

## CHRONIC KIDNEY DISEASE AND ITS MODERN LABORATORY DIAGNOSIS

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**Abstract:**The article discusses modern approaches to the diagnosis of chronic kidney disease. Attention is focused on the determination of cystatin C for the purpose of early diagnosis of this pathology.

**Keywords:**chronic kidney disease, creatinine, cystatin C, glomerular filtration rate.

**INTRODUCTION:**Chronic non-communicable diseases are of medical and socio-economic importance due to an increase in population mortality, reduction or loss of working capacity and deterioration in the quality of life.

Kidney diseases occupy an important place in the structure of chronic non-communicable diseases due to their high prevalence in the population, which is confirmed by numerous large-scale international studies. Chronic kidney disease (CKD) is comparable in prevalence to such socially significant diseases as coronary heart disease, hypertension and diabetes mellitus [1].

**MATERIALS AND METHODS:**Chronic kidney disease (CKD) is a supra-nosological concept that unites all patients with signs of kidney damage and/or decreased function that persist for 3 or more months.

The causative factor for CKD can be many diseases of the urinary and other body systems that occur with kidney damage:

- diseases occurring with predominantly damage to the glomeruli (chronic glomerulonephritis), tubules and interstitium (chronic pyelonephritis, interstitial nephritis);
- obstructive nephropathies: urolithiasis, hydronephrosis, tumors of the genitourinary system;
- congenital kidney diseases (polycystic kidney disease, renal hypoplasia, Fanconi syndrome, Alport syndrome, diffuse mesangial nephrosclerosis, nail-patella syndrome and other congenital skeletal diseases combined with nephropathy);
- rheumatic diseases (SLE, systemic scleroderma, periarteritis nodosa, Wegener's granulomatosis, hemorrhagic vasculitis), occurring with kidney damage;
- metabolic diseases (diabetes mellitus, amyloidosis, gout, cystinosis, hyperoxaluria);
- primary vascular lesions: malignant hypertension, renal artery stenosis, hypertension (essential hypertension).

**RESULTS AND DISCUSSION:**If the GFR is preserved or increased, or if it is 60 GFR<90 ml/min/1.73 m<sup>2</sup>, the diagnosis of CKD can be made if there are signs of kidney damage:

When GFR <60 ml/min/1.73 m<sup>2</sup>, CKD is diagnosed in the absence of markers of kidney damage.

**Table 1. Classification of CKD by GFR level**

Designation	Characteristics of kidney function	GFR level (ml/min/1.73 m <sup>2</sup> )
c1	High and optimal	>90*
c2	Slightly reduced	60–89*
c3a	Moderately reduced	45–59

c36	significantly reduced	30–44
c4	Sharply reduced	15–29
c5	End-stage renal failure (dialysis or kidney transplant)	<15

\* in the absence of signs of kidney damage, eGFR categories c1 or c2 do not meet the CKD criteria

The “gold standard” for assessing renal function is determining GFR by measuring the clearance of exogenous substances by glomerular filtration: inulin, drugs [ $^{51}\text{Cr}$ ] - EDTA (ethylenediamine tetraacetic acid) [ $^{99\text{m}}\text{Tc}$ ] - DTPA (diethylenetriamine pentaacetic acid), [ $^{125}\text{I}$ ] - iothalamate. These substances are injected into the blood and allow the function of each kidney to be assessed. However, technical complexity, the need to introduce a foreign substance into the blood, and high cost limit their use. In modern nephrology, the filtration capacity of the kidneys is usually assessed by the level of creatinine concentration in the serum or by using formulas that are based on calculating the concentration of creatinine (in clinical practice, the CKD-EPI formula is most widely used for calculating GFR) [2].

However, it is known that creatinine is not a specific marker of kidney damage. Its level varies due to age and gender, the level of metabolism in muscle tissue. It should be noted that the kidneys have a large functional reserve and in the initial stages of kidney damage the level of this marker does not change. Also, with a decrease in glomerular filtration, there is a compensatory increase in the secretion of creatinine by the proximal tubules, which indicates the presence of a “blind spot” of creatinine in the early stages of CKD. Changes in creatinine are inertial, therefore, in acute conditions (acute renal failure), creatinine does not reflect renal function accurately enough until some time has passed after the onset of the condition [3].

There are also non-renal factors that affect serum creatinine concentrations. These are ethnicity, the presence of chronic diseases, and consumption of meat. In addition, some drugs (for example, trimethoprim, cimetidine) inhibit creatinine secretion, but do not change GFR.

**CONCLUSION:** It should be noted that the calculation of the GFR level based on cystatin C is more accurate and more consistent with the GFR value determined according to the “gold standard” [4].

It must be emphasized that cystatin C is a marker not only for disorders of renal functions. Elevated levels of cystatin C are associated with the risk of cardiovascular events (myocardial infarction), cardiac remodeling, and help predict the onset of microalbuminuria in patients with essential hypertension [5].

Timely diagnosis and timely treatment of CKD is an important factor in preventing complications caused by impaired renal function, which allows reducing the costs of renal replacement therapy, as well as reducing overall mortality and increasing life expectancy of the population. Even a slight decrease in kidney function can be associated with serious pathophysiological consequences for the body and for health in general.

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