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Acute kidney injury in acute coronary syndrome

M.V. Menzorov*, A.M. Shutov, V.S. Sakharov, V.N. Kabanova

Ulyanovsk State University, Ulyanovsk, Russia

Abstract

Acute kidney injury is a common complication of acute coronary syndrome that aggravates its prognosis. The article presents the current criteria and stratification of the acute kidney injury severity, its place in the structure of cardiorenal syndromes, renal and cardiorenal continuums. The data on the frequency, severity and clinical variants of complications in acute coronary syndrome, myocardial infarction, unstable angina pectoris are presented. The risk factors for the development of acute kidney injury and its contrast-induced variant are described. The data on the significance of acute renal dysfunction in changing the trajectory of cardiovascular disease, worsening the immediate and long-term prognosis, the development and progression of chronic kidney disease, ischemic complications are presented. The effect of mechanical and pharmacological reperfusion on the incidence of acute kidney injury is described. Promising approaches to the diagnostics of acute kidney injury, including the significance of biomarkers and the problems associated with their use, are outlined. The article presents data on the role of radiopaque agents in the development of acute kidney injury, describes the difference between contrast-induced nephropathy, its contrast-associated, post-contrast and contrast-induced variants. The current approaches to the prevention and treatment of acute kidney injury from the point of view of various professional communities are outlined. Approaches to risk stratification and the possibility of using risk scales are described. The main measures for the prevention and treatment of acute kidney injury, depending on its severity, the place of renal replacement therapy are presented. The paper presents the current hydration regimens and describes the principles of their modification depending on the clinical characteristics of patients, proposed by experts from the Scientific Society of Nephrology of Russia and the consensus of the American College of Radiology, the US National Kidney Foundation.

Keywords: acute kidney injury, acute coronary syndrome, contrast-induced acute kidney injury, biomarkers of acute kidney injury.

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Criteria and stratification of the severity of acute kidney injury

Acute kidney injury (AKI) is a relatively new concept proposed by the Acute Dialysis Quality Initiative (ADQI) in 2004 [1]. The first clinical guidelines (KDIGO[1] Clinical Practice Guideline for Acute Kidney Injury) were developed and presented in 2012 [2]. To date, they have become generally accepted and are used as a reference for the recommendations of the Scientific Society of Nephrologists of Russia [3, 4].

AKI is currently defined as "an increase in serum creatinine of 0.3 mg/dL (≥ 26.5 μmol/L) over 48 h, an increase in serum creatinine by up to ≥1.5 times compared with baseline (if known or suspected to have occurred within the previous 7 days), or urine volume <0.5 mL/kg/h for 6 h" [2, 3].

In patients with a history of chronic kidney disease (CKD), AKI is diagnosed against CKD, whereas in its absence, AKI is diagnosed *de novo*. If kidney damage develops before hospital admission, then prehospital AKI is recorded; if it occurs during treatment, hospital-acquired AKI is registered [2].

AKI severity stratification is based on the degree of change in serum creatinine levels and/or diuresis over time, as shown below [2, 3]:

– Stage 1 is defined as an increase in serum creatinine level ≥26.5 μmol/L or 1.5–1.9 times from the baseline or with a decrease in the volume of urine excreted of <0.5 mL/kg/h for 6–12 h.

– Stage 2 is defined as an increase in serum creatinine by 2–2.9 times or a decrease in diuresis < 0.5 mL/kg/h for ≥12 h.

– Stage 3 is defined as an increase in serum creatinine ≥3 times or >353.6 μmol/L, at the initiation of renal replacement therapy (in patients aged <18 years, with a decrease in glomerular filtration

For correspondence: menzorov.m.v@yandex.ru

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[1] KDIGO: Kidney Disease: Improving Global Outcomes.

rate <35 mL/min/1.73 m2), a decrease in urine output <0.3 mL/kg/h for ≥24 h, or anuria ≥ 12 h.

AKI within the renal and cardiorenal continua

According to modern concepts, AKI and CKD are mutually related and are parts of the "renal continuum" (Fig. 1).

AKI is characterized by a rapid decline in renal function that occurs within 7 days [2]. CKD is a decrease in kidney function or the presence of structural changes and markers of kidney damage for ≥3 months [6]. The term "acute kidney disease" is currently interpreted in different ways.

In the KDIGO (2012) guidelines, acute kidney disease is defined as AKI, glomerular filtration rate <60 mL/min/1.73 m^2, presence of markers of kidney damage detected within ≤3 months, a decrease in glomerular filtration rate of ≥ 35%, or an increase in the level serum creatinine >50% for ≤3 months [2].

In the concerted position of nephrologists in Europe and North America, published in 2020, it is proposed to exclude the use of AKI as a synonym for acute kidney disease, indicating AKI only as a pathology that developed within 7 days [7].

A similar approach is presented in the updated Russian guidelines for AKI (2020) that have been developed by the Scientific Society of Nephrologists of Russia; Associations of Nephrologists and Anesthesiologists-Resuscitators of Russia; and National Society of Specialists in Hemapheresis and Extracorporeal Hemocorrection. The guidelines state that an acute kidney disease is understood as a pathological condition that has not resolved in up to 7 days, AKI lasting 7–90 days, and characterized by persistent signs of kidney damage or dysfunction of varying severity [8].

Given the strong correlation between the heart and kidneys in the performance of their physiological functions, in 2008, Ronco proposed the term "cardiorenal syndrome," that was subsequently finalized by the experts of the ADQI group [9]. According to modern concepts, cardiorenal syndrome is a group of conditions, wherein cardiac dysfunction leads to the deterioration of kidney function and vice versa.

Currently, five types of syndromes are known depending on the order and rate of lesion development [10]:

– Type 1: acute cardiorenal syndrome, wherein an acute deterioration in heart function leads to AKI.

– Type 2: chronic cardiorenal syndrome, wherein chronic dysfunction of the heart causes CKD.

– Type 3: acute renocardial syndrome, wherein AKI causes damage and/or dysfunction of the heart.

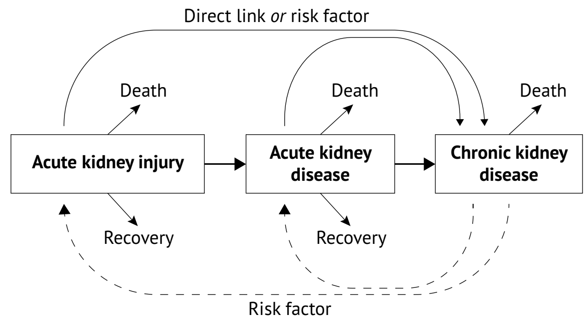

Fig. 1. Kidney continuum [5]

– Type 4: chronic renocardial syndrome, wherein CKD results in heart damage and/or dysfunction.

– Type 5: secondary cardiorenal syndrome, wherein systemic diseases/conditions lead to simultaneous damage and/or dysfunction of the kidneys and heart.

Patients with acute coronary syndrome (ACS) can have both type 1 and type 3 cardiorenal syndromes; however, in practice, it is not always possible to establish a causal relationship and the order of organ damage [11, 12].

Taking into account the commonality of risk factors; the relationship of damage to the cardiovascular system and kidneys; and their cross-negative effect on the prognosis, cardiorenal relationships are now considered a continuous chain of events, called the cardiorenal continuum [5, 13].

AKI in ACS

A recent meta-analysis that included 36 cohort studies and more than 100,000 patients showed that AKI occurred in 16% of patients with ACS [14]. According to the American NCDR Cath-PCI registry (approximately 680,000 were examined), wherein radiopaque contrast agent was injected into all patients with ACS and percutaneous coronary intervention was performed, AKI was determined in 8% [15]. According to the results of a meta-analysis by Vandenberghe et al., wherein contrast-induced nephropathy was the criterion for study exclusion, the complication was diagnosed in 15% of cases [16]. In the case of cardiogenic shock, the incidence of acute kidney dysfunction varies between 20% and 35% but can reach ≥50% [17–19].

The prevalence of AKI (10%–37%) in myocardial infarction does not depend on the presence or absence of *ST*-segment elevation on the electrocardiogram [15, 20]. Timely restoration of coronary blood flow in the infarct-associated artery is accompanied by a decrease in the incidence of acute kidney dysfunction, regardless of whether mechanical or pharmacological reperfusion is used [21]. Several studies have revealed a trend toward an increase in AKI probability when using a pharmacoinvasive strategy compared with primary percutaneous coronary intervention [22, 23]. The incidence of AKI in ACS without *ST*-segment elevation on the electrocardiogram is comparable with that following conservative therapy and percutaneous coronary intervention [24]. The prevalence of acute kidney dysfunction in unstable angina is approximately two times lower than that in myocardial infarction [15].

Single-center studies performed in the Russian Federation revealed a slightly higher incidence (19%–37%) of AKI in ACS that can be attributed to the higher prevalence of risk factors and diseases associated with AKI development [23, 25, 26].

In ACS, nonsevere stage 1 AKI predominates (2/3 of cases), and the incidence of a severe variant requiring renal replacement therapy is <3% of the total cases [16, 20, 27].

Worldwide, a decreasing trend in the prevalence of AKI in patient with ACS was noted; an exception was patients with senility and centenarians as they showed a reverse trend [27].

Risk factors and AKI-related illnesses in ACS are well understood. According to most researchers, these include age >60 years, female sex, CKD, hypertension, postinfarction cardiosclerosis, cerebrovascular diseases, and anemia [15, 28, 29]. AKI is associated with decreased left ventricular ejection fraction; high levels of troponin, glucose, leukocytes, and C-reactive protein; use of angiotensin-converting enzyme inhibitors; diuretics; and nonuse of statins. It occurs following a transfusion of erythrocyte mass for bleeding and is often accompanied by severe acute heart failure [15, 16, 29, 30].

AKI in ACS is associated with the deterioration in the immediate and long-term prognosis. This is primarily due to an increase in hospital mortality and case fatality rate [15, 16, 20, 29–32]. Even a small increase in blood serum creatinine by 0.1 mg/dL is associated with an increased risk of death [31]. Acute deterioration of kidney function is accompanied by the increased incidence of recurrent myocardial infarctions, revascularizations, and bleeding [33, 34]. AKI in patients with ACS contributes to CKD development and progression [31]. According to Nemoto et al., every sixth patient who received percutaneous coronary intervention has a continuous deterioration in renal function by >25% in the next 6–8 months [35].

The results of the randomized placebo-controlled study ELIXA demonstrated a twofold increase in creatinine levels in 1% of the patients after ACS during 108 weeks of followup [36]. According to a large American database, every third patient following AKI diagnosis develops CKD within a year [37], whereas repeated AKI episodes increase this probability [38]. In fact, kidney function deterioration changes the course of cardiovascular diseases, which is indicated in the position agreement of European cardiologists [39].

Modern role of AKI biomarkers

The current KDIGO criteria do not allow early diagnostics of AKI and has become the grounds for the search for biochemical markers by analogy using troponin in myocardial infarction. To date, several studies have tried to assess the diagnostic significance of AKI biomarkers [40, 41], and the most promising, according to experts, are neutrophil gelatinase-associated lipocalin, KIM-1 (AKI-1 molecule), cystatin C, liver fatty acid-binding protein, lysosomal enzyme N-acetyl-b-D-glucosaminidase, interleukin-18, tissue metalloproteinase inhibitor-2, and insulin-like growth factor binding protein 7 [42].

In ACS, biomarkers have demonstrated conflicting results, i.e., in some studies, they provided good early diagnostics of AKI and predicted poor outcomes [43–45], whereas in other studies, their value has not been confirmed [25, 44–46]. These dissimilarities were probably due to the differences in the pathogenesis and mechanisms of development of acute kidney dysfunction [47, 48].

The current KDIGO criteria are functional; thus, AKI diagnosed based on them is not always accompanied by an increase in the contents of biomarkers and vice versa [48]. Based on data obtained, in 2019, ADQI experts proposed in a consensus document to modify the existing KDIGO system, supplementing it with biomarkers [42]. In their opinion, AKI should be diagnosed based on standard criteria or increased levels of biomarkers or a combination of these approaches [42].

In the elaborated stratification system, authors proposed dividing each of the existing severity levels into subgroups A (no increase in biomarkers) and B (increased biomarkers), introducing an additional stage 1S, i.e., without changes in functional criteria but with increased levels of biomarkers [42].

The authors acknowledged considerable gaps in knowledge about AKI biomarkers that must be filled in future studies, including the issue of verification of their limit values [42]. The above suggestions can be presented in the current guidelines for diagnostics and treatment of AKI.

Significance of radiopaque agents in AKI development

Coronary angiography and percutaneous coronary intervention are integral components of the diagnostics and treatment of patients with ACS. Radiopaque contrast agents used in this case may lead to AKI development [2–4]. According to experts from KDIGO and the Scientific Society of Nephrology of Russia, contrast-induced AKI, an AKI variant, should be diagnosed based on standard criteria after excluding alternative causes [2–4, 6, 49, 50].

Besides radiopaque agents, other factors that may be significant in AKI development in ACS include hemodynamic and drug effects, oxidative stress, endothelial dysfunction, and electrolyte disturbances [51]. In most cases, assessing the contribution of contrast agents and distinguishing between contrast-induced AKI and AKI associated with other causes are difficult.

A large randomized trial could answer the question about the role of radiopaque agents in AKI development in ACS; however, it is not feasible for ethical reasons because the control group would be devoid of endovascular intervention. Meanwhile, some single-center retrospective studies have established that the administration of radiopaque agents did not lead to an increase in the incidence of AKI in ACS [21]. In a large meta-analysis (>140,000 participants), the incidence of AKI was identical in patients who underwent computed tomography with and without contrast [52].

Given the conflicting opinions about the role of contrast agents in AKI development, in 2020, the American College of Radiology and the National Kidney Foundation issued a joint statement on the use of radiopaque agents [53]. Experts suggested that the traditional term “contrast-induced nephropathy” should be abandoned because it is misleading about the role of the contrast agent, which is often a “companion” but not a “culprit” in AKI development. They proposed using the terms “contrast-associated AKI” or “postcontrast AKI,” that suggest a link rather than causality. The term “contrast-induced AKI” should be used when all AKI causes other than radiopaque agents have been ruled out.

Currently, with the administration of contrast agents, contrast-associated AKI develops more often, with a prevalence of <5%, initial glomerular filtration rate of >60 mL/min/1.73 m^2, and no more than 30% with glomerular filtration rate <30 mL/min/1.73 m^2. The incidence of contrast-induced AKI at a baseline glomerular filtration rate of >30 mL/min/1.73 m^2 was lower than previously thought of <2% [53].

AKI prevention and treatment

According to the current recommendations of KDIGO and the Scientific Society of Nephrologists of Russia on AKI prevention, modifiable risk factors (such as arterial hypotension; iatrogenic hypoperfusion of the kidneys; an uncontrolled low-sodium diet accompanied by hypovolemia and intake of diuretics against it; and use of angiotensin-converting enzyme, angiotensin II receptor antagonists; and non-steroidal antiinflammatory drugs), nonmodifiable risk factors (age > 65 years, male sex, and Negroid race), and associated clinical diseases/conditions (CKD; diabetes mellitus; anemia; arterial hypertension; clinically significant heart failure; and bilateral renal artery stenosis) must be identified promptly and eliminated to the extent possible [2, 4].

Russian experts have proposed a risk stratification system for AKI development based on the following parameters [4]:

– A low risk is verified in the absence of risk factors and associated clinical conditions.

– A moderate risk is detected if 1 or 2 risk factors (or associated clinical conditions) or CKD stages 1–2 are identified.

– A moderate–high risk is verified in the presence of three risk factors (or associated clinical conditions) or CKD stages 3–5, or in the combination of one or two risk factors (or associated clinical conditions) with CKD stages 1–2.

– A high risk is detected if one or two risk factors (or associated clinical conditions) are simultaneously present and CKD stages 3–5, or a combination of three risk factors (or associated clinical conditions) with CKD stages 1–2.

– A very high risk is determined when three risk factors (or associated clinical conditions) are combined with CKD stages 3–5.

Patient management should be based on the identified risk and AKI stage [2]. According to the KDIGO (2012) recommendations, the management of patients at high risk and in any AKI stage should include the following:

– Withdrawal of all nephrotoxic drugs.

– Provision of volumetric status, perfusion pressure, and hemodynamic monitoring.

– Control of serum creatinine, urine volume, and glycemia.

– Use, if the clinical situation allows, of diagnostic approaches alternative to radiopaque methods.

In stage 1 AKI, invasive testing should be minimized. In stage 2, additional dose adjustment of drugs is required. In Russian recommendations, special attention is paid to the temporary withdrawal of angiotensin-converting enzyme inhibitors and angiotensin II receptor antagonists with the transition to other drug groups at high risk of AKI development [4]. Unlike AKI stages 1 and 2, stage 3 or the presence of extrarenal complications often requires renal replacement therapy. The indications are determined based on an analysis of the clinical situation and dynamics of laboratory data [2].

When predicting contrast-induced AKI, patient-related risk factors (age > 75 years, CKD, AKI history, diabetes mellitus, hypovolemia, dehydration, shock of various etiologies, hypotension, severe heart failure, low left ventricular ejection fraction, myocardial infarction up to a day, multiple myeloma, anemia, and use of nephrotoxic drugs) should be considered, as well as factors associated with endovascular procedures (large volume of surgery, intra-arterial administration of radiopaque agents, their repeated use within 24 h, use of high-osmolar drugs, and complications from previous use of radiopaque agents) [49].

For a quick analysis of the probability of developing contrast-induced AKI in percutaneous coronary intervention, risk assessment models have been developed and validated. The most studied and recommended of these is the scale by Mehran et al. [54].

Hydration with an isotonic solution of sodium chloride or sodium bicarbonate is the most reasonable in the prevention of contrast-induced AKI [2, 4, 49, 53]. Experts from the Scientific Society of Nephrologists of Russia propose the intravenous administration regimen with 3 mL/kg for 1 h before the administration of a radiopaque agent and at a rate of 1 mL/kg/h for 6 h after its use [4].

The joint statement of the American College of Radiology and the National Kidney Foundation on the use of radiopaque contrast agents recommends intravenous prophylactic hydration for all patients with glomerular filtration rate <30 mL/min/1.73 m^2 and may be considered when the glomerular filtration rate decreases to <45 mL/min/1.73 m^2. Isotonic sodium chloride solution should be administered at a rate of 1–3 mL/kg/h 1 h before or within 3–12 h after the use of radiopaque agents, and in the case of clinically significant heart failure, the infusion rate must be decreased to 0.5 mL/kg/h [53].

Attitudes toward the role of HMG-CoA reductase inhibitors[1] (statins) in the prevention of contrast-induced AKI are currently ambiguous. According to European experts, as set out in the guidelines for myocardial revascularization (2018), highdose statins may be useful in this situation [55]. Experts from KDIGO and the Russian Scientific Society of Nephrology believe in the insufficiency of evidence for the inclusion of statins in the recommendations for the prevention of contrast-induced AKI [2, 4].

Undoubtedly, AKI should be detected promptly to implement measures for its prevention and treatment. Meanwhile, even in developed countries, AKI is not diagnosed in 20% of patients. In 50% of cases, it is diagnosed late [56].

[2]HMG-CoA reductase — 3-hydroxy-3-methylglutaryl-coenzyme A reductase

Conclusion

AKI is a common complication of ACS that adversely affects the prognosis because of an increased risk of ischemic events, bleeding, CKD onset and/or progression, and increased case fatality rate and mortality. Despite the established recommendations, including Russian ones, AKI is not detected promptly in many patients. The current criteria based on the dynamics of creatinine and diuresis do not allow early AKI diagnosis. The use of biochemical markers to improve it has not yet been mentioned in the recommendations because of significant gaps in knowledge and the lack of limit values that have been validated. The previously assumed leading role of radiopaque agents in AKI development in patients with ACS has now been revised downward.

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Author detalis

Maksim V. Menzorov, M.D., D. Sci. (Med.), Prof., Depart. of Therapy and Occupational Diseases, Ulyanovsk State University, Ulyanovsk, Russia; menzorov.m.v@yandex.ru; ORCID: http://orcid.org/0000-0002-6000-4850

Alexander M. Shutov, M.D., D. Sci. (Med.), Prof., Head of Depart. of Therapy and Occupational Diseases, Ulyanovsk State University, Ulyanovsk, Russia; amshu@mail.ru; ORCID: http://orcid.org/0000-0002-1213-8600

Vladimir S. Sakharov, PhD Stud., Depart. of Therapy and Occupational Diseases, Ulyanovsk State University, Ulyanovsk, Russia; ar74pq@gmail.com; ORCID: http://orcid.org/0000-0001-6993-5830

Vera N. Kabanova, PhD Stud., Depart. of Therapy and Occupational Diseases, Ulyanovsk State University, Ulyanovsk, Russia; kabanona2016@gmail.com; ORCID: http://orcid.org/0000-0003-0203-2535