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Assessment of the Functional State of the Kidneys in Children Against the Background of Community-Acquired Pneumonia

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Abstract Acute respiratory infections, including pneumonia, often lead to kidney damage, which often determines the course and prognosis of the primary disease. [3,7] Patients with community-acquired pneumonia, the diagnosis of AKI may be difficult due to the lack of information on premorbid serum creatinine levels. [10] A short episode of AKI in terms of long-term prognosis is associated with an increased risk of developing CKD. [8] In our research, we investigated the prevalence and prognostic factors of AKI in children with community-acquired pneumonia.

Keywords Community-acquired pneumonia, Acute kidney injury (AKI), Chronic kidney disease (CKD), Serum creatinine (Cr) and cystatin C (CysC)

1. Introduction

Pneumonia is the leading single infectious cause of death among children worldwide. According to WHO, 740,180 children under five years of age died from pneumonia in 2019, accounting for 14% of all deaths in children under five years of age, and among children aged one to five years, pneumonia accounted for 22% of all deaths. [1] In recent years, morbidity and mortality due to pneumonia have been increasing, while acute kidney injury (AKI) remains one of the causes of high mortality in pneumonia. The kidneys are sensitive to hypoxia, ischemia, infectious-toxic influences, nephrotoxic effects of drugs and disorders of systemic hemodynamics. Therefore, during severe community-acquired pneumonia (CAP), there is a high probability of developing renal dysfunction. [7]

2. Aims

The main purpose of our study was to assess the functional state of the kidneys in children with community-acquired pneumonia.

3. Method

Our research was conducted at the 1st City Children's Clinical Hospital of Tashkent in the period 2024-2025. The study included children aged 5 to 7 years with community-acquired pneumonia, the main group (MG) of 120 patients, and the control group (CG) of 20 healthy children. The diagnosis of community-acquired pneumonia was made based on clinical symptoms and chest radiography. All study participants had their serum creatinine (Cr) and cystatin C (CysC) levels measured. Acute kidney injury (AKI) was defined as an increase in serum creatinine concentration of 26.5 μmol/L (0.3 mg/dL) within 3 days; or increase in serum creatinine to ≥1.5 times baseline within the prior seven days; or urine volume ≤0.5 mL/kg/hour for six hours (Kidney Disease Improving Global Outcomes (KDIGO) classification, 2012.) [2]

4. Results

The results of the study (Table 1) showed that the initial blood creatinine concentration in children with community-acquired pneumonia was comparable to that of healthy children (46.85±12.46 μmol/L vs. 42.40±9.76 μmol/L, the difference was not significant). At the same time, a different pattern was observed in the analysis of serum cystatin C concentration: overall, the average cystatin C level in the serum of children with pneumonia was significantly higher than that of the control group (0.88±0.12 mg/L vs. 0.52±0.09 mg/L, p<0,001) Quantitative analysis showed that the cystatin C concentration in the blood of 39 (32.5%) children with pneumonia exceeded the control values, with an average of

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0.95±0.24 mg/L. Among them, 5 children had cystatin C levels exceeding the reference values (1.2 mg/L), with an average of 1.25±0.11 mg/L. Thus, already from the first day, nearly a third (32.5%) of children with community-acquired pneumonia showed signs of kidney dysfunction, according to the serum cystatin C concentration level.

Table 1. Indicators of kidney function in children with community-acquired pneumonia during the first days of hospitalization

Group Indicator	Creatinine, µmol/L	Cystatin C, mg/L
Community-Acquired Pneumonia (n=120)	46.85±12.46	0.88±0.12**
Control Group (n=20)	42.40±9.76	0.52±0.09

Note: * - statistical significance compared to the control group (KG).
One sign – p<0.05, two sign – p<0.01, three sign – p<0.001.

It should be noted that creatinine and cystatin C are endogenous markers of impaired glomerular filtration rate (GFR) in the kidneys. [2,4] Determining serum creatinine concentration and its dynamics is the most common method for assessing kidney function. Creatinine is freely filtered in the glomerular apparatus, with no reabsorption or metabolism occurring in the kidneys. Creatinine clearance is used to calculate GFR. [8] Cystatin C is synthesized by all nucleated cells at a constant rate and has 100% clearance. Since the concentration of cystatin C in serum (sCysC) and urine (uCysC) is not dependent on age, gender, diet, hydration, muscle mass, or metabolic characteristics of the patient, it can be used to more accurately assess renal concentrating ability compared to serum creatinine levels. [4,10]

Daily determination of blood creatinine concentration in children with community-acquired pneumonia allowed identifying those with acute kidney injury (AKI). The study results showed that acute kidney injury was detected in 27 (22.5%) children, of which 20 (74.1% of all children with AKI) were diagnosed with stage 1 AKI, and 7 children (25.9% of all children with AKI) were diagnosed with stage 2 AKI.

In the present study, we analyzed the relationship between the development of acute kidney injury (AKI) and the baseline levels of creatinine and cystatin C in the blood of children with pneumonia (Table 2). The conducted research showed that the serum creatinine concentration did not differ between children with pneumonia who developed AKI and those without AKI (49.25±16.25 µmol/L in children without AKI and 52.36±10.10 µmol/L in children with AKI, intergroup differences – not significant). No differences were found in the creatinine concentration levels in children with pneumonia complicated or uncomplicated by AKI compared to the control group (46.85±12.46 µmol/L, 49.25±16.25 µmol/L, and 52.36±10.10 µmol/L, respectively, intergroup differences – not significant).

At the same time, the analysis showed that the average cystatin C concentration in the blood of children with pneumonia complicated by AKI was significantly higher than in the control group (1.15±0.16 mg/L vs. 0.52±0.09 mg/L, p<0.001), and also higher than in children with uncomplicated pneumonia (1.15±0.16 mg/L vs. 0.59±0.14 mg/L, p<0.01). In children with pneumonia without the development of AKI, the cystatin C concentration at admission (0.59±0.14 mg/L) did not differ from the value in the control group.

Table 2. Baseline kidney function indicators in children with community-acquired pneumonia depending on the development of acute kidney injury (AKI)

Group Indicator	Creatinine, µmol/L	Cystatin C, mg/L
Community-Acquired Pneumonia (n=120)	46.85±12.46	0.88±0.12**
Community-Acquired Pneumonia without AKI (n=93)	49.25±16.25	0.59±0.14
Community-Acquired Pneumonia with AKI (n=27)	52.36±10.10	1.15±0.16***^^
Control Group (n=20)	42.40±9.76	0.54±0.09

Note: * - statistical significance compared to the Control Group (CG),
^ - statistical significance compared to children without AKI.
One sign – p<0.05, two sign – p<0.01, three sign– p<0.001.

In our study, the median cystatin C concentration in the serum of children with community-acquired pneumonia was 0.72 mg/L. The risk of developing acute kidney injury (AKI) in patients with an initial cystatin C concentration of less than 0.72 mg/L was 13.3% (8 out of 60), while in those with a cystatin C concentration greater than 0.72 mg/L, the risk was 31.7% (19 out of 60). The relative risk was 2.38 (chi-square = 5.78, p = 0.017).

5. Conclusions

Thus, the conducted study showed that in children with community-acquired pneumonia, 32.5% (39 children) of cases exhibited impaired kidney function from the first days of the illness, manifested by an increase in serum cystatin C levels. Acute kidney injury was registered in 27 (22.5%) children. In children with pneumonia complicated by AKI, the average cystatin C concentration in the blood was significantly higher than in those without AKI (p<0.001).

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