AJPMHS GLOBAL RESEARCH NETWORK

AMERICAN Journal of Pediatric Medicine and Health Sciences
Volume 3, Issue 12, 2025 ISSN (E): 2993-2149

Assessment of Changes in Kidney Dysfunction During the Acute Phase of Myocardial Infarction

Muxamedova Nurxon, Gadaev Abdigaffor, Abduvalieva Iqbolxon, Gadaeva Nilufar
Tashkent State Medical University

Abstract: This article compares changes in the acute phase of myocardial infarction, comparative studies were conducted on changes in renal glomeruli and tubules using troponin, creatinine, cystatin C, neutrophil gelatinase-associated lipocalin (NGAL), and kidney injury molecule-1 (KIM-1). The obtained results indicate that NGAL, compared to cystatin C and KIM-1, allows for earlier detection of acute kidney injury that develops during the acute phase of myocardial infarction.

Keywords: Myocardial infarction, ECG, EchoCG, KIM -1, NGAL, cystatin S, renal dysfunction.

Introduction. Acute kidney damage in myocardial infarction is one of its complications, and according to some literature, it is observed in 10-60% of patients [3,4]. The addition of renal dysfunction dramatically worsens the course of myocardial infarction, increases the risk of death, and increases the cost of treatment. [1].

The development of acute renal failure in myocardial infarction is associated with decreased renal perfusion and decreased filtration due to decreased cardiac output [6]. At the same time, damage to the renal tubules further complicates the process [6].

As is known, creatinine is widely used to detect it. However, for a number of reasons (patient age, sex, muscle mass, etc.), it does not allow for early diagnosis of changes in the functional state of the kidneys. Therefore, in recent years [12], the use of highly sensitive specific biomarkers has made it possible to detect changes in the kidneys early [14]. However, their diagnostic criteria have not been studied in detail so far.

Recent studies have shown that acute kidney injury following myocardial infarction is a risk factor for both short- and long-term adverse outcomes. The 10-year mortality rate from myocardial infarction is 15% in mild acute kidney injury, 23% in moderate acute kidney injury, and 33% in severe acute kidney injury [7].

Therefore, the study of markers that confirm acute kidney injury in dynamics is of great practical importance. Biological markers are used in practice to assess the risk of developing the disease or its diagnosis, and to assess the effectiveness of treatment [19].

It is known that the biomarkers widely used in practice are blood creatinine and albuminuria. Although they indicate worsening renal dysfunction, changes are observed in the late stages of this complication. [22].

The end in years balls filtration calculation with together kidneys dysfunction in determining to practice battalion wide applicable biomarker cystatin - C is considered This protein is 122 amino acids consists of is 13 kDa in weight and that is cysteine proteases inhibitor is counted. Cystatin -

S in the body all including liquids in the urine both exists [20] and his/her permanence the organism control as impossible from proteolysis protection does Cystatin -C from creatinine different dying work expulsion row factors, in particular age, sex, tumors to increase, plus muscle mass, inflammation to the process related not. But him/her high-level man gender, height length and excessive body even with weight dependency about separate in research cited [16]. Cystatin -S tubules to the void come out his/her proximal in part complete metabolism occurs. Because of this his/her in the blood indicator balls reversed by filtration proportional because conclusion if it is released will be.

Available information kidney dysfunction in determining Cystatin -C is one in line from creatinine both parallel use to the goal suitable that it is shows. Kidney diseases epidemiology researcher by the community (CKD - EPI). 1.73m2 body to the surface one less than 60 ml per minute when creatinine and together from cystatin -S use in diagnosis importance about research passed [24].

Measured together with creatinine and cystatin -C, it excludes all causes, including cardiovascular disease. the indicator is 85 per 1.73 m^2 body surface per minute ml when it is less than, the death rate increases in parallel [25].

Therefore, studies show that estimation of glomerular filtration rate using cystatin-S and creatinine is reliable in diagnosing renal dysfunction. Also, the risk stratification of blood creatinine, cystatin-C values, and the ratio of albumin to creatinine in urine more clearly confirm the development of renal failure [27].

Neutrophil gelatinase-associated lipocalin (NGAL) is a 25-kDa protein consisting of 128 amino acids. NGAL is found mainly in the monomeric form in biological fluids. In animals with ischemic nephrotoxic acute renal failure, elevated levels of this protein were detected in their serum and urine. Therefore, NGAL has been considered a primary biomarker of ischemic kidney damage. This protein is mainly produced in the ascending limb of the renal tubule of Genie and in the collecting ducts [28].

Currently, NGAL is not only a reliable marker of acute kidney injury, but also has been shown to be important in the diagnosis of SCD in two types of rats [26]. It has also been shown to be elevated in the blood of patients with rapidly developing SCD [14]. In addition, elevated levels of NGAL in the blood and urine have been found in a number of other kidney diseases, including IgA nephropathy, autosomal polycystic kidney disease, and diabetic nephropathy [15].

KIM-1 is a transmembrane lipoprotein with a molecular weight of 90 kDa and contains extracellular mucin and immunoglobulin domains. Its levels are unchanged in normal kidneys but are elevated in the proximal tubules after ischemia. The extracellular domain of KIM-1 is readily detectable in urine shortly after ischemic kidney injury [13].

In rats, KIM-1 knockdown of renal epithelial cells induces interstitial inflammation with fibrosis, leading to anemia, proteinuria, hyperphosphatemia, and hypertension, which can lead to death. This may provide a useful insight into the mechanisms underlying human renal disease [21].

Therefore, sustained KIM-1 production is a marker of advanced renal fibrosis, a mechanism of acute and chronic damage, and an increase in SCC. Retrospective studies of patients with diabetes-related proteinuria have shown that elevated urinary KIM-1 levels were normalized after treatment with angiotensin-converting enzyme inhibitors or a low-salt diet. This suggests that this lipid may play an important role in assessing the efficacy of treatment [10,11].

Although a number of studies have been conducted in recent years on the importance of the above-mentioned biochemical markers in the early detection of renal dysfunction in the acute phase of myocardial infarction, they have not been studied in a comparative manner. Troponins, which are considered important diagnostic markers of myocardial infarction, have been reported in the literature to change not only in this disease, but also in a number of other pathological

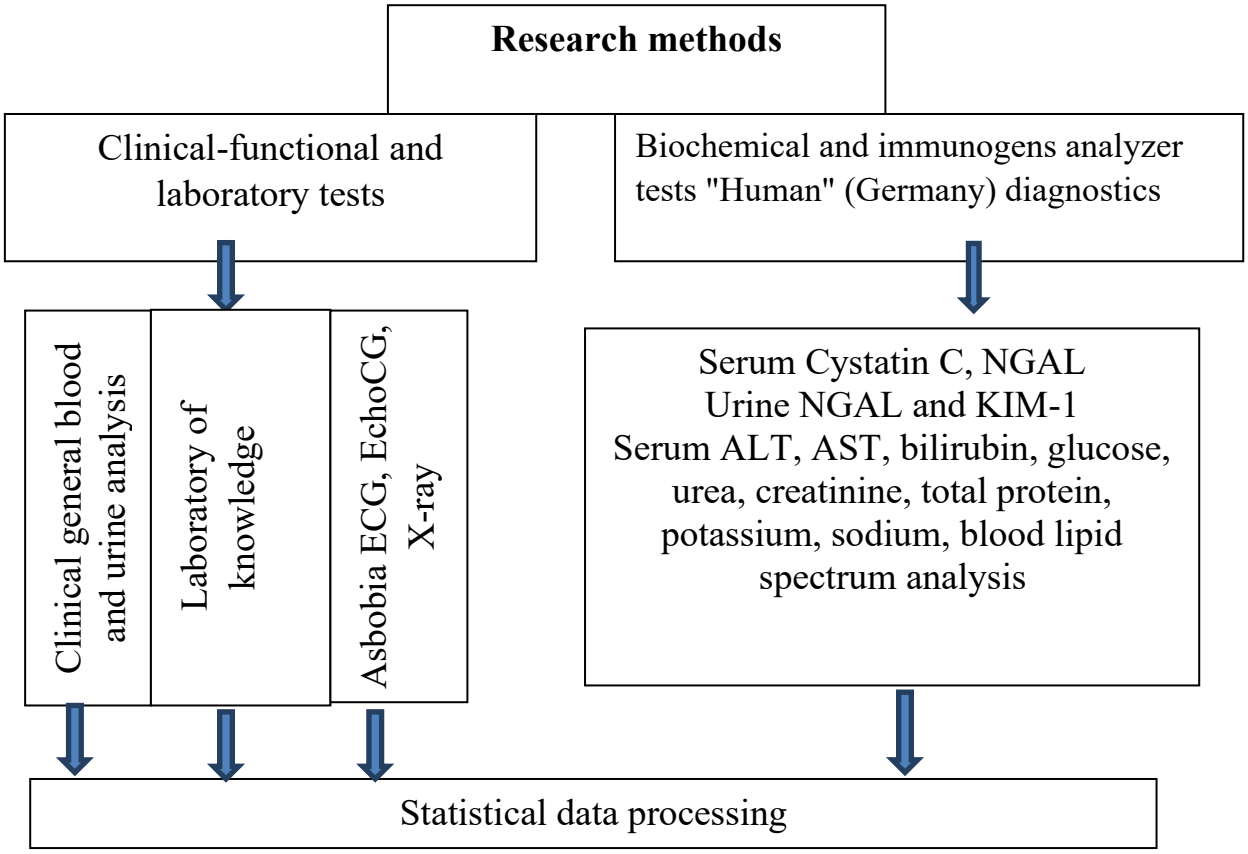
conditions, including nephropathies. From this point of view, studying the relationship between troponin and other markers of renal dysfunction is of great practical importance.

The aim of the study was to compare changes in renal glomeruli and tubules during the acute phase of myocardial infarction using troponin, creatinine, cystatin-C, neutrophil gelatin-associated lipocalin-NGAL, and renal injury molecules - KIM-1.

Materials and methods

Based on the goals and objectives set for our study, 60 patients with a diagnosis of acute myocardial infarction treated in the cardioresuscitation department of the Multidisciplinary Clinic of the Tashkent Medical Academy in 2024 were included in our study - the main group, and 30 patients with stable angina pectoris of functional classes III-IV, who received treatment in the cardiology department, were included in the control group. Of the patients in the main group, 38 (63.3%) were men and 22 (36.7%) were women. The average age was 60.5±7.4. The control group included 16 men and 14 women, and their average age was 63.6±5.9 years. The main group of patients, in turn, was divided into two subgroups based on electrocardiographic changes. The first group consisted of 32 patients with ST segment elevation on electrocardiography, their average age was 57.4±3.68. Of these, 23 were men and 9 were women. The second group consisted of 28 patients without ST segment elevation on electrocardiography, their average age was 63.8±2.87 (women-13, men-15). The patients were prescribed standard treatment for acute myocardial infarction (antiischemic, ACE inhibitor or sacubitril/valsartan, β -blockers, statin, antiplatelet agents, anticoagulants).

Included 30 patients with stable angina pectoris of functional class III-IV. This group of patients received standard treatment for ischemic heart disease.



All subjects were examined before the start of treatment, 3 days later, and 3 months later.

The inspection plan included the following:

Patient complaints, anamnesis, objective examination;

The re-examination was carried out on the 3rd day of their treatment.

Determination of laboratory parameters. *Complete blood count.* The main part of the complete blood count was performed on the "Mythic-22 " Orphee (Switzerland) hematological analyzer. It determined the hemoglobin content, erythrocyte, leukocyte, platelet, and hematocrit indices. The leukocyte formula was determined by staining the blood smear using the Romanovsky-Giemza method and examining it under a "BAUSCH-LOMB" (Russia) microscope. counted. Erythrocytes sinking speed Panchenko blood was mixed with 5% sodium citrate solution in a 100 mm tube.

Biochemical parameters of blood - serum alanine transferase (ALT), aspartate aminotransferase (AST), bilirubin, glucose, urea, creatinine, total protein, potassium, sodium, blood lipid spectrum analysis were performed on the Mindry BA-88 biochemical analyzer using reagents from the company "Human" (Germany).

Serum cystatin C levels were determined using the ELISA method on a semi-automatic Mindray MR 96A device. Detection range: 0.313-20 ng/ml, Sensitivity: 0.188 ng/ml.

Serum NGAL levels were determined using the ELISA method on a semi-automatic Mindray MR 96A device. Detection range: 0.313-20 ng/ml, Sensitivity: 0.188 ng/ml.

Serum NGAL levels were determined using the ELISA method on a semi-automatic Mindray MR 96A device. Detection range: 0.156-10 ng/ml, Sensitivity: 0.094 ng/ml.

Discussion of research results. We studied the role of pathological changes and laboratory markers in the development of acute kidney injury 72 hours after hospitalization in patients with acute myocardial infarction. The obtained laboratory analysis results are presented in Table 1 below.

Table 1. Changes in laboratory indicators in dynamics in patients with acute myocardial infarction.

№	Indicators	ST with segment elevation, n =32		ST segment elevation, n=28	
		Initially	After 72 hours	Initially	After 72 hours
1	Creatinine in blood, μmol /l	88.6±9.39	124.1±8.4*	8 0. 3 ±1 1. 26	96.3±9.4
2	Renal dysfunction, hKFT (1.72 m ^{2} body surface per minute, ml)	86.0±5.36	61.4±4.6**	97 ± 5.01	80.3±6.1*
3	Cardio troponin, ng/ml	29.3±0.93	42.2±1.4***	13.67±0.45	17.1±1.6*
4	ALT, U/L	29.2±6.55	40.9±6.4	22. 04 ±5. 45	26.44±4.87
5	AST, U/L	43.68±6.83	60.4±5.7	2 4. 4 ±5. 21	28.8±4.3
6	Urea, μmol /l	8.0±0.52	11.2±1.2*	7.4 ± 0.62	8.8±0.72
7	C-reactive protein, mg/ml	15.0±1.45	22.6±1.7**	11.2±1. 2	13.44±1.4

Note: * - differences are significant compared to baseline (*- P <0.05, ** - P <0.01, *** - P<0.001).

As shown in the table, the level of cardiotropin increased significantly (p<0.001) from 29.3±0.93 ng/ml to 42.2±1.4 ng/ml in the group with ST elevation. In the second group, that is, in those without ST elevation, its average level increased from 13.67±0.45 ng/ml to 17.1±1.6 ng/ml, and a significant difference was noted (p<0.05).

Serum creatinine increased 1.4 times from 88.6±9.39 μmol/l to 124.1±8.4 μmol/l in the first group, and a significant difference was detected ($p<0.05$). In the second group, its level increased 1.19 times from 80.3±11.26 μmol/l to 96.3±9.4 μmol/l ($p>0.05$). The glomerular filtration rate, calculated by creatinine, decreased significantly ($p<0.01$) from 86.0±5.36 ml per minute per 1.72 m^2 body surface area to 61.4±4.6 ml per minute. In patients without ST elevation, the eGFR decreased from 97±5.01 ml per minute per 1.72 m2 of body surface area to 80.3±6.1 ml, a significant difference ($p<0.05$) was noted.

In the first group, the level of ALT changed from 29.2±6.55 U/L to 40.9±6.4 U/L, and the level of AST changed from 43.68±6.83 U/L to 60.4±5.7 U/L, and no significant difference was detected between them. In the second group, no significant changes were detected in the dynamics of ALT and AST. The level of urea in serum increased in both groups from 8.0±0.52 μmol/L to 11.2±1.2 ($p<0.05$) and from 7.4±0.62 to 8.8±0.72 μmol/L ($p>0.05$), respectively. C-reactive protein levels significantly increased in patients with ST-segment elevation (from 15.0±1.45 mg/ml to 22.6±1.7 mg/ml, $p<0.01$). No significant difference was found in the group of patients without ST-segment elevation (from 11.2±1.2 mg/ml to 13.44±1.4, $p>0.05$).

Also, dynamic echocardiography was performed in the groups involved in the study, and its results are presented in Table 2.

Table 2. Changes in echocardiography indicators in dynamics in patients with acute myocardial infarction.

№	Indicators	ST with segment elevation, n =32		ST segment elevation, n=28	
		Initially	After 72 hours	Initially	After 72 hours
1	End diastolic volume, ml	171.7±4.91	176.8±4.4	158.9±6.72	159.7±5.4
2	End systolic volume, ml	85. 5 ±2.79	87.4±3.1	70. 8 ±4.08	71.2±4.2
3	End diastolic size, cm	5.5±0.06	5.5±0.07	5.3±0.07	5.2±0.04
4	End systolic size, cm	4. 5 ±0.06	4.4±0.06	4. 2 ±0.09	4.3±0.1
5	Bleeding fraction, %	44.7 ±1.6	4 3.1±1.4	48.3±2.0	48.9±2.0
6	Interventricular barrier thickness, mm	15.1±0.49	15.1±0.5	13.4±0.45	13.4±0.45
7	Left ventricular posterior wall thickness, mm	12.3±0.2	12.2±0.3	11.9±0.23	11.8±0.2

As shown in the table, no statistically significant changes were detected in the dynamics of the main group of patients during the observation period. The left ventricular end-diastolic volume in both groups changed from 171.7±4.91 ml to 176.8±4.4 ml and from 158.9±6.72 ml to 159.7±5.4 ml, respectively. The ejection fraction changed from 44.7±1.6% to 43.1±1.4% in those with ST segment elevation, while in the second group it initially increased from 48.3±2.0% to 48.9±2.0% after 3 days. The results obtained confirm that after the treatment procedures in patients with myocardial infarction, the functional state of the heart is stabilized and the risk of acute severe complications is reduced.

We also studied the dynamics of the main markers of acute kidney injury. Figures 1, 2 and 3 below show the results obtained.

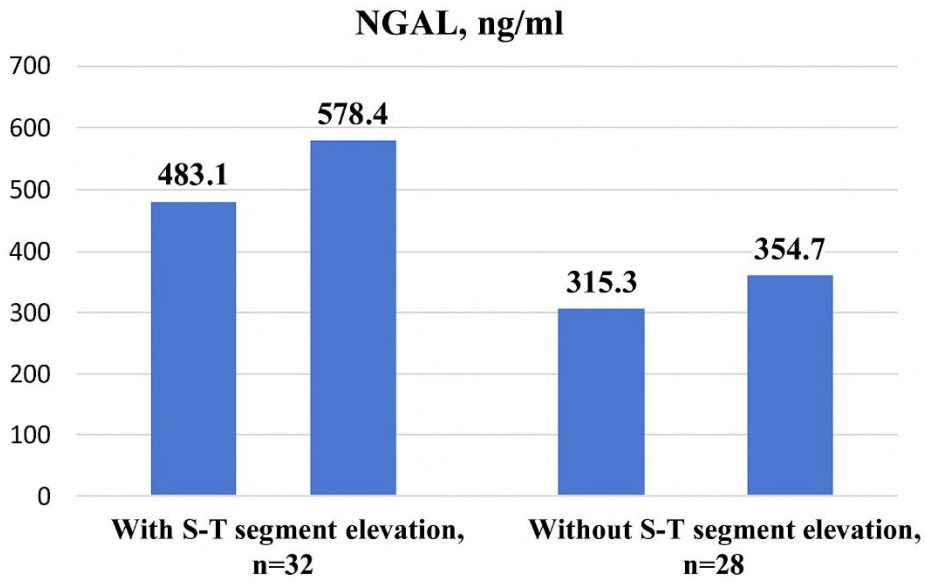


Figure 1. Dynamic changes in NGAL levels in patients with acute myocardial infarction

As shown in the figure, in patients with ST segment elevation, the urinary level of neutrophil gelatinase-associated lipocalin (NGAL) was 483.1±34.57 ng/ml, and when re-examined 72 hours after treatment, its level was 578.4±24.5 ng/ml, which increased by 1.2 times. When they were compared, a significant difference ($p<0.05$) was noted. In the second group, its level increased by 1.12 times from 315.3±32.5 ng/ml to 354.7±28.4 ng/ml during 3-day dynamic observation, but no significant difference was detected. We also studied the dynamics of KIM-1 indicators in the main group of patients in our study. The results are presented in Figure 2 below.

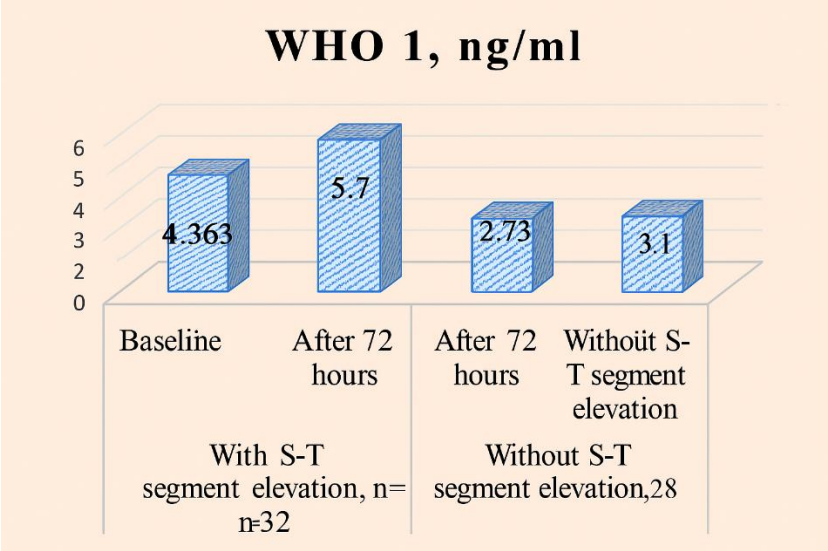


Figure 2. Changes in KIM-1 levels over time in patients with acute myocardial infarction.

KIM-1 levels in the first group were initially 4.363±0.147 ng/ml. On the third day of treatment, its level was 5.7±0.15 ng/ml, and when compared, a significant ($P<0.001$) difference was noted. In patients without ST segment elevation, the average urinary KIM-1 level was initially 2.713±0.089 ng/ml and after three days it was 3.1±0.1 ng/ml.

A reliable ($P<0.01$) difference was observed when the obtained results were compared. Cystatin-S indicators, which are considered to be the main markers in the assessment of the functional state of the kidneys in patients with advanced myocardial infarction, were also evaluated dynamically. The obtained results are presented in Figure 3.

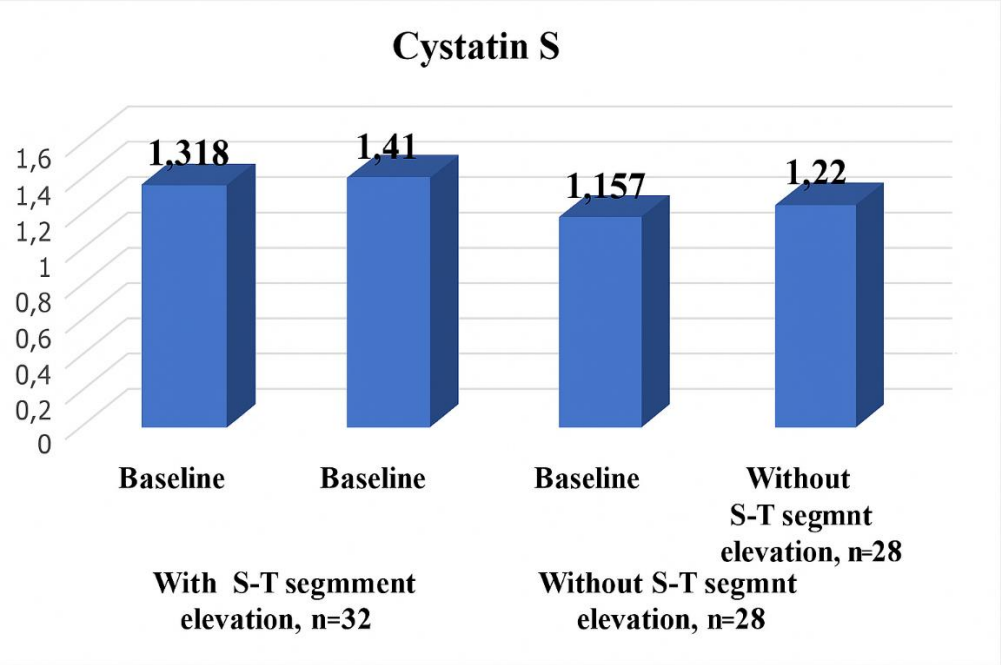


Figure 3. Changes in cystatin C indicators in dynamics in patients with acute myocardial infarction.

As shown in the figure, cystatin C levels increased in dynamics from 1.318±0.034 mg/l to 1.4±0.03 mg/l in patients with ST segment elevation, and a significant ($P<0.05$) difference was noted. In the second group, its level was initially 1.157±0.034 mg/l and after 3 days was 1.22±0.04 mg/l. In both groups of patients, cystatin C levels did not increase as significantly as NGAL and KIM-1 levels.

Conclusion

The kidneys are the most commonly affected target organ in patients with acute myocardial infarction. The results we have found in our study confirm this. In this group of patients, the presence of any comorbidities accelerates the process in the kidneys and leads to the development of acute kidney injury. Therefore, the detection of the above-mentioned markers in the acute period of the disease requires dynamic monitoring of the functional state of the kidneys and the implementation of nephroprotective therapy.

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