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Features of the Immune Response and Clinical Course of Community-Acquired Pneumonia in Children with Congenital Heart Defects

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Abstract Congenital heart defects remain one of the leading causes of morbidity, disability, and mortality in children worldwide. The presence of congenital cardiovascular abnormalities significantly increases the risk of infectious and inflammatory complications, particularly community-acquired pneumonia, which is characterized by a severe clinical course and high probability of adverse outcomes. This article is devoted to the study of the features of the immune response and the clinical manifestations of community-acquired pneumonia in children with congenital heart defects. A prospective clinical study included 140 children aged from 0 to 3 years who underwent treatment at the Republican Specialized Scientific and Practical Medical Center of Pediatrics and the Regional Children's Multidisciplinary Medical Center of Fergana. The study assessed clinical symptoms, anamnestic data, laboratory and instrumental parameters, as well as indicators of cellular and humoral immunity. Special attention was paid to the analysis of cytokine status, including the determination of serum levels of TNF-α, IL-1β, IL-4, IL-6, IL-8, and IFN-γ using enzyme-linked immunosorbent assay. The obtained results demonstrated that community-acquired pneumonia in children with congenital heart defects is accompanied by a more severe clinical course characterized by respiratory failure, prolonged intoxication syndrome, severe dyspnea, cyanosis, myocardial dysfunction, and significant hemodynamic disturbances. The majority of hospitalized patients were children of the first year of life, among whom ventricular septal defect, atrial septal defect, and Tetralogy of Fallot predominated. Echocardiographic examination revealed a decrease in myocardial contractility, enlargement of heart chambers, increased pulmonary artery pressure, and more frequent occurrence of pericardial effusion in children with combined pathology.

Keywords Congenital heart defects, Community-acquired pneumonia, Children, Immune response, Cytokines, Inflammatory process, Hemodynamic disorders, TNF-α, IL-6, IL-8, IL-1β, Immunoregulation, Respiratory failure

1. Introduction

Despite modern advances in cardiology and cardiac surgery, congenital heart defects remain a frequent cause of disability and mortality in childhood and include a wide range of phenotypes that differ in morphology, physiology, and other factors, which necessitates a differentiated study of the mechanisms underlying the development of this pathology. Congenital malformations are the most common pathology among newborns, one-third of which are congenital heart defects (CHD). In Uzbekistan, about 10 thousand children with CHD who require surgery are born annually. Of these, from 50 to 70% require emergency care. [1,2] Mortality in such CHDs is extremely high: by the end of the first week, 29% die; by the first month, 42%; by the first year of life, 87%; by the first month, 42%; by the first year of life, 87%. The study of the incidence rate, types of CHD, as well as risk factors for their development may serve as a basis for the development of therapeutic and preventive programs aimed at reducing this pathology in the region. Moreover, a further increase in the prevalence of CHD is predicted. The increase in the proportion of children with CHD is also facilitated by modern surgical treatment methods, which ensure the survival of children with almost all defects and, as a consequence, the rapid growth of the population of adolescents and adults with operated CHD. [3,4]

The article examines the features of the immune response and the clinical course of community-acquired pneumonia in children with congenital heart defects. Changes in the indicators of cellular and humoral immunity, as well as their

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relationship with the severity of the disease, duration of the course, and outcomes, are analyzed. Particular attention is paid to the role of immune dysfunction in the development of complicated forms of pneumonia. [5,6,9]

The obtained data make it possible to deepen the understanding of the pathogenetic mechanisms of the disease in this category of patients and can be used to optimize diagnosis, predict the course of the disease, and determine treatment strategies. [10,11,12]

Objective of the study: To investigate the features of the immune response and the clinical course of community-acquired pneumonia in children with congenital heart defects in order to identify factors influencing the severity of the disease and the development of complications.

2. Materials and Methods of the Study

In accordance with the objectives set, we conducted a prospective study of 140 children with congenital heart defects (CHD) and community-acquired pneumonia who were treated at the Republican Specialized Scientific and Practical Medical Center of Pediatrics and at the Regional Children's Multidisciplinary Medical Center of Fergana. The children were aged from 0 to 3 years, including 60 patients with CHD and 60 children with CHD and community-acquired pneumonia (comparison group). All children with CHD admitted to the RSSPMC of Pediatrics were registered with a cardiac surgeon. The reason for admission to the RSSPMC of Pediatrics was a sharp deterioration in the child's condition, weakness accompanied by dyspnea, severe anxiety, cyanosis of the skin, and increased body temperature. Clinical and anamnestic data, chest X-ray results, and laboratory parameters were taken into account. Immunological studies were also performed. The quantitative levels of cytokines – IL-1β, IL-4, IL-6, IL-8, TNF-α, and IFN-γ in blood serum were determined by enzyme-linked immunosorbent assay using the semi-automatic immunoassay analyzer "Multiskan FC" (Finland).

3. Results and Discussion

In children of the CHD group, the following were observed: ventricular septal defect (VSD) in 16 children, including 12 aged 0–1 year, 3 aged 1–2 years, and 1 child aged 2–3 years; atrial septal defect (ASD) in 20 children, including 10 aged 0–1 year, 6 aged 1–2 years, and 4 aged 2–3 years. Tetralogy of Fallot (TOF) was identified in 11 children, including 9 aged 0–1 year, 1 child aged 1–2 years, and 1 child aged 2–3 years.

As can be seen from the table, among the majority of children with CHD and community-acquired pneumonia, ventricular septal defect (VSD) was the most common and was observed most frequently in children under 1 year of age – 26 children (43.3%). At the age of 1–2 years, there were 4 children (6.7%), and at the age of 2–3 years – 8 children (13.3%). Atrial septal defect (ASD) was diagnosed less frequently — in 10 children (16.7%), and Tetralogy of Fallot — in 5 patients (8.3%). Other forms of CHD with community-acquired pneumonia occurred only in isolated cases (Table 1).

Analysis of the anamnestic data showed that in children with CHD and community-acquired pneumonia, 2–3 days elapsed from the onset of the disease to the moment of hospitalization. The overwhelming majority of children with CHD and community-acquired pneumonia – 73% (n=44) were hospitalized on the 3rd–5th day from the onset of the disease after unsuccessful treatment at home. Analysis of the medical history data revealed that 67.5% (n=41) of children received treatment at the pre-hospital stage, while 31.7% (n=19) did not seek medical care before hospitalization and received no treatment.

During examination of patients in the hospital, the general condition of the patients on the day of admission was assessed as extremely severe in 23.3% (n=14), severe in 61.7% (n=37), and moderately severe in 15% (n=9) of children.

Analysis of the clinical data revealed that the main complaints of the parents of patients with CHD and community-acquired pneumonia hospitalized in the inpatient department were cough in 100% (n=60), dyspnea in 94.1% (n=96), elevated body temperature in 81.4% (n=83) of children, decreased appetite in 92.2% (n=55), and lethargy in 97.1% (n=58). The clinical picture of patients with CHD was mainly characterized by lethargy, decreased appetite, cyanosis of the nasolabial triangle, dyspnea, moist cough, and moist rales. The frequency of these symptoms was higher in the group of children with CHD and community-acquired pneumonia (Table 2). Signs of hypoxia in the form of cyanosis of the nasolabial triangle were observed in 73.5% (n=44) of patients; in the remaining children, cyanosis occurred during crying and anxiety, with a duration of 6.1±0.4 days. Mixed-type dyspnea involving accessory respiratory muscles was observed in 94.1% (n=56) of patients. The duration of dyspnea was 4.3±0.3 days. Percussion changes in the lungs in 84.3% (n=51) were local in nature and manifested as shortening of the pulmonary sound, while in 33.3% (n=20) a boxed percussion sound was observed. On auscultation, harsh breathing was detected in 86.3% (n=52) of cases, and weakened breathing in 48.0% (n=29). Moist rales, sometimes combined with crepitation, were heard in 99.0% (n=59) of patients, with a duration of 8.9±0.3 days. Dry rales were heard in 63.7% (n=38) of patients, with a duration of 9.5±0.4 days.

In children, heart rate indicators demonstrated compensatory phenomena in the form of tachycardia in the group of children with CHD and community-acquired pneumonia (heart rate of more than 170 beats/min in 68.4% (41), 130–160 beats/min in 18.3%, and 100–110 beats/min in 13.3% of children), whereas in children with isolated CHD, heart rate values were within the age norm.

Table 1. Distribution of patients by age and types of CHD with community-acquired pneumonia

CHD with Community-Acquired Pneumonia	Number	0–1 years	1–2 years	2–3 years
Ventricular Septal Defect (VSD)	38	26	4	8
Atrial Septal Defect (ASD)	10	6	4	—
Tetralogy of Fallot	5	5	—	—
Patent Ductus Arteriosus (PDA)	3	2	1	—
Pulmonary Artery Stenosis (PAS)	1	—	1	—
Double Outlet Right Ventricle (DORV)	2	1	1	—
Total Anomalous Pulmonary Venous Drainage (TAPVD)	1	1	—	—
TOTAL	**60**	**41**	**11**	**8**

Table 2. Duration of clinical symptoms in children

Clinical Symptoms	CHD (n=60)		CHD with Community-Acquired Pneumonia (n=60)		P
	n = 60	%	n = 60	%	
Lethargy	33	55.8	58	97.1	
Decreased appetite	48	80.8	55	92.2	
General weakness	36	60.8	53	88.2	
Headache	15	25.6	25	42.3	
Temperature					
Febrile	10	16.7	6	10.8	
Subfebrile	27	45.8	42	70.6	
Cough					
Dry	20	34.2	5	8.8	
Moist	39	65.8	55	91.2	
Dyspnea	42	70.0	56	94.1	
Cyanosis of the nasolabial triangle	28	47.5	44	73.5	
Percussion					
Shortening of percussion sound	38	63.3	51	84.3	
Boxed percussion sound	22	36.7	20	33.3	
Auscultation					
Harsh breathing	51	85.8	52	86.3	
Weakened breathing	9	14.2	29	48.0	
Rales					
Dry	44	74.2	38	63.7	
Moist	53	89.2	59	99.0	
Muffled heart sounds	39	65.0	52	86.3	
Heart Rate (beats/min)					
100–110	9	15	–	–	
110–130	12	20	8	13.3	
130–160	39	65	11	18.3	
More than 170	–	–	41	68.4	
Less than 90	–	–	–	–	

A comparative analysis of echocardiographic parameters in children with congenital heart defects and in patients with combined CHD and community-acquired pneumonia demonstrated the presence of significant intergroup differences presented in Table 3.

It was established that the group with CHD and community-acquired pneumonia demonstrated a more pronounced decrease in myocardial contractility. The left ventricular ejection fraction was lower in children with a concomitant inflammatory process and amounted to 53.6 ± 6.9%, whereas in children with isolated CHD this parameter reached 61.2 ± 4.8% ($p<0.001$).

Table 3. Echocardiographic Parameters at Admission in the Examined Children

Parameter	CHD (n=60)	CHD+ Community-Acquired Pneumonia (n=60)	P
Left ventricular ejection fraction (%)	61.2 ±4.8	53.6 ±6.9	<0.001
LV end-diastolic dimension (mm)	37.5 ±5.9	41.8 ±6.7	0.002
LV end-systolic dimension (mm)	23.6 ±3.8	28.2 ±4.9	<0.001
Interventricular septum thickness (mm)	6.4 ±1.1	7.1 ±1.3	0.01
Left ventricular posterior wall thickness (mm)	6.2 ±1.0	6.9 ±1.2	0.02
Pulmonary artery pressure (mm Hg)	27.8 ±7.6	35.9 ±9.8	<0.001
Right ventricular size (mm)	17.9 ±3.1	21.7 ±3.9	<0.001
Left atrial size (mm)	19.8 ±3.7	23.5 ±4.6	0.001
Tricuspid regurgitation velocity (m/s)	2.4 ±0.3	3.0 ±0.5	<0.001
Pericardial effusion (%)	8.3% (5/60)	33.3% (20/60)	<0.001

The study of the linear dimensions of the left ventricle revealed a tendency toward its enlargement in the second group. The end-diastolic dimension and end-systolic dimension were higher in children with CHD + community- acquired pneumonia (41.8 ±6.7 mm and 28.2 ±4.9 mm, respectively) compared with the first group (37.5 ±5.9 mm and 23.6 ±3.8 mm), and the differences were statistically significant.

The indicators of interventricular septal thickness and left ventricular posterior wall thickness also demonstrated a moderate increase in the second group, which may reflect structural remodeling of the myocardium against the background of the inflammatory process.

Additionally, children with combined pathology showed increased pressure in the pulmonary artery system, as well as enlargement of the right heart chambers and the left atrium, indicating more pronounced hemodynamic disturbances. Blood flow velocity during tricuspid regurgitation was higher in patients of the second group, indirectly indicating increased pulmonary hypertension. It should also be noted separately that the presence of pericardial effusion was recorded significantly more often in children with CHD complicated by inflammation compared with patients without signs of an inflammatory process.

Thus, the obtained data indicate that the addition of an inflammatory component in children with congenital heart defects is associated with deterioration of the functional state of the myocardium, remodeling of the heart chambers, and more pronounced hemodynamic disturbances.

The analysis of cytokine status revealed pronounced changes in the immune response in children with community -acquired pneumonia compared with practically healthy children, as well as its aggravation when CHD was combined with viral pneumonia (CHD with community-acquired pneumonia).

Pro-inflammatory cytokines (TNF-α, IL-1β, IL-6, IL-8) in children with CHD were significantly elevated relative to the control group ($p<0.05–0.001$), indicating activation of the systemic inflammatory response. The most pronounced increase was observed in the group of children with CHD and community-acquired pneumonia, where the levels of all the above inflammatory mediators exceeded the values of both healthy children and patients with isolated CHD ($p1<0.01–0.001$).

Thus, the concentrations of TNF-α and IL-1β increased more than twofold in CHD and reached maximum values, reflecting a high degree of cytokine-mediated inflammatory reaction. A similar trend was noted for IL-6, which is a key marker of inflammatory activity and severity of the infectious process.

The level of IL-8, responsible for neutrophil chemotaxis, also increased significantly in CHD and especially in CHD with community-acquired pneumonia, indicating enhanced recruitment of innate immune cells to the inflammatory focus and more pronounced damage to lung tissue.

The anti-inflammatory cytokine IL-4 demonstrated a different pattern of changes: in CHD, its concentration increased significantly compared with normal values, which can be regarded as a compensatory response of the body aimed at limiting inflammation. However, with the addition of community-acquired pneumonia, the IL-4 level decreased compared with the CHD group ($p1<0.01$), indicating disruption of the balance between pro- and anti-inflammatory mechanisms.

The analysis of cytokine status revealed pronounced changes in the immune response in children with community -acquired pneumonia compared with practically healthy children, as well as its aggravation when CHD was combined with viral pneumonia (CHD with community-acquired pneumonia).

In children with CHD, a pronounced imbalance of cytokine regulation is formed, characterized by activation of pro-inflammatory mediators. When CHD is combined with community-acquired pneumonia, this imbalance becomes more pronounced, reflecting a more severe course of the disease and exhaustion of the anti-inflammatory mechanisms of the immune response.

Thus, the combination of congenital heart defects and community-acquired pneumonia creates a pathogenetically unfavorable background that determines the severity of the clinical course and an increased risk of complications. The

obtained results emphasize the importance of early detection of immunological disorders and the development of personalized approaches to the management of such patients.

4. Conclusions

1. In children with congenital heart defects, community -acquired pneumonia is characterized by a more severe clinical course accompanied by pronounced respiratory failure, intoxication syndrome, and a high frequency of hospitalization in severe condition. In the structure of congenital heart defects among the examined patients, ventricular septal defect, atrial septal defect, and Tetralogy of Fallot predominated, while children of the first year of life represented the most vulnerable group.
2. The addition of community-acquired pneumonia in children with CHD leads to a significant deterioration of hemodynamic parameters: a decrease in myocardial contractile function, enlargement of the heart chambers, increased pulmonary artery pressure, and a higher incidence of pericardial effusion.
3. Clinical and functional changes are accompanied by pronounced activation of the systemic inflammatory response, manifested by a significant increase in the levels of pro-inflammatory cytokines (TNF-α, IL-1β, IL-6, IL-8).
4. A disturbance of immune regulation was revealed, characterized by an imbalance between pro- and anti-inflammatory cytokines: against the background of increased IL-4 in isolated CHD, its relative decrease was observed when combined with pneumonia, indicating exhaustion of compensatory anti-inflammatory mechanisms.
5. A relationship was established between the severity of the cytokine response and the severity of the clinical course of the disease, confirming the significant role of immune disorders in the pathogenesis of community -acquired pneumonia in children with CHD.
6. The obtained data substantiate the need for early diagnosis, comprehensive assessment of immune status, and a differentiated approach to the treatment of this category of patients in order to reduce the risk of complications and mortality.

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