

## GLOMERULONEPHRITIS

Xolmurotova Arofat Baxrom qizi  
Tashkent Pediatric Medical Institute  
3rd year 2 student of Pediatrics

Scientific leader Dzhabbarova Akida Mannapovna

### Abstract:

This article presents ideas about the development, causes, symptoms, diagnosis, treatment, and complications of glomerulonephritis.

**Keywords:** glomerulonephritis, kidney, tissue, oliguria, micromacrohematuria.

Glomerulonephritis (Greek: glomerulo - kidney ball, nephritis - kidney inflammation, glomerular nephritis) is an immunoinflammatory kidney disease, which is mainly caused by damage to the kidney balls. Interstitial tissue and renal tubules are sometimes involved in the process. Glomerulonephritis can occur as an independent disease or develop in certain systemic diseases (infective endocarditis, hemorrhagic vasculitis, systemic erythematosus). The clinical picture of glomerulonephritis consists of urinary, edema and hypertensive syndromes. In glomerulonephritis, urinalysis, Zimnitsky and Reberg samples, renal ultrasound and Doppler ultrasound of renal vessels have diagnostic value.

In many cases, the development of glomerulonephritis is associated with an excessive immune response of the body to antigens of an infectious nature. In addition, there is an autoimmune form of glomerulonephritis, in which kidney damage occurs as a result of the destructive effect of autoantibodies. In glomerulonephritis, antigen-antibody complexes accumulate in the capillaries of the kidney balls, as a result of which the primary urine production process is disturbed, water, salts and metabolic products are retained in the body, and the level of antihypertensive factors decreases. All this leads to the development of arterial hypertension and kidney failure.

Glomerulonephritis in children ranks second among acquired kidney diseases after urinary tract infection. According to statistics of urology, glomerulonephritis is the most common cause of premature disability of patients due to the development of chronic kidney failure. Acute glomerulonephritis can develop at any age, but usually the disease occurs in patients under 40 years of age.

Symptoms of acute diffuse glomerulonephritis often appear one or three weeks after an infectious disease caused by streptococci (angina, pyoderma, tonsillitis). Three main symptoms are characteristic for acute glomerulonephritis:

Urine (oliguria, micro- or macrohematuria);

Swelling;

Hypertonic.

Acute glomerulonephritis in children develops rapidly, passes cyclically and usually ends with recovery. When acute glomerulonephritis develops in adults, the disease manifests itself in a dim form, characterized by changes in urine, the absence of general symptoms, and a tendency to transition to a chronic form. Glomerulonephritis begins with an increase in body temperature (there is a possibility of severe hyperthermia), general weakness, nausea, loss of appetite, headache and pain in the back. The patient turns pale, his eyelids are swollen. In acute glomerulonephritis, a decrease in diuresis is observed in the first 3-5 days. Then the amount of urine excreted increases, but its relative density decreases. Another permanent and indispensable sign of glomerulonephritis is hematuria (blood in the urine). Microhematuria develops in 83-85% of cases. In 13-15% of cases, macrohematuria can develop, in which the color of "meat wash" is typical for urine, sometimes it can be black or dark brown. One of the most obvious symptoms of glomerulonephritis is swelling of the face, which is expressed in the morning and decreases during the day. It should be noted that the accumulation of up to 2-3 liters of fluid in the muscles and subcutaneous fat can occur without swelling. In chubby children of preschool age, the only sign of swelling is sometimes thickening of the subcutaneous tissue. 60% of patients with acute glomerulonephritis develop hypertension, and in severe cases it can last for several weeks. In 80-85% of cases, acute glomerulonephritis causes damage to the cardiovascular system of children.



Central nervous system dysfunction and liver enlargement are possible. There are two main variants of acute glomerulonephritis:

Typical (cyclic). Rapid onset and acute manifestation of clinical symptoms are characteristic;

Hidden (acyclic). It is an obscure form of glomerulonephritis, characterized by a gradual onset and a weak expression of symptoms. Due to its late diagnosis and tendency to transition to chronic glomerulonephritis, it poses a significant risk.

If acute glomerulonephritis goes well, timely diagnosis and treatment, the main symptoms (edema, arterial hypertension) will disappear within 2-3 weeks. Complete recovery is noted after 2-2.5 months.

The following variants of chronic glomerulonephritis are distinguished:

Nephrotic (urinary symptoms predominate);

Hypertensive (increased blood pressure is noted, urinary syndrome is weakly expressed);

Mixed (combination of hypertensive and nephrotic syndromes);

Latent or hidden (a very common form characterized by the absence of arterial hypertension and tumors in weakly expressed nephrotic syndrome);

Hematuric (the presence of erythrocytes in the urine is noted, there are no or weakly expressed other symptoms). Relapse is characteristic for all forms of glomerulonephritis. Clinical symptoms of an attack of the disease are similar to the first episode of acute glomerulonephritis or completely repeat it. The probability of relapse increases in the spring-autumn period and begins 1-2 days after the trigger.

Latent or hidden (a very common form characterized by the absence of arterial hypertension and tumors in weakly expressed nephrotic syndrome);

Hematuric (the presence of erythrocytes in the urine is noted, there are no or weakly expressed other symptoms). Relapse is characteristic for all forms of glomerulonephritis. Clinical symptoms of an attack of the disease are similar to the first episode of acute glomerulonephritis or completely repeat it. The probability of relapse increases in the spring-autumn period and begins 1-2 days after the trigger.



## References

1. Monarch Disease Ontology release 2018-06-29sonu — 2018-06-29 — 2018.
2. А.В.Жаров, В.П.Шишков и др. Патологическая анатомия сельскохозяйственных животных. — М.: Колос, 1995.
3. Д.О.Журов, И.Н.Громов. Морфологические изменения в почках цыплят при нефрозно-нефритной форме инфекционного бронхита (other). — 2021. — ISSN 2078-0109. Архивировано 19 марта 2023 года.
4. Д.О.Журов, И.Н.Громов, А.С.Алиев, А.К.Алиева. Патоморфологическая и дифференциальная диагностика болезней кур, протекающих с поражением почек. — Витебская государственная академия ветеринарной медицины, 2017. — ISBN 978-985-512-943-2. Архивировано 19 марта 2023 года.
5. Фозиноприл у детей. Руководство UptoDate. <https://www.uptodate.com>.
6. Эналаприл у детей. Руководство UptoDate <https://www.uptodate.com>.