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DIABETIC NEPHROPATHY IN PEDIATRICS

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Abstract
The article provides information about the morphology and function of the kidneys in children with obesity. The influence and interrelationship of numerous pathogenetic factors affecting the functioning of the kidney is discussed. The peculiarity of obesity nephropathy in children compared with the adult body was noted.

Keywords: Obesity, children, nephropathy, method, treatment.

Introduction

According to the World Health Organization (WHO), obesity is an excessive accumulation of adipose tissue in the body, which disrupts metabolism [1]. Obesity is detected in 1/4 of the population of Western Europe and in 1/3 of the population of North America. The term "obesity epidemic" has become firmly established in the specialized literature. The National Health and Nutrition Examination Survey (NHANES) indicates that the prevalence of obesity is increasing in all age groups, affecting both sexes, and being traced across ethnic and racial groups. Many factors are involved in the development of obesity, including genetic and environmental changes, metabolic disorders, lifestyle features and eating habits. However, more than 90% of obesity cases are idiopathic; less than 10% are associated with hormonal or genetic disorders [2].

MATERIALS AND METHODS

Currently, there is no universally recognized definition of childhood obesity. Diagnosing obesity in children as a pathological condition causes certain difficulties. To assess the trophic status of the child, pediatricians compare the patient's body weight with his height. The assessment is carried out according to centile tables compiled taking into account age and gender. The degree of deviation of body weight from the average value at a given body length in the direction of decrease (body weight deficit) or increase (excess body weight) is expressed as a percentage. In this case, the value of body weight corresponding to the median of the 4th corridor (the average between the values of the left and right interval edges of the 4th corridor) is taken as 100% [5].

RESULTS AND INFORMATION

To date, a large amount of data has been accumulated on fetal programming of pathology manifested in adulthood and old age. About 20 years ago, Barker and colleagues at the

University of Southampton found that the geographic areas with high natal, postnatal and infant mortality rates are the same as those with relatively high mortality rates from coronary heart disease in middle-aged individuals. Since then, many studies have been conducted proving an inverse relationship between birth weight and such adult pathological conditions as insulin resistance, cardiovascular diseases, including stroke, obesity, and possibly breast cancer and atopy [2]. The so-called "prenatal program" is proposed - a set of factors that adversely affect the fetus. The study of the consequences of malnutrition at the early stages of development made it possible to formulate the concept of "nutritional programming".

Morphological changes in the nephron in obesity are similar to those in oligomeganephronia. In advanced cases, the development of secondary FSGS is possible [4]. This form is distinguished by the fact that it is not characterized by massive proteinuria corresponding to nephrotic syndrome, and there is practically no edema. In severely obese individuals with preserved renal function, morphological changes are found in biopsies, including glomerulomegaly, hypertrophy and fusion of podocytes, expansion of the mesangial matrix, and proliferation of mesangial cells [2]. Glomerulomegaly is a primary histopathological sign that distinguishes GPO from primary FSGS [12]. Thickening of the glomerular basement membrane (GBM), which was previously considered an early manifestation of hyperglycemia and diabetic nephropathy, may be an additional pathological finding in obesity. GBM thickening is found on biopsy in patients with nephrosclerosis associated with essential arterial hypertension and in GPO patients with normal glycemia. The thickness of GBM directly correlates with cholesterol and triglyceride levels [3].

Leptin is an adipokine belonging to the 1st class of cytokines. It is produced mainly by visceral adipose tissue, as well as subcutaneous fat, fatty deposits in the pericardium, placenta, and stomach [2]. Leptin receptors are found on endothelial and smooth muscle cells. Leptin induces oxidative stress in endothelial cells. It has been proven that chronic hyperleptinemia leads to arterial hypertension. It is hypothesized to be due to stimulation of the sympathetic nervous system, suppression of natriuresis, and inhibition of nitric oxide (NO) production by endothelial cells. Leptin induces the growth of glomerular endothelial cells and increases the production of transforming growth factor (TGF-α1). A 72-hour infusion of recombinant leptin stimulated TGF-alfa1 expression and increased the total number of proliferating cells; a prolonged three-week infusion caused an increase in type IV collagen in the glomeruli. In mesangial cells, leptin stimulates the production of type I collagen, contributing to the development of glomerulosclerosis and proteinuria. In patients with type 2 diabetes mellitus, hyperleptinemia was positively correlated with the degree of proteinuria and negatively correlated with the glomerular filtration rate.

The key link linking obesity and hypertension is called an increase in tubular sodium reabsorption. An important determinant of tubular reabsorption is glomerular hyperfiltration. Obviously, obese patients are not homogeneous in the nature of changes in

intrarenal hemodynamics. Thus, during the examination of adult men with obesity (BMI > 36) by Israeli nephrologists (Rabin Medical Center), two groups of patients differed in the level of sodium filtration fraction (FFNa). In both groups, GFR significantly exceeded that in the group of people with normal body weight. In the group of patients with a high FFNa value, postglomerular oncotic pressure was 13% higher, and fractional excretion of lithium (a marker of proximal sodium reabsorption) was 33% lower than in the control group. In the second group with normal FFNa levels, postglomerular oncotic pressure and fractional lithium excretion remained normal. The authors believe that the mechanism of hyperfiltration in severe obesity is heterogeneous. Previous observations of these authors in patients with severe obesity have shown that renal plasma plasma (RPT) changes to a lesser extent compared to GFR. RTP values in different individuals range from normal to increased to the same extent as GFR.

CONCLUSION

In pediatrics, it is practically important to identify a risk group for the formation of nephropathy, obesity, metabolic syndrome and cardiorenal syndrome. Children born with low birth weight by gestational age, children with signs of early obesity, children from families with obesity, impaired carbohydrate metabolism and arterial hypertension should be included in the risk group. Further research will make it possible to individualize the approach to each overweight child, diagnose and correct the leading link of impaired metabolism.

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