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DEVELOPMENT OF NEPHROPATHY IN THE CONTEXT OF OVERWEIGHT AND OBESITY: A LITERATURE REVIEW

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Relevance. Overweight and obesity are considered important risk factors not only for metabolic syndrome, arterial hypertension and cardiovascular diseases, but also for the development of chronic kidney disease. In obesity, insulin resistance, dyslipidemia, chronic low-grade inflammation, endothelial dysfunction and glomerular hyperfiltration are formed. These changes contribute to microalbuminuria, a decrease in renal filtration function and the gradual development of nephropathy.

Aim of the Review. The aim of this literature review is to analyze the role of lipid profile abnormalities, adiponectin levels and early laboratory markers of renal injury in the development of nephropathy among individuals with overweight and obesity.

Literature Review. According to the literature, obesity-related nephropathy develops through several pathogenetic mechanisms. Increased body weight leads to a higher hemodynamic load on the kidneys and initially causes glomerular hyperfiltration. Over time, increased intraglomerular pressure, podocyte injury, changes in the glomerular basement membrane and proteinuria may occur. If these processes persist, they may result in glomerulosclerosis and tubulointerstitial fibrosis.

Disorders of lipid metabolism play a significant role in the development of nephropathy in obesity. Dyslipidemia is usually characterized by increased levels of total cholesterol, triglycerides and low-density lipoprotein cholesterol (LDL-C), together with decreased levels of high-density lipoprotein cholesterol (HDL-C). The formation of an atherogenic lipid profile negatively affects renal microcirculation, aggravates endothelial dysfunction and creates conditions for sclerotic changes in the glomerular capillaries. Elevated triglycerides and LDL-C are especially important, as they activate oxidative stress and inflammatory processes in renal tissue.

Changes in the lipid profile are also associated with early laboratory signs of nephropathy. The literature indicates that increased triglyceride and LDL-C levels may be accompanied by microalbuminuria and an elevated albumin-to-creatinine ratio. A decrease in HDL-C reflects weakening of protective anti-atherogenic mechanisms. Therefore, in patients with obesity, evaluation of renal function should not be limited to creatinine and urea; lipid profile parameters should also be included in the comprehensive assessment.

Adipokines, particularly adiponectin, have an important role in the pathogenesis of obesity. Adiponectin is a biologically active substance produced by adipose tissue. It improves insulin sensitivity and has anti-inflammatory, anti-atherogenic and nephroprotective properties. Under physiological conditions, adiponectin supports endothelial function, reduces oxidative stress and limits fibrotic processes in renal tissue.

In overweight and obesity, adiponectin levels usually decrease. Reduced adiponectin contributes to insulin resistance, dyslipidemia, arterial hypertension and chronic inflammatory activity. Low adiponectin levels may be associated with endothelial dysfunction, podocyte injury and the development of albuminuria in the renal glomeruli. Therefore, adiponectin can be considered a promising additional biomarker for the early assessment of obesity-related nephropathy.

The main laboratory criteria for detecting the early stages of renal injury include microalbuminuria, albumin-to-creatinine ratio, cystatin C and glomerular filtration rate (GFR). Microalbuminuria reflects early damage to the glomerular filtration barrier. Cystatin C is considered a more sensitive marker than creatinine for detecting early changes in renal filtration function. A decrease in GFR indicates a reduction in the functional reserve of the kidneys.

At the same time, assessment of nephropathy risk in patients with obesity should include a combined analysis of body mass index, waist circumference, arterial blood pressure, glucose level, total cholesterol, triglycerides, LDL-C, HDL-C, adiponectin, creatinine, urea, microalbuminuria, albumin-to-creatinine ratio, cystatin C and GFR. Such an integrated approach makes it possible to detect renal injury at the subclinical stage.

Conclusion. The analysis of the literature shows that the development of nephropathy in overweight and obesity is closely associated with metabolic, hemodynamic, inflammatory and lipid-related disturbances. Dyslipidemia, particularly increased triglycerides and LDL-C together with decreased HDL-C, negatively affects renal microcirculation and the glomerular filtration barrier.

A decrease in adiponectin levels in obesity enhances insulin resistance, endothelial dysfunction and chronic inflammation, thereby creating conditions for the development of nephropathy. Therefore, adiponectin may be regarded as an important additional biomarker in the assessment of obesity-related renal injury.

For early detection of nephropathy in individuals with overweight and obesity, it is advisable to evaluate renal markers together with lipid profile parameters and adiponectin levels. A comprehensive analysis of these indicators may improve the identification of renal injury at an early stage and support timely preventive measures.

Keywords: Overweight, obesity, nephropathy, lipid profile, adiponectin, microalbuminuria, cystatin C, glomerular filtration rate, dyslipidemia.