MODERN METHODS OF EARLY DIAGNOSIS OF GLOMERULONEPHRITIS

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Abstract: Etiology of glomerulonephritis, development of the disease, consequences of the disease, modern methods of early diagnosis of the disease, development of treatment measures for the disease

Key words: Diffuse glomerulonephritis, infection, progressive, nephritogenic, streptococcus,

One of the most common types of kidney diseases is an allergic autoimmune inflammatory disease, which is mainly caused by damage to the glomerular vessels. Its main clinical types are acute, chronic and very rapidly progressive glomerulonephritis.

In the etiology of diffuse glomerulonephritis, infection, toxic substances, and some exogenous allergens that trigger immune mechanisms play a key role. The role of a known etiological factor can be determined in 80-90% of patients with acute Diffuse Glomerulonephritis, in 5-10% of patients with chronic and rapidly progressive types. In the remaining patients, the cause of Diffuse Glomerulonephritis remains undetermined. A hemolytic streptococcus (types 4, 12) is important in the development of diffuse glomerulonephritis, and it is considered a specific "nephritogenic" strain.

In most cases, Diffuse Glomerulonephritis develops directly with the involvement of immune mechanisms. Post-streptococcal acute Diffuse glomerulonephritis is the development of acute transient allergic nephritis associated with the formation of antibodies 10-12 days after angina or pharyngitis.

Chronic Diffuse Glomerulonephritis is often associated with the gradual formation of antigen and antibody-capturing immune complexes and their deposition on the surface of the glomeruli and insufficient elimination. In acute Diffuse Glomerulonephritis and some chronic Diffuse Glomerulonephritis, the disease is caused by the formation of antibodies against the basement membrane of glomeruli.

The mechanism of the formation of immune complexes can be imagined as follows: streptococcal toxins affect the kidney tissue (probably the basement membrane of the glomeruli), as a result of which a changed protein is formed, which serves as an antigen, to which anti-kidney antibodies are produced. Formed antibodies (IgG, IgM) interact with antigens in the blood, then in the form of immune complexes (antigen - antibody - complement) they settle on the basal membrane of the balls and

cause the development of immune inflammation. Thus, diffuse glomerulonephritis is considered a disease of immune complex genesis.

Body cooling plays an important role in the pathogenesis of diffuse glomerulonephritis.

It is known to develop most rapidly in cold weather (autumn and winter) and especially in cold wet climates. Ventilation can change the course of the body's reactivity and immunological reactions through a reflex disturbance of the blood supply and trophism of the kidney.

Therefore, it can play the role of a decisive factor in a previously sensitized organism. In addition to the infectious etiology of glomerulonephritis, it is also assumed to be of a serum and vaccine nature.

In Diffuse Glomerulonephritis from a clinical point of view

* proteinuria,
* hematuria,
* swelling,
* arterial
* hypertension,

It is manifested by a violation of kidney function.

The diagnosis of "acute glomerulonephritis" is made based on anamnesis (recent infectious diseases), clinical picture (edema, arterial hypertension) and laboratory data. The analysis results are characterized by the following changes:

• Micro- or macrohematuria. In macrohematuria, the urine takes on a black, dark brown or "meat wash" color. In microhematuria, there is no change in the color of urine. In the first days of the disease, mainly new erythrocytes are detected in the urine, and then dehydrated ones.

• moderate albuminuria (usually in the range of 3-6%) is noted for 2-3 weeks;

• According to the results of urine sediment microscopy, there are granular and hyaline cylinders in microhematuria, and erythrocytes in macrohematuria;

• Nocturia, decreased diuresis when performing Zimnitsky's test. The preservation of the concentration ability of the kidneys is confirmed by the high relative density of urine;

• According to the results of the study of endogenous creatinine clearance, a decrease in the filtration capacity of the kidneys;

According to the results of general blood analysis, leukocytosis and increase of ECHT are determined in acute glomerulonephritis. Biochemical blood analysis confirms high levels of urea, cholesterol and creatinine, AST and ASL-O titer. Acute azotemia is characteristic (increased amount of residual nitrogen).

An ultrasound examination of the kidneys and an ultrasound dopplerography of the renal vessels are performed. If the results of laboratory studies and UTT are

suspicious, a kidney biopsy is taken to confirm the diagnosis of glomerulonephritis and the obtained material is examined morphologically.

Acute glomerulonephritis is treated in a hospital. Diet No. 7, bed routine is prescribed. Patients are prescribed antibacterial therapy (ampicillin + oxacillin, penicillin, erythromycin), non-hormonal (cyclophosphamide, azathioprine) and hormonal (prednisolone) drugs to correct immunity. The complex of treatment measures includes the treatment of inflammation (diclofenac) and symptomatic therapy to reduce swelling and blood pressure.

Further treatment in sanatoriums is recommended. After acute glomerulonephritis, patients are under the supervision of a nephrologist for two years. In the treatment of chronic glomerulonephritis, a complex of treatment measures similar to acute glomerulonephritis is carried out during the disease attack. During the period of remission, the treatment scheme is determined based on the presence and severity of symptoms.

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