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LABORATORY NEPHROTIC FORM OF GLOMERULONEPHRITIS IN CHILDREN IMPORTANCE OF DIAGNOSES

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Annotation. In this article bacute course of glomerulonephritis in children, diagnosisandImportance of laboratory diagnostics in nephrotic forminformation about.The word glomerulonephritis (glomerulo-ball) nephritis is derived from the word nephron, which means inflammation of the nephron. It is considered a serious disease and occurs in children of all ages, especially from 5 to 20 years old, on average it is more common from 7 to 12 years old.

Key words: Kidney, infectious-allergic , nephrotic, nephritic, hematuria, acute and chronic.

Today, 10-15 adult patients per 10,000 people suffer from glomerulonephritis. According to the rate of detection among all pathologies of the kidney, this disease takes the 3rd place. Glomerulonephritis can be diagnosed among patients of any age, but most often the disease occurs among people under 40 years of age.

Among male populations, this disease occurs 2-3 times more often. In children, among all kidney diseases, glomerulonephritis ranks second. According to statistics, this pathology is the most common cause of disability that develops due to chronic kidney failure. Approximately 60% of patients with acute glomerulonephritis develop hypertension. In 80 percent of children, the acute form of this disease causes various cardiovascular diseases[1,5].

Recently, the rate of detection of glomerulonephritis among the population of different countries is increasing. This is due to the deterioration of the environment, as well as the general reduction of immunity among the population, which is a consequence of not fulfilling the recommendations for a healthy lifestyle. The basis for the development of respiratory failure in children with glomerulonephritis, as well as the development of arterial hypertension, is the retention of sodium and water. Water is stored in tissues, so swelling occurs. For a certain time, the liquid is stored only in the tissues. However, it quickly moves to this gap and fills them. Thus, excess body fluid accumulates in the pleural cavity, pericardial cavity, abdominal cavity, etc. This, in turn, causes other symptoms. For example, fluid accumulated in the pericardial space compresses the heart, which leads to dyspnea and bradycardia (slow heartbeat). Shortness of breath also causes fluid to accumulate in the lungs, which causes venous stasis in the circulatory system. Thus, nephrogenic elements (kidney) pulmonary edema[2,6].

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At the same time, the first important mechanism of swelling is proteinuria. The body's loss of proteins leads to fluid in the bloodstream and tissue impregnation with this fluid (ie, swelling). Normal albumin (a high molecular weight protein) keeps the blood fluid in the blood vessels. But with glomerulonephritis, there is a large loss of these proteins in the urine, as a result of which their concentration in the blood serum drops. Fewer albumins remain in the blood, and the more fluid that passes from the bloodstream into the tissues, the greater the swelling[3,4].

Nephrotic syndrome is a symptom complex characterized by protein in the urine, a decrease in the concentration of protein in the blood, and swelling. The most noticeable and obvious sign of nephrotic syndrome is proteinuria or protein in the urine. The daily loss of protein exceeds 3.5 grams, which means a large loss of proteins by the body. In this case, proteinuria is mainly due to albumin, high molecular weight proteins. Thus, in the human body there are two types of proteins (consisting of two parts) - albumin and globulins. The first part is high-density proteins that retain water in the blood serum, that is, they maintain oncotic pressure. The second part of proteins is involved in the maintenance of immunity, and the first does not have such an effect on oncotic pressure. Thus, albumin traps water in the blood. Therefore, when a large amount is excreted in the urine, water from the blood enters the tissues. This is the main mechanism of tumor formation. The more albums there are, the more swelling occurs. There fore, such swelling occurs in nephrotic syndrome[9,10].

The second sign of nephrotic syndrome is hypoalbuminemia and hyperlipidemia. First, it reduces the concentration of proteins in the blood, and secondly, it increases the concentration of lipids (fat) in the blood.

Diagnosis. There may be a decrease in diuresis, abdominal urination, the appearance of swelling or pastosity of the face, an increase in blood pressure (usually). A set of studies: measurement of blood pressure, blood pressure, total blood volume, determination of daily proteinuria, the amount of protein flow and the assessment of proteinogram, blood lipids. A thorough physical and clinical laboratory examination is designed to establish the possible cause of CGN - a general or systemic disease. Ultrasound of the kidneys (X-ray) allows you to determine the size and density of the kidneys. Assessment of kidney function - Reberg-Tareev test, determination of the concentration of urea and / or creatinine in the blood. A kidney biopsy confirms the diagnosis[7,8].

Laboratory data:

• In the blood - an increase in the average level of ESR (with secondary CGN, a significant increase can be detected depending on the underlying disease), an increase in the level of CEC, antistreptolysin O, a decrease in the level of complement blood (immunocomplex CGN) and an increase in IgA in Berger's disease increase

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• Total protein and albumin concentrations (significantly - with nephrotic syndrome), increased concentration of α2 - and β - globulins in nephrotic syndrome, hypogammaglobulinemia. If there is a secondary CGN due to systemic diseases of the connective tissue (lupus nephritis), γ-globulins may be elevated. Hyper and dyslipidemia (nephrotic form).

• Reduction of GFR, urea and creatinine, anemia in the blood, metabolic acidosis, hyperphosphatemia, etc. (CRF or ARF with CRF).

• Erythrocytes in urine, proteinuria (mass with nephrotic syndrome), leukocytosis, cylindrical - granular, soft (with nephrotic syndrome).

Instrumental data

•With ultrasound examination or examination, the size of the kidney is normal or reduced (with CRF), the contours are smooth, the echogenicity is expanding

• Radiology of chest organs - enlargement of heart borders (arterial hypertension)

• ECG - signs of left ventricular hypertrophy

• Kidney biopsy (light, electron microscope, immunofluorescence examination) allows to eliminate the morphological form, activity of CGN, kidney diseases with similar symptoms. Decrease in diuresis, appearance of abdominal swelling or facial pastosis, increase in blood pressure (usual) may occur[4,5].

Differential diagnosis: chronic pyelonephritis, acute glomerulonephritis, nephropathy of pregnancy, chronic tubulo-interstitial nephritis, alcoholic kidney damage, amyloidosis and diabetic nephropathy, as well as diffuse diseases of the renal tissue (mainly SLE) and systemic vasculitis. pyelonephritis, interstitial nephritis, lipoid nephrosis in SGN; hemorrhagic diathesis; metabolic disorders and dysmetabolic nephropathy; carried out with sarcoidosis and other tumor diseases[7].

Complex of studies: measurement of blood pressure, UAC, OAM, determination of daily proteinuria, amount of protein flow and evaluation of proteinogram, blood lipids. A thorough physical and clinical laboratory examination is designed to determine the possible cause of CGN - general or systemic disease. Kidney ultrasound (x-ray) allows to determine the size and density of the kidneys. Assessment of kidney function - Reberga - Tareev test, determination of carbamide and creatinine concentration in blood. A kidney biopsy confirms the diagnosis[1,3].

Prevention. Timely detection and treatment of chronic infection, adequate therapy of AGN, rational use of gamma globulins, nephrotoxic drugs and other blood products. consists of Consequence. It depends on the clinical forms of the disease. With nephrotic syndrome with minimal changes in the glomerular membrane, the outcome is favorable (80-90% of patients are cured). But in most cases, SGN is a progressive disease leading to indolent renal sclerosis.

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