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ACUTE GLOMERULONEPHRITIS IN CHILDREN: OPTIMIZING TREATMENT OF ITS VARIOUS FORMS

M.Sh. Ganieva

Department of Hospital and Emergency Pediatrics,

Andijan State Medical Institute

ABSTRACT: Objective: The aim of this study is to determine optimal approaches to the treatment of various forms of glomerulonephritis (GN) in children based on an analysis of morbidity in the Andijan region. Methods: The study analyzed the clinical and laboratory characteristics, prevalence, and course of acute glomerulonephritis in 126 children treated at the Andijan Regional Children's Multidisciplinary Medical Center over a 10-year period. Diagnosis was verified based on anamnesis, extrarenal symptoms (edema, hypertension), and renal symptoms (hematuria, proteinuria). Results: A link between GN and previous infection was established in 90.8% of patients, with a confirmed streptococcal etiology in 64.5%. The disease manifested as typical acute nephritic syndrome in 69.1% of patients, while 22% presented with an atypical onset. Treatment involved a combination of antibiotics, anticoagulants (heparin), and antiplatelet agents. For patients with Nephrotic Syndrome (NS), corticosteroid therapy was the standard; however, due to high relapse rates and steroid resistance, cytostatics (such as 6-mercaptopurine) and pulse therapy were employed for optimizing outcomes. Additionally, specific therapeutic regimens were identified for hepatitis-associated nephritis (using interferons) and cases associated with tuberculosis infection. Conclusion: The study indicates an increase in the number of patients with GN accompanied by nephrotic syndrome, hematuria, and hypertension. Effective management requires an individualized selection of basic therapy, including corticosteroids and cytostatics, as well as screening for hepatitis and tuberculosis to rule out secondary causes.

Keywords: Acute glomerulonephritis, children, nephrotic syndrome, corticosteroids, cytostatics, hematuria, streptococcal infection, treatment optimization, Andijan region.

INTRODUCTION

Glomerulonephritis (GN) ranks among the most serious kidney diseases occurring in childhood in terms of prognosis. Concepts regarding the etiology and pathogenesis of GN have undergone significant changes in the past decade. Currently, most clinicians understand AGN as an immune complex lesion of the glomerular apparatus of the kidneys, which develops after a bacterial or viral infection, or against the background of autoimmune diseases.

In the typical course of AGN, it most often manifests itself as acute nephritic syndrome (arterial hypertension, edema, macro- and microhematuria, minor proteinuria), as well as impaired renal function in the acute period of the disease, up to acute renal failure (ARF). The clinical picture may also include cardiovascular changes, angiospastic retinopathy, and encephalopathy, which are associated with volume overload and pose a life-threatening risk. In patients with atypical OPIGN, extrarenal manifestations are absent or very minor and short-lived [1,4,6]. The course of

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glomerulonephritis is determined primarily by its morphological basis. Therefore, the treatment approach is based on immunomorphological data, clinical findings, and the outcomes of previous treatments [2, 3, 5]. It is known that the outcome of GN is determined by the severity of clinical symptoms, particularly edema, hematuria, and hypertension. In recent years, according to literature data and an analysis of childhood morbidity in the Andijan region (according to data from the nephrology department of the Andijan Regional Children's Medical Center).

Objective of the study: To determine optimal approaches to the treatment of various forms of glomerulonephritis in children**.**

MATERIAL AND METHODS

We analyzed the prevalence, clinical and laboratory characteristics, and course of acute gastritis in 126 children treated at the Andijan Regional Children's Multidisciplinary Medical Center over the past 10 years. The diagnosis of AGN was verified based on the anamnesis (the connection of the disease with a previous infection, hypothermia, the absence of any kidney diseases in the past, as well as hereditary and congenital renal pathology in blood relatives), identification of extrarenal (edema and arterial hypertension) and renal symptoms (hematuria, proteinuria and oliguria).

RESULTS AND DISCUSSION

There were, on average, 1.4 times more boys than girls—89 (57.9%) and 37 (42.1%), respectively. The age at the time of illness was as follows: 15.3% of patients were under 7 years old, 42% were 7–12 years old, and 42.7% were over 12 years old. A link between glomerulonephritis and a previous infection was demonstrated in 89 of 126 (90.8%) patients. Combined bacteriological and serological studies confirmed the streptococcal etiology of GN in 191 of 296 (64.5%) patients. In this case, streptoderma was detected in 97 (29.8%) children, acute streptococcal tonsillitis in 81 (24.9%) patients, scarlet fever in 20 (6.1%) patients. In 22 (6.7%) children, the etiology of the primary infectious process could not be established, Although the nature of the infection and the elevated titer of antistreptococcal antibodies (antistreptolysin-O) in the blood suggested a streptococcal etiology for these diseases. Thus, 10 children showed signs of submandibular lymphadenitis, 6 had sinusitis, 4 had pulpitis, and 2 had otitis. When acute streptococcal infection of the skin and oropharynx occurred in children, the treatment, as a rule, did not follow the 10-day regimens of antibacterial therapy with penicillins or macrolides followed by mandatory bicillin prophylaxis. In the case of streptoderma, treatment errors were the most common: antibiotics were prescribed rarely and for shortened courses; local therapy was used more often, while bicillin prophylaxis of immune complex complications was not carried out.

In 10 other patients (3.1%), the development of AGN was preceded by acute diarrheal syndrome of unspecified etiology. Yersiniosis was diagnosed and laboratory-confirmed in one patient.

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Regarding ARI, it preceded the onset of nephritic syndrome in 56 children (20.3%), characterized by fever and symptoms of intoxication.

Hypothermia as a trigger for the development of GN occurred in 30 (9.1%) children. However, most of these patients also showed a significant increase (1.5-2 times higher than normal) in antistreptococcal antibody levels in their blood tests. This allows us to speak about the presence of a latent streptococcal infection, during the course of which the above conditions could play the role of a trigger factor for glomerulonephritis.

In 58 of 126 (69.1%) patients, GN had a typical course, with the development of acute nephritic syndrome. The "clear" period between the onset of the first symptoms of the disease and the preceding infection ranged from 10 to 28 days. It was longest in cases of streptoderma and relatively short in cases of acute respiratory infections.

Edema in the acute period of glomerulonephritis was recorded in all 126 children; in 48 of them (14.7%) it was not only widespread, but also significant, up to the appearance of hydrothorax, ascites, and free fluid in the pericardial cavity.

Arterial hypertension (AH) was observed in 59 (48.8%) patients, in 42 of them systolic blood pressure (SBP) was increased to 130–150 mm Hg, diastolic (DBP) – to 90–95 mm Hg. However, in 12 (10.7%) observations AH progressed more malignantly, reached 170–180/100 mm Hg and more, was torpid to the antihypertensive drugs used, contributing to the development of such a serious complication as angiospastic encephalopathy (renal eclampsia).

The development of renal eclampsia was preceded by insomnia, headache, nausea, repeated vomiting (28.8%), lumbar pain in 32.5% of children, bradycardia, which was subsequently joined by anxiety, tonic-clonic seizures, and depression of consciousness.

During the advanced stages of the disease, moderate dilution anemia, leukocytosis with a neutrophilic shift, and an increase in ESR to 20–30 mm/hour were observed. Dysproteinemia due to moderate hypoalbuminemia (at least 35 g/L) and hyperglobulinemia were also observed in 23% of cases.

In 84 (71.8%) patients, a transient (within 1–2 weeks) increase in blood urea levels to 9–15 mmol/l and creatinine to 110–140 μmol/l was recorded, which was considered as an acute renal dysfunction. In cases of severe hypervolemia and oliguria, relative hyponatremia and hyperkalemia were observed, which, like azotemia, were eliminated as diuresis was restored.

Urine sediment in 100% of cases showed moderate proteinuria (no more than 50 mg/kg per day), macro- or microhematuria, and abacterial leukocyturia. Urine specific gravity was normal in all patients during the acute phase of the disease.

In 28 of 126 (22%) patients, GN had an atypical onset, characterized only by slight eyelid swelling and predominantly by abnormal urine sediment in the form of microhematuria (68% of cases), less commonly macrohematuria (32% of cases), and proteinuria up to 0.5–1 g/L. However, their primary renal functions were preserved.

Treatment of patients with AGN was carried out taking into account existing recommendations and included bed rest, a salt-free diet with limited animal proteins until the elimination of

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extrarenal symptoms and azotemia, and penicillin antibiotics. Concurrently, agents aimed at correcting coagulation disorders were used. Unfractionated heparin was administered at 200–300 U/kg per day for 3–4 weeks, followed by gradual tapering. Along with direct anticoagulants, antiplatelet agents were used to improve blood rheology.

Arterial hypertension, hyperkalemia, oliguria, pre- and eclampsia served as the basis for the prescription of loop diuretics (furosemide) and antihypertensive drugs.

During the implementation of the specified therapy, extrarenal manifestations of AGN were stopped in a relatively short time - in 58.9% of children within 7-10 days and in 41.1% - 14-15 days after the start of treatment, which is generally characteristic of the typical form of acute glomerulonephritis.

It was established that the average age of these children at the onset of the disease was 13 ± 0.8 years (minimum age 7 years, maximum 15 years). Moreover, a link between GN and a previous acute infection was proven in 100% of cases, but with streptococcal diseases – only in 16 (42.1%) patients. In 20 patients (52.6%), glomerulonephritis was manifest at onset, with renal dysfunction in the acute phase; in the remaining 18 (34.2%), it was asymptomatic (atypical). Also noteworthy was the duration of macrohematuria—11–14 days—in 28 patients (73.7%), and the severity of proteinuria in the acute phase—2.5–3 g/L—in 55.3% of children.

The results show a marked increase in the incidence of glomerulonephritis in children with nephrotic syndrome (NS) and congenital nephrotic syndrome. NS is characterized by alternating relapses and remissions and high sensitivity to corticosteroids (CST) [8,9,11,12]. Although the literature reports a reasonable rate (up to 50%) of spontaneous remission, in our practice this rate was significantly low. According to the classification of NS, there are hormone-sensitive, non-relapsing, chronically relapsing, frequently relapsing, and steroid-resistant variants [13, 14,15,16].

Since the standard treatment regimen for a child with newly diagnosed nephrotic syndrome included corticosteroids, we used prednisolone at a dose of 1.5-2 mg/kg for up to 3-4 weeks as the basic drug in the treatment of these children. Remission was observed in 90% of patients, and the prednisolone dose was then reduced to a maintenance dose of 0.3-0.5 mg/kg per day, administered using an intermittent dosing regimen—3 days a week on and 4 days off. However, relapses were observed in 75% of patients with this regimen. Prednisolone administered every other day for up to six months proved more effective, even after remission had occurred. Early discontinuation of prednisolone increased the risk of relapse, possibly due to steroid suppression of the pituitary-adrenal axis and relative adrenal insufficiency.

Based on literature and our own data, the lack of response to glucocorticoid therapy is only partially associated with a violation of its pharmacokinetics. The absence of dynamics during an 8-week course of prednisolone therapy allows us to consider these patients as steroid-resistant and, unlike children with nephrotic syndrome with minimal changes, their morphological changes are different. However, in 50% of cases, a late response to treatment with prednisolone was observed, and in 30%, the response occurred against the background of treatment with immunosuppressants.

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Based on the results of a one-year follow-up, 35% of patients experienced two relapses, 25% experienced one, and 19% had no relapse. Among patients with frequent relapses, the first relapse occurred within 6 months. They were prescribed prednisolone on an alternating schedule, i.e., every other day at a dose of 0.3-0.5 mg/kg per day for up to 6 months followed by a reduction and complete discontinuation over 2-3 months. In 5 patients who failed to respond to prednisolone, we used pulse therapy, i.e., prednisolone injections at 15-30 mg/kg for 3 days. However, hypertension was observed as a side effect in 2 children. Hormone resistance in patients warranted the inclusion of cytostatics in the treatment regimen, including cyclophosphamide or chlorambucil (chlorbutin) [1,2,3,11,15], among others. Of the cytostatics used, azathioprine caused side effects such as nausea, vomiting, and dizziness; children tolerated 6-mercaptopurine better. The use of prednisolone in combination with prednisolone led to the onset of remission (as a result of the transformation of steroid-resistant variants into steroid-sensitive ones) or to an extension of remission. The initial dose of 6-Mercaptopurine – 2-4 mg/kg per day – was administered for 8 weeks, then reduced to half the dose and continued for another 8 weeks. The duration of remission ranged from 1 to 5 years. We also used levamisole as a non-specific T-lymphocyte stimulator at a dose of 2.5 mg/kg every other day. Since the use of the latter is often accompanied by leukopenia (neutropenia), we performed weekly blood tests. Analysis of the observation results showed a 28% reduction in the relapse rate.

In 25% of the patients we observed, complications of corticosteroid therapy such as secondary Itsenko-Cushing syndrome, hirsutism, hypertension, infections, etc. were observed. Although, according to C. Ponticelli and P. Passerini (2), pulse therapy with methylprednisolone for NSMI in adults is effective in 100% of cases, in 30% of children the response was negative. At the same time, 20% of patients, although remission was achieved with pulse therapy, experienced relapses 3-4 weeks after completion of treatment without a maintenance dose of prednisolone. In 2% of patients with nephrotic syndrome who did not respond to corticosteroids 3-4 weeks after the start of treatment, the "butterfly" sign, hepatomegaly, cardiomegaly, and carditis were observed, which are known to be signs of SLE or other collagen diseases. These children continued their examination and treatment in the cardiology and rheumatology department. In all these cases, LE cells were not detected. According to our clinic's data, 30% of patients with nephrotic syndrome relapsed within 7-8 years, and 12% relapsed within 10 years, although the incidence of relapses decreased over time. Ten percent of patients developed so-called acquired resistance to CST, i.e., a condition characterized by frequent relapses. According to the literature, this is associated with the development of FSGS. At the same time, 13% of patients, on the contrary, responded to lower doses of steroids compared to standard [3, 6, 8, 10].

Long-term treatment with CST, especially with frequent relapses, is difficult due to the large number of side effects: 35% of patients have osteoporosis, hypocalcemic and hypokalemic syndrome, 2 patients have psychosis, 8 have growth retardation, 40% have arterial hypertension, 90% have Cushing's syndrome, et c. When the severity of side effects increases, the question of continuing or discontinuing CST becomes increasingly pressing: to leave only symptomatic treatment (diuretics, sodium restriction, etc.), counting on spontaneous remission, or to obligatory include cytostatic drugs: azathioprine, 6-Mercaptopurine, chlorbutin, etc. [5,7,12]. In 56% of cases, we decided in favor of the latter.

Although there are significant treatment challenges for patients with nephrotic syndrome and their tendency toward relapse, the overall prognosis is favorable in most cases. According to J.

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Cameron et al. (4), patient survival reaches 10 years. In 95% of cases, the causes of death were infections, cardiovascular and pulmonary diseases, and chronic renal failure. We observed the latter in 28% of patients.

An analysis of the incidence patterns in the nephrology department of the Regional Medical and Medical Center revealed that one of the most common forms of acute nephropathy (ANP) over the past five years is nephrotic syndrome with hematuria and hypertension. Its pathogenesis is based on an immune complex mechanism that develops in response to toxins from nephritogenic strains of β-hemolytic streptococci.

The diagnosis of the disease was confirmed by determining the titer of antibodies to streptolysin-O (O-antistreptolysins), nicotinamide adenine nucleotidase (anti-NADase) and deoxyribonuclease-B (anti-DNase) of streptococci in the blood. In the clinical presentation of the disease, the predominant renal symptoms are oliguria, hematuria, pain in the lumbar region, abdominal pain, and azotemia; extrarenal symptoms include swelling, typically localized in the eyelids and face in the morning, headaches, increased blood pressure, and pallor. Treatment for this category of patients also presents certain difficulties.

We use the following antibacterial therapy regimen: penicillin and semisynthetic penicillins for 2-3 short 7-10-day courses, followed by a transition to bicillin. Antihistamines and medications that improve renal blood flow (heparin, curantil, and trental) are used for pathogenetic therapy.

Symptomatic therapy: diuretics for severe edema, hypertension (furosemide, uregit, amiloride, triampur, etc.), antihypertensive drugs (papaverine, dibazol, reserpine, capoten, captopril) for persistent and severe hypertension.

Of the 30% of cases of hepatosplenomegaly observed in patients, HbSAg was detected by ELISA in 10%. Based on literature data on so-called hepatitis-associated nephritis, we included interferons, immunomodulin 0.5-1 ml intramuscularly, cobavit, lipoic acid, and Essentiale Forte in the treatment regimen for these patients. The prognosis for the disease is 85% recovery, 15% chronicity, and 1.8% fatality.

Three patients with AGN and hematuria had a history of contact with tuberculosis patients, as well as positive Mantoux tests and X-ray signs of bronchial adenitis. In all cases, urine tests for bronchial tuberculosis were negative. In consultation with a pediatric phthisiologist, these patients were prescribed a course of treatment with specific anti-tuberculosis drugs. The outcome of the disease is recovery in 90% of cases.

CONCLUSION

Based on the above research results, we made the following conclusions**:**

1. The number of patients with glomerulonephritis with nephrotic syndrome, hematuria, and hypertension has increased in recent years;

2. A regimen of basic therapy—corticosteroids and cytostatics—must be individually selected for each specific case of nephrotic syndrome.

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3. Identification of so-called hepatitis-associated nephritis and the prescription of specific therapy for these patients;

4. Hematuric variants of glomerulonephritis require a more in-depth study of the causes of the disease to exclude tuberculosis infection and collagenoses.

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