

Modern Approaches to The Diagnosis And Evaluation Of Acute Kidney Injury In Obstetric Pathologies

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Abstract – Prerenal AKI is caused by renal hypo perfusion. With the timely restoration of normal renal perfusion, rapid normalization of renal function is observed. The causes of perennial AKI may be due to a decrease in effective arterial volume: myocardial, alular, pericardial, rhythm and conduction disorders; pulmonary hypertension, pulmonary thromboembolism, mechanical ventilation; systemic vasodilatation; sepsis, liver failure, anaphylaxis; renal vasoconstriction: nor epinephrine, ergotamine, liver diseases; under the influence of pharmacological drugs: angiotensin converting enzyme inhibitors (ACE inhibitors) or angiotensin II receptor blockers, no steroidal anti-inflammatory drugs (NSAIDs). The development of AKI can be observed with general hypervolemia, but with a decrease in arterial blood volume, which is observed in chronic heart failure, nephritic syndrome, cirrhosis of the liver and sepsis.

Keywords – Cardiac Surgery, Pregnant Women, Nephrotoxic Drugs, Therapeutic Technologies.

Renal damage to the kidneys includes glomerular, interstitial, and tubular damage. In addition, there are two additional variants of AKI — vascular renal damage and caused by intratubular obstruction. Acute tubular necrosis (OTN) is the most common cause of renal AKI, accounting for 70%. There is ischemic and toxic REL. The share of ischemic REL in the structure of causes is 50-60%, and in 20-45% of cases it is caused by sepsis [1]. Toxic tubulonecrosis accounts for 20% of AKI cases.

Ischemic tubular necrosis: Ischemic kidney injury and perennial AKI represent two stages of the pathological process. In severe hypo perfusion, the tubular cells are damaged, and renal dysfunction persists. Risk factors for the development of ischemic REL include the presence of previous kidney disease — CKD, atherosclerosis, diabetes mellitus; cardiac surgery. Ischemic OTN can develop in the absence of hypotension in cases of impaired renal auto regulation: in the elderly, with severe atherosclerosis, arterial hypertension and endovascular lesions, or in the presence of previous CKD.

Nephrotoxic OTN develops under the influence of endogenous toxins (hemo-and myoglobulinuria in massive hemolytic causes prevail or rhabdomyolysis, respectively) or exogenous toxins. The spectrum of exogenous agents has changed significantly in recent years, with antimicrobial agents, radio contrast agents, and chemotherapeutic agents prevailing.

The main causes of the development of renal AKI: damage to large renal vessels: thrombosis, atheroembolism, thromboembolism, dissection, vacuities (Takayasu's disease); classic nodular polyarteritis; thrombosis, compression; glomerular apparatus damage: primary glomerulonephritis (GN), GN in systemic diseases and vacuities (systemic lupus erythematosus, Wegener's granule-matosis, microscopic polyangiitis, Charge-Strauss syndrome, hemorrhagic vasculitis—Schonlein — Henoch

purpura, cryo—globulinemic vacuities); GN in infectious endocarditis; malignant hypertension; gestosis of pregnant women; scleroderma kidney; hypercalcemia; drugs; radiocontrast substances; hematological: hemolytic-uremic syndrome (thrombotic thrombocytopenic purpura), disseminated intravascular coagulation, high viscosity syndrome.

Pathological conditions characterized by a predominant lesion of the tubules (often with the development of OTN): ischemia caused by renal hypo perfusion; exogenous toxins: antibiotics, antitumor drugs, radiocontrast agents, NSAIDs, diuretics, *α*-methyldopa, allopurinol, azathioprine, etc.; endogenous toxins (myoglobin, hemoglobin, uric acid, light chain myeloma). Acute lesions of the interstitial apparatus: interstitial nephritis (antibiotics, NSAIDs, etc.); infections (viruses, bacteria, fungi); acute cellular reaction of kidney transplant rejection; infiltrative processes (lymphomas, leukemia, sarcoidosis).

Post renal AKI is the result of urinary system obstruction (MVD), which can occur at the level of the bladder or urethra (obstruction of the lower MVD) or at the level of the ureters and kidneys (obstruction of the upper MVD). In unilateral obstruction, AKI syndrome usually does not develop with preserved contralateral kidney function.

The main reasons for the development of post renal AKI: obstruction of the upper parts of the MVS (bilateral obstruction or obstruction of a single kidney, papillary necrosis, blood clots, germinating kidney carcinoma, retroperitoneal tumor, retroperitoneal fibrosis, endometriosis); lower parts of the MVS (neurogenic bladder, bladder carcinoma, blood clots, concretions); prostate (prostate cancer, adenoma prostate gland); urethra (strictures, pharoses, concretions).

Post renal AKI can occur with both complete and partial obstruction. In the first case, anuria is observed, in the second — distal phenomena (frequent urination, false urges, nocturnal, a feeling of incomplete emptying of the bladder). Partial obstruction may occur with or without oliguria.

The clinical picture of AKI is non-specific, depending on the etiology and the underlying disease that caused it. Often, latent development under the guise of the underlying disease, so early diagnosis is possible only if diuresis, creatinine and urea levels in the blood plasma are monitored.

The algorithm for diagnosing AKI is as follows:

1. Diagnosis of AKI based on the increase in blood creatinine level to 258.8 μmol/l during the acceptable period for AKI (3 months) and a decrease in diuresis.
2. Elimination of CKD (normal kidney functions 3 months ago — blood creatinine level 75.5 μmol/L).
3. Clarification of the mechanism of AKI: renal damage (absence in the anamnesis and during the follow-up examination of the prerenal and postrenal causes of AKI, taking potentially nephrotoxic drugs-NSAIDs).
4. Identification of the cause of renal AKI (presumably NSAIDs).
5. Determination of severity (stage) AKI in accordance with the clinical recommendations for the diagnosis and treatment of AKI (2014) — the 3rd degree of severity (increased blood creatinine more than 3 times from the original).

Diagnosis: Acute renal failure (AKI), drug-induced (NSAID), 3rd degree of severity, uncomplicated.

In accordance with the recommendations of KDIGO (2012): "Patients with AKI should be monitored for 3 months to assess the degree of recovery of kidney function, a repeat episode of AKI, or deterioration of the course of previously existing CKD" [5]. At the same time, the level of creatinine and the volume of urine are monitored. Patients are recommended to be divided into groups according to the degree of risk of developing AKI. Patients should be examined to identify the reversible causes of AKI in order to eliminate them (for example, postrenal).

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The incidence of AKI in the general population is continuously increasing and reaches 0.25%, which is comparable to the incidence of acute myocardial infarction. AKI remains an important cause of both end-stage renal failure and the less severe stages of chronic kidney disease. In addition, AKI (in the near or long term) may be considered an important determinant of cardiovascular

risk. Despite the continuous improvement of therapeutic technologies, primarily methods of renal replacement therapy, there is no significant improvement in the results of treatment of AKI. The outcomes of severe variants of AKI remain unsatisfactory, and the mortality rate in them can reach 70% or more. At the same time, even a short-term, transient increase in Scr is associated with an increase in the duration of hospitalization, as well as with an increase in mortality over time. AKI therapy requires huge material costs, which place a heavy burden on all health care funding systems. All this makes us consider AKI as one of the most important medical and social problems.

As noted above, AKI is a relatively new concept, which has largely replaced the usual phrase AKI. To date, some doctors believe that such a replacement is purely terminological in nature. However, the reasons for the development of the concept of AKI are much deeper and are determined by the logic of the development of modern medicine. The main reason for the creation of this concept was the accumulation of information that even a slight transient increase in the concentration of creatinine in the blood serum (Run) is associated with a sharp increase in mortality. Such an increase in mortality is observed both in the early and long-term period. At the same time, the fatal outcome is not always determined by "renal" causes.

In nephrology, as in other areas of medicine, there are a lot of conditional terms that do not reflect the essence of the pathological processes that they characterize. In the vast majority of cases, these terms have historical roots and it is not possible to abandon them. However, when it comes to introducing new terms, especially those borrowed from foreign literature, to translate them into Russian, it is necessary not only to know the grammar of a foreign language, but also to understand the meaning of the term, since literal translation is in most cases impossible.

When using the RIFLE system, nephrologists encountered a number of problems. As mentioned above, even minimal changes in Scr (less than 44 $\mu\text{mol} / \text{L}$) have been shown to be associated with increased mortality among hospitalized patients. In addition, there were certain difficulties in assessing the severity of kidney damage at a particular time in a particular patient, for example, with an increase in the concentration of serum creatinine in a patient, the stages of AKI from R to F could be consistently detected. Finally, we emphasize a very important point from our point of view. As the developers noted, "the criteria of the OPN [RIFLE-criteria, auth.] can be applied to all forms of AKI in patients in critical conditions, with the exception of primary kidney diseases, such as glomerulonephritis».

Moreover, it requires the doctor to pay attention to the patient's kidney function and, especially, to be wary of possible changes in their condition after any effects, including medical ones. Unfortunately, as many nephrologists know, some of our colleagues do not pay enough attention even to the size of the minute diuresis, and we sometimes get patients in extremely serious condition, although timely adoption of fairly simple measures could prevent such cases. Therefore, the KDIGO classification is also of great preventive value. Therefore, the concept of AKI and the stratification of its severity occupy a place in medicine close to the concept of CKD. Although it is unfortunate that many of the provisions of the KDIGO Recommendations on AKI do look unnecessarily formalized.

It also seems that the available methods of treating AKI, especially RRT, used for AKI, are approaching their limit and breakthroughs in this direction are not expected. Therefore, at present, early detection, primary and secondary prevention of AKI is becoming the main direction. This is possible only on the basis of common approaches to the definition and stratification of the severity of this condition.

Treatment of patients with AKI is very expensive and puts a heavy burden on society. It is important that AKI can cause CKD, and in patients who have had AKI, the cardiovascular risks increase dramatically.

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