Renal Function in Children With Congenital Heart Defect

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Abstract- Congenital heart defects (CHDs) are the most common type of congenital birth defect. The study aimed to assess the effect of CHD on renal involvement in comparison with the controls. This case control study conducted on 140 children with CHD and 70 healthy children in the years of 2022 and 2023. CHD diagnosis and based on the level of O2 saturation divided in CCHD and aCCHD. Potassium, sodium, hemoglobin in blood and creatinine and albumin in urine were measured in all participants. Glomerular filtration rate (GFR) is considered as a marker of renal involvement. The data analyzed with SPSS 22 and the level of significant considered equal or less than 0.05. Sex distribution was similar in groups of participants (X2=0.976, *P*=0.614). About 36.10% of the children with CCHD had renal involvement when this rate was 6.30% and 2.90% in aCCHD and the controls respectively (X2=36.154, *P*<0.001). Pairwise comparisons showed (C.C=0.350, *P*<0.001), (C.C=0.394, *P*<0.001), (C.C=0.082, *P*=0.317) for the pairs of CCHD-aCCHD, CCHD-Controls and aCCHD-Controls, respectively. The children with CCHD had odds ratio of 19.179 and the children with aCCHD had an odds ratio of 2.297 related to the reference (controls) group. The study concluded that children with cyanotic congenital heart disease (CCHD) exhibited a significantly higher risk of renal involvement compared to those with acyanotic congenital heart disease (aCCHD) and healthy controls. The chronic hypoxemia characteristic of CCHD may lead to compensatory erythrocytosis, resulting in elevated hemoglobin levels, which in turn may contribute to renal impairment through mechanisms such as increased blood viscosity, reduced renal perfusion, and glomerular injury.

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Introduction

Congenital heart diseases (CHDs) are the most common congenital anomalies worldwide and remain a leading cause of childhood mortality (1). Globally, CHD prevalence ranges from 0.4% to 5%, with most estimates falling between 0.9% and 1.4% (2-4). However, regional variation exists for instance, a Finnish study reported prevalence rates ranging from 1.95 to 12.5 per 1,000 live births (4). In Iran, the true burden of CHD is difficult to assess due to the absence of a national registry system (5). CHDs result from multifactorial causes, involving complex interactions between genetic predispositions and environmental exposures. Among the notable risk factors are maternal health conditions during pregnancy. Specifically, substance use and addiction during pregnancy have been found to be more prevalent among mothers of children diagnosed with CHD (6,7). Based on their physiological and anatomical features, CHDs are broadly categorized into cyanotic (CCHD) and acyanotic (aCCHD) types (3,8). Cyanotic CHDs impair oxygenation, resulting in systemic desaturation and clinical cyanosis. These defects are often accompanied by abnormal blood flow and may trigger arrhythmias, which can further aggravate hypoxemia, although they are not the primary cause of cyanosis (9). Medical and surgical advancements have significantly improved survival in CHD patients, with more than 97% now surviving into

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adulthood (10,11). As a result, there is a growing need to monitor and manage long-term comorbidities particularly renal complications (12,13). Renal involvement is more common in CCHD, likely due to chronic hypoxemia, which leads to erythrocytosis, increased blood viscosity, and changes in renal hemodynamics. Gupte *et al*., (14) described structural renal changes in children with CCHD, including glomerulomegaly, glomerulosclerosis, periglomerular fibrosis, and interstitial damage. Further histopathological studies by Perlov *et al*., (15) revealed two distinct patterns of glomerular injury: a vascular pattern characterized by glomerular capillary dilation and hyperfiltration due to increased shear stress and nitric oxide production, and a non-vascular pattern marked by mesangial cell proliferation and matrix expansion (15,16). These alterations are thought to be mediated in part by circulating megakaryocytes that bypass the lungs through right-to-left shunts and release cytokines such as platelet-derived growth factor (PDGF) and transforming growth factor-beta (TGF-β), contributing to glomerular injury (16). Over time, these changes may result in glomerulosclerosis, proteinuria, and progression to renal dysfunction. Indeed, chronic kidney disease (CKD) has been reported in 46.5% of a cohort of 359 children with CCHD, underscoring the high renal risk in this population (17). Given these concerns, the present study aims to identify key factors contributing to renal complications in children with congenital heart disease.

Materials and Methods

This case-control study was carried out at the pediatric clinic of Zahedan University of Medical Sciences, Iran, over a two-year period from 2021 to 2023. A total of 140 children under the age of 18 who had been diagnosed with symptoms or asymptomatic CHD included in the study. Based on their clinical evaluations and results from arterial blood gas (ABG) tests, the children were divided into two groups: those with cyanotic CHD (CCHD), which included 61 children, and those with acyanotic CHD (aCCHD), which included 79 children. To provide a comparison, the study also included a control group of 70 healthy children, matched by age, who had no history or signs of CHD

Criteria

Inclusion criteria

Age less than 18 years old, confirmed diagnosis of Congenital Heart Disease (CHD) and willing to sign a written consent form to be part of the study

Exclusion criteria

Children were not included in the study if they had previously undergone heart reconstructive surgery or had experienced a fever or signs of infection such as painful urination, frequent urination, or unusual discharge within the past two weeks. Other reasons for exclusion included recent use (within the last two weeks) of antibiotics or non-steroidal anti-inflammatory drugs (NSAIDs), being pregnant, or having a known diagnosis of cancer, diabetes, or any heart condition other than congenital heart disease. Children with known kidney disease, a family history of inherited kidney problems, or obesity were also excluded. Additionally, those taking medications that could harm the kidneys such as certain contrast agents used in imaging tests were not eligible to participate in the study.

Measurements

The urine and blood samples, each consisting of 2 cc, were transported to the laboratory at Ali Ibn Abitalib Hospital for analysis. Urinary albumin levels were assessed using an automated analyzer kit (Albumin Pars Azmun, Tehran, Iran), while urinary creatinine levels were determined using dedicated kits (Creatinine Pars Azmun, Tehran, Iran). Potassium levels were measured using the Easy LytePlus device (manufactured in the USA), and hemoglobin levels were analyzed using the Cell Count device, Mindray BC-5800 (manufactured in China).

GFR

For children, the most commonly employed creatinine-based equation to estimate Glomerular Filtration Rate (GFR) is the revised bedside Schwartz formula: [(k×Height (cm)) / Scr (mg/dL)]. This method is endorsed by the National Kidney Disease Education Program for GFR calculations. The value of k is set at 0.45 for participants younger than 1 year and at 0.55 for children aged 1 to 15 years.

This study received ethical approval from the Research Ethics Board of the Deputy of Research at Zahedan University of Medical Sciences, Zahedan, Iran, and adhered to the principles outlined in the Declaration of Helsinki. It was assigned the study code IR.ZAUMS.REC.1398.378.

Statistical analysis

Statistical analyses were conducted using SPSS 20 (SPSS Inc, Chicago, IL, USA). Continuous variables were presented as mean±SD and compared using either one-way analysis of variance or Kruskal-Wallis tests,

depending on the distribution of the data. Logistic regression was employed to assess the impact of variables, with statistical significance set at $P<0.05$.

Results

Our study included 140 patients with congenital heart disease, classified into two groups: CCHD (n=61, 43.5%) and aCCHD (n=79, 56.5%). Within the CCHD group, 39 patients (63.9%) had tetralogy of Fallot (TOF), 5 (8.2%) had double outlet right ventricle (DORV), 6 (9.8%) had transposition of the great arteries (TGA), 5 (8.2%) had tricuspid atresia (TA), and 6 (9.8%) had pulmonary atresia with ventricular septal defect (PAVSD). In the aCCHD group, 48 patients (60.8%) had ventricular septal defect (VSD), 14 (17.7%) had atrial septal defect (ASD), 9 (11.4%) had patent ductus arteriosus (PDA), 5 (6.3%) had pulmonary stenosis (PS), and 3 (3.8%) had aortic stenosis (AS). All continuous variables, including urinary potassium, urinary creatinine, urinary microalbumin, urine albumin-to-creatinine ratio, and hemoglobin levels, showed a non-normal distribution ($P<0.05$, Shapiro-Wilk test). The mean ages of the children were 50.9±51.49 months in the CCHD group, 59.85±39.68 months in the aCCHD group, and 55.94±66.41 months in the control group, with no statistically significant difference observed among the groups (F=0.487, $P=0.615$). Table 1 depicted the gender distribution among participants (CCHD, aCCHD, and controls), showing a similar distribution across groups ($\chi^2 = 0.976$, $P=0.614$). Table 2 presented the incidence of renal involvement among the three groups, revealing that 36.10% of children with CCHD had renal involvement, compared to 6.30% in aCCHD and 2.90% in controls ($\chi^2=36.154$, $P<0.001$). Pairwise comparisons in Table 2 showed contingency coefficients (C.C) and P of (C.C=0.350, $P<0.001$) for CCHD vs. aCCHD, (C.C=0.394, $P<0.001$) for CCHD vs. controls, and (C.C=0.082, $P=0.317$) for aCCHD vs. controls. Table 3 displayed the variations in potassium, creatinine in urine, microalbumin in urine, albumin/creatinine ratio in urine, and hemoglobin among patient groups. It indicated significantly higher potassium levels in CCHD patients (4.72 vs. 4.30, $P<0.001$), higher creatinine in urine in aCCHD children (2.3 vs. 8.54, $P<0.001$), higher microalbumin in urine in CCHD children (14.42 vs. 10.08, $P=0.003$), higher albumin/creatinine ratio in urine in CCHD children (9.42 vs. 2.22, $P<0.001$), and higher hemoglobin levels in CCHD children (15.95 vs. 10.90, $P<0.001$) compared to their respective counterparts. Table 4 presented the results of univariate and multivariate logistic regression analyses to assess the impact of various factors on renal involvement. In the univariate analysis, children with CCHD had an odds ratio of 19.179, and those with aCCHD had an odds ratio of 2.297 compared to the reference (controls) group. The odds ratios for urine creatinine, urine albumin, and albumin/creatinine ratio were 0.935, 1.013, and 1.090, respectively. In the multivariate logistic regression, children with CCHD had an odds ratio of 95.686, and those with aCCHD had an odds ratio of 7.215 compared to the reference group. The odds ratios for urine creatinine, urine albumin, and albumin/creatinine ratio were 1.04, 0.910, and 1.029, respectively.

Table 1. Sex distribution of participants

Groups of participants	Statistics	Gender		Total	X2	*P*
		Girls	Boys			
CCHD	n	25	36	61	0.976	0.614
	%	41.0%	59.0%	100.0%		
aCCHD	n	33	46	79		
	%	41.8%	58.2%	100.0%		
Control	n	34	36	70		
	%	48.6%	51.4%	100.0%		
Total	n	92	118	210		
	%	43.8%	56.2%	100.0%		

CCHD: cyanotic congenital heart defect, aCCHD: acyanotic congenital heart defect

Table 2. Comparing renal involvement in the groups of participants

Participants	Statistics	Renal involvement		Total	X2	*P*	Participants	C.C	*P*
		Yes	No						
CCHD	n	22	39	61	36.154	<0.001	aCCHD	0.350	*P*<0.001
	%	36.10%	63.90%	100.00%			Control	0.394	*P*<0.001
aCCHD	n	5	74	79			Control	0.082	0.317
	%	6.30%	93.70%	100.00%					
Control	n	2	68	70					
	%	2.90%	97.10%	100.00%					
Total	n	29	181	210					
	%	13.80%	86.20%	100.00%					

CCHD: cyanotic congenital heart defect, aCCHD: acyanotic congenital heart defect

Table 3. Comparison of laboratory variables between cyanotic and acyanotic patients

Variables	Patients	Mean	SD	Median	Mean Rank	Sum of Ranks	Mann-Whitney U	*P*
k	cyanotic	4.72	0.53	4.60	93.61	5710.50	99.50	*P*<0.001
	acyanotic	4.30	0.32	4.30	52.65	4159.50		
Urine Cr	cyanotic	2.30	1.72	2.00	40.53	2472.50	581.50	*P*<0.001
	acyanotic	8.54	6.88	6.00	93.64	7397.50		
Urine Microalbumin	cyanotic	14.42	7.77	10.90	82.15	5011.00	1699.00	*P*=0.003
	acyanotic	10.08	2.04	10.20	61.51	4859.00		
Urine albumin / Cr	cyanotic	9.42	6.84	8.40	101.16	6171.00	539.00	*P*<0.001
	acyanotic	2.22	2.04	1.46	46.82	3699.00		
Hb	cyanotic	15.95	1.50	15.50	110.00	6710.00	0.00	*P*<0.001
	acyanotic	10.90	0.49	11.00	40.00	3160.00		

K: Potassium, CCHD: cyanotic congenital heart defect, aCCHD: acyanotic congenital heart defect , Cr: creatinine, Hb: Hemoglobin

Table 4. Univariate and Adjusted Logistic Regression Analysis of Factors Associated with Renal Involvement in Pediatric Patients with Congenital Heart Disease

	Univariate					Adjusted				
Factors	**B**	**S.E.**	**Wald**	**Sig.**	**OR**	**B**	**S.E.**	**Wald**	**Sig.**	**OR**
Control(ref)			26.165	<0.001	1			18.446	<0.001	1
CCHD	2.954	0.765	14.894	<0.001	19.179	4.561	1.476	9.546	0.002	95.686
aCCHD	0.832	0.853	0.950	0.330	2.297	1.976	1.332	2.201	0.138	7.215
Urine Cr	-0.067	0.024	7.698	0.006	0.935	0.045	0.033	1.910	0.167	1.046
Urine Microalbumin	0.013	0.038	0.118	0.731	1.013	-0.095	0.066	2.059	0.151	0.910
Urine albumin /Cr	0.087	0.030	8.338	0.004	1.090	0.029	0.061	0.226	0.635	1.029

CCHD: cyanotic congenital heart defect, aCCHD: acyanotic congenital heart defect , Cr: creatinine

Discussion

The study assessed renal function in 210 children, including 140 CHD and 70 healthy controls. Renal involvement was observed in 36.1% of children with cyanotic CHD, 6.3% in acyanotic CHD, and 2.9% in controls. Children with CCHD showed higher levels of microalbuminuria, urinary albumin-to-creatinine ratio, and hemoglobin, while urinary creatinine was more elevated in the aCCHD group. Logistic regression revealed significantly increased odds of renal involvement in children with CCHD (OR=95.69) and aCCHD (OR=7.22) compared to healthy controls.

Additionally, comorbidities and structural abnormalities outside the heart have been observed in children with CHD, controlled as an exclusion criterion because influencing disease progression and treatment approaches (18). In patients with CHD, age has emerged as an independent risk factor for renal complications. The prevalence of chronic kidney disease (CKD) in children with CHD ranges from 2 to 3 percent during childhood and increases to 7 to 14 percent in adolescence and early adulthood. Unlike their peers without CHD of similar age, children with CHD do not exhibit comparable rates of renal impairment, as age correlates directly with disease duration and polycythemia (19,20).

Research on renal function in CHD patients remains limited (21). Studies by Talolena *et al*., (22) and Zheng *et al*., (23) underscore that patients with CHD, particularly those with cyanotic CHD (CCHD), face a heightened risk of renal diseases compared to controls. For instance, Talolena *et al*., (22) conducted a study involving 78 children, including 44 with CCHD and 34 controls, revealing a significant association between CCHD and nephropathy incidence in children. Furthermore, their findings indicated variability in nephropathy incidence among different CHD subtypes. Gillesén *et al*., (4) reported a 6.44-fold higher risk of kidney damage among CHD patients compared to controls. Fang *et al*., (17) found renal involvement in 46.5 percent of the 359 CHD patients included in their study. Amozgar *et al*., (22) have confirmed that children with cyanotic congenital heart disease (CCHD) are at increased risk of kidney damage, which correlates more closely with the duration of cyanosis, a proxy for age. Khadijeh *et al*., (24) conducted a study involving 55 children, including 22 with acyanotic CHD (aCCHD), 22 with cyanotic CHD (CCHD), and 11 controls. Their findings indicated that children with CCHD faced a greater risk of renal impairment compared to both controls and those with

aCCHD. Amornchaicaroensuk *et al*., (25) investigated renal function in older children and adults with CHD, employing measurements such as glomerular filtration rate (GFR), urinary protein/creatinine ratio, and urinary albumin/creatinine ratio. They concluded that the CCHD group exhibited a higher prevalence of impaired renal function than the aCCHD group. Mohamed (18) found no association between the degree of glomerular filtration rate (GFR) and the presence of cyanosis in their study. Hamed *et al*., (26) evaluated 49 patients with congenital heart disease (CHD), dividing them based on significant albuminuria. Initially, they reported a higher risk of renal involvement in children with cyanotic CHD (CCHD). However, they observed no statistically significant differences in GFR markers or urinary albumin/creatinine ratio (Alb/Cr) between children with CCHD who had significant albuminuria and those who did not. Maleki *et al.*, (27) found that the GFR of severe CCHD and acyanotic CHD (aCCHD) groups did not differ significantly from that of the mild CCHD group. In contrast, the current study observed a declining trend in GFR among children with CCHD, regardless of age, compared to both aCCHD groups and controls. According to Mohamed *et al*., (18), infants under one year old with aCCHD did not exhibit abnormal GFR values, whereas 15.4% of infants in the same age group with CCHD showed statistically different abnormal values compared to controls. In patients older than one year, there exists a notable disparity in glomerular filtration rate (GFR) between those with acyanotic CHD (aCCHD) and cyanotic CHD (CCHD), with abnormal GFR observed in 8.3% of aCCHD patients and 25% of CCHD patients exhibiting GFR values outside the normal range. These findings underscore the significant impact of CHD, particularly CCHD, on renal function as evidenced by several cited studies. Hypoxemia in CCHD impairs effective blood pumping by the heart, leading to blood congestion and elevated pressure in the main vein supplying the kidneys, thereby causing renal congestion. Reduced oxygenated blood flow further compromises kidney function. Congenital cardiomyopathy (CCHD), or critical CHD, reduces systemic oxygen delivery to the body. Notably, the most complex lesion categories—severe non-conotruncal and conotruncal lesions—demonstrate the highest susceptibility to kidney damage (CKD). This aligns with prior research and ongoing investigations highlighting a robust association between anatomical complexity, cyanosis, and renal dysfunction (28-30). Chronic low oxygen levels in the blood, known as hypoxemia, originate in the arteries and can indicate issues with blood circulation or respiratory function (31,32). In cases of congenital heart disease (CHD), renal dysfunction is a significant consequence of prolonged hypoxia. Persistent hypoxia in critical CHD stimulates erythropoietin production, leading to erythrocytosis and increased blood viscosity (hyperviscosity). This hyperviscosity can elevate the filtration fraction, hydraulic pressure within the glomerulus, and resistance in the glomerular efferent arteriole, thereby increasing glomerular oncotic pressure. Other factors contributing to changes in renal function include polycythemia, alterations in renal blood flow, intraglomerular hemodynamic shifts, and activation of neurohormonal pathways. Cyanosis, a manifestation of chronic hypoxia in individuals with critical CHD, can lead to kidney pathologies such as glomerulosclerosis due to abnormalities in hydrostatic pressure (32). The primary cause of elevated hemoglobin levels in blood is typically attributed to low oxygen levels. In the context of chronic hypoxemia, children with congenital heart disease (CCHD) often experience increased hemoglobin (Hgb) levels to compensate for insufficient oxygen delivery. Elevated hemoglobin levels have been associated with progressive declines in renal function and are indicative of early-stage cardio-renal risks in chronic illness. In the current study, significantly higher hemoglobin levels were observed in children with CCHD compared to both those with non-CCHD and controls. Moreover, children with renal dysfunction exhibited notably elevated hemoglobin levels compared to those without renal complications. The term "electrolyte" encompasses a wide range of electrically charged minerals and compounds present in blood, urine, tissues, muscles, and other bodily fluids. These substances play crucial roles in various essential physiological processes, particularly those involving myocardial function—the muscular tissue of the heart (33,34). Maintaining electrolyte balance is critical for safeguarding cellular function, tissue perfusion, energy production, muscle contraction, and acid-base equilibrium, thereby ensuring overall bodily homeostasis (35). The prevalence of potassium imbalances in patients with impaired kidney function underscores the kidneys' crucial role in regulating potassium homeostasis (36). Individuals with renal impairment may experience either hyperkalemia or hypokalemia, with hyperkalemia typically stemming from decreased kidney function (37). Our study revealed that children with congenital heart disease (CCHD) and those with renal dysfunction exhibited significantly elevated potassium levels above the normal range. Consequently, elevated potassium levels, known as hyperkalemia, can persist in the bloodstream and pose

health risks (38). The primary findings of our study align closely with those of previous research, albeit with slight variations attributable to patient age, methodological differences, and the severity of cyanosis. Specifically, our study highlights that children with CCHD face a heightened absolute risk of kidney disease compared to those with less severe forms of congenital heart disease (aCCHD).

Limitations

While this study provides valuable insights into renal function among children with congenital heart disease (CHD), several limitations should be considered. Firstly, the sample size, although adequate for detecting significant trends, may not fully represent the diverse spectrum of CHD severity and associated renal outcomes, it was due to lack of cooperation in clinical process by some of the participants that was the main cause of low sample size. And then, the study's observational design limits the establishment of causal relationships between CHD and renal dysfunction, warranting further prospective studies to validate these associations rigorously.

Based on the findings from this study, it is evident that children with congenital heart disease (CHD), particularly those with cyanotic congenital heart defects (CCHD), face a significant risk of renal dysfunction. The research underscored that factors such as chronic hypoxia, elevated hemoglobin levels, and disturbances in potassium balance play pivotal roles in contributing to renal complications in these patients. Our results highlight the importance of monitoring renal function closely in children with CCHD, as early detection and intervention could potentially mitigate the progression of kidney disease. Further studies addressing the specific mechanisms and optimal management strategies are warranted to improve outcomes and quality of life for these vulnerable patient populations.

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