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IMMUNOLABORATORY DIAGNOSTICS OF GLOMERULONEFRITIS IN CHILDREN

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Annotation. Chronic glomerulonephritis(CGN, sluggish glomerular disease, a group term that includes diseases of the glomeruli of the kidneys with a common immune mechanism of impairment and a gradual deterioration in kidney function with the development of renal failure. Statistics - 13 per 10,000 population -50 cases Primary CGN occurs 2 times more often in men, than in women, the second is due to the underlying disease, can develop at any age, but most often in children 3-7 years old and in adults 20-40 years old.

Keywords. Kidneys, immunology, urine, hematuria, acute and chronic.

Chronic glomerulonephritis (CGN) is a group of primary glomerulopathies characterized by persistent, progressive immune inflammation, sclerotic and destructive nephron damage, subsequent development of tubulointerstitial sclerosis, and partial chronic renal failure. Etiology. SGN is a chronic disease with a primary, genetic predisposition. Trigger factors include:

1) irrational drug therapy (long-term use of nephrotic drugs, polypharmacy);
2) foci of chronic infection;
3) persistent viral infections;
4) excessive antigenic stress (recurrent and recurrent infections, repeated injections of immunoglobulins, irrational transfer of immunization)[3]. Pathogenesis. The basis of the disease is an immunopathological process:

1) as a result of the instability of the nephron membranes in kidney dysplasia, there is an influx of renal antigens into the bloodstream, damage to the histohematological barrier as a result of various factors. As a result, autoantibodies, cytotoxic lymphocytes, immune complexes are formed that damage the basement membrane of the tubules and glomeruli, the complement system is activated;

2) as a result of a violation of immunological reactivity leads to an autoimmune process in intact kidney tissues (with systemic diseases).

Classification. In international practice, the morphological classification of SGN is used, the following morphological forms are distinguished:

1) webbed;
2) membranous-proliferative GN;
3) focal segmental glomerulosclerosis;

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4) mesangioproliferative (IgA-nephropathy);

5) fibroplastic (the beginning of the above forms - SGN)[1,2].

The SGN transition can be as follows:

1) drug varying duration or recurrent SGN with spontaneous remission; 2) the flow of continuous activity of the persistent process;

3) progressive, rapidly developing SGN (after 2-5 years) is combined with the development of SGN. The clinical picture of SGN. It is more difficult to determine the onset of the disease in the hematuric form (microhematuria is detected suddenly). Complaints about fat content, normal blood pressure, transient edema, dysuria and intoxication are not observed. Sometimes they reveal a change in skin color, pain in the abdomen and back, fatigue, headache. The main symptom of the disease is persistent hematuric syndrome. Hematuria is well known. Kidney function is not impaired. The swelling proteinuric form of CGN often has an acute onset. After a respiratory infection, tonsillitis, vaccination, colds, sometimes acute nephritis, the clinic without external causes begins with massive proteinuria[5,6].

The main symptoms of the disease are:

1) massive proteinuria (more than 3% in urinalysis, more than 2.5 g/milk);

2) different development of edema;

3) hypoproteinemia, hyperlipidemia. Arterial hypertension and azotemia may occur, which quickly disappear during treatment. The azo-secretory function of the kidneys persists for a long time, but later SBE develops[4].

The mixed form of SGN begins as acute nephritis. characteristic: 1) pain in the abdomen and back, dysuria;

2) obvious tumors;

3) arterial hypertension (headache, dizziness, lethargy or agitation, decreased vision, sometimes facial paralysis, vomiting, hyperreflexia, ataxia and focal or general convulsions). Blood pressure rises from the age norm. Hypertensive angioretinopathy is detected when examining the fundus. the following are typical:

1) changes in the urine (hematuria, severe proteinuria);

2) hypoproteinemia, hyperlipidemia;

3) kidney biopsy reveals proliferative-fibroplastic glomerulitis[10]. With this form of the disease, SBE develops after 1-2 years. External CIS is carried out in the same way as clinical and laboratory studies for AGN. In nephrotic forms of the disease, additional markers of viral hepatitis B, cytomegalovirus are checked. In special nephrological centers, a kidney biopsy is performed, as a result of which the morphological type of kidney damage is determined, the expediency of prescribing glucocorticoids and immunosuppressants is determined, and the outcome of the disease is organized[8,9].

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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.

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].

Treatment of SGN is determined individually depending on the course and form of the disease, the presence of symptoms of renal failure, comorbidities and complications of the disease. Basic therapy consists of diet, daily regimen, herbal medicine and infection treatment. The diet depends on the functional state of the kidneys. With a recurrence of the hematuric form of CGN, the diet is prescribed in the same way as with AGN. In hematuric and mixed forms of the disease, a low-salt, low-protein diet is recommended. Expansion of the diet and increase in protein content is carried out gradually. In the acute phase of the disease, bed rest is recommended[8]. 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.

Recommendations

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