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VARIANTS OF THE COURSE OF GLOMERULONEPHRITIS AND THEIR ETIOPATHOGENESIS

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Abstract
Glomerulonephritis is an immune-inflammatory kidney disease with predominant damage to the glomeruli, but also involving the tubules and interstitial tissue. Glomerulonephritis, by its development mechanism, belongs to the group of infectious-allergic diseases. The term "infectious-allergic" reflects the formation of infectious allergy in combination with various non-immune damage to the organ.

Keywords: Autoantibodies, immune inflammation, lupus erythematosus, infective endocarditis, group A streptococcus.

Introduction

Glomerulonephritis is an immune-inflammatory disease with predominantly affecting the glomeruli of the kidneys, as well as involving the tubules and interstitial tissue. Glomerulonephritis , by its development mechanism, belongs to the group of infectious-allergic diseases. The term "infectious-allergic" reflects the formation of an infectious allergy in combination with various non-immune damage to the organ. There are also autoimmune forms of the disease, which are caused by damage to the renal tissue by autoantibodies , i.e. antibodies to the organ itself.

Glomerulonephritis is an independent disease, but can also occur in many systemic diseases, such as systemic lupus erythematosus, hemorrhagic vasculitis, infective endocarditis, etc.

Prevalence of glomerulonephritis

Glomerulonephritis is one of the most common kidney diseases in children, leading to the development of chronic renal failure and early disability. In terms of prevalence, it is the second most common acquired kidney disease in childhood after urinary tract infection.

Acute glomerulonephritis can develop at any age, but most patients are under 40 years of age.

Causes of glomerulonephritis

The development of glomerulonephritis is associated with acute and chronic diseases of various organs, mainly of streptococcal origin.

The most common causes of glomerulonephritis are:

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- tonsillitis;
- scarlet fever;
- purulent skin lesions (streptoderma);
- pneumonia;

The development of glomerulonephritis can also be caused by acute respiratory viral infections, measles, and chickenpox.

Etiological factors also include cooling of the body in a humid environment ("trench" nephritis). Cooling causes reflex disorders of the blood supply to the kidneys and affects the course of immunological reactions.

There are reports of a causal role for microorganisms such as Staphylococcus aureus , streptococcus pneumoniae , Neisseria meningitidis , malaria plasmodium, Toxoplasma gondii and some viruses.

Usually, the onset of the disease is preceded by a streptococcal infection in the form of pharyngitis, tonsillitis, scarlet fever, skin lesions - impetigo-pyoderma by 1-3 weeks. It has been established that acute glomerulonephritis is usually caused only by " nephritogenic " strains of group A β-hemolytic streptococcus.

It is believed that if an outbreak of streptococcal A infection in a children's group is caused by nephritogenic strains, then 3-15% of infected children develop nephritis, although among adults and children around the sick child, approximately 50% have changes in the urine, i.e. they are likely to suffer from torpid (low-symptom , asymptomatic) nephritis.

Among children who have had scarlet fever, 1% develop acute glomerulonephritis during hospital treatment and in 3-5% of children treated at home. A respiratory viral infection in a child with chronic tonsillitis or carriage of cutaneous nephritogenic streptococcus A can lead to activation of the infection and cause the development of acute glomerulonephritis.

Glomerulonephritis (GN) is an immune-inflammatory kidney disease that primarily affects the glomeruli, but also involves the tubules and interstitial tissue. The main clinical types of GN are acute, chronic, and rapidly progressive. GN are independent nosological entities, but can also occur with many systemic diseases: systemic lupus erythematosus, hemorrhagic vasculitis, subacute bacterial endocarditis, etc.

In the etiology of GN, infections (most clearly in acute poststreptococcal GN), toxic substances (organic solvents, alcohol, mercury, lead, etc.), exogenous antigens acting with the involvement of immune mechanisms, including within the framework of immediate-type hypersensitivity (atopy), and rarely endogenous antigens - DNA, uric acid, tumor (Table 1) - play a role. The etiological factor can be established in 80-70% of patients with acute GN and in 5-10% of patients with chronic GN.

In other patients, the cause of the disease remains unknown. In the vast majority of cases, GN develops with the participation of immune mechanisms. In acute post-streptococcal GN, this is the formation of antibodies 10-12 days after pharyngitis or tonsillitis; in chronic GN, this is more often the slow formation of immune complexes containing an antigen and antibodies to it, deposited in the glomeruli when the mechanisms for their removal are insufficient; in most patients with rapidly progressing GN, this is the formation of antibodies to glomerular tissue (the basement membrane of the capillaries). Immune complexes and antibodies to the basement membrane can be detected

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and identified in kidney tissue during immunohistochemical examination. The renal glomerulus responds to immune damage with two types of pathological reactions: proliferation of renal glomerular cells (mesangial , endothelial, epithelial) and the production of intercellular substance by these cells. Both processes stimulate cytokines (primarily interleukin-1, tumor necrosis factor, platelet-derived growth factor, transforming growth factor beta), which are secreted by cells infiltrating the renal glomerulus. The development of glomerulonephritis is always associated with a chronic or acute infection localized in various organs and, as a rule, of a streptococcal nature. Most often, glomerulonephritis develops against the background of Staphylococcus aureus , Neisseria meningitidis , streptococcus pneumoniae , Toxoplasma gondii , plasmodia malaria infection, and invasion of some viruses. In some cases, glomerulonephritis develops as a result of vaccination, chemical poisoning, or consumption of products containing preservatives.

The leading immunopathological process in glomerulonephritis is considered to be the formation of so-called immune complexes in the blood or kidneys. Moreover, the main antigen is usually endostreptolysin A of nephritogenic streptococci. At the very beginning of the disease, a typical picture is formed in the blood: an increase in immune complexes and a decrease in C3 complement, while C1, C2 and C4 remain normal. Plus, an increase in antibodies to O-anti- streptolysins (streptolysin-0), anti- NADase B (deoxyribonuclease B) or anti- NADase (nicotinamide adenine nucleotidase) of streptococci is detected in the blood serum. During a kidney biopsy at the first stage of the disease, approximately from the 28th day to the 42nd day, glomerular damage from 80 to 100% is detected in the examined material. lumpy granular deposits of immunoglobulin G and C3 complement are formed along the basement membranes of the glomerular capillaries and mesangium . A tubulointerstitial component is detected in a third of patients. It should be noted that in the acute form of the disease, a characteristic picture of proliferative endocapillary glomerulonephritis . However, after a maximum of two and a half months, these deposits are no longer detectable. However, the thickness of the mesangial matrix and the number of mesangial cells can remain high for several years. Almost anyone can get glomerulonephritis, but men under forty and children are most susceptible to it. At the same time, in children, glomerulonephritis is the most common of all kidney diseases, which leads to the development of renal failure and / or early disability and is second in prevalence, second only to urinary tract infections.

Symptoms of glomerulonephritis

Acute diffuse glomerulonephritis develops 6-12 days after an infection, usually streptococcal (angina , tonsillitis, pyoderma); the most nephritogenic is group A β-hemolytic streptococcus, especially strains 12 and 49. The following symptoms are characteristic:

- hematuria (often macrohematuria);
- swelling;
- oliguria;
- increased blood pressure.

In children, acute glomerulonephritis usually has a cyclical course, with a rapid onset, in most cases ending in recovery. In adults, a latent variant with changes in urine without general symptoms is more common, gradually becoming chronic. The first signs of acute glomerulonephritis appear 1-3 weeks after an infectious disease or exposure to other factors. The disease begins with general weakness, headache , nausea, back pain, chills , loss of appetite. There

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may be increases in body temperature to very high numbers. Pallor of the face, swelling of the eyelids, a sharp decrease in the amount of urine excreted are noted. A decrease in urine volume can last 3-5 days, after which diuresis increases, but the relative density of urine, according to analysis data, decreases.

Another characteristic sign is the presence of blood in the urine - hematuria. Urine takes on the color of "meat slops" or becomes dark brown or black. In cases of microhematuria, the color of urine may not change. At the beginning of the disease, fresh erythrocytes predominate, later mainly leached ones are released.

Edema is one of the most characteristic symptoms of glomerulonephritis. It is usually located on the face, appears in the morning, and decreases by evening. Before visible edema develops, about 2-3 liters of fluid can be retained in the muscles and subcutaneous tissue. Edema is more difficult to detect in obese preschool-age children; sometimes it is determined only by some compaction of the subcutaneous tissue. Hypertension (increased blood pressure) is observed in about 60% of cases of the disease. In severe cases of glomerulonephritis, increased blood pressure can last for several weeks. Cardiovascular damage in acute cases of glomerulonephritis is observed in 80-85% of children.

There may be an enlargement of the liver, changes in the function of the central nervous system. With a favorable course of the disease and timely diagnosis and treatment, edema disappears in 2-3 weeks, blood pressure normalizes. Recovery from acute glomerulonephritis usually occurs in 2-2.5 months.

There are two most characteristic forms of acute glomerulonephritis:

1. Cyclic form (starts violently)
2. The latent form (characterized by a gradual onset) is common, and its diagnosis is of great importance, since in this form the disease often becomes chronic.

Any acute glomerulonephritis that does not end without a trace within a year should be considered as having become chronic. The following clinical forms of chronic glomerulonephritis are distinguished :

1. The nephrotic form is the most common form of primary nephrotic syndrome.
2. Hypertensive form. For a long time, arterial hypertension predominates among the symptoms, while the urinary syndrome is poorly expressed.
3. Mixed form. In this form, nephrotic and hypertensive syndromes are present simultaneously.
4. Latent form. This is a fairly common form; it usually manifests itself only as a mild urinary syndrome without arterial hypertension and edema.

hematuric form is also distinguished , since in some cases chronic glomerulonephritis can manifest itself as hematuria without significant proteinuria and general symptoms. All forms of chronic glomerulonephritis may periodically give relapses, very reminiscent of or completely repeating the picture of the first acute attack of diffuse glomerulorephritis . Exacerbations are especially often observed in the fall and spring and occur 1-2 days after exposure to an irritant, most often a streptococcal infection.

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