Original Article

Patterns of Congenital Heart Diseases in Pediatric Population: Findings from 2D- Echocardiography at University of Benin Teaching Hospital, Benin City, Edo State, Nigeria

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ABSTRACT

Congenital heart disease (CHD) is the most common congenital anomaly worldwide and a major contributor to childhood morbidity and mortality. In Nigeria, data on the spectrum and determinants of CHDs remain limited. The study aimed to describe the spectrum, severity and associated socio-demographic factors of congenital heart diseases among children presenting to the University of Benin Teaching Hospital, Benin City, Nigeria. This was a prospective cross- sectional study conducted between February and September 2025. Children aged 0-16 years with suspected CHD were consecutively recruited from pediatric cardiology clinic and children emergency ward. Data were collected using structured questionnaires and diagnoses confirmed using 2D echocardiography. CHDs were classified as mild, moderate or severe using ICD-10 criteria. Data were analyzed using SPSS version 26.0 with chi-square tests and multivariate logistic regression used to assess associations and predictors. A total of 168 children with CHDs were studied, with an equal male- to – female ratio. Acyanotic CHDs predominated (82.1%) with atrial septal defects (34.0%) and ventricular septal defects (31.6%) being the most common lesions. Tetralogy of Fallot (11.6%) was the most frequent cyanotic CHD. Nearly half of the children (47.6%) had severe CHDs. Maternal education, marital status, tribe, place of residence were significantly associated with CHD occurrence ($p < 0.005$). On multivariate analysis, children residing in urban areas were less likely to have CHDs compared with those in rural areas (OR=0.39, $p = 0.019$).

Keywords: Disease severity, Congenital heart disease, Echocardiography, ICD- 10 classification.

INTRODUCTION

Congenital heart diseases are the most common birth defects and account for a third of all major congenital abnormalities in the world.[1] Congenital heart disease (CHD) is defined as a gross structural abnormality of the heart or intrathoracic great vessels present at birth that is actually or potentially of functional significance.[2] These anomalies usually result from defective morphogenesis during embryogenesis, leading to a wide spectrum of structural and functional cardiac lesions. The malformations may be limited to the cardiovascular system (nonsyndromic) or occur in association with anomalies of other systems as part of defined syndromes (syndromic).[3]They are a diverse group of disorders with varying aetiology, symptomatology, severity and outcome.[3] CHD is among the cause of morbidity and mortality in children.

Most of the data about CHD globally are extrapolated from high- income countries and quality of regional data from low and middle – income countries (LMIC) are lacking.[4] Adequate

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documentation of births and mortality is essential to drive a policy shift that would affect access to care for children with heart disease and to fight the perception that CHD rate in LMIC are lower than high- income countries. Globally, the prevalence of congenital heart diseases is estimated to be 10-12 per 1000 live births and represents approximately 1.35 million live births each year.[5] Reported figures vary worldwide, and a meta-analysis by van der Linde found that the highest CHD birth prevalence was in Asia, 9.3 per 1000 live births and the lowest in Africa, 1.9 per 1000 live births.[1] The prevalence of CHD in LMIC countries, especially in Africa, is underestimated owing to a paucity of data, lack of priorization of cardiac services, access to care in general, poor health infrastructure, lack of trained personnel, limited resources and early infant mortality before diagnosis.[7] In Nigeria, several hospital – based studies have provided insight into the burden of CHD, though with varying prevalence estimates. Sadoh et al., Ekure et al., Chinawa et al., Otaigbe et al documented prevalence of 14.4, 6.6,2.2,3.5 per 1000 in Benin, Lagos, Enugu, Port-Harcourt Nigeria respectively.[8,9,10,11] More recent studies in Jos by Ige et al[12] and Asaba by Okwundika et al[13] have reinforced that the true burden is probably underestimated because of methodological differences between studies and lack of echocardiographic detection of asymptomatic disease. There is also marked regional inequality in mortality with mortality rate correlating closely with the country –level socioeconomic indicators. These variations highlight the need for more robust, prospective and echocardiography-based studies to ascertain the true spectrum of CHDs in Nigeria. Improving mortality from CHD will be integral to achieving the Sustainable Development Goals (SDGs), including SDG 3.2 to reduce neonatal mortality and SDG 3.4 to reduce premature deaths from non-communicable diseases.[14]

The aim of this study therefore was to investigate the spectrum and associated risk factors of congenital heart diseases in children presenting to University of Benin Teaching Hospital, Benin City using 2D echocardiography. Specifically, this study determines the distribution of different types of congenital heart disease among children attending UBTH Benin, classified CHDs according to severity (mild, moderate, severe) using ICD-10 diagnostic criteria and identified maternal, perinatal and neonatal predictors CHD in the study population

MATERIALS AND METHODS

This was a descriptive cross-sectional study. Two hundred children with signs and symptoms suggestive of CHD between the ages of 0 month to 16 years were enrolled in the study. The study was carried out between Feb - Sept 2025. The participants were recruited consecutively from the paediatric cardiology clinic and children emergency ward of University of Benin Teaching Hospital Benin, Edo state Nigeria. Approval was obtained from the hospital's Research Ethics Committee (NHREC-UBTH-HREC/24/12/2022B). Informed written consent and assent were obtained from the parents/caregivers before enrollment into the study.

Two Resident Doctors in pediatrics at UBTH served as research assistants and were trained by the lead investigator for three days on the study protocol, administrations of questionnaires, and the required clinical examinations including echocardiogram findings.

Data collection

A semi-structured questionnaire developed specifically for this study was used to obtain basic bio-demographic and clinical data about the children and their parents from the mothers/caregivers. This information was documented by the trained assistants. Demographic data such as maternal age, parity, tribe, highest level of education, gestational age at delivery, birth weight, age of the child, age at onset of symptoms, age at diagnosis. Clinical signs and symptoms were corroborated with the case notes.

A 2D- Echocardiogram was performed by the lead researcher, who has been trained in and routinely performs pediatric echocardiography on all the enrolled patients using GE-vivid T9 matrix. A diagnosis of CHD was made in any child with single or multiple structural heart defects. The CHDs were classified as mild, moderate or severe lesions according to the ICD-10 diagnostic codes;

Mild lesions included small atrial septal defect

(ASD) 3-5mm in diameter and small ventricular septal defect (VSD) less than 3mm in diameter. Other mild lesions included pulmonary stenosis with peak gradient <30mmHg and bicuspid aortic valve without aortic stenosis or incompetence.

Moderate lesions were large ASD >5mm in diameter; moderate sized PDA and VSD measuring 1.5-3mm and 3-6mm respectively; complex forms of VSD associated with other forms of CHD; non-critical coarctation of the aorta, moderate pulmonary stenosis with peak gradient of 30-60mmHg and mild to moderate aortic stenosis with ≤50mmHg peak gradient.

Severe lesions included all cyanotic CHD as determined by the presence of central cyanosis which is defined as oxygen saturation <95% in the absence of the other causes, and acyanotic CHD such as large VSD >6mm, large PDA >3mm, atrioventricular septal defects (AVSD), severe pulmonary stenosis with peak gradient >60mmHg, severe aortic stenosis >50mmHg peak gradient and any critical CHD such as severe duct- dependent lesions requiring urgent surgical intervention for survival. These included hypoplastic left heart syndrome (HLHS), critical coarctation of the aorta (CoA), critical aortic stenosis, tricuspid atresia without shunt defects, total anomalous pulmonary venous connection (TAPVC) and severe tetralogy of Fallot (TOF).

Data Analysis

Data were processed and analyzed using SPSS version 26.0. Means and standard deviations were used as summary indices for numerical data such as age while non- numerical data such as gender, type and severity of CHD were presented as frequencies or percentages or as charts. The unpaired student's t-test was used to test the difference between means of continuous variables such as age, weight and gestational ages of children.

Analysis of variance (ANOVA) was used to compare means of more than two groups while the chi-squared test was used to compare non- numerical characteristics such as gender, place Multivariate analysis was also performed to determine the association of CHD with maternal age and neonatal gestational age and weight. A 95% confidence interval was used in this study as the interval estimate and a p-value of ≤ 0.05 was considered statistically significant.

RESULTS

Socio-demographic characteristics of children with CHDs

Socio-demographic characteristics of the study participants are shown in Table 1. From the table, there was equal number of male and female participants. Greater proportions of the participants aged between 2 months and 5 years. Majority of the children (146/168;86.9%) were delivered at term and most (156/168;92.9%) of the children were singleton babies. A greater proportion (74/168;44%) had normal birth weights of 2.5 – 3.4kg. Onset of symptoms were in the post-neonatal period in 50% of the participants.

Table 2 shows socio-demographic characteristics of mothers of children with/without CHDs. A greater proportion of the mothers in both the observed group (100/168;59.5%) and control group (30/50;60.0%) were aged 20 - 34 years. Majority of the mothers in both the observed group (97/168;57.7%) and control group (24/50;48.0%) were educated up to the secondary level. Greater proportions of mothers in both the observed group (140/168;83.3%) and control group (38/50;76.0%) were not exposed to passive smoking. Greater proportions of mothers in both the observed group (142/168;84.5%) and control group (40/50;80.0%) had no antenatal infection. Additionally, greater proportion of mothers in both the observed group (163/168;97.0%) and control group (47/50;94.0%) were not obese. A greater proportion of the mothers in both the observed group (151/168;89.9%) and control group (43/50;86.0%) had folic acid supplementation.

From Figure I, exactly half (50.0%) of the children had isolated CHDs and the other half (50.0%) had multiple CHDs.

Table 3 presents the type of CHDs among the study participants. Approximately one-third of the children with CHDs had ASD (73;34%), 31.6%(68) had VSD, nearly 12%(25) had tetralogy of Fallot (TOF), approximately 7%(15) had patent ductus

arteriosus (PDA), 7.0%(15) had complete AVSD, 2.3%(5) and 1.9%(4) had tricuspid atresia and transposition of the great arteries (TGA) respectively; while pulmonary stenosis, tricuspid regurgitation, common atrium, patent foramen ovale (PFO), and hypoplastic left heart syndrome each accounted for less than 1%. Table 4 shows that majority of male (74; 88.1%) and female children (64; 76.2%) had an acyanotic CHD.

Based on ICD-10 classification, approximately forty-eight percent (47.62%) of the children with CHDs (isolated or multiple) had a severe type, approximately thirty-one percent (30.95%) had the moderate type, while 21.4% had a mild type (Figure II).

Table 5 shows association between socio-demographic characteristics and CHDs in children. There was no evidence of association between maternal age and CHD (χ^2 = 0.028, p = 0.870). All (100%) of the mothers who had a tertiary education had children with a CHD. There was a statistically significant association between maternal highest level of education and having a child with CHD (χ^2 = 21.363, p < 0.001). There was a statistically significant association between marital status and having a child with CHD (χ^2 = 127.963, p = 0.009). There was a statistically significant association between place of residence and having a child with CHD (χ^2 = 7.234, p = 0.027). There was no evidence of association between child's position and having a child with CHD (χ^2 = 4.571, p = 0.102). There was no evidence of association between passive smoking and having a child with CHD (χ^2 = 1.383, p = 0.297). There was no evidence of association between antenatal infection and having a child with CHD (χ^2 = 0.572, p = 0.515). All (100%) of the mothers who had diabetes had children with a CHD. There was no evidence of association between time of commencement of folic acid and having a child with CHD (χ^2 = 6.363, p = 0.095).

Table 6 presents predictors of occurrence of CHDs in children. The predictor variables in the model accounted for 10.8% of the variation observed in the outcome variable (occurrence of CHD in a child). Respondents that are aged ≥ 35 years are 1.057 (95% CI = 0.509 – 2.196, p = 0.882) more likely to have children with CHDs than those aged ≤ 34 years, although this effect was not statistically significant. Respondents that reside in urban areas are 0.392 (95% CI = 0.179 – 0.858, p = 0.019) times less likely to have children with CHDs than those who resided in rural areas. This effect was statistically significant at p < 0.05. The 2nd or 3rd child was 0.986 (95% CI = 0.289 – 3.361, p = 0.982) times less likely to have a CHD than the 1st child, whereas the 4th child or lower was 2.123 (95% CI = 0.702 – 6.423, p = 0.183) times more likely to have a CHD than the 1st child, although this effect was not statistically significant. Respondents who did not have antenatal infection were 1.247 (95% CI = 0.512 – 3.033, p = 0.627) times more likely to have children with CHDs than those who had antenatal infection, although this effect was not statistically significant. Respondents who were not obese were 2.324 (95% CI = 0.488 – 11.054, p = 0.289) times less likely to have children with CHDs than those that were obese, although this effect was not statistically significant.

Table 1: Socio-demographic characteristics of children with CHDs

Socio-demographic characteristics	Frequency (n = 168)	Percent (%)
Gender		
Male	84	50.0
Female	84	50.0
Age		
Neonate	21	12.5
2 - 11 months	58	34.5
1 - 5 years	65	38.7
6 - 10 years	16	9.5
> 10 years	8	4.8
Maturity		
Preterm	22	13.1
Term	146	86.9
Plurality		
Single	156	92.9
Multiple	12	7.1
Birth weight		
< 2.5 Kg	50	29.8
2.5 - 3.4 Kg	74	44.0
3.5 - 4.4 Kg	42	25.0
≥ 4.5 Kg	2	1.2
Onset of symptoms		
1st month	47	28.0
Late neonatal period	37	22.0
Post-neonatal period	84	50.0
Age at diagnosis		
Neonatal age	59	35.1
Infancy	69	41.1
After infancy	40	23.8

Table 2: Socio-demographic characteristics of mothers of children with/without CHDs

Socio-Demographic characteristics	Observed group		Control	
	Frequency (n = 168)	Percent (%)	Frequency (n = 50)	Percent (%)
Maternal age				
< 20 years	3	1.8	0	0
20 - 34 years	100	59.5	30	60.0
≥ 35 years	65	38.7	20	40.0
Maternal highest level of education				
No formal			1	2.0
Primary	17	10.1	8	16.0
Secondary	97	57.7	24	48.0
Tertiary	54	32.1	17	34.0
Maternal tribe				
Benin	86	51.2	24	48.0
Esan	18	10.7	0	0
Hausa	6	3.6	0	0
Igbo	21	12.5	13	26.0
Yoruba	5	3.0	8	16.0
Other	32	19.0	5	10.0
Marital status				
Single	19	11.3		
Married	145	86.3	48	96.0
Separated	0	0	1	2.0
Divorced	3	1.8	1	2.0
Widowed	1	0.6		
Place of residence				
Rural	53	31.5	9	18.0
Semi-urban	75	44.6	20	40.0
Urban	40	23.8	21	42.0
Child's position				
1st	69	41.1	14	28.0
2nd - 3rd	72	42.9	30	60.0
≥ 4th	27	16.1	6	12.0
Passive smoking				
Yes	28	16.7	12	24.0
No	140	83.3	38	76.0
Antenatal infection				
Yes	26	15.5	10	20.0
No	142	84.5	40	80.0
Maternal diabetes				
Yes	7	4.2	0	0
No	161	95.8	50	100.0
Maternal obesity				
Yes	5	3.0	3	6.0
No	163	97.0	47	94.0
Folic acid supplementation				
Yes	151	89.9	43	86.0
No	17	10.1	7	14.0
Time of commencement of folic acid; n = 151				
1st trimester	73	48.3	29	67.4
2nd trimester	71	47.0	14	32.6
3rd trimester	7	4.6	0	0

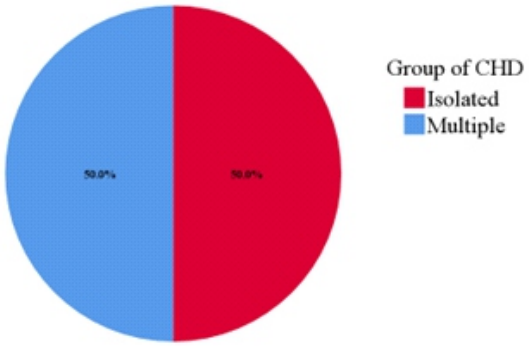

Figure 1: Pie chart showing Stratification of CHDs as isolated or multiple

Table 3: Type of Congenital Heart Diseases

Type of CHD	Frequency (n)	Percent (%)
Atrial septal defect	73	34.0
Ventricular septal defect	68	31.6
Tetralogy of Fallot	25	11.6
Patent ductus arteriosus	15	7.0
Complete AVSD	15	7.0
Tricuspid atresia	5	2.3
TGA	4	1.9
Pulmonary stenosis	3	1.4
Tricuspid regurgitation	2	0.9
Common atrium	2	0.9
Patent foramen ovale	2	0.9
HLHS	1	0.5

*AVSD: Atrioventricular septal defect; TGA: Transposition of the Great Arteries; HLHS: Hypoplastic left heart syndrome

Table 4: Proportions of children with CHDs stratified by gender and class of CHD

Gender	Class of CHD		Total
	Cyanotic	Acyanotic	
Male	10 (11.9%)	74 (88.1%)	84 (100%)
Female	20 (23.8%)	64 (76.2%)	84 (100%)

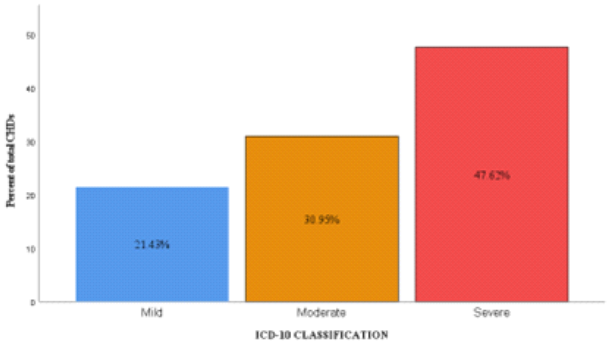

Figure 2: Bar chart showing ICD-10 classification of CHDs

Table 5: Association between socio-demographic characteristics and CHDs in children

Variables	CHD status of child		Test statistic	P value
	Has CHD Freq. (%)	Has no CHD Freq. (%)		
Maternal age				
≤ 34 years	103 (77.4%)	30 (22.6%)	0.028	0.870
≥ 35 years	65 (76.5%)	20 (23.5%)		
Maternal highest level of education				
Less than tertiary	114 (69.5%)	50 (30.5%)	21.363	**< 0.001***
Tertiary	54 (100.0%)	0		
Tribe				
Benin	86 (96.6%)	3 (3.4%)	127.963	**< 0.001***
Esan	18 (27.7%)	47 (72.3%)		
Others	64 (100.0%)	0		
Marital status				
Never married	19 (100%)	0	6.195	**0.009***
Married now or before	149 (74.9%)	50 (25.1%)		
Place of residence				
Rural	53 (85.5%)	9 (14.5%)	7.234	**0.027***
Semi-urban	75 (78.9%)	20 (21.1%)		
Urban	40 (65.6%)	21 (34.4%)		
Child's position				
1st	69 (83.1%)	14 (16.9%)	4.571	0.102
2nd - 3rd	72 (70.6%)	30 (29.4%)		
≥ 4th	27 (81.8%)	6 (18.2%)		
Passive smoking during pregnancy				
Yes	28 (70.0%)	12 (30.0%)	1.383	0.297
No	140 (78.7%)	38 (21.3%)		
Antenatal infection				
Yes	26 (72.2%)	10 (27.8%)	0.572	0.515
No	142 (78.0%)	40 (22.0%)		
Maternal diabetes				
Yes	7 (100.0%)	0	2.152	0.356
No	161 (76.3%)	50 (23.7%)		
Maternal obesity				
Yes	5 (62.5%)	3 (37.5%)	0.997	0.388
No	163 (77.6%)	47 (22.4%)		
Folic acid supplementation				
Yes	151 (77.8%)	43 (22.2%)	0.592	0.445
No	17 (70.8%)	7 (29.2%)		
Timing of commencement folic acid				
1st trimester	73 (71.6%)	29 (28.4%)	6.363	0.095
2nd trimester	71 (83.5%)	14 (16.5%)		
3rd trimester	7 (100.0%)	0		
Not applicable	17 (70.8%	7 (29.2%)		

* Significant p-value

Table 6: Predictors of occurrence of CHDs in children

Predictors	B (regression coefficient)	Odds ratio	95% CI for OR		P-value
			Lower	Upper	
Maternal age					
≤ 34 years	1				
≥ 35 years	0.055	1.057	0.509	2.196	0.882
Place of residence					
Rural	1				
Semi-urban	-1.243	0.289	0.114	0.731	**0.009***
Urban	-0.936	0.392	0.179	0.858	**0.019***
Child's position					
1st child	1				
2nd /3rd child	-0.014	0.986	0.289	3.361	0.982
4th child or lower	0.753	2.123	0.702	6.423	0.183
Passive smoking during pregnancy					
Yes	1				
No	0.423	1.527	0.644	3.621	0.336
Antenatal infection					
Yes	1				
No	0.220	1.247	0.512	3.033	0.627
Maternal obesity					
Yes	1				
No	0.843	2.324	0.488	11.054	0.289
Folic acid supplementation					
Yes	1				
No	0.557	0.573	0.188	1.745	0.327

R^2 (Nagelkerke)= 0.108; p-value = 0.065 ; * Significant p value

DISCUSSION

Findings from this study showed that most mothers of children with CHDs were within the 20-34 years age group, which corresponds to peak reproductive period. This finding is consistent with reports from several Nigerian studies where the majority of CHD cases occurred among mothers in this age range.[6,8] the predominance of CHD in this age group suggests that maternal age alone may not be a significant determinant of CHD in Nigeria. The study revealed that most mothers were married at the time of pregnancy which was similarly documented across different regions of Nigeria. Although marital status is not a direct risk factor for CHD but it may influence maternal access to healthcare services, antenatal and postnatal care utilization.

Most mothers in this study had attained at least secondary education. This finding aligns with studies conducted in urban tertiary hospitals in Nigeria, where higher levels of maternal education have been reported among caregiver of children with CHD.[7,8,9] Despite this relatively high educational attainment, delayed diagnosis of CHD persisted, indicating systemic healthcare limitations rather than maternal ignorance. A high proportion of mothers attended antenatal care during pregnancy. This finding is comparable with previous Nigerian studies that reported good ANC attendance among mothers of children with CHD[8,15] However, the continued occurrence of late CHD diagnosis despite ANC attendance highlights deficiencies in routine antenatal screening, limited availability of fetal echocardiography and inadequate expertise in prenatal detection of cardiac anomalies. The study showed equal sex predilection for CHD. This is consistent to the findings by Ekure et al[19] and Sadoh et al.[20] However, Adebayo et al[16] and Sani et al[6] reported Male predominance. Moreso, Ogunkule et al [21] noted that there is lesion specific to females like ASD and PDA.

The present study demonstrated an equal distribution of isolated and multiple congenital heart diseases. This finding differs slightly from the pattern reported in many Nigerian studies, where isolated congenital heart defects predominate as reported by Sani et al[6] in Northern Nigeria and Adebayo et al[16] in South- west Nigeria. Similarly, Ekure et al[17] reported a predominance of isolated defects in Lagos Nigeria. In contrast, the relatively high proportion of multiple CHDs observed in the present study is comparable to the finding by Ogunkunle et al[21] that documented multiple defects among children presenting late or with severe

symptoms as referral to specialist centers. This suggests a possible referral bias as the study center is a tertiary institution that gets referrals for complex CHDs.

This study shows that acyanotic CHDs predominated with ASD (34%) and VSD (31.6%) accounting for almost two- thirds of all cases. This pattern indicates that left- to -right shunt lesions remain the most frequently diagnosed CHDs in the study population. The predominance of ASD over VSD may reflect increased echocardiographic detection of secundum ASCDs which often presents later in childhood and are more likely to be picked up during evaluation for recurrent respiratory infections or incidental murmurs. TOF was the most common cyanotic CHD (11.6%) followed by PDA and AVSD. TOF has consistently been reported as the leading cyanotic lesion due to its relatively better survival beyond infancy compared with other critical cyanotic CHDs, increasing the likelihood of hospital presentation and diagnosis.

Many Nigerian studies similarly report that septal defects are the most common CHDs among children. A multicenter Nigerian echocardiographic study reported that VSD was the most prevalent CHD (46.6%) followed by PDA and ASD in their cohort of children with CHDs;[22] also a systematic review of 17 Nigerian studies (1964-2015) found that VSD accounted for 40.6% of CHD cases with ASD at 11.3% and that TOF was the most common cyanotic CHD (11.8%).[23] Previous studies by Sadoh et al[20] in Benin city reported that isolated VSD constituted 55.1% of defects with TOF (28.6%) and other lesions less frequent.

This study showed that acyanotic CHDs predominated in both genders though cyanotic CHDs were relatively more frequent among females than males. Several hospital-based echocardiographic studies in Nigeria have shown that left- to- right shunt lesions are predominant and these defects arise from relatively common errors in cardiac septation and ductal closure during embryogenesis. Abdulkadir[22] reported that over 70% of CHDs were acyanotic with no marked gender disparity in overall distribution, although subtle variations existed for specific lesions. The slightly higher proportion of cyanotic CHDs among females observed in this study have been documented in some Nigerian studies; Sadoh et al.[20] in Benin city reported a female predominance among children with cyanotic CHD particularly TOF and truncus arteriosus. Similarly, studies from Lagos and Enugu [24,25] have noted female predominance in cyanotic lesions while males tended to predominate in acyanotic defects such as VSD and PDA. These variations have been attributed to biological factors, referral bias and differences in survival patterns. Conversely, a Nigerian study by Sani et al. reported male predominance in cyanotic CHDs particularly TOF suggesting that gender distribution may vary by region and sample size. The absence of consistent gender pattern across Nigerian studies suggests that sex may not be a strong independent determinant of CHD class.

The above study showed that according to ICD- 10, severe CHDs constituted the largest proportion of cases (47.6%) followed by moderate (30.9%) suggesting a predominance of moderate-to- severe CHDs in this study population. Several studies in Nigeria (Benin, Lagos, Enugu, Kano)[20,24,10,6] have shown that severe diseases constituted between 40-55% of diagnosed cases closely aligning with the 47.6% severe category observed in this study. The relatively low proportion of mild CHDs in this study mirrors Nigerian findings and is often attributed to late presentation, lack of routine neonatal cardiac screening and limited access to echocardiography resulting in under-diagnosis of small septal defects and other mild lesions. In West Africa, similar trends are indicated. Study by Anim et al.[26]in Ghana reported over half of children diagnosed with CHDs had lesions classified as severe reflecting referral bias toward symptomatic and life- threatening conditions. In Senegal, Diop et al.,[27] observed that critical and severe CHDs predominated particularly cyanotic lesions and large left-to- right shunts, due to delayed diagnosis and limited pediatric cardiology services at the primary and secondary healthcare levels. These findings are comparable to the severity distribution observed in the present study.

This study found that maternal education, tribe, marital status and place of residence were significantly associated with CHD while maternal

age, birth order, passive smoking, antenatal infections, maternal diabetes, obesity and folic acid supplementation showed no statistically significant associations. The absence of statistical association between maternal age and CHD in this study aligns with findings from Sadoh et al.[20] in Benin city and Animasahun et al.[24] in Lagos who reported no clear relationship between advanced maternal age and CHD occurrence. However, this contrast with global data suggesting increased risk with advanced maternal age. These discrepancies may be attributed to younger maternal age distribution, under-reporting of maternal age extremes. The strong association between low maternal education and CHD is consistent with Nigerian studies that highlight the influence of socioeconomic factors on congenital anomalies. Ekure et al[24] in Lagos reported that children with CHD were more likely to be born to mothers with low educational attainment reflecting poor health literacy and reduced antenatal care utilization. Similarly, Abdulkadir's systematic review emphasized maternal education as a proxy for socioeconomic status influencing detection and outcomes of CHD in Nigeria.

The statistically significant association between tribe and CHD observed in the study is similar to findings reported by Sani et al.[6] in Kano. This may not be true genetic susceptibility and may just be a reflection of regional population dominance and referral bias. Marital status was significantly associated with CHD in this study, with children of never- married mothers all having CHD. Comparable findings were reported by Okoromah et al.[28] in Lagos, who observed poorer health outcomes among children born to unmarried mothers largely due to limited social support and delayed healthcare-seeking behavior. In Nigeria, marital status is closely linked to socioeconomic stability and access to antenatal care. The significant association between rural/ semi- urban residence and CHD mirrors reports from Benin city, Ilorin and Zaria[20,21,29] where children with CHD were more frequently from non-urban settings. This may be attributed to limited antenatal services, delayed diagnosis and poor referral networks in rural areas while Urban residence is associated with better access to prenatal screening and early diagnosis. The lack of a significant association between birth order and CHD is consistent with findings from Animasahun et al.,[24] and Sadoh et al.,[20] who reported inconsistent or weak relationships between parity and CHD. This suggests that parity alone may not be a strong determinant of CHD risk in Nigerian population. This study found no significant association between CHD and passive smoking or antenatal infections similar to observations by Sani et al. and Ekure et al.[24] Nigerian authors have noted that smoking prevalence among women are under-reported while antenatal infections are frequently self- reported without laboratory confirmation, limiting accurate risk assessment. The absence of a significant association between folic acid supplementation or timing of initiation and CHD is similar to findings from Ilorin and Benin City where late antenatal booking and inconsistent supplementation were common.[20,29]

CONCLUSION

1. Acyanotic CHDs predominated with ASD and VSD accounting for nearly two- thirds of all cases while TOF was the most common cyanotic lesion
2. There was an equal gender distribution overall though cyanotic CHDs were relatively more frequent among females
3. Almost half of the children had severe CHDs based on ICD- 10 classification reflecting a predominance of moderate- severe disease
4. Socio- demographic factors- maternal education, marital status, tribe and place of residence were significantly associated with CHD occurrence.

Recommendations

1. Strengthen Early detection: Routine cardiovascular screening during antenatal and postnatal care should be improved.
2. Improve access to care in rural and semi- urban areas: Decentralization of pediatric cardiology services and strengthening referral networks are needed to reduce delays in diagnosis
3. Enhance maternal education and health literacy: health education programs targeting women of reproductive age should emphasize early antenatal booking and recognition of warning signs of congenital anomalies

4. Further research: Larger, multicentered, population- based studies to better estimate the true prevalence and risk factors of CHDs in Nigeria.

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