

## GLOMERULONEPHRITIS IN CHILDREN: RISK FACTORS, COURSE, PROGNOSIS

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### **Annotation**

The nephritic form of GN proceeds with maximum, moderate and minimal activity, the manifestations of which determine the severity of hematuria, proteinuria, arterial hypertension, edema, and impaired renal function. Several risk factors play a role in the development of the nephritic form of GN: perinatal factors, gastrointestinal diseases, giardiasis, chronic foci of infection, heredity aggravated by renal pathology or arterial hypertension, damage to the kidney tissue long before the development of GN, which is manifested by minimal urinary syndrome. A combination of various risk factors determines the severity of kidney damage. The etiological factor in the manifestation of the nephritic form of GN is most often  $\beta$ -hemolytic streptococcus.

**Keywords:** Children, glomerulonephritis, risk factors, connective tissue, dysplasia, immunology.

### **Introduction**

Glomerulonephritis (GN) is one of the severe kidney diseases in children, characterized by the frequent development of complications and progression to chronic renal failure (CRF) [1,2]. Many researchers attach importance to nephritogenic strains of streptococcus in the development of GN [2,3]. However, the reasons for the development of various clinical manifestations, the severity of the disease in individual children remain unclear.

**The aim of the study** was to assess the role of risk factors on the course and outcome of post-streptococcal AGN in children.

**Materials and research methods.** Under observation were 230 children aged 2–17 years with AGN, manifested by the nephritic form. Research methods included parental questionnaires, urinalysis, clinical and biochemical blood tests, examination of the functional state of the kidneys, hemostasiogram, immunogram (immunoglobulins of three classes, circulating immune complexes - CEC, complement), antistreptolysin O titer (ASL-O), throat swab and from the skin (with streptoderma), instrumental methods: ultrasound examination of the

abdominal organs and kidneys, ECG, according to indications esophagogastroduodenoscopy (EGDS).

**Results.** The factors immediately preceding the development of GN in 26.1% of children were acute tonsillitis, 20.3% - streptoderma, 1.3% - purulent otitis media, 2.17% - pneumonia, 25.83% - acute respiratory viral infection. 12.5% had multiple dental caries. In 38 (16.5%),  $\beta$ -hemolytic streptococcus was isolated from the pharynx. In 210 (91.3%), an increase in the ASL-O titer in the blood serum was found. According to the clinical and laboratory manifestations of the disease, 3 variants of the course of GN were identified: 1) with maximum activity of the renal process (58 children - 25.2%), 2) with moderate activity (141 patients - 61.3%), 3) with minimal activity (31 children - 13.5%). In variant I of the course of AGN in children, edema was observed on the face, trunk and extremities, increased blood pressure (BP) (within 140/90–185/105 mm Hg), macrohematuria. Urinary syndrome was manifested by proteinuria (1–2 g/l), erythrocyturia - all over the entire field of view. Gross hematuria persisted for 10–12 days, arterial hypertension for 8–15 days, and edema for 9–11 days. In variant II, edema was localized mainly on the face, disappeared on the 5<sup>th</sup>–6<sup>th</sup> day, blood pressure was in the range of 135/90–150/95 mm Hg. Art., returned to normal on the 6–8<sup>th</sup> day, changes in urine tests were in the form of proteinuria  $0.99 \pm 0.13$  g/l, erythrocyturia in large quantities. In variant III, there was a slight increase in blood pressure (up to 130/85 mm Hg) within 1–2 days, pastosity on the eyelids, urinary syndrome (proteinuria - from traces to 0.66 g/l, erythrocytes in large numbers). Patients with maximal and moderate GN activity had statistically significantly lower levels of total protein, higher levels of urea and serum creatinine than their levels in children with minimal activity and in healthy children. Serum cholesterol levels were slightly elevated only in children with maximal GN activity. Immunogram parameters in patients with AGN statistically significantly differed from those in children of the control group. The highest levels of IgA and IgM, CEC and lower levels of IgG were detected in children with the maximum activity of the renal process. All patients with AGN showed an increase in ASL-O titer, the most pronounced increase in its level was noted at maximum activity. Acute renal failure was detected in 47 (19.6%) children with AGN at the onset of the disease, of which 31 (53.4%) had a maximum activity and 16 (11.3%) had a moderately pronounced activity of the renal process. With minimal GN activity, none of the children showed renal failure, however, in some patients there was a violation of the partial functions of the kidneys. Kidney dysfunction in 57.9% of children with AGN was manifested by a decrease in glomerular filtration, in 53.7% - a decrease in concentration function, in 41.7% - an increase in the level of urea, in 39.2% - an increase in the level of serum creatinine. The level of urea ( $r=+0.53$ ) and serum creatinine ( $r=0.47$ ) closely correlated with the degree of AGN activity.

**Conclusions.** The nephritic form of AGN proceeds with maximum, moderate and minimal activity, the manifestations of which determine the severity of hematuria, proteinuria, arterial hypertension, edema, and impaired renal function.

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