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A Modern Approach to the Correction of Lipid Metabolism in Kidney Disease

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Annotation. Chronic kidney disease — a proven risk factor of the development and progression of lipid metabolism disorders. The basis of these disorders — an increase in blood plasma cholesterol, triglycerides, low density lipoproteins and decreased levels of high density lipoproteins, apo AI and apo AII. There has been a decrease in the activity of enzymes: lipoprotein lipase, hepatic triglyceride lipase, lecithin-cholesterol acyltransferase. The use of lipid modifying drugs — statins, fibrates, nicotinic acid was proposed.

Key words: chronic kidney disease, cholesterol, triglycerides, low density lipoproteins, high density lipoproteins, apo AI, apo AII, lipoprotein lipase, hepatic triglyceride lipase, statins, fibrates, nicotinic acid.

Introduction

Chronic kidney disease (CKD) is a violation of homeostasis caused by an irreversible decrease in the mass of active kidney nephrons, which occurs with all progressive kidney diseases and is manifested by a multi-symptom complex that reflects the participation of almost all organs and systems of the patient in this process. CKD is defined as kidney damage or decreased function for 3 months or more, regardless of diagnosis. Such a time limit (criterion of "persistence") was chosen as a time parameter for determining CKD because, in these terms, acute variants of the development of kidney dysfunction, as a rule, end in recovery. Chronic kidney disease is an important medical and social problem of our time, the prevalence of which reaches 5–11% in the general population [1, 2]. A number of factors have a significant impact on the development and progression of chronic kidney dysfunctions in a particular population, including: an increase in the age of the population, the incidence of certain infections, alcohol and smoking, the state of the environment, climate, dietary habits, genetic characteristics of the population, etc. About 40% of adults have an increased risk of developing CKD, among which a significant number of patients with arterial hypertension, metabolic syndrome and diabetes mellitus, which leads to a sharp decrease in the quality of life, high mortality, as well as the need for expensive replacement therapy in the terminal stage - dialysis and kidney transplant. The rapidly increasing number of patients with endstage renal disease (ESRD) requires a constant increase in the cost of dialysis and kidney transplantation. Despite the fact that only a small proportion of patients with CKD require renal replacement therapy (RRT), the cost of RRT is significant and becomes burdensome even for countries with highly developed economies. CKD is a generic term and an independent diagnosis. In addition to the variety of etiological factors characteristic of CKD, most chronic kidney diseases have a single mechanism of progression, and morphological changes in

the kidneys in renal failure are of the same type. Ultimately, they come down to the predominance of fibroplastic processes with the replacement of functioning nephrons with connective tissue and wrinkling of the kidneys, which leads to the death of nephrons. Therefore, CKD is now a global public concern. Modern international recommendations suggest classifying CKD into five stages, taking into account the glomerular filtration rate (GFR) [3], since GFR has an independent diagnostic and prognostic value. In addition, the new guidelines suggest dividing stage 3 CKD into stages 3a and 3b because renal prognosis is not the same in groups of people with stage 3 CKD with GFR from 59 to 45 ml/min/1.73 m2 and from 44 to 30 ml/min/1.73 m2. GFR at the level of 90 ml/min is taken as the lower limit of normal. GFR value < 60 ml/min was chosen due to the corresponding death of more than 50% of nephrons. Lipid metabolism disorders in CKD In patients diagnosed with CKD, one of the risk factors for this disease is the development and progression of lipid metabolism disorders [4–6]. According to numerous clinical studies, hyperlipidemia ranks first among metabolic disorders in CKD [7]. The assumption about the relationship between lipid accumulation and kidney disease was first made in 1860 by Rudolf Virchow [8], who in his lectures at the Institute of Pathology in Berlin noted "fatty degeneration of the renal epithelium as a stage of Bright's disease" (the historical designation of glomerulonephritis, described in the 19th century by the British scientist Richard Bright, one of the founding fathers of nephrology) [9]. In 1982, an article by J. Moorhead et al. was first published in the Lancet. [10], in which the authors proposed the hypothesis of lipid nephrotoxicity, which served as an incentive for further research on lipids in kidney disease. This was the first publication to introduce the concept that compensatory hepatic lipoprotein synthesis in response to urinary albumin excretion may lead to progressive kidney disease and that the pathogenesis of atherosclerosis and glomerulosclerosis in renal injury may share a common pathway. In this process, persistent albuminuria stimulates an excess of lipoprotein synthesis in the liver, thereby disrupting the lipid synthesis cycle. It has been suggested that many of the diseases of the glomerular and tubulointerstitial apparatus are associated with atherosclerosis (the term "glomerular atherosclerosis" has been proposed), including dyslipidemias. Since then, numerous clinical and laboratory studies have confirmed the hypothesis that hyperlipidemia is the result of compensatory synthesis of hepatic lipoproteins in response to urinary albumin excretion and contributes to the progression of atherosclerosis and glomerulosclerosis [11]. Chronic kidney disease associated with pathology of lipid metabolism Hyperlipidemia may exert its influence on the progression of renal damage in several ways. 1. By developing intrarenal atherosclerosis. 2. Through the toxic effect of lipids on the structures of the nephron. The main mechanisms of CKD progression associated with lipid metabolism differ depending on the stage of the process. At the same time, there are some common features of development, which are based on elevated levels of cholesterol, TG, LDL and low levels of HDL in blood plasma [12, 13]. In nephrological patients, dyslipidemia has been shown to damage the endothelium of glomerular capillaries and deposition of lipids in mesangial cells that bind and oxidize LDL, stimulating mesangial proliferation and the development of glomerulosclerosis [14]. Hyperlipidemia increases the activation of mesangial cells with LDL receptors, which leads to stimulation of cell proliferation and an increase in the synthesis of macrophages, chemotaxis factors, extracellular matrix components, plasminogen activator-1, reactive oxygen species, etc. [15–17]. At the same time, LP deposited in the basement membrane of cells bind glycosaminoglycans and thereby increase the permeability of the membrane for proteins. As a result of this process, LP filtered in the glomeruli settle in the tubules of the kidneys, which initiates tubulointerstitial processes and sclerosis. In the future, an increased content of lipids leads to their capture by the epithelium of the tubules and deposition inside the cells. The deposition of lipids in mesangiocytes and tubular epithelium gives the cells a characteristic foamy appearance. This leads to their dystrophy and atrophy with the accumulation of lipid material in the intercellular space [18] The morphological substrate of CKD is glomerulosclerosis, which is characterized, regardless of the primary pathology of the kidneys, by mesangial sclerosis, expansion of the extracellular matrix, which includes laminin, fibronectin, heparan sulfate proteoglycan, type IV collagen, and interstitial collagen (normally absent in the glomeruli). An increase in the extracellular matrix that replaces a functionally active tissue is a complex process involving various growth factors, cytokines, and heat shock proteins. It has been established that in most patients with GFR of about 25 ml/min and below, terminal chronic renal failure occurs regardless of the nature of the disease. There is an adaptive response of intrarenal hemodynamics to the loss of mass of active nephrons. This is manifested in a decrease in resistance in the afferent and efferent arterioles of functioning nephrons, leading to an increase in the rate of intraglomerular plasma flow, that is, to glomerular hyperperfusion and an increase in hydraulic pressure

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in their capillaries. As a result, hyperfiltration occurs, and subsequently - glomerulosclerosis. Dysfunction of the tubular epithelium is closely associated with the development of tubulointerstitial fibrosis. The tubular epithelium is capable of synthesizing a wide range of cytokines and growth factors. In response to damage or overload, it enhances the expression of adhesion molecules, the synthesis of endothelin and other cytokines that contribute to tubulointerstitial inflammation and sclerosis. Any damage to the vessel wall stimulates platelet aggregation with the release of thromboxane, a powerful vasoconstrictor that plays an integral role in the development of arterial hypertension. Increased platelet reactivity and aggregation stimulates hyperlipidemia, the combination of which with arterial hypertension is accompanied by even more pronounced changes in the glomeruli. Metabolism of essential lipids in CKD 1. Total cholesterol in CKD In patients with CKD, the nature of dyslipidemia differs depending on the stage of the process [19-21]. It is believed that the greatest damage to the glomeruli of the kidneys causes a high level of total serum cholesterol. The hypercholesterol diet in experimental animals causes the appearance of lipid deposits in the glomeruli, monocytic infiltration and mesangial hypercellularity, as well as an increase in the mesangial matrix. Simultaneously with an increase in the level of total cholesterol, proteinuria and the number of sclerosed glomeruli increase. Even just hypercholesterolemia leads to the development of proteinuria, uremia and glomerulosclerosis and an increase in intraglomerular pressure. Clinical studies have shown that hyperlipidemia in any nephropathy accelerates the progression of renal failure. The rate of progression depends on the level of total serum cholesterol. 2. Triglycerides in CKD Among the possible mechanisms of uremic hypertriglyceridemia, various processes are discussed: changes in enzyme activity, disturbances in the receptor apparatus, and a decrease in HDL metabolism. It has been shown that already in the early stages of CKD, the level of plasma triglycerides increases, reaching maximum values in patients with nephrotic syndrome and in patients receiving renal replacement therapy [22]. This occurs due to a decrease in the activity of enzymes such as lipoprotein lipase (LPL) (EC 3.1.1.34) and hepatic triacylglycerol lipase (PTHL) (EC 3.1.1.3). The mechanism of action of lipoprotein lipase is to cleave triglycerides of the largest and lipid-rich lipoproteins in blood plasma - HM and VLDL. Chylomicrons circulating in the blood are gradually released from triacylglycerols (as a result of contacts with lipoprotein lipase) and are converted into residual chylomicrons, which contain very little triacylglycerols and a lot of cholesterol. Residual chylomicrons (partial and whole) are taken up by the liver cells. Hepatic triglycerol lipase catalyzes the hydrolysis of triglycerides to LDL, resulting in the formation of LDL. The hydrolytic action of the enzyme is carried out on the endothelial surface of the liver capillaries during the passage of blood through them (this determined the name of the enzyme). The hydrolysis of triacylglycerols is catalyzed by three lipases: triacylglycerol-, diacylglycerol- and monoacylglycerol lipase. The activity of the last two enzymes is 10–100 times higher than the activity of triacylglycerol lipase, which is a regulatory enzyme. The action of triacylglycerol lipase is to cleave triacylglycerols with the formation of 1,2-diacylglycerol and free fatty acids. As a result of a decrease in the activity of these enzymes, TG accumulates in the composition of HM and VLDL. Due to a decrease in the activity of lipases, the breakdown of TH to free fatty acids, which are necessary to meet the energy needs of the body, is disrupted, which may affect the development of protein-energy deficiency syndrome in patients on hemodialysis [23]. 3. High-density lipoproteins in CKD Characteristic of CKD is a decrease in the concentration of HDL. This is facilitated by the low concentration and activity of lecithin-cholesterolacyltransferase, which leads to disruption of the synthesis and transport of HDL and the rapid breakdown of HDL [22, 24]. In the later stages, but still in the pre-dialysis period, patients usually have elevated LDL levels and low HDL levels. In patients with significant proteinuria and nephrotic syndrome, lipid metabolism disorders are also expressed due to an increase in LDL, hypertriglyceridemia and hypercholesterolemia. Hypoalbuminemia, often associated with end-stage renal disease and CKD, also has the potential to contribute to a decrease in HDL. This is explained by the fact that HDL receive a significant amount of cholesterol from albumin, which acts as a carrier of free cholesterol from peripheral tissues to HDL, at the same time, defective oxidized forms of HDL are formed, which, in turn, acquire pro-oxidant and pro-inflammatory properties. In addition, the severity of hypercholesterolemia inversely correlates with the degree of hypoalbuminemia, which is explained by the compensatory nature of the increase in LP synthesis in the liver in nephrotic syndrome. The reduced catabolism of LP is also important due to a decrease in the activity of lipoprotein lipases. 4. Low density lipoproteins in CKD Mesangial cells, which have receptors for low-density lipoproteins, bind and oxidize them, which triggers a cascade of cytokine production that stimulates mesangial proliferation and the development of glomerulosclerosis. In parallel, the production of protective proteoglycans and

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collagenolytic enzymes that regulate the formation of the mesangial matrix decreases, the phagocytic properties of mesangiocytes are weakened, and the mesangium is “overloaded” with macromolecules. Lipoproteins deposited in the basement membrane of cells bind negatively charged glycosaminoglycans and neutralize its negative charge, increasing the permeability of the membrane for proteins. In addition, lipoproteins filtered in the glomeruli, deposited in the tubules of the kidneys, induce tubulointerstitial processes, interstitial sclerosis and the development of renal failure. Current guidelines suggest LDL-C < 2.5 mmol/L for CKD patients with GFR 30–60 ml/min/1.73 m2 and < 1.8 mmol/L for CKD patients with GFR < 30 ml as targets /min/1.73 m2. 5. Apolipoproteinins A-I and A-II in CKD Apolipoproteins A-I and A-II are part of HDL. ApoA-I is an LCAT activator and a ligand for HDL receptors, while apoA-II is an activator of hepatic lipase. In patients with ESRD, the concentrations of these apoproteins are significantly reduced. LCAT is the main determinant of the formation and content of HDL in blood plasma. In patients with ESRD, its activity is reduced. Deficiency of this enzyme can lead to a decrease in plasma HDL levels and impaired HDL conversion in CKD, which is accompanied by a significant increase in free serum cholesterol and a decrease in the concentration of esterified cholesterol. 6. Lipid peroxidation in CKD Activation of lipid peroxidation in the membranes of endothelial cell structures leads to the loss of their functional activity and is one of the mechanisms for the development of kidney diseases, which determines the degree of intoxication in CKD [19]. At the stage of severe CKD, lipoproteins undergo modification and become oxidized LDL (ox-LDL). Ox-LDL promote adhesion of monocytes to the endothelium of glomerular capillaries and affect tubular epithelial cells [20]. They bind to receptors in the mesangium and, through a number of cellular and molecular mechanisms, enhance inflammatory and fibrogenic processes in it. The cytotoxic effect of ox-LDL is manifested in the induction of podocyte apoptosis with the loss of nephrin and damage to the glomerular barrier. Features of correction of lipid disorders in CKD Lipid-lowering therapy in CKD patients today is the most important element of the nephroprotective strategy, designed not only to prevent, but also to slow down the progression of nephrosclerosis, preventing or postponing the development of renal failure. The principles of drug therapy are common for hyperlipidemias of any etiology, and the principle is the start of treatment already in the early stages of CKD. Lipid-correcting drugs include statins, fibrates, nicotinic acid, bile acid sequestrants, omega-3 polyunsaturated fatty acids, and antioxidants. Statins are structural inhibitors of the enzyme hydroxy-methylglutaryl-CoA reductase (HMG-CoA reductase), the main enzyme that regulates cholesterol biosynthesis in hepatocytes. As a result of a decrease in the intracellular cholesterol content, the liver cell increases the number of membrane receptors for LDL on its surface. Receptors "recognize", bind and remove atherogenic LDL particles from the bloodstream and thus reduce the concentration of cholesterol in the blood. Along with the lipid-lowering effect, statins have pleiotropic effects. In particular, they improve the function of the endothelium, reduce the level of C-reactive protein, a marker of an inflammatory response in the vascular wall, inhibit platelet aggregation, and weaken the proliferative activity of smooth muscle cells of the vascular wall. Statins are classified according to how they are made. Thus, lovastatin, simvastatin and pravastatin are naturally synthesized compounds derived from the waste products of certain fungal species, while fluvastatin, atorvastatin and rosuvastatin are synthesized drugs. Statins are most effective at lowering LDL cholesterol levels. The effect of statins on LDL-C levels is dose-dependent. Each doubling of the statin dose results in an additional 6% reduction in LDL-C (the 6% rule). Statins have little effect on TG and HDL cholesterol levels. As a rule, they reduce the level of triglycerides by 1015% and increase the level of HDL-C by 8-10%. These drugs not only contribute to the normalization of the lipid profile and thus prevent the development of atherosclerosis, but also, by reducing the accumulation of lipids in the kidney tissue, inhibit the proliferation of mesangial cells and the development of glomerulosclerosis. In addition to statins, mono- and combined lipid-lowering therapy using drugs from different groups is relevant. According to the clinical guidelines for the management of CKD in conditions of dyslipidemia, a special approach to the pharmacological correction of lipid metabolism disorders is recommended [25, 26]. In 2013 (KDIGO Clinical Practice Guideline for Lipid Management in Chronic Kidney Disease) recommendations for the treatment of lipid disorders in CKD were presented [27-35]. Thus, dyslipidemia is closely associated with the progression of CKD. Its influence is due to both atherosclerotic lesions of the renal vessels and the direct nephrotoxic effect of lipids. Lipid-lowering therapy in patients with CKD has the main goal of preventing the development and progression of CKD itself. Identification of the initial manifestations of lipid metabolism disorders in patients with chronic renal diseases makes it possible to identify high-risk groups with an unfavorable outcome of CKD, and timely

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prescribed therapy to prevent the development of the disease.

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