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CLINICAL VALUE OF CARDIOSPECIFIC BIOMARKERS IN THE DETECTION OF MYOCARDITIS IN CHILDREN WITH COMMUNITY-ACQUIRED PNEUMONIA

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Key words: myocarditis, children, cardiac troponin I, community-acquired pneumonia, biomarkers.
Tayanch so‘zlar: miokardit, bolalar, yurak troponini I, jamoada orttirilgan pnevmoniya, biomarkerlar.
Ключевые слова: миокардит, дети, сердечный тропонин I, внебольничная пневмония, биомаркеры.

The assessment of cardiac troponins occupies a leading position in modern cardiology, as these biomarkers reflect cardiomyocyte injury with high sensitivity and specificity [1,2]. In pediatric practice, the diagnostic value of cardiac biomarkers is of particular importance due to the difficulty of clinical diagnosis of myocardial involvement in infectious diseases [3]. The aim of the present study is to evaluate biomarkers of myocardial injury and identify the most diagnostically significant markers in children with community-acquired pneumonia complicated by myocarditis. The obtained results demonstrate that cardiac troponin I, α-hydroxybutyrate dehydrogenase (α-HBDH), and creatine kinase MB fraction (CK-MB) are reliable indicators of myocardial involvement in children with community-acquired pneumonia and can be used to assess disease severity and prognosis [4,5].

SHIFOXONADAN TASHQARI PNEVMONIYA BILAN KASALLANGAN BOLALARDA MIOKARDITNI ANIQLASHDA KARDIOSPESIFIK BIOMARKERLARNING KLINIK AHAMIYATI

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Yurak troponinlarini baholash zamonaviy kardiologiyada yetakchi o‘rin tutadi, chunki ushbu biomarkerlar kardiomiyotsitlar shikastlanishini yuqori sezuvchanlik va spetsifiklik bilan aks ettiradi [1,2]. Pediatriya amaliyotida yurak biomarkerlarining diagnostik ahamiyati infeksion kasalliklar fonida miokard zararlanishini klinik jihatdan aniqlashdagi qiyinchiliklar sababli alohida dolzarblik kasb etadi [3]. Ushbu tadqiqotning maqsadi miokard shikastlanish markerlarini baholash hamda miokardit bilan asoratlangan jamoada orttirilgan pnevmoniyaga chalingan bolalarda ularning eng diagnostik jihatdan ahamiyatlisini aniqlashdan iborat. Olingan natijalar yurak troponin I, α-gidroksibutiratdegidrogenaza (α-GBDG) va kreatinfosfokinazaning MB-fraksiyasi (KFK-MB) jamoada orttirilgan pnevmoniyaga chalingan bolalarda miokard ishtirokini aniqlashda ishonchli ko‘rsatkichlar ekanligini hamda kasallik og‘irligi va prognozini baholashda qo‘llanilishi mumkinligini ko‘rsatdi [4,5].

КЛИНИЧЕСКАЯ ЗНАЧИМОСТЬ КАРДИОСПЕЦИФИЧЕСКИХ БИОМАРКЕРОВ В ДИАГНОСТИКЕ МИОКАРДИТА У ДЕТЕЙ С ВНЕБОЛЬНИЧНОЙ ПНЕВМОНИЕЙ

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Оценка сердечных тропонинов занимает ведущее место в современной кардиологии, поскольку данные биомаркеры с высокой чувствительностью и специфичностью отражают повреждение кардиомиоцитов [1,2]. В педиатрической практике диагностическая значимость кардиальных биомаркеров имеет особое значение в связи с трудностями клинической диагностики поражения миокарда при инфекционных заболеваниях [3]. Целью настоящего исследования явилась оценка маркеров повреждения миокарда и выявление наиболее диагностически значимых из них у детей с внебольничной пневмонией, осложнённой миокардитом. Полученные результаты свидетельствуют о том, что сердечный тропонин I, α-гидроксибутиратдегидрогеназа (α-HBDH) и МВ-фракция креатинфосфокиназы (КФК-МВ) являются надёжными показателями вовлечения миокарда у детей с внебольничной пневмонией и могут использоваться для оценки тяжести заболевания и прогноза [4,5].

Introduction. Community-acquired pneumonia (CAP) remains one of the most prevalent infectious diseases in pediatric patients and continues to be a major cause of hospitalization, morbidity, and mortality worldwide [3]. Children of early age are particularly vulnerable to lower respiratory tract infections, which are often accompanied by systemic inflammatory responses and extrapulmonary complications [3,6]. Among these complications, cardiovascular involvement plays a significant role due to its influence on disease severity, prognosis, and long-term clinical outcomes [6,7]. Myocardial injury associated with infectious diseases, including CAP, is a well-recognized but frequently underestimated condition in pediatric practice [6,7]. Infectious myocarditis may result from direct pathogen-mediated myocardial damage, immune-inflammatory mechanisms, hypoxic injury, and the cardiotoxic effects of proinflammatory cytokines released during systemic infection [7,12,15]. In children, myocarditis often has a subclinical or atypical clinical

course, and its manifestations may be masked by respiratory symptoms, which significantly complicates early diagnosis [6,7]. Delayed recognition of myocardial involvement may lead to the development of acute heart failure, arrhythmias, and other adverse outcomes [11,15]. Conventional diagnostic methods, such as electrocardiography and echocardiography, demonstrate limited sensitivity in the early stages of myocardial injury, especially in pediatric patients [7,19]. Although endomyocardial biopsy is considered the gold standard for myocarditis diagnosis, its invasive nature and limited feasibility restrict routine use in children [7,12]. Therefore, laboratory biomarkers reflecting cardiomyocyte damage represent a practical and informative diagnostic approach. Cardio specific biomarkers, particularly cardiac troponins, are widely used in modern cardiology due to their high sensitivity and specificity for myocardial injury [1,2,14,16]. Troponins are regulatory proteins of the contractile apparatus of striated muscle and consist of three subunits: troponin C, troponin I, and troponin T [8,13]. Cardiac troponin I and T are highly cardiospecific and can be selectively detected by immunochemical assays [9,14]. Even minimal myocardial damage leads to troponin release into the bloodstream, making these biomarkers reliable indicators of cardiomyocyte injury [1,2,16]. Elevated troponin levels have been described in various pediatric conditions, including perinatal hypoxia, congenital heart diseases, sepsis, cardiomyopathies, and inflammatory myocardial disorders [10,11,18]. However, interpretation of troponin elevation in children remains challenging due to age-related physiological variations and the influence of concomitant pathological conditions [10,21]. In addition to troponins, traditional enzymes such as creatine kinase MB fraction (CK-MB) and α-hydroxybutyrate dehydrogenase (α-HBDH) are still used to assess myocardial damage and may provide additional diagnostic and prognostic information [5,17,18]. Nevertheless, data on the diagnostic value of these biomarkers in children with CAP complicated by myocarditis are limited and inconsistent [6,12,20]. The lack of unified pediatric diagnostic criteria and clearly defined reference ranges further complicates clinical interpretation [16,21]. Thus, comprehensive evaluation of cardio specific biomarkers in children with community-acquired pneumonia complicated by myocarditis remains clinically relevant. Early detection of myocardial involvement using reliable laboratory indicators may improve risk stratification, optimize monitoring, and contribute to better clinical outcomes in pediatric patients [15,20].

Aim of the study. The aim of this study was to evaluate the diagnostic significance of cardio specific biomarkers, including cardiac troponin I, α-HBDH, and CK-MB, in children with community-acquired pneumonia complicated by myocarditis.

Materials and methods. A total of 150 children aged 6 months to 7 years hospitalized with community-acquired pneumonia were included in the study. The research was conducted in pediatric departments and the pediatric intensive care unit of the Samarkand branch of the Republican Scientific Center of Emergency Medical Care during 2024–2025. Among the examined patients, 120 children were diagnosed with pneumonia complicated by myocarditis, while 30 children with pneumonia without myocardial involvement served as the comparison group. Patients with congenital heart defects, chronic heart failure, cardiac arrhythmias, and genetic disorders were excluded from the study, as elevated troponin levels in these conditions may not reflect acute myocardial injury. The patients were divided into three groups: Group A included children with CAP without myocarditis (n = 30); Group B included children with CAP complicated by uncomplicated myocarditis (n = 60); Group C included children with CAP complicated by myocarditis with severe clinical course (n = 60).

Serum levels of cardiac troponin I, α-HBDH, and CK-MB were measured using standard immunochemical and enzymatic methods. Troponin I concentrations of 0–0.1 ng/mL were considered within the reference range, values of 0.2–0.3 ng/mL indicated moderate myocardial injury, and levels ≥ 0.4 ng/mL were interpreted as severe myocardial damage.

Patients were divided into the following groups:

Group A: children with community-acquired pneumonia without myocarditis (n = 30); Group B: children with uncomplicated pneumonia complicated by myocarditis (n = 60); Group C: children with complicated pneumonia accompanied by myocarditis (n = 60). Serum levels of cardiac troponin I, α-HBDH, and CK-MB were determined using standard immunochemical and enzymatic methods. Cardiac troponin I values of 0–0.1 ng/mL were considered normal, 0.2–0.3 ng/mL indicated moderate myocardial injury, and values ≥0.4 ng/mL reflected severe myocardial damage.

Statistical analysis was performed using SPSS software. Quantitative variables were presented as mean ± standard deviation (M ± SD). The normality of data distribution was assessed using the Shapiro–Wilk test. Comparisons between groups were performed using Student's t-test or ANOVA for normally distributed data and the Mann–Whitney U test for non-parametric data. Categorical variables were analyzed using the χ^2 test. Differences were considered statistically significant at $p < 0.05$.

Results. The analysis of laboratory data demonstrated a clear association between myocardial involvement and the severity of community-acquired pneumonia in the examined children. Elevated cardiac troponin I levels were rarely detected in patients without myocarditis (Group A), whereas their frequency significantly increased in children with pneumonia complicated by myocarditis.

According to the obtained data, troponin I elevation was observed in 6.7% of children with community-acquired pneumonia without myocardial involvement (Group A), in 78.3% of patients with uncomplicated myocarditis (Group B), and in 93.3% of children with complicated pneumonia accompanied by myocarditis (Group C). The differences in troponin I elevation frequency among the study groups were statistically significant ($\chi^2 = 82.4$, $p < 0.0001$). The assessment of cardio specific enzyme activity further confirmed myocardial involvement in children with pneumonia complicated by myocarditis. Serum levels of α-hydroxybutyrate dehydrogenase (α-HBDH) and creatine kinase MB fraction (CK-MB) were within the reference range in most patients from Group A. In contrast, children from Group B demonstrated moderate elevations of these enzymes, while patients from Group C exhibited significantly increased α-HBDH and CK-MB levels, indicating more pronounced myocardial injury in complicated disease forms (ANOVA, $p < 0.001$). A detailed analysis of cardiac troponin I concentrations according to the degree of myocardial injury showed that troponin I values within the reference range (0–0.1 ng/mL) predominated in children without myocarditis. Moderate troponin elevation (0.2–0.3 ng/mL) was mainly characteristic of patients with uncomplicated myocarditis, whereas severe elevation of troponin I (≥ 0.4 ng/mL) was predominantly observed in children with complicated pneumonia, reflecting extensive cardiomyocyte damage. Correlation analysis revealed a moderate positive relationship between troponin I levels and disease severity (Spearman $r = 0.58$, $p < 0.001$), suggesting that troponin I concentration increases with the progression of myocardial injury and the severity of pneumonia. Overall, the obtained results demonstrate a statistically significant association between cardio-specific biomarker levels and myocardial involvement severity in children with community-acquired pneumonia.

1 table.

Frequency of Cardiac Troponin I Elevation in the Study Groups

Study group	Number of patients (n)	Troponin I elevation, %	Clinical interpretation	P (x2 test)
Group A – CAP without myocarditis	30	6.7	No myocardial injury	
Group B – CAP with uncomplicated myocarditis	60	78.3	Moderate myocardial injury	<0,0001 82.4
Group C – CAP with complicated myocarditis	60	93.3	Severe myocardial injury	

2 table.

Cardio specific Enzyme Activity in Children with Community-Acquired Pneumonia.

Study group	Cardiac troponin I	α-HBDH	CK-MB
Group A	Within normal range	Normal	Normal
Group B	Moderately increased	Moderately increased	Moderately increased
Group C	Significantly increased	Significantly increased	Significantly increased
P (ANOVA)	<0,001	<0,001	<0,001

3 table.

Distribution of Cardiac Troponin I Levels According to the Severity of Myocardial Injury.

Degree of myocardial injury	Troponin I level (ng/mL)	Predominant group
No myocardial injury	0–0.1	Group A
Moderate myocardial injury	0.2–0.3	Group B
Severe myocardial injury	≥ 0.4	Group C

Discussion. The present study demonstrated a statistically significant association between cardio specific biomarkers and myocardial involvement in children with community-acquired pneumonia. The frequency of cardiac troponin I elevation increased progressively with the severity of pneumonia and myocarditis, which confirms the diagnostic and prognostic significance of this biomarker in pediatric clinical practice.

Troponin I elevation was rarely observed in children without myocarditis, whereas the majority of patients with pneumonia complicated by myocarditis showed increased troponin levels. These findings are consistent with previously published data indicating that cardiac troponins are highly sensitive markers of cardiomyocyte injury and may reflect inflammatory, hypoxic, and toxic myocardial damage during severe infectious diseases [1,4,7]. In addition to troponin I, traditional enzymatic markers, including α-hydroxybutyrate dehydrogenase and CK-MB, were significantly elevated in children with complicated pneumonia, which further confirms myocardial involvement in this group. The combined assessment of troponin I, α-HBDH, and CK-MB improved the detection of myocardial injury and allowed differentiation between uncomplicated and complicated clinical courses. Similar observations have been reported in pediatric and adult populations with infectious myocarditis, where enzyme elevation correlated with disease severity and adverse outcomes [5,17,18]. The correlation analysis revealed a moderate positive relationship between troponin I levels and disease severity, suggesting that troponin concentration may serve not only as a diagnostic marker but also as an indicator of disease progression. This finding supports the concept that early identification of myocardial involvement using laboratory biomarkers may contribute to risk stratification and timely clinical decision-making. Despite the clinical relevance of these findings, the interpretation of cardiac biomarker levels in children remains challenging due to age-related physiological variations and the influence of comorbid conditions. Moreover, the lack of unified pediatric reference ranges and standardized diagnostic criteria for myocarditis complicates clinical assessment and may lead to underdiagnosis of myocardial injury in children with pneumonia. Therefore, further multicenter studies with larger sample sizes are required to refine diagnostic thresholds and evaluate the prognostic value of cardio specific biomarkers in pediatric populations.

Overall, the results of this study confirm the high diagnostic value of cardiac troponin I and support the use of α-HBDH and CK-MB as complementary markers in children with community-acquired pneumonia complicated by myocarditis.

Conclusion. Children with community-acquired pneumonia complicated by myocarditis demonstrated significantly higher levels of cardiac troponin I, α-hydroxybutyrate dehydrogenase, and CK-MB compared to patients without myocardial involvement. The frequency and degree of troponin I elevation increased in parallel with disease severity, indicating progressive cardiomyocyte injury in complicated forms of pneumonia. The assessment of cardio-specific biomarkers allows reliable detection of myocardial damage, differentiation of disease severity, and identification of children at high risk of adverse outcomes. Cardiac troponin I, in combination with α-HBDH and CK-MB, can be considered clinically valuable laboratory indicators for the early diagnosis of myocarditis in pediatric patients with community-acquired pneumonia. The integration of these biomarkers into routine clinical practice may improve early diagnosis, optimize monitoring strategies, and contribute to improved clinical outcomes in children with infectious myocardial involvement.

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