

Clinical Approach to Diabetic Nephropathy

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ABSTRACT The review presents modern information about the main mechanisms of the development and progression of diabetes. Diabetic nephropathy (DN), which is the same in both types of diabetes mellitus (DM), occurs. However, with type 2 diabetes, additional damaging factors such as obesity, dyslipidemia, hyperuricemia lead to the formation of DN and the development of end-stage renal failure.

Microalbuminuria is an early sign of DN (UIA). Active treatment of DN at the stage of MAU leads to regression and remission of laboratory signs of DN in 40-50% of patients after 2 years of treatment. Prognostic factors of DN remission are high. signal control of glycemia, control of arterial hypertension, especially when using renin blockers, the angiotensin system can lead to the reverse development of morphological changes in kidney tissues with DN. Only with maintaining normoglycemia for a long time (more than 10 years).

Keywords: diabetes mellitus in children; diabetic nephropathy; glomerulosclerosis; microalbuminuria; glycemic control; glycated hemoglobin.

The official representative of the World Health Organization recognized diabetes as an incurable disease. The modern level of medical science and clinical practice emphasizes the need to pay the patient for himself and take responsibility for his health. The global growth of humanity, the lifestyle cut off from nature leads to the development of many factors that cause diabetes.

Diabetic nephropathy (DN) is one of the most serious complications. Diabetic nephropathy (DN) can develop in these early stages, leading to sudden death of patients and death from cell failure. [3, 4].

Distribution: DN is constantly growing, DN is characterized by the pathology of diabetes, damage to arteries, arterioles, glomeruli, resulting in the rapid development of metabolic diseases due to the

deposition of carbohydrates and lipids in the parenchyma and tubules of the kidney. [4-7]. Today, the term "diabetic nephropathy", "diabetic glomerulosclerosis" leads to already developed morphophysiological changes. [1,2].

It is common to distinguish three stages of DN: microalbuminuria (MAU); the stage of proteinuria with conservation of kidney function and the stage of chronic kidney disease

The frequency of detection of DN is closely related to the duration of DM, and this relationship is more pronounced in type 1 diabetes (insulin-dependent) [5,6]. Often, the development of type 1 DN in patients with long-term diabetes is 5-6% under the age of 10, 20-25% under the age of 20, 35-40% under the age of 30, 45% under the age of 40, maximum. The minimum peak in the development of DN corresponds to periods of 15 to 20 years of the presence of SD. With type 2 diabetes, the frequency of DN is the same as the duration of SD [7]. The formation and development of kidney damage in diabetes mellitus is constantly progressive. Among the pathogenetic theories of gofactor processes, chemical, hemodynamic and genetic are recognized as important metabolic [5]. According to the theory of metabolism, the most important mechanism for the formation of DN is hyperglycemia, which causes a series of biochemical disorders, for example, a) approximate glycation of proteins with subsequent non-enzymatic cumulative reversible (Schiff bases), partially reversible (Amadori products) and finally the final products of irreversible glycation (CNG), which lead to harmful effects on the kidneys b) activation of the following protein S kinase due to the direct glucotoxic effect of glucose, with an increase in lipid peroxidation processes, poisons with a cytotoxic effect [9]; c) activation of the polyol pathway of glucose metabolism (the conversion of glucose to sorbitol in the presence of the enzyme, aldose reductase, and intracellular myo-inositol reserves).

As a result of the slow non-enzymatic soy, the breakdown of hemoglobin with glucose is formed with glycosides. The reflected lysed hemoglobin (HbA1c) is different from the average blood sugar concentration measurements over a long period (up to 3 months) of blood glucose concentration. it gives an idea about its level in the blood during the study. HbA1c Mayer is formed as a result of the reaction between hemoglobin and blood glucose and reflects them irreversibly combined with glucose molecules in the center of blood hemoglobin. An increase in the amount of glucose in the blood with diabetes significantly accelerates this reaction, which leads to an increase in the level of HbA1c in the blood. The average life span of erythrocytes containing hemoglobin is 120-125 days. That is why the level of HbA1c reflects the average level of glycemia for about 3 months. The higher

its level, the higher the last level of glycemia in 3 months and, accordingly, the risk of developing complications. HbA1s values are normal. 4 to 5.9%. With diabetes, its level increases, which indicates a greater risk of retino-development. pathology, DN and other complications. The International Diabetes Federation recommends keeping the level of HbA1c below 6.5%. The value of HbA1s exceeds 8% means that the diabetes is almost uncontrollable and the therapy has been changed and should be changed .

Early works showed the possibility of previewing. prevention of the development of DN with ideal compensation was carbohydrate exchange in patients with type 1 diabetes DCC T study, in patients with type 2 diabetes - study UKPDS [4, 9]. The latter showed that a 1% decrease in HbA1c was associated with a decrease in the risk of stroke by 12%, myocardial infarction by 14%, and atherosclerosis of peripheral vessels by 43% . HbA1c with a high affinity for oxygen leads to a decrease in the partial pressure of acid in the blood and tissue ischemia. Therefore, raising the level of HbA1c always leads to a significant increase in the risk of developing such complications of diabetes as acute cerebral circulation disorders, myocardial infarction, atherosclerosis [10].

It should be noted that glycation of almost all tissues occurs with diabetes. Glycation of proteins interferes with their normal functioning due to molecular disruption of the functional structure, change in enzymatic activity and disruption of receptor interaction. CNG is not only the development of intracellular and extracellular complexes with proteins, as well as with lipids and nucleic acid Tami, contributes to the development of diabetic complications . The interaction of AGEs with receptors (RCPG) has been identified, leading to internal disturbances in localized biomembranes. Triple cellular signaling, oxidative stress, release of anti-inflammatory and prosclerotic cytokines, free radicals that play an important role in the formation of medical science. play. Influenced by the mechanism of gene expression by KKE. Initially, these compounds bind to monocytes, macrophages, endothelial cells, which are localized with a special CPGR, which mediates signal transmission, a means of increasing the formation of free radicals with oxygen. the expression of many genes is damaged. It is clear, the compression of various proteins that activate, the effect of AGE can be stopped or blocked by the use of antibodies against CPGR or antibodies against AGE [10]. In the kidney, AGEs are formed in the basement membrane of glomeruli, which contains albumin, IgG, etc., which leads to its thickening, deposition in it, immune complexes - gradually formed, increasing changes in the structure and properties of collagen, kidney glomeruli genes, basement membrane and other components of the glomerular matrix. Hyperglycemia is the leading factor in the formation of

diabetic angiopathy, including DN. factors that cause thrombotic occlusion are the development of capillaries and coagulopathy. also, endothelial dysfunction is correlated with early recovery and early inflammatory response in patients with type 1 diabetes [35]. Relative or absolute non-insulin sufficiency leads to impaired synthesis of nitrogen oxides (NO), easy formation of large amounts of oxidized LDL, formation of active macrophages, oxidative stress that provokes and aggravates inflammatory reactions and stimulating thrombosis. Endothelial hyperproduction of various growth factors in response to cells glycosylated proteins or cytokines

The genetic theory of DN shows that the location is an important factor in its development, turning and development. A large number of established single nucleotide polymorphisms play a leading role in the genetic predisposition to the formation of LTOs. It was found that the risk of developing DN increases several times with the heredity of risk alleles in the susceptibility loci of different genes, AC E, IL, TNFb, COL 4A1, eNOS, SOD 2, APOE, etc. Identification of variants at the biomarker level of these genes can provide identification of individuals at high risk of developing vitia DN, in treatment, diagnosis and prevention of diseases in the early stages [5,9].

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