z-C., Chen J-X., Zou R.* Role and mechanisms of SGLT-2 inhibitors in the treatment of diabetic kidney disease. Front Immunol. 2023 Sep 21:14:1213473.doi: 10.3389/fimmu.2023.1213473. eCollection 2023.

37. *Davies M., Aroda V.R., Collins B.S.* Management of Hyperglycemia in Type 2 Diabetes, 2022. A Consensus Report by the American Diabetes Association (ADA) and the European Association for the Study of Diabetes (EASD). Diabetes Care. 2022 Nov 1;45(11):2753-2786.doi: 10.2337/dci22-0034

38. *Draznin B., Aroda V.R., Bakris G., Benson G. et al.* Cardiovascular disease and risk management: standards of medical care in diabetes-2022. Diabetes Care. 2022;45 (Supplement_1): S144–S174. doi: 10.2337/dc22-S010. [DOI] [PubMed] [Google Scholar]

39. ESC Scientific Document Group 2019. ESC Guidelines for the diagnosis and management of chronic coronary syndromes: The Task Force for the diagnosis and management of chronic coronary syndromes of the European Society of Cardiology (ESC). European Heart Journal 2020, 41 (3): 407–7. DOI: 10.1093/eurheartj/ehz425.

40. ESC Guidelines for the management of acute coronary syndromes 2023. Eur Heart J Acute Cardiovasc Care. 2024 Feb 9. 13(1). 55-161.doi: 10.1093/ehjacc/zuad107.

41. *Fishman S.L., Sonmez H., Basman C. et al.* The role of advanced glycation end-products in the development of coronary artery disease in patients with and without diabetes mellitus: a review. Mol Med. 2018;24(1):59. doi: 10.1186/s10020-018-0060-3. [DOI] [PMC free article] [PubMed] [Google Scholar]

42. *Fox B.M, Gil H-W., Kirkbride-Romeo L.* Metabolomics assessment reveals oxidative stress and altered energy production in the heart after ischemic acute kidney injury in mice. Kidney Int. 2019 Mar;95(3). 590-610.doi: 10.1016/j.kint.2018.10.020. Epub 2019 Jan 30.

43. GBD 2017 Causes of Death Collaborators. Global, regional, and national age-sex-specific mortality for 282 causes of death in 195 countries and territories, 1980–2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet, 2018; 392: 1736–1788. https://doi.org/10.1016/s0140-6736(18)32203-7

44. *Hostalek U.* Global epidemiology of prediabetes – present and future perspectives. Clin Diabetes Endocrinol. 2019;5:5. https://doi.org/10.1186/ s40842-019-0080-0.

45. *Jankauskas S.S., Kansakar U., Varzideh F., et al.* Heart failure in diabetes. Metabolism 2021:154910. [DOI] [PMC free article] [PubMed]

46. KDIGO 2022 Clinical Practice Guideline for Diabetes Management in Chronic Kidney Disease. Kidney Disease: Improving Global Outcomes (KDIGO) Diabetes Work Group. Kidney Int. 2022 Nov;102(5S):S1-S127.doi: 10.1016/j.kint.2022.06.008.

47. *Kim N.H., Seo M.H., Jung J.H.* 2023 Diabetic Kidney Disease Fact Sheet in Korea. Diabetes Metab J. 2024 May;48(3):463-472.doi: 10.4093/dmj.2023.0310. Epub 2024 Mar 19.

48. *Krittanawong C., Britt W.M., Rizwan A.* Clinical Update in Heart Failure with Preserved Ejection Fraction.

Curr Heart Fail Rep. 2024 Oct;21(5):461-484.doi: 10.1007/s11897-024-00679-5. Epub 2024 Sep 3.

49. *Lagosz P., Biegus J., Urban S.* Renal Assessment in Acute Cardiorenal Syndrome. Biomolecules. 2023 Jan 27;13(2):239.doi: 10.3390/biom13020239.

50. *Laichuthai N., Ghani M., Kosiborod M.* Newly Discovered Abnormal Glucose Tolerance in Patients With Acute Myocardial Infarction and Cardiovascular Outcomes: A Meta-analysis. Diabetes Care. 2020 Aug;43(8):1958-1966.doi: 10.2337/dc20-0059.

51. *Larsson J., Auscher S., Shamoun A., Pararajasingam G., Heinsen L.J., Andersen T.R. et al.* Insulin resistance is associated with high-risk coronary artery plaque composition in asymptomatic men between 65 and 75 years and no diabetes: a DANCAVAS cross-sectional sub-study. Atherosclerosis. 2023;385:117328. doi: 10.1016/j.atherosclerosis.2023.117328. [DOI] [PubMed] [Google Scholar]

52. *Lee S., Sohn J.Y., Lee H.J., Susie Yoon.* Effect of pre-operative carbohydrate loading on aspiration risk evaluated with ultrasonography in type 2 diabetes patients: a prospective observational pilot study. Sci Rep. 2022 Oct 20;12(1):17521.doi: 10.1038/s41598-022-21696-1.

53. *Lee W-C., Fang C-Y., Chen C-J.* The Clinical Outcomes Based on the Achievement of Low-Density Lipoprotein Cholesterol Targets after ST Elevation Myocardial Infarction. J Clin Med. 2019 Dec 28;9(1):79.doi: 10.3390/jcm9010079.

54. *Li L., Zhao M., Zhang Z.* Prognostic significance of the stress hyperglycemia ratio in critically ill patients. Cardiovasc Diabetol. 2023 Oct 13;22(1):275.doi: 10.1186/s12933-023-02005-0.

55. *Li W., Chen D., Peng Y., Lu Z., et al.* NHANES III. Association Between Metabolic Syndrome and Mortality: Prospective Cohort Study. JMIR Public Health Surveill. 2023 Sep 5:9:e44073.doi: 10.2196/44073.

56. *Li Y., Li X., Zhang Y.* Impact of glycemic control status on patients with ST-segment elevation myocardial infarction undergoing percutaneous coronary intervention. BMC Cardiovasc Disord. 2020 Jan 30;20(1):36. doi: 10.1186/s12872-020-01339-x

57. *Lin H., Guo X., Wang M.* Risk factors and early prediction of cardiorenal syndrome type 3 among acute kidney injury patients: a cohort study. Ren Fail. 2024 Dec;46(1):2349113.doi: 10.1080/0886022X.2024.2349113. Epub 2024 May 9.

58. *Lullo L., Reeves P.B., Bellasi A.* Cardiorenal Syndrome in Acute Kidney Injury. Semin Nephrol. 2019 Jan;39(1):31-40. doi: 10.1016/j.semnephrol.2018.10.003

59. *Ma C.X., Ma X.N., Guan C.H.* Cardiovascular disease in type 2 diabetes mellitus: progress toward personalized management. Cardiovascular Diabetology (2022) 21:74 https://doi.org/10.1186/s12933-022-01516-6

60. *McCallum W., Sarnak M.J.* Cardiorenal Syndrome in the Hospital. Clin J Am Soc Nephrol. 2023 Jul 1;18(7):933-945.doi: 10.2215/CJN.0000000000000064. Epub 2023 Jan 13.

61. *Mosenzon O., Alguwaihes A., Jose Leon J.L.A.* CAPTURE: a multinational, cross-sectional study of cardiovascular disease prevalence in adults with type 2 diabetes across 13 countries. Cardiovasc Diabetol. 2021 Jul 27;20(1):154.doi: 10.1186/s12933-021-01344-0.

62. *Nguyen L., Borghi C., Evans M., et al.* Prevalence and impact of newly diagnosed dysglycaemia in acute coronary syndrome: Insights from the EUROASPIRE V survey. Eur J Prev Cardiol. 2020;27(1):8-19. doi:10.1177/2047487319881446.

63. *Pandey R.K., Lwin B.B., Vashishta A.* Acute Hyperglycemia and Its Impact on Mortality of Acute Coronary Syndrome Patients: A Systematic Review. Cureus. 2024 Aug 7;16(8):e66365. doi: 10.7759/cureus.66365. eCollection 2024 Aug

64. *Pepe M., Addabbo F., Cecere A..* Acute Hyperglycemia-Induced Injury in Myocardial Infarction. Int J Mol Sci. 2024 Aug 4;25(15):8504.doi: 10.3390/ijms25158504.

65. *Pickering W., Blunt R., Than P.* Acute Kidney Injury and mortality prognosis in Acute Coronary Syndrome patients: A meta-analysis. Nephrology (Carlton). 2018 Mar;23(3):237-246. doi: 10.1111/nep.12984.

66. *Ploumen E., Buiten R., Kok M..* Three-year clinical outcome in all-comers with "silent" diabetes, prediabetes, or normoglycemia, treated with contemporary coronary drug-eluting stents: From the BIO-RESORT Silent Diabetes study. Catheter Cardiovasc Interv. 2020 Aug;96(2):E110-E118.doi: 10.1002/ccd.28536. Epub 2019 Oct 17.

67. *Qi L., Liu H., Cheng L.* Impact of Renal Insufficiency on Prognosis of Patients with Acute Coronary Syndrome. Int J Gen Med. 2021 Nov 26:14:8919-8927. doi: 10.2147/IJGM.S334014. eCollection 2021

68. *Ramírez-Guerrero G., Ronco C., Reis T.* Cardiorenal Syndrome and Inflammation: A Forgotten Frontier Resolved by Sorbents? Cardiorenal Med. 2024;14(1):454-458.doi: 10.1159/000540123. Epub 2024 Aug 2.

69. *Rodrigues A.S., Morato de Castilho F., Ribeiro A.J.* Association between Hemodynamic Profile at Hospital Admission and Mortality in Acute Heart Failure Patients Included in the Best Practice in Cardiology Program in Brazil. Arq Bras Cardiol. 2024 Jun 24;121(5):e20230699.doi: 10.36660/abc.20230699. eCollection 2024.

70. *Roumeliotis S., Veljkovic A., Georgianos P.* Association between Biomarkers of Oxidative Stress and Inflammation with Cardiac Necrosis and Heart Failure in Non-ST Segment Elevation Myocardial Infarction Patients and Various Degrees of Kidney Function. Oxid Med Cell Longev. 2021 Nov 1:2021:3090120. doi: 10.1155/2021/3090120. eCollection 2021.

71. *Ruiz H.H., Ramasamy R., Schmidt A.M.* Advanced glycation end products: building on the concept of the “Common Soil” in metabolic disease. Endocrinology. 2020; 161(1). [DOI] [PMC free article] [PubMed]

72. *Sadeghi R., Aghajani M.H., Parandin R..* Leuko-Glycemic Index in the Prognosis of Acute Myocardial Infarction; a Cohort Study on Coronary Angiography and Angioplasty Registry. Arch Acad Emerg Med. 2023 Sep 13;11(1):e63.doi: 10.22037/aaem.v11i1.2085. eCollection 2023.

73. *Saluja S.* Foot ulceration and its association with mortality in diabetes mellitus: a meta-analysis / S. Saluja, S.G. Anderson, I. Hambleton, et al. Diabet Med. – 2020. – Vol. 37(2). – pp. 211-218. doi: 10.1111/dme.14151

74. *Santosa Y., Harca A.D., Yuwono A.* Is It Safe to Do Percutaneous Coronary Intervention in Moderate to Severe Chronic Kidney Disease Patients? A Prospective Cohort Study. Cureus. 2022 Oct 14;14(10):e30312.doi: 10.7759/cureus.30312. eCollection 2022 Oct.

75. *Savic L., Mrdovic I. 1 2, Asanin M.* The Impact of Kidney Function on the Slow-Flow/No-Reflow Phenomenon in Patients Treated with Primary Percutaneous Coronary Intervention: Registry Analysis. J Interv Cardiol. 2022 Nov 30:2022:5815274.doi: 10.1155/2022/5815274. eCollection 2022.

76. *Scheen M., Giraud R., Bendjelid K.* Stress hyperglycemia, cardiac glucotoxicity, and critically ill patient outcomes current clinical and pathophysiological evidence. Physiol Rep. 2021 Jan;9(2):e14713.doi: 10.14814/phy2.14713.

77. *Shan Liu.* Heart-kidney interactions: mechanistic insights from animal models. Am J Physiol Renal Physiol. 2019 May 1;316(5):F974-F985.doi: 10.1152/ajprenal.00624.2017. Epub 2019 Mar 6.

78. *Shirani J., Meera S., Dilsizian V.* The Cardiorenal Axis: Myocardial Perfusion, Metabolism, and Innervation. Curr Cardiol Rep. 2019 May 20;21(7):60. doi: 10.1007/s11886-019-1147-3

79. *Shu J., Matarese A., Santulli G.* Diabetes, body fat, skeletal muscle, and hypertension: the ominous chiasmus? J Clin Hypertens (Greenwich) 2019;21(2):239–242. doi: 10.1111/jch.13453. [DOI] [PMC free article] [PubMed] [Google Scholar]

80. *Shu J., Santulli G.* Update on peripheral artery disease: epidemiology and evidence-based facts. Atherosclerosis. 2018;275:379–381. doi: 10.1016/j.atherosclerosis.2018.05.033. [DOI] [PMC free article] [PubMed] [Google Scholar]

81. *Son H.E., Moon J.M., Park J-M.* Additive harmful effects of acute kidney injury and acute heart failure on mortality in hospitalized patients. Kidney Res Clin Pract. 2022 Mar;41(2):188-199. doi: 10.23876/j.krcp.21.111. Epub 2021 Dec 1.

82. *Stampouloglou P., Anastasiou A., Bletsa E.* Diabetes Mellitus in Acute Coronary Syndrome. Life (Basel). 2023 Nov 19;13(11):2226. doi: 10.3390/life13112226.

83. *Stefanos Roumeliotis 1, Andrej Veljkovic 2, Panagiotis I Georgianos.* Association between Biomarkers of Oxidative Stress and Inflammation with Cardiac Necrosis and Heart Failure in Non-ST Segment Elevation Myocardial Infarction Patients and Various Degrees of Kidney Function. Oxid Med Cell Longev. 2021 Nov 1:2021:3090120. doi: 10.1155/2021/3090120. eCollection 2021.

84. *Suzuk R., Kazumori K., Usui T.* Prevalence and predictors of clinical inertia in patients with type 2 diabetes who were treated with a single oral antidiabetic drug. J Diabetes Investig. 2023 Jan;14(1):81-91.doi: 10.1111/jdi.13923. Epub 2022 Oct 13.

85. *Thygesen K., Alpert J.S., Jaffe A.S., Chaitman B.R., Bax J.J., Morrow D.A., White H.D;* ESC Scientific Document Group. Fourth universal definition of myocardial infarction.Eur Heart J. 2019;40(3):237-269. doi: 10.1093/eurheartj/ehy462.

86. *Timmis A., Vardas P., Townsend N., Torbica A., Katus H., De Smedt D., et al.* European Society of Cardiology: cardiovascular disease statistics 2021. Eur Heart J, 2022; 43:716–799. https://doi.org/10.1093/eurheartj/ehab892

87. *Veiga G.L., Costa Aguiar Alves B., Perez M.M., et al.* Kidney Diseases: The Age of Molecular Markers. Adv Exp Med Biol. 2021;1306:13-27. doi: 10.1007/978-3-030-63908-2.

88. *Vrints Ch., Andreotti F., Koskinas K.C., et al.* 2024 ESC Guidelines for the management of chronic coronary syndromes. Eur. Heart J. 2024.

89. *Weferling M., Liebetrau C., Kraus D.* Definition of acute kidney injury impacts prevalence and prognosis in ACS patients undergoing coronary angiography. BMC Cardiovascular Disorders volume 21, Article number: 183 (2021)

90. *Wu L., Rodriguez M., Hachem K.E.* Diuretic Treatment in Heart Failure: A Practical Guide for Clinicians. J Clin Med. 2024 Jul 30;13(15):4470. doi: 10.3390/jcm13154470.

91. *Xu C., Tsihlis G., Chau K.* Novel Perspectives in Chronic Kidney Disease-Specific Cardiovascular Disease. Int J Mol Sci. 2024 Feb 24;25(5):2658. doi: 10.3390/ijms25052658.

92. *Xu F., Wang G., Ye N.* Mild renal insufficiency and attributable risk of adverse In-hospital outcomes in patients with Acute Coronary Syndrome from the improving care for Cardiovascular Disease in China (CCC) project. BMC Nephrol. 2022 Jan 13;23(1):29.doi: 10.1186/s12882-022-02663-4.

93. *Yang J., Chen S., Wang Y., et al.* Prevalence of prediabetes among patients with coronary heart disease in China: A meta-analysis. Diabetes Res Clin Pract. 2020;165:108233. doi:10.1016/j.diabres.2020.108233

94. *Yaqub S., Hashmi S., Kashif Kazm M.* A Comparison of AKIN, KDIGO, and RIFLE Definitions to Diagnose Acute Kidney Injury and Predict the Outcomes after Cardiac Surgery in a South Asian Cohort. Cardiorenal Med. 2022;12(1):29-38.doi: 10.1159/000523828. Epub 2022 Mar 3.

95. *Zeng M., Sun E., Zhu L. et al.* Influence of prediabetes on the prognosis of patients with myocardial infarction: a meta-analysis. Diabetol Metab Syndr. 2024 Jul 12;16(1):160. doi: 10.1186/s13098-024-01381-1.

96. *Zeru M.A., Tesfa A., Mitiku A.* Prevalence and risk factors of type-2 diabetes mellitus in Ethiopia: systematic review and meta-analysis. Sci Rep. 2021 Nov 5;11(1):21733.doi: 10.1038/s41598-021-01256-9.

97. *Zhan K, Weng L., Qi L. et al.* Effect of Antidiabetic Therapy on Clinical Outcomes of COVID-19 Patients With Type 2 Diabetes: A Systematic Review and Meta-Analysis. Ann Pharmacother. 2023 Jul;57(7):776-786. doi: 10.1177/10600280221133577.

98. *Zhou M., Liu J., Hao Y., Liu J., Huo Y., Smith S.C. Jr. et al.* Prevalence and inhospital outcomes of diabetes among patients with acute coronary syndrome in China: findings from the Improving Care for Cardiovascular Disease in China-Acute Coronary Syndrome Project. Cardiovasc Diabetol. 2018;17(1):147. doi: 10.1186/s12933-018-0793-x.

99. *Zhu J., Liu W., Li J. et al.* Small Increase in Serum Creatinine within 48 h of Hospital Admission Is an Independent Predictor of In-Hospital Adverse Outcomes in

Patients with ST-Segment Elevation Myocardial Infarction Undergoing Primary Percutaneous Coronary Intervention: Findings from the Improving Care for Cardiovascular Disease in the China Project. Cardiol Res Pract. 2023 Mar 28:2023:1374206.doi: 10.1155/2023/1374206. eCollection 2023.

100. *Zweck E., Hassager C., Beske R.P.* Microaxial Flow Pump Use and Renal Outcomes in Infarct-Related Cardiogenic Shock: A Secondary Analysis of the DanGer Shock Trial. Circulation. 2024 Dec 17;150(25):1990-2003. doi: 10.1161/CIRCULATIONAHA.124.072370. Epub 2024 Oct 27

References: [1-20]

1. Arsenicheva O.V. Prediktory ostrogo povrezhdeniya pochek u bol'nykh infarktom miokarda s podyeomom segmenta ST, oslozhnyeonnym kardiogennym shokom, perenyeosshikh chreskozhnoe koronarnoe vmeshatel'stvo [Predictors of acute kidney injury in patients with ST-segment elevation myocardial infarction and complicated cardiogenic shock who underwent percutaneous coronary intervention]. *Meditsinskii vestnik Juga Rossii* [Medical Bulletin of South Russia]. 2022. 13(3):118-126. DOI 10.21886/2219-8075-2022-13-3-118-126 [in Russian]

2. Barbarash O.L., Voevoda M.I., Galstjan G.R., Shestakova M.V. Prediabet kak mezhdistsiplinarnaya problema: opredelenie, riski, podkhody k diagnostike i profilaktike sakharnogo diabeta 2 tipa i serdechno-sosudistyh oslozhnenij. Rossijskij kardiologicheskij zhurnal. 2019;(4):83-91. https://doi.org/10.15829/1560-4071-2019-4-83-91 [in Russian]

3.Bojcov S.A., Barbarash O.L., Vajsman D.Sh., Galjavich A.S. i dr. Klinicheskaja, morfologicheskaja i statisticheskaja klassifikacija ishemicheskoj bolezni serdca: konsensus Rossijskogo kardiologicheskogo obshhestva, Rossijskogo obshhestva patologoanatomov i specialistov po medicinskoj statistike. 2020. 24 p. [in Russian]

4.Dedov I.I., Mel'nichenko G.A. Jendokrinologija. Nacional'noe rukovodstvo / M.: GJeOTAR-MEDICINA, 2024. – 433 s. [in Russian]

5.Demchuk O.V., Sukmanova I.A. Prognozirovanie chastoty razvitija hronicheskoj bolezni pochek u pacientov s infarktom miokarda i ostrym povrezhdeniem pochek. Rossijskij kardiologicheskij zhurnal. 2023;28(6):5247. doi:10.15829/1560-4071-2023-5247. [in Russian]

6.Zykov M.V., D'jachenko N.V., Velieva R.M., Kashtalap V.V., Barbarash O.L. Vozmozhnosti sovmestnogo ispol'zovanija shkaly GRACE i razlichnyh indeksov komorbidnosti dlja povyshenija jeffektivnosti ocenki riska gospital'noj letal'nosti u bol'nyh s ostrym koronarnym sindromom. Terapevticheskij arhiv. 2022;94(7):816–821. DOI: 10.26442/00403660.2022.07.201742[in Russian]

7.Demchuk O.V., Sukmanova I.A. Ostroe pochechnoe povrezhdenie u pacientov s infarktom miokarda i chreskozhnymi koronarnymi vmeshatel'stvami: laboratornye markery i vlijanie na chastotu povtornyh serdechno-sosudistyh sobytij. Kompleksnye problemy serdechno-sosudistyh zabolevanij. 2022;11(4):25-34. https://doi.org/10.17802/2306-1278-2022-11-4-25-34 [in Russian]

8.Koziolova N.A., Poljanskaja E.A., Mironova S.V. Saharnyj diabet 2 tipa i mikrososudistye oslozhnenija u bol'nyh stabil'noj ishemicheskoj bolezn'ju serdca: rasprostranennost', prognoz i vybor antitromboticheskoj terapii. Juzhno-Rossijskij zhurnal terapevticheskoj praktiki. 2022;3(4):7-24. https://doi.org/10.21886/2712-8156-2022-3-4-7-24 [in Russian]

9.Kompanec N.V., Shhukin Ju.V., Limareva L.V., Komarova M.V. Prognosticheskoe znachenie nejtrofil'nogo zhelatinaza-associirovannogo lipokalina i cistatina S u pacientov s hronicheskoj serdechnoj nedostatochnost'ju i perenesennym infarktom miokarda. Rossijskij kardiologicheskij zhurnal. 2021;26(1):4126. doi:10.15829/1560-4071-2021-4126 [in Russian]

10. Leonova M.V. Ingibitory natrij-gljukoznogo kotransportera 2: mehanizmy kardiorenal'noj zashhity. Consilium Medicum. 2024;26(4):225–231. DOI: 10.26442/20751753.2024.4.202763 [in Russian]

11. Martynov S.A., Shamhalova M.Sh. Rol' ingibitorov natrij-gljukoznogo kotransportera 2 v zamedlenii progressirovanija pochechnoj nedostatochnosti u pacientov s saharnym diabetom 2 tipa // Jeffektivnaja farmakoterapija. 2021. T. 17. № 5. S. 16–23. [in Russian]

12. Maslennikova O.M., Ojnotkinova O.Sh., Starodubova A.V., Mackeplishvili S.T. Obzor rekomendacij Evropejskogo obshhestva kardiologov 2024 g. po vedeniju hronicheskih koronarnyh sindromov: chto novogo. Jeffektivnaja farmakoterapija. 2024; 20 (31): 9–22. [in Russian]

13. Nikitina E.A. Kliniko-funkcional'nye i prognosticheskie osobennosti reabilitacii pacientov s ostrym koronarnym sindromom i saharnym diabetom 2 tipa : dissertacija ... kandidata medicinskih nauk : 14.01.05 / Nikitina Elena Aleksandrovna; [Mesto zashhity: FGBOU VO «Permskij gosudarstvennyj medicinskij universitet imeni akademika E.A. Vagnera» Ministerstva zdravoohranenija Rossijskoj Federacii]. - Kirov, 2019. - 102 s. : il. [in Russian]

14. Nikulina N.A., Akulova E.A., Docenko Je.A., Krivelevich N.B., Sejfidinova S.G., Kovalev A.A., Zinkevich D.D., Ponomarenko D.A., Tishkov S.P., Koval'chuk A.N., Repina Ju.V. Tranzitornaja giperglikemija pri ostryh formah ishemicheskoj bolezni serdca. Problemy zdorov'ja i jekologii. 2023;20(2):49–58. DOI: https://doi.org/10.51523/2708-6011.2023-20-2-07 [in Russian]

15. Popyhova Je.B., Stepanova T.V., Lagutina D.D., Kirijazi T.S., Ivanov A.N. Rol' saharnogo diabeta v vozniknovenii i razvitii jendotelial'noj disfunkcii. Problemy Jendokrinologii. 2020;66(1):47-55.https://doi.org/10.14341/probl12212 [in Russian]

16. Reznik E.V., Nikitin I.G. Kardiorenal'nyj sindrom u bol'nyh s serdechnoj nedostatochnost'ju kak jetap kardiorenal'nogo kontinuuma (chast' I): opredelenie, klassifikacija, patogenez, diagnostika, jepidemiologija (obzor literatury). Arhiv# vnutrennej mediciny. 2019; 9(1):5-22. DOI: 10.20514/2226-6704-2019-9-1-5-22 [in Russian]

17. Rizaeva M.A. Narushenija uglevodnogo obmena pri infarkte miokarda i vozmozhnosti gipoglikemicheskoj terapii. Amaliy va tibbiyot fanlari ilmiy jurnali. vol. 3 No. 5 (2024):. 153-155 [in Russian]

18. Sejtmaganbetova N.A., Veklenko G.V., Hibina M.A., Zhaubatyrova A.A., Kurmasheva G.S. Sostojanie funkcii pochek u pacientov s ostrym koronarnym sindromom: retrospektivnoe issledovanie // West Kazakhstan Medical Journal. 2020. №1 (62). S. 60-67. [in Russian]

19. Chazova I.E., Shestakova M.V., Zhernakova Ju.V. Evrazijskie rekomendacii po profilaktike i lecheniju serdechno-sosudistyh zabolevanij u bol'nyh s diabetom i prediabetom (2021). Evrazijskij kardiologicheskij zhurnal. 2021; (2):6-61, https://doi.org/10.38109/2225-1685-2021-2-6-61 [in Russian]

20. Chistova N.P. Rol' polimorfizmov genov-kandidatov jendotelial'noj disfunkcii i metabolicheskih narushenij v razvitii serdechno-sosudistyh zabolevanij pri vozdejstvii proizvodstvennyh faktorov. Med. truda i prom. jekol. 2022; 62(5): 331–336. https://elibrary.ru/jdniwu https://doi.org/10.31089/1026-9428-2022-62-5-331-336 [in Russian]

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