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Evaluation of Kidney Function in Chronic Kidney Disease

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Abstract: Chronic non-communicable diseases associated with high treatment costs, disability, severe complications and mortality. One of the main ones among them are kidney diseases. The problem of chronic kidney disease (CKD) is socially significant, which has a high prevalence, mortality, and at the terminal stage requires high-cost methods of substitution therapy - dialysis, kidney transplantation.

Dudko M.Yu., Kotenko O.N. [7] conducted a study to assess the prevalence of CKD among the adult population of Moscow. The authors found a high prevalence of CKD among the population of this city. At the same time, the analysis of the prevalence of CKD by stages showed a high proportion of early stages of CKD with significantly lower detectability of late stages.

In the Kyrgyz Republic in 2018, a total of 172.9 cases of acute and chronic glomerulonephritis (GLN) per 100 thousand population were registered, 52.9 cases per 100 thousand population were detected for the first time. The authors came to the conclusion that timely diagnosis, comprehensive support of patients suffering from GLN, can significantly improve the prognosis of the outcome of these diseases [2].

The prevalence of CKD was 18.7-21.9 cases per 100 thousand population. The high prevalence of CKD in Tajikistan dictates the revision of some tasks of nephrological care [1]. Vyalkova A.A. [5] provides the results of the analysis of large-scale studies (ACCOMPLISH, ADVANCE, ALTITUDE, CARRESS-HF, ONTARGET, ROADMAP), where it was confirmed that CKD has a high prevalence (10-15%) and occupies one of the leading places in the overall structure of mortality and morbidity of the population. In these studies, CKD is defined as a supranosological concept characterized by structural damage to the kidneys and/or a decrease in their function for 3 or more months, regardless of the nosological diagnosis of chronic renal disease.

Clinical, laboratory and instrumental examination: increased albuminuria / proteinuria, erythrocyturia, cylindruria, leukocyturia, changes in the electrolyte composition of blood and urine, kidney abnormalities, cysts, hydronephrosis, changes in the size and shape of the kidneys and others. CKD is established even in the absence of any markers of renal damage, but a decrease in glomerular filtration rate (GFR) of less than 60 ml/min/1.73 m2 was detected, which also persists for 3 months or more [8; 22].

Renal function is a GFR level below 90 ml/min/1.73 m2. Informative and accessible methods are used to assess kidney function by GFR in adults: the Cockcroft-Gault formula, which reflects GFR at the initial stages of CKD, and the MDRD formula, which more accurately reflects the function at the 3-5 stages of CKD. Another method of calculating GFR for CKD is presented according to CKD-ERI [19; 31].

Formulas for calculating GFR according to Schwartz: GFR = k x body length (cm) x 80 / plasma creatinine (mmol/l), where k = 0.55 (for children from 2 to 12 years), k = 0.55 (for girls from 13 to 18 years), k = 0.77 (for boys from 13 to 18 years old). Barrat GFR calculation formula: for young children: GFR = 0.55 x body length (cm) / plasma creatinine (mg%); for older children: GFR = 0.45 x body length (cm) / plasma creatinine (mg%) [5]. Diagnosis of CKD to determine the prognosis and rate of its progression against the background of therapy, great importance is attached to the assessment of albuminuria / proteinuria. The previous gradations of albuminuria were left at the London conference of KDIGO in 2009: < 30; 30-299; > 300 mg of albumin/g of creatinine in urine. To assess urinary albumin excretion, instead of normal, micro, macroalbuminuria/proteinuria, the following are proposed: "optimal" level (<10 mg/g), "high normal" (10-29 mg/g), "high" (30-299 mg/g), "very high" (300-1999 mg/g) and "nephrotic" (>2000 mg/g) and their use for predicting the risk of general and cardiovascular mortality. In 2007, in the ICD-10, the term "chronic renal failure" was replaced by "CKD" (code N18) and corresponding codes were assigned to each of its stages: C1 - N18.1; C2 - N18.2; C3a and C3b - N18.3; C4 - N18.4; C5 - N18.5. To indicate the etiology of CKD, in addition to these codes, appropriate disease codes should be used; code N18.9 indicates cases of CKD with an unspecified stage [18].

Renal disorders detected by imaging and/or morphological studies; renal functional disorders detected by changes in renal functional parameters in urine and/or blood tests. To determine the stage of CKD, the evaluation of the tubule function and GFR is carried out with the calculation according to Schwartz [5]. The concept of 5-stage CKD was formulated by experts of the National Kidney Foundation (National Kidney Foundation/ Kidney Disease Outcomes Quality Initiative) USA in 2002 and received recognition from the world medical community [27]. In 2012, improved recommendations for the diagnosis, classification, and treatment of CKD were published depending on the level of GFR and albuminuria [26].

The fundamental difference between the concept of CKD and CRF is the inclusion in this category of patients, including those with a normal functional state of the kidneys. Two main reasons gave rise to the concept of CKD: the presence of common mechanisms of progression of nephropathy and high cardiovascular morbidity and mortality in patients with CKD. In his opinion, for the first time in the world, a generally recognized classification has appeared that allows us to look at a patient with CKD from a single position [23].

As a result of research by English authors Wall N.A. et al. [30] it was found that significant renal dysfunction (eGFR <60 ml/min/1.73 m2, CKD stage G3-5) was detected in 6% of the UK population, and its prevalence increases markedly with age, affecting more than 30% of people aged 75 years and older. According to the authors, a decrease in eGFR or an increase in albuminuria is associated with mortality regardless of all causes and progression to severe CKD - end-stage renal failure (ESRD), the annual mortality from which exceeds 10%.

The results of large population studies (HOPE, PREVENT, LIFE) revealed a direct link between a decrease in GFR, albuminuria and cardiovascular morbidity (CVD). About 40-80% of patients with CKD at the predialysis stage had CVD. Death from CVD in dialysis patients occurred in 40-50% of cases, which is 10-20 times higher than in the general population. As the stages of CKD progressed, the severity and frequency of cardiovascular disorders increased [11; 26].

According to Weber B.R. et al. [29] hemodynamically significant renal artery stenosis, referred to by the term "ischemic kidney disease", is one of the leading causes of irreversible deterioration of kidney function in the elderly and often remains unrecognized until ESRD. It can be accelerated in these patients by frequently used elderly medications and diagnostic interventions.

It is clear that ischemic kidney disease is inherent mainly in patients with widespread atherosclerosis. In ischemic kidney disease, it is reasonable to eliminate iatrogenic factors aggravating renal dysfunction - nonsteroidal anti-inflammatory drugs, loop diuretics in large doses causing relative hypovolemia, but primarily ACE inhibitors and angiotensin II receptor blockers. They are often prescribed without proper control to elderly patients with arterial hypertension [25].

CKD, characterized by a persistent decrease in GFR and, therefore, associated with the greatest deterioration in the long-term prognosis, are more common among the elderly [20].

Treatment of an elderly CKD patient is associated with significant difficulties. They are the primary group that should avoid polypragmasia. Nutritional status disorders remain a particular problem for them, often occurring in this age group and with preserved kidney function, but always increasing significantly with the progression of CKD. To denote the mal-nutrition syndrome, the term "protein-energy deficiency" is also used for elderly patients with CKD. Currently, successful experience has been accumulated in the use of amino acid ketoanalogs for the prevention and correction of protein-energy deficiency in patients with CKD. In the economic analysis of the Italian researcher Scalone L. et al. (2010), it was proved that the use of a low-protein diet with the appointment of ketoanalogs of amino acids makes it possible to safely postpone the start of dialysis for an average of 1 year and leads to savings of 30 thousand euros per elderly patient with CKD for 3 years [21].

According to the results of her research, M.A. Daminova [6] concludes that the definition of CKD and classification by stages in children at the moment do not differ from those in adults. Currently, it is known that the development of CKD in children is promoted by genetic, endogenous, demographic (gender, age) and a complex of exogenous factors. Due to the fact that in childhood it is possible to reverse the development of chronic kidney damage and restore organ function, early detection, timely treatment is an important prerequisite for preventing or distancing from its unfavorable outcome.

The same author [6] in his other work emphasizes that in nephrology there are 4 groups of risk factors that affect the development and course of CKD in children: factors affecting the development of CKD: risk factors that initiate CKD: risk factors that lead to the progression of CKD: risk factors of the terminal stage of CKD.

Each individual responds to the action of certain risk factors by changing the functional state of his body. The development of a non-specific adaptive reaction with the formation of certain prenosological conditions is the same for all. The peculiarity of the concept of risk factors in prenosological diagnostics is that the intensity of any risk factor can be investigated in relation to various functional states. Risk factors for the development of maladaptation are also risk factors for diseases. Diseases arise through the transition of prenosological states to premorbid, and then to nosological. The relationship between risk factors and disease is carried out through functional states, reflecting stresses and overstrain of regulatory mechanisms with subsequent disruption of homeostasis and compensation. At the current level of medical development, it is possible to identify factors that can provoke the development of diseases of the urinary system [13].

Seema Sharma et al. [28] studies were conducted to identify the risk factors causing CKD and assess the impact of counseling on the quality of life of 52 patients with CKD. A validated QOL questionnaire was conducted. The authors found that arterial hypertension was the cause of CKD in 48.1% of patients, diabetes in 7.6%, hypertension together with diabetes in 32.7%, glomerulonephritis in 5.8%, immunological causes in 5.8% of patients. There was a statistically significant improvement in social functioning, emotional, mental and self-assessment parameters, which indicated the influence of counseling. The authors concluded that consulting a clinical pharmacist helps in improving the quality of life in the studied patients.

Evaluation of the relationship of serum concentrations of angiopoietin-like proteins of types 3 and 4 (APPB 3 and 4) with the development of renal dysfunction in 158 patients aged 21-80 years with rheumatoid arthritis on the background of metabolic changes with a disease duration of 9 years. Negative correlations of average strength between the calculated GFR according to the CKD-EPI formula and the level of APPB3 and APPB4 were revealed. It was found that the content of APPB4 in patients with rheumatoid arthritis is directly influenced by two factors - renal dysfunction and the presence of metabolic syndrome [3].

Another study [11] proved that impaired renal function is strongly interrelated with the prognosis of heart failure in patients with systolic dysfunction of the left ventricle, unfavorable course of coronary artery disease, arterial hypertension. The close relationship between changes in the kidneys and

organs of the cardiovascular system led to the conclusion that there is a clinical and pathogenetic community - the "cardiorenal continuum".

Sibireva O.F. et al. [17] patented an invention, the essence of which is that the prognosis of the nature of the progressive course of CKD is determined using coagulation characteristics of blood: activated partial thromboplastin time (APTT), prothrombin time (PV), thrombin time (TV), level of antithrombin III (ATIII), Willebrand factor (PV), endothelin-1 (E-1), D-dimer. With ACTV values ranging from 28 to 35 seconds, PV from 12 to 15 seconds, TV from 9 to 13 seconds, ATIII level from 29 to 70%, PV level from 105 to 115%, E-1 no more than 0.32 fmol/ml and D-dimer from 245 to 520 ng FEI/ ml predict a slow progression of the course of CKD. With the same values of APTT, PV, TV, ATIII level, and an EF level of more than 115%, E-1 more than 0.32 fmol/ml, D-dimer more than 620 ng FEI/ml, accelerated progression of CKD is predicted. The use of the claimed method makes it possible to increase the accuracy and reliability of the diagnosis of CKD.

In another study, the author [12] studied the diagnostic and prognostic value of the biomarkers of kidney damage NGAL (lipocalin associated with neutrophil gelatinase), KIM-1 (kidney damage molecule-1) and L-FABP (hepatic form of fatty acid binding protein) in patients with CKD. The most studied of them is NGAL, the increase in the content of which in the urine reflects the severity of the course of CKD. The increased level of NGAL urine is also considered as a prognostic criterion that allows identifying patients with a high risk of an unfavorable course of the disease. The increased level of KIM-1 urine in patients with CHF makes it possible to identify individuals with tubulointerstitial kidney damage that has an unfavorable prognostic value, and assess their risks of overall mortality and re-hospitalization for CHF.

According to Brendan Nichols et al. [24] risk variants of apolipoprotein L1 (APOL1) increase the risk of kidney disease in African Americans. It was found that patients developed collapsing focal segmental glomerulosclerosis while taking interferon, which carried the high-risk APOL1 genotype. This discovery increased the likelihood that interferons and pattern molecule recognition receptors that stimulate interferon production may contribute to APOL1-associated kidney diseases. Overexpression of APOL1 risk variants is more harmful to cells than overexpression of wild-type APOL1 protein. It has been proven that protivoviral pathways can be important inducers of kidney disease in people with the high-risk APOL1 genotype, and identifies potential targets for treatment.

Thus, foreign researchers have shown the importance of the problem of CKD for different segments of the world's population. A classification is proposed, criteria and markers for the diagnosis of CKD are defined, and the effectiveness of some tests for the diagnosis of this supranozologic condition is shown. In the next part of our review, we found it necessary to provide an analysis of scientific sources of domestic researchers on the problem of CKD in humans.

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