Birth prevalence of congenital heart disease among newborns in a tertiary hospital in Benin City, Nigeria

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Birth prevalence of congenital heart disease among newborns in a tertiary hospital in Benin City, Nigeria

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Abstract

Background: Congenital heart disease (CHD) is an important cause of childhood morbidity. The birth prevalence and distribution of CHD among neonates in a tertiary hospital in Nigeria was determined.

Methods: This descriptive, cross-sectional study involved consecutive neonates in the neonatal and postnatal wards of the hospital. Bedside echocardiography was conducted on all neonates. Data entry and analysis was done with IBM-SPSS version 20.0.

Results: A total of 2 849 neonates were recruited, consisting of 1 482 (52.0%) males. Forty-one neonates had CHD, giving a birth prevalence of 14.4/1 000 live births. Of the 41 with CHD, 21 (51.2%) were male. Thirty-six (87.8%) neonates had acyanotic CHD, of which the commonest was isolated ventricular septal defect [11 (26.8%)]. Transposition of the great arteries [3 (7.3%)] was the commonest cyanotic CHD.

Conclusion: The birth prevalence of 14.4/1 000 live births in this study is high and buttresses the need for strengthening existing cardiac services in Nigeria.

Keywords: congenital heart disease, neonates, echocardiography

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Congenital heart diseases (CHDs) are structural abnormalities of the heart and or intrathoracic vessels and are the most common and important congenital anomalies.[1] The global incidence of CHD is reported to range from 4.5 to 9.7/1 000 live births.[2,3] Birth prevalence is different from region to region. It is reported to be highest among Asians and lowest in Africans.[2,3]

Most Nigerian studies on CHD are hospital-based audits of echocardiography laboratory findings.[4-7] The study on birth prevalence of CHD by Gupta and Antia[4] in 1967 found an incidence of 3.5/1 000 live births, but this study was limited by the use of only clinical screening of the newborn for the detection of CHD. There was no echocardiographic confirmation. This method would have missed out some of the minor and asymptomatic CHDs, thus giving a lower-than-expected birth prevalence value. Furthermore, this study was done over four decades ago and may not represent the current prevalence and distribution of CHDs in Nigeria.

The birth prevalence of CHD is influenced by the method of recruitment. Higher figures are obtained with echocardiographic screening of children, as it picks up nearly all cases of CHD.[2] The availability of portable and mobile echocardiography machines that can be deployed by the bedside of the child makes echocardiographic screening achievable and accurate. The use of echocardiographic screening for disease conditions is known to produce a more accurate prevalence of diseases. An example is the echocardiographic screening for rheumatic heart disease compared to the use of clinical screening where a four- to 10-fold increase in prevalence was observed with echocardiographic screening over the clinical method.[8]

Newborn screening captures the population of newborns with CHD, which may manifest only after discharge from hospital, and those who might die from the condition having escaped detection. This is particularly important because as many as 62% of deliveries in Nigeria occur outside healthcare facilities and are therefore unattended by skilled personnel.[9]

There is a paucity of studies on the birth prevalence of CHD in Nigeria, especially that determined by echocardiographic screening. Knowledge of the current birth prevalence will assist health planners to formulate appropriate policies to address the disease. This study was therefore conducted to determine the

birth prevalence of CHDs and the type in newborns in a tertiary centre in Nigeria.

Methods

The study was carried out in the postnatal and neonatal wards of the University of Benin Teaching Hospital (UBTH), Benin City, Edo State. Benin is the capital of Edo State. It is a cosmopolitan city located in the south-south geopolitical zone of Nigeria and is situated in the rainforest belt. It is 122 m above sea level with an estimated population of 1 085 676 (2006 census).

The UBTH is a 700-bed centre that provides primary, secondary and tertiary healthcare services to the entire Edo State and neighbouring states of Delta, Ondo and Kogi. Babies delivered vaginally and who are stable are discharged after 48 hours while those born by caesarian section are discharged after five days from the postnatal wards. This provides ample opportunity for their echocardiographic screening before they go home. The neonatal wards have a capacity for 50 beds. The annual birth rate in the UBTH is 2 000 births.

Ethical approval was obtained from the ethics committee of the UBTH with protocol number ADM/E 22 A/VOL VII/1380. Written informed consent was obtained from parents/guardians of the newborns.

This descriptive, cross-sectional study was carried out over 16 months (January 2017 to April 2018). The study participants were consecutive live newborns delivered in the UBTH and recruited from the postnatal and neonatal wards. All neonates were recruited irrespective of gestational age, APGAR score, maternal illnesses and mode of delivery.

The neonates who fulfilled the following criteria were recruited for the study: all newborns admitted into the postnatal wards after delivery, all sick babies admitted into the neonatal wards including those with congenital anomalies, and all babies of mothers with acute or chronic illnesses.

Neonates with the following criteria were excluded from the study: newborns with patent foramen ovale of ≤ 3 mm in size were not considered as significant CHD, and preterm newborns (< 37 completed weeks) with haemodynamically insignificant patent ductus arteriosus (PDA) were also not counted as having CHD. The following features characterised haemodynamically significant PDAs: those with PDA diameter > 1.5 mm, the ratio of left atrium:aortic diameter > 1.4, mitral valve E and A velocity ratio > 1, and those with flow reversal in the descending aorta.[10] Neonates with tiny muscular ventricular septal defects (VSDs) were equally excluded.

The age at recruitment, gestational age, gender and birth weight of each neonate recruited for the study were documented. Other information that was sought for included parental age. The socio-economic status of the family was determined using the method described by Olusanya *et al*.[11] The maternal age was categorised into < 40 and ≥ 40 years, while the paternal age was categorised into < 50 and ≥ 50 years. A full clinical examination was carried out on each neonate with the emphasis on the cardiovascular system.

The gestational age was determined by dates and early ultrasound where available. The babies were weighed using an infant weighing scale and the crown–heel length was taken in the supine position using a non-elastic tape. The gestational maturity was determined using the Dubowitz and Dubowitz method.[12]

Each neonate after feeds, while lying quietly or asleep, had a screening echocardiogram using standard views including two-dimensional, M-mode, colour flow and spectral Doppler. Analysis of the report was done according to the recommendations of the American Society of Echocardiography.[13] A Sonosite Micromaxx model with an 8-MHz probe was used to interrogate the heart from the apical, subcostal, parasternal and suprasternal views. The echocardiograms were done by the principal investigator who is trained in echocardiography.

Each newborn diagnosed with CHD was referred to the paediatric cardiology unit of UBTH for a comprehensive echocardiographic study and further management, including preparing for surgical intervention if indicated.

Statistical analysis

All collected data were checked for completeness, coded and analysed using the IBM-SPSS version 20.0 (Chicago, Illinois). The birth prevalence of CHD was expressed as number per 1 000 live births. The difference in birth prevalence between types of CHD was tested with the *Z*-test.

The relationship between variables such as gender, socio-economic class, parental age categories and the presence of CHD and its types was tested with the chi-squared test in a bi-variate analysis. The difference in mean values such as the mean gestational ages of newborns with different types of CHD was compared using the student's *t*-test or one-way ANOVA where more than two means were compared. The level of significance was set at $p < 0.05$.

Results

A total of 2 849 babies were recruited during the study period. There were 1 482 (52.0%) males with a male:female ratio of 1.1:1. They were recruited between 24 hours and 18 days of life with a mean age of 2.2 ± 1.7 days. The mean weight was 2.85 ± 0.73 kg with a range of 0.25–7.5 kg. Their mean length was 46.94 ± 4.32 cm with a range of 26–65 cm. The mean occipitofrontal circumference (OFC) was 33.79 ± 2.84 cm and ranged between 20 and 56 cm. The details of the sociodemographic and clinical characteristics of the newborns are shown in Table 1.

Table 1. The demographic characteristics of the study population

Characteristics	*Frequency*	*Percentage*
Maturity (*n* = 2 724)		
Term	2107	77.4
Preterm	594	21.8
Post-term	23	0.8
Gestational maturity (*n* = 2 702)		
Normal for gestational age	2306	85.3
Large for gestational age	121	4.5
Small for gestational age	275	10.2
Birth weight categories (*n* = 2 805)		
Normal birth weight	2195	78.3
Low birth weight	449	16.0
Very low birth weight	132	4.7
Extremely low birth weight	29	1.0
Socio-economic class (*n* = 2 734)		
High class	1612	59.0
Middle class	619	22.6
Low class	503	18.4

Table 2. Distribution of CHD by gender in the study population

Types of CHD	*Male,* n *(%)*	*Female,* n *(%)*	*Total*
Isolated VSD	7 (63.6)	4 (36.4)	11
Isolated ASD	2 (28.6)	5 (71.4)	7
Isolated PDA	3 (60.0)	2 (40.0)	5
Isolated AVSD	2 (40.0)	3 (60.0)	5
TGA	1 (33.3)	2 (66.7)	3
Common atrium	0 (0.0)	2 (100.0)	2
Cor triatriatum	1 (100.0)	0 (0.0)	1
PTA	1 (100.0)	0 (0.0)	1
TOF	0 (0.0)	1 (100.0)	1
Mixed CHD	5 (100.0)	0 (0.0)	5

VSD = ventricular septal defect, ASD = atrial septal defect, PDA = patent ductus arteriosus, AVSD = atrioventriculoseptal defect, TGA = transposition of the great arteries, PTA = persistent truncus arteriosus, TOF = tetralogy of Fallot. The breakdown of mixed CHD is in the text.

There were 2 438 fathers with available data on age. Of these, 2 361 (96.8%) were < 50 and 77 (3.2%) were ≥ 50 years. The maternal age ranged between 15 and 60 years with a median age of 31 years, while the fathers' ages ranged between 18 and 71 years with a median age of 37 years. Of the 2 774 mothers whose ages were available, 2 617 (94.7%) were < 40 while 147 (5.3%) were ≥ 40 years.

There were 41 newborns with CHD, giving a birth prevalence of 14.4/1 000 live births (95% CI: 10–18.8/1 000). Of the 41 newborns, 21 (51.2%) were male and 20 (48.8%) were female. Thirty-six (87.8%) had acyanotic CHD while five (12.2%) had cyanotic CHD. The commonest CHD was isolated VSD, which comprised 11 (26.8%) of the CHDs. Transposition of the great arteries [TGA, 3 (7.3%)] was the most prevalent cyanotic CHD. There were five (12.2%) cases of mixed CHD with a prevalence of 1.8/1 000 live births (95% CI: 0.2–3.4/1 000). The mixed CHD consisted of one case each of VSD + ASD (atrial septal defect), ASD + PDA (patent ductus arteriosus) and VSD + ASD + PDA and two cases of VSD + PDA. The distribution of the other CHD cases is shown in Table 2.

Five (12.2%) of the CHD cases were severe CHD, giving a prevalence of 1.8/1 000 live births (95% CI: 0.2–3.4/1 000). There was one case each of tetralogy of Fallot (TOF) and persistent truncus arteriosus (PTA) and three cases of TGA. Seventeen patients (41.5%) had minor CHDs constituting 6.0/1 000 live births (95% CI: 3.2–8.8/1 000). One neonate had dextrocardia but with no associated CHD.

Babies with CHD were significantly lighter in weight than those without CHD ($p = 0.001$). The mothers of babies with CHD were significantly older than those without CHD ($p = 0.038$) and similarly, the fathers of babies with CHD were significantly older than fathers of babies without CHD ($p = 0.016$). The difference in mean values of the length, occipitofrontal circumference (OFC) and gestational age between babies with CHD and those without CHD are shown in Table 3.

Table 3. Comparison of means of some variables between babies with and without CHD

Variable	*CHD Mean (SD)*	*No CHD Mean (SD)*	t-*test*	p-*value*
Birth weight (kg)	2.57 ± 0.75	2.87 ± 0.73	2.61	0.009*
Length (cm)	46.4 ± 4.3	47.1 ± 4.3	1.03	0.300
Occipitofrontal circumference (cm)	34.0 ± 3.6	33.8 ± 2.8	0.45	0.65
Gestational age (weeks)	36.4 ± 3.6	37.4 ± 3.3	1.92	0.055
Maternal age (years)	33.1 ± 4.8	31.5 ± 4.9	2.08	0.038*
Paternal age (years)	39.5 ± 6.2	37.4 ± 5.5	2.44	0.016*

*Indicates significant values.

Of the newborn variables evaluated for their relationship with CHD, low-birth-weight babies had significantly more cases of CHD than babies with normal birth weight ($p = 0.31$). Similarly, post- and preterm babies had significantly more CHDs than term babies ($p = 0.0019$). The details of the relationships between newborn and parental variables with CHD are shown in Table 4.

Although there were no genetic studies done, 12 babies had clinically diagnosed Down syndrome, representing an incidence of 1:237 live births.

Discussion

The birth prevalence of CHD of 14.4/1 000 live births in this study is higher than the reported range of 4.5–9.4/1 000 live births in a meta-analysis of global birth prevalence from articles published in 2010 and earlier.[1] The birth prevalence is however comparable to the 13.8/1 000 live births obtained from the birth registry of CHD in Castellon province of the Valencia region[14] of Spain, and the 13.7/1 000 live births recorded as the overall birth prevalence in Norway.[15] These high values speak to the ability of echocardiographic screening to accurately identify children with CHD even though some of the CHDs may be clinically silent.

The birth prevalence in this study is higher than the 8.07/1 000 live births recorded in a similarly conducted study in North India.[16] The difference in prevalence is mainly due to exclusion of a large number of minor CHDs in the Indian study. For instance, ASDs ≤ 5 mm in dimension were excluded, whereas in our study, those ≤ 3 mm were excluded, allowing more cases in our study to be recruited, with the attendant higher birth prevalence.

While the overall prevalence in this study is high, the prevalence of severe CHD of 1.6/1 000 live births is low and consistent with other previous studies. The implication of this is

Table 4. Distribution of CHD by neonatal and parental characteristics

Variable	*CHD* n *(%)*	*No CHD* n *(%)*	*Total*	p-*value*
Gestation ($n = 2\,724$)				
Term	22 (1.0)	2085 (99.0)	2107	0.009
Preterm	18 (3.0)	578 (97.0)	594	
Post-term	0 (0.0)	23 (100.0)	23	
Gestational maturity ($n = 2\,848$)				
Normal for gestational age	31 (I.3)	2275 (98.7)	2306	0.106
Large for gestational age	1 (0.8)	120 (99.2)	121	
Small for gestational age	8 (2.9)	267 (97.1)	275	
Birth weight categories ($n = 2\,805$)				
Normal birth weight	24 (1.1)	2171 (98.9)	2195	0.0043
Low birth weight	14 (3.1)	435 (96.9)	449	
Very low/extremely low birth weight	2 (1.2)	159 (98.8)	161	
Maternal age categories ($n = 2\,774$)				
< 40 years	36 (1.3)	2591 (98.7)	2627	0.0010
≥ 40 years	5 (3.4)	142 (96.6)	147	
Paternal age categories ($n = 2\,438$)				
< 50 years	34 (1.4)	2327 (98.6)	2361	0.21
≥ 50 years	3 (3.9)	74 (96.1)	77	
Socio-economic class ($n = 2\,734$)				
High class	21 (1.3)	1591 (98.7)	1612	0.27
Middle class	7 (1.1)	612 (98.9)	619	
Low class	11 (2.1)	492 (97.8)	503	

that the observed increase is caused by minor and asymptomatic CHDs of septal defects and PDA. Similar findings have been noted in other studies, further emphasising the advantage of echocardiographic screening.

The most prevalent CHD was isolated VSD, followed by isolated ASD. This finding is in agreement with other previous studies that showed VSD to be the most prevalent CHD and acyanotic CHD sub-group.[1,14,15] The commonest cyanotic CHD in this study was TGA. This is similar to some previous studies,[16,17] but at variance with other studies that showed TOF as the commonest cyanotic CHD.[1,4-7,14]

It is possible that because the screening in our study was at birth, neonates with TGA were detected before they presented later in the neonatal period with symptoms, whereas most of these other studies were audits of echocardiography laboratory findings in which children with TGA may not have been represented, having died from their condition before accessing diagnostic facilities. In our setting, such babies, because of lack of funds, would have passed on or been missed altogether if delivered outside the hospital setting.

The birth prevalence of 14.4/1 000 live births in this study is at variance with the earlier report of low prevalence among Africans compared to Caucasians and Asians. The earlier studies in which low prevalence was reported among Africans could have been due to the method of recruitment. Previous studies utilised the method of clinical evaluation of possible CHDs, with echocardiographic confirmation of such CHDs.[4-7] This method could have excluded asymptomatic cases or ones with delayed presentation later in life. It would seem from this study that the birth prevalence of CHD in Africans compares with that of other races.

Babies with CHD in this study were significantly lighter in weight than those without the condition. Similarly, there were more low-birth-weight babies with CHD than those without CHD, although the difference did not reach statistical significance. These findings are consistent with previous work.[18] Although children without CHD were also taller than those with CHD, the difference did not reach statistical significance. Some previous studies have shown this difference to be significant.[18] It has been shown that lower birth weight predicted poorer five-year survival after surgery.[19]

The parents of babies with CHD were significantly older than those of children without CHD. This finding is similar to other studies that have described increasing risk of having babies with CHD with increasing maternal and paternal age.[20,21] It is thought to be caused by increased mutations in the germ cell line due to cumulative cell replications.

One limitation of this study is the relatively small sample size. The absence of some CHDs such as hypoplastic left heart syndrome, double-outlet right ventricle and Ebstein's anomaly is also a limitation, which may be due to the low incidence of such CHDs, as seen in previous studies.[2,3,5] It is possible that the screening of a larger population of neonates would yield such CHDs. Another limitation is the small number of CHDs such as TGA, PTA and TOF, which makes the determination of their specific prevalences difficult.

Conclusion

The birth prevalence of CHDs in this study was high and we found that the prevalence of CHDs in Africans was comparable to that of other races. This information is important for health policy formulators in preparing for interventions for the CHDs in our health facilities.

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