CONGENITAL HEART DISEASE IN CHILDREN

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Relevance of the topic

Congenital Heart Disease (CHD) comprises a spectrum of structural cardiovascular anomalies resulting from disrupted embryogenesis during weeks 3 to 8 of gestation, representing one of the most prevalent classes of congenital birth defects in pediatric populations. Globally, the overall birth prevalence of CHD is estimated between 8.0 and 9.4 per 1,000 live births. Approximately 25% of these cases (2.5 per 1,000 live births) present as severe or critical structural lesions requiring urgent surgical or transcatheter intervention during early infancy.

In advanced healthcare systems, 1-year survival rates for non-critical structural defects reach approximately 97.0%, while survival for critical lesions exceeds 83.0% (with 18-year survival reaching 95.0% and 69.0%, respectively). Conversely, in low- and middle-income countries (LMICs), 47.0% to 63.0% of pediatric CHD cases are diagnosed after irreversible secondary complications such as severe pulmonary arterial hypertension or Eisenmenger syndrome have already developed, with up to 49.4% of patients presenting with overt congestive heart failure at primary evaluation.

Congenital heart lesions are broadly categorized by their systemic oxygen saturation and hemodynamic impact into acyanotic and cyanotic defects.

Acyanotic lesions preserve systemic saturation (SpO2 > 95%) while increasing pulmonary flow or outflow resistance. These include Ventricular Septal Defects (VSD, 30–35% of cases), which cause high-pressure shunting and risk pulmonary arterial hypertension; Atrial Septal Defects (ASD, 7–10%), leading to right-heart volume overload that often remains subclinical until early adulthood; Patent Ductus Arteriosus (PDA, 5–10% in term infants and up to 45% in low-birth-

weight neonates), which elevates pulmonary pressures and left-heart preload; and Coarctation of the Aorta (CoA, 5–7%), characterized by juxtaductal narrowing that creates a systolic pressure gradient exceeding 20 mmHg between upper and lower extremities.

Cyanotic lesions involve right-to-left shunts or structural discordance that route deoxygenated blood into systemic circulation (SpO2 < 85%-90%). Major forms include Tetralogy of Fallot (TOF, 7–10%), defined by VSD, RVOT obstruction, overriding aorta, and right ventricular hypertrophy; Transposition of the Great Arteries (d-TGA, 5–7%), characterized by parallel circulatory loops requiring urgent mixing via ASD or PDA for postnatal survival; and Hypoplastic Left Heart Syndrome (HLHS, 2–3%), where severe left-heart underdeveloped anatomy leaves systemic perfusion entirely reliant on right ventricular output through a ductus arteriosus.

Materials and Methods

A prospective observational cohort study was conducted at the Bukhara Regional Children's Medical Association to evaluate the clinical features, hemodynamic parameters, and diagnostic findings in pediatric patients with congenital heart disease (CHD). The study included 150 children aged 0–18 years who were divided into three groups: the main group consisted of 60 children with CHD and laboratory-confirmed COVID-19, the comparison group included 60 children with CHD without COVID-19, and the control group comprised 30 clinically healthy children without structural cardiovascular abnormalities. All participants underwent clinical examination, including assessment of medical history, cardiovascular and respiratory symptoms, heart rate, blood pressure, oxygen saturation, and physical development. Cardiovascular evaluation included electrocardiography and transthoracic echocardiography to determine the type and hemodynamic characteristics of CHD. Laboratory investigations included complete blood count, biochemical parameters, and inflammatory markers. Written informed consent was obtained from the parents or legal guardians of all participating children, and the study was conducted in accordance with relevant

ethical requirements.

Results

A total of 150 pediatric subjects were analyzed across the three designated study cohorts: the Main Group (n=60), the Comparison Group (n=60), and the Control Group (n=30). At baseline, the mean age was 24.5±14.2 months in the Main Group, 25.1±13.8 months in the Comparison Group and 23.8±12.5 months in the Control Group ($p = 0.884$). Sex distribution was comparable across cohorts, with males representing 53.3% (n=32), 51.7% (n=31) and 50.0% (n=15) of subjects in the Main, Comparison and Control groups, respectively ($\chi^2= 0.141$, $p = 0.932$). No statistically significant inter-group differences were observed regarding age, sex or body weight, confirming adequate baseline comparability between the cohorts.

Echocardiography provided the principal method for confirming the diagnosis and characterizing the anatomical substrate of CHD. The examination allowed differentiation between septal defects, abnormalities of the cardiac valves and outflow tracts, and more complex congenital cardiac lesions. The clinical significance of individual defects was determined not only by their anatomical type but also by their hemodynamic consequences.

Transthoracic echocardiographic evaluation demonstrated significant hemodynamic derangements in both patient groups relative to healthy controls. Systemic arterial oxygen saturation (SpO_2) was significantly lower in the Main Group (88.4±6.2%) and Comparison Group (89.1±5.8%) compared to the Control Group (98.2±1.1%; $p<0.001$). Mean pulmonary artery pressure (PAP) was markedly elevated in the Main Group (42.6±8.4 mmHg) and Comparison Group (41.8±7.9 mmHg) relative to controls (16.2±2.8 mmHg; $p < 0.001$). Left ventricular ejection fraction was mildly diminished in patient cohorts (58.2±5.4% in Main Group vs. 57.9 ±6.1% in Comparison Group) compared to controls (64.5±3.2%; $p < 0.001$). Additionally, the pulmonary-to-systemic blood flow ratio averaged 2.1±0.4 in the Main Group and 2.0±0.5 in the Comparison Group, reflecting substantial pulmonary hyperperfusion prior to intervention compared to

physiological baseline levels (1.0 ±0.1; $p < 0.001$).

Conclusion

Congenital Heart Disease remains a major public health consideration in pediatric cardiology. Modern early screening strategies combining fetal ultrasonography, post-natal pulse oximetry, and color Doppler echocardiography allow for timely anatomical repair. Continued research focused on reducing perioperative cerebral hypoxia, refining tissue-engineered prosthetic valves, and expanding pediatric cardiac care in resource-limited settings is critical to improving lifelong functional outcomes for affected children.

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