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Abstract

Background: Congenital Heart Disease (CHD) is the commonest group of congenital malformations and affects 7-8 per 1000 live born newborns. Nevertheless, it is estimated that more than 50% of babies with undiagnosed CHD are not detected by routine neonatal cardiac examination.

Aim: To find the incidence of CHD in newborns and to determine the accuracy of pulse oximetry for detecting clinically unrecognized critical congenital heart disease (CCHD) in the newborns.

Methods: Pulse oximetry was performed on clinically normal newborns within 4 hours of first day of life. Inclusion criteria: All newborns who were admitted in postnatal ward & Neonatal Intensive care unit (NICU). Exclusion criteria: babies and neonates with a prenatal diagnosis of duct dependent circulation. If oxygen saturation (SpO_2) was below 90%, then echocardiography was performed.

Results: During the study period, 4926 live born neonates were examined. Nine out of 12 neonates with SpO2<90% had CCHD. Four neonates had tetralogy of Fallot (TOF), two had tricuspid atresia, two had transposition of great arteries (TGA) and one had truncus arteriosus. The incidence of CHD was 33.49 per 1000 live births and CCHD was 1.82 per 1000. A pulse oximetry cut-off value of below 90% for detecting CCHD showed 90% sensitivity, 99.94% specificity, 75% positive predictive value (PPV) and 99.98% negative predictive value (NPV).

Conclusion: Pulse oximetry is safe, feasible and noninvasive and also used to screen for CCHD. It is the nice method to detect the CHD along with the physical examination of neonates by medical personal.

MeSH: Newborn, Screening, Heart defect, Pulse Oximetry, Incidence

Introduction

Congenital cardiovascular malformations are the commonest type of birth defects, occurring in 7 to 8 per 1000 live births and are responsible for most mortality in the first year of life.[1,2] One quarter of these children will have complex congenital heart disease (CCHD), which requires surgery or catheter intervention in the first year of life.[3] Neonates with such unrecognized CCHD can manifest with profound metabolic acidosis, intracranial hemorrhage, hypoxic-ischemic encephalopathy, necrotizing enterocolitis, cardiac failure, cardiovascular collapse and even death. Early surgical interventional therapies have contributed to declining levels of morbidity and mortality due to CCHD. Delayed detection or failure to diagnose CCHD continues to cause poor neurological outcomes and avoidable deaths.[4] Heart murmurs are one of the clinical finding of noncritical heart disease typically diagnosed on cardiac examination. However, many times these may be absent because of the underlying anatomy, prolonged decline of pulmonary vascular resistance or reduced ventricular function.[5-8] Pulse oximetry is a universal tool for detecting and differentiating preductal

and post-ductal hypoxemia.[9] Large number of studies have reported a high sensitivity and specificity for pulse oximetry for early detection of CCHD in newborn babies.[5-12] Pulse oximetry screening could provide improvements in CHD case detection and management. Clinical evaluation though mandatory can miss out the diagnosis as the findings or may be too subtle in the initial 24 hours of life. This study was designed to find the incidence of CHD and early detection of CCHD by pulse oximetry in a rural hospital.

Material & Methods

This hospital-based prospective study was conducted in the Neonatology Unit of Pediatric Department at Acharya Vinoba Bhave Rural Hospital (AVBRH), Sawangi Meghe, Wardha from August 2013 to February 2015. All newborns delivered in these hospital including preterm babies were included in the study. Neonates with a prenatal diagnosis of duct dependent circulation by fetal echocardiography and out-born babies were excluded. An informed consent was obtained from one of the parents (preferably by mother) before initial screening and the purpose of the screening was explained to them. The measurements of SpO2 were performed using a Massimo Single Extraction Technology (SET) handheld pulse oximeter with a neonatal reusable Nellcor SpO2 sensor OXI-A/N probe. SpO2 was measured within the first 4 hours of life by a trained social worker on all the limbs of the newborn. The pulse oximetry probe was held manually to the palm or wrist and to the sole, following a random order and measured the saturation (Figure 1).

Figure 1. Measurement of SpO2 by Pulse oximetry in newborns

Any screening saturation SpO2 ≥95% and saturation difference between right upper limb and lower limb was <3% was considered as normal. If the saturation difference between right upper limb and lower limb was ≥3%, then echocardiography was done. If the SpO2 was between 90-94%, clinical examination was performed. If suspicious of CHD, the neonate was referred for echocardiography. If no suspicion of CHD, then saturation was repeated after 6 hours and echocardiography done if SpO2 ≤95. When the saturation was ≤90%, echocardiography was performed. The definition which was used in our study is as follows:

- **CCHD** defines as lesions that include cyanotic defects such as pulmonary atresia, TGA, truncus arteriosus, Fallot's tetralogy (TOF), total anomalous pulmonary venous return (TAPVR), and tricuspid atresia, as well as left-sided obstructive lesions, including critical aortic stenosis, coarctation of the aorta, interrupted aortic arch, and hypoplastic left heart syndrome.
- **Non-critical CHD** was defined as any ASD >5mm, PDA>2mm, VSD, valvular pulmonary stenosis, aortic stenosis and pulmonary artery branch stenosis.
- **Normal variants** were patent foramen ovale or ASD <5 mm and PDA <2 mm.

For all newborns, clinical examination was performed with a special emphasis to signs and symptoms related to the cardiovascular system to detect CHD. The presence of central cyanosis, abnormal peripheral pulses, abnormal precordium, heart murmurs on cardiac auscultation, tachypnoea, and chest retractions were considered as positive clinical examination findings more suggestive of CHD.[9] The neonates with positive clinical examination also underwent echocardiography.

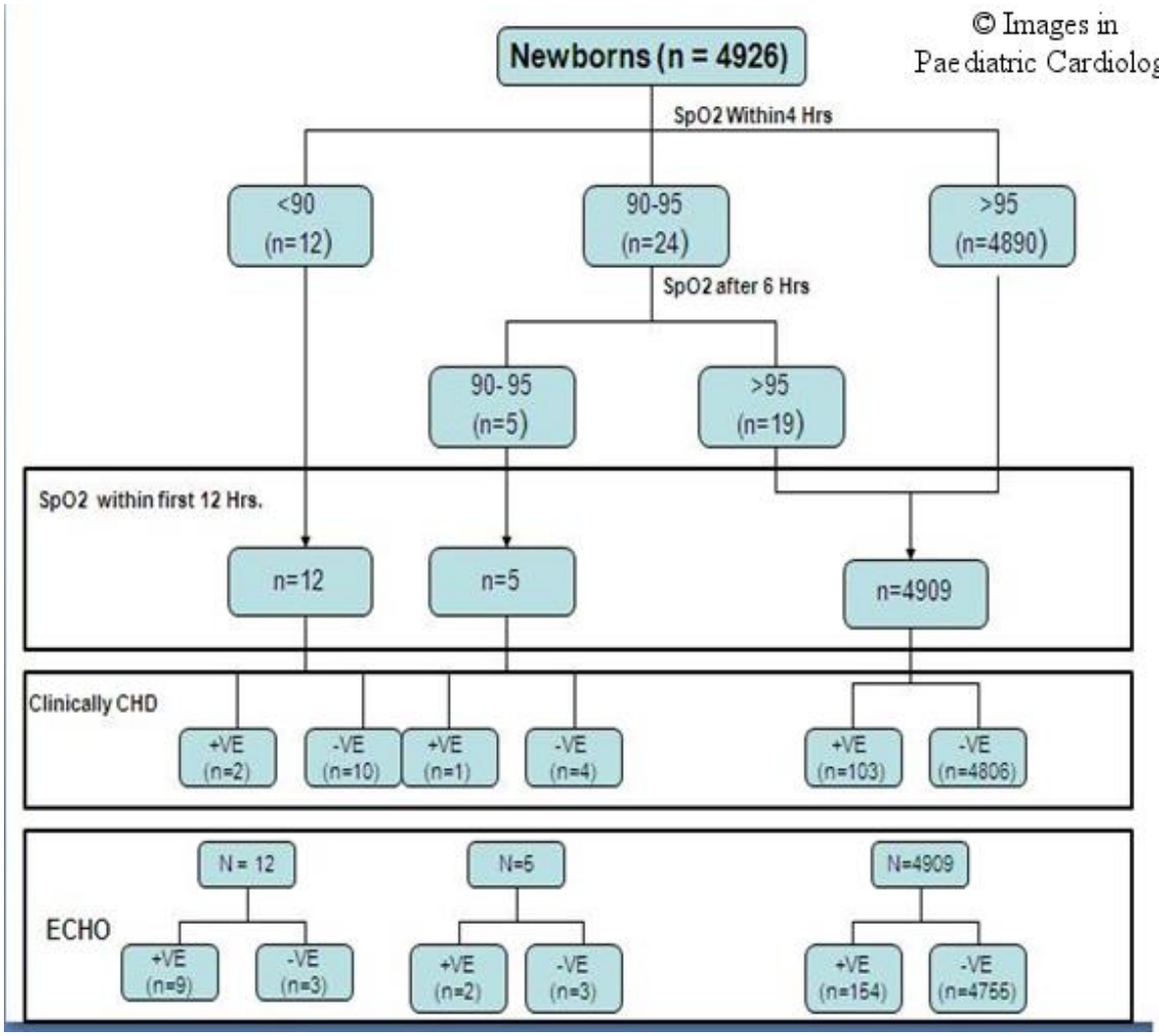
The study protocol was approved by the ethical committees of the JNMC Institute.

Statistical analysis: All statistical analyses were performed using SPSS 16 statistical software. The diagnostic accuracy of pulse oximetry for detecting the CCHD was measured by computation of sensitivity, specificity, positive predictive values and negative predictive values.

Results

A total number of 4926 (Male: 2510; Female: 2416) live born neonates were examined at AVBRH, Sawangi Meghe. Pulse Oximetry screening was performed on all newborns. There were 12 neonates with saturation < 90%, 24 neonates with saturation between 90-95% and 4890 neonates with saturation >95%. Nine out of 12 neonate with SpO2<90% had CCHD (TOF:4, Tricuspid atresia:2, TGA:2 and one had truncus arteriosus). One neonate who had severe coarctation of aorta was not diagnosed by pulse oximetry. Out of 24 with saturation 90-95%, 19 neonates showed normal saturation when repeated after 6 hours of initial measurement. Five neonates still showed afall in SpO2. Of these two had CHD, one a VSD and one am atrioventricular septal defect. A flowchart showing the distribution of neonates based on pulse oximetry screening for detection of CCHD in shown in figure 2.

Figure 2. Flowchart showing distribution of neonates based on pulse oximetry screening for detection of CCHD conducted in the SNCU.



There were 165 neonates with CHD in this study period, the incidence was 33.49 per 1000 live births and CCHD was 1.82 per 1000. A total of 106 patients (2.1%) had positive clinical evaluation, the most common being a murmur. Clinical evaluation was positive in only 3 patients with CCHD and 32 patients with non-critical CHD. The common non-critical CHD were PDA (n=89), VSD (n=48) and ASD (n=17). For detecting CCHD, a cut-off value of below 90% saturation showed 90% sensitivity, 99.94% specificity, 75% PPV and 99.98% NPV. Whereas cut-off value of below 95% saturation showed 90% sensitivity, 99.84% specificity, 52.94% PPV and 99.98% NPV. The sensitivity of the clinical signs for diagnosing CHD was 30%, 99.84% specificity, 48.11% PPV and 97.63% NPV.

Discussion

Congenital heart disease is one of the commonest human malformations, accounting for 10% of infant deaths and about 50% of deaths from malformations.[1] Delayed diagnosis of CHD is associated with a worse preoperative condition.[2] In our study the incidence was 33.49 per 1000 live birth which is higher than the earlier reported study from various population-based studies.[3-5]

PDA was the most common CHD followed by VSD found in our study. Fernanda Cruz De et al[9] reported 9 (0.23%) cases with CHD in their study in 4,027 newborns. In another study done by Mathur NB et al[11], found 72(7.57%) cases with CHD out of 950 screened cases. The incidence of CCHD varies from study to study, depending on the CCHD definition and the number of different cardiac defects included in the study.[10]T he incidence of CCHD was 1.82 per 1000 live birth in our

study agrees with a previous study.[6-7] This is mostly because of detection of cardiac lesions due to use of echocardiography for screening of all suspicious babies. All live newborns were eligible for this study, irrespective of gestational age or admission to a NICU. The initial presentation of CHD could mimic septicemia, respiratory distress or other conditions.[11-14] The symptoms of progressive cardiac failure, such as sweating, feeding difficulty, fast breathing and failure to thrive, are nonspecific in early neonatal periods. The cardiovascular examination of the neonate is important for diagnosing CHD especially murmurs, cyanosis and abnormal heart rate.

Ainsworth et al reported that the neonatal examination detects only 44% of cardiac malformations which present in infancy. He also concluded that if a murmur is heard there is a 54% chance of there being an underlying cardiac malformation. Bakr AF et al[12] detected 46% CHD with clinical examination and 31% by pulse oximetry detecting, resulting in a combined sensitivity of 77% with 99.7% specificity.

Screening newborns with non-invasive measurement of oxygen saturation has been accepted for early detection of CCHD. Vaidyanathan B et al[3] mentioned a poor sensitivity for pulse oximetry, for detection of CHD. Koppel RI et al[6] reported the effectiveness of pulse oximetry screening for CHD in asymptomatic newborns with sensitivity of 60%; specificity: 99.95%; PPV: 75%; NPV: 99.98% and accuracy of 99.97%. Our study showed a very good sensitivity and but PPV was less than optimal to detect CCHD. Results of present study were comparable to the study done by Arlettazet et al.[15]

Similarly, de Wahl Granelli A et al[16]found that systematic screening for CCHD with high accuracy required a latest generation pulse oximeter, and saturation from the right hand and one foot substantially improves the CCHD detection (Sensitivity: 98.5%; specificity: 96.0%; PPV: 89.0% and NPV: 99.5%).

Thangaratinam et al[17] completed a meta-analysis that included 13 studies and reported a specificity of 99.9% and a false positive rate (FPR) of 0.05%. Riede et al[2] reported foot pulse oximetry in 41,445 births and demonstrated a sensitivity of 77.8% and FPR of 0.1%. In contrast, de-Wahl Granelli et al[4] conducted simultaneous pre-ductal and post-ductal oximetry in 39,821 births with a sensitivity of 62% and a FPR of 0.17 %. Whereas Richmond et al[5] showed that repeat pulse oximetry brought their FPR down from 5% to 1%. In our study, for detecting CCHD, pulse oximetry cut-off value of below 90% for detecting CCHD showed 90% sensitivity, 99.94% specificity, 75% PPV and 99.98% NPV.

Pulse oximetry is a noninvasive, inexpensive, and useful tool that will detect the CCHD cases. Earlier diagnosis may lead to earlier interventions and improved neonatal outcomes. However, studies have mentioned the limitations of clinical examination in early detection of infants with CCHD. The clinical examination limitations include lack of specificity of neonatal heart murmurs, absence of any cardiac findings including murmur in nearly half of all infants with CCHD, and limited pediatrician experience in discriminating innocent from pathological murmurs. Also, studies have clearly reported that the visual assessment of cyanosis is suboptimal.[19-20] Echocardiography is the gold standard for diagnosis of CHD but it is not feasible and readily available as a routine screening tool in developing countries.[21-23] This study suggests that the presence of abnormal clinical signs such as murmurs should warrant an urgent cardiac evaluation.

Also, pulse oximetry may potentially emerge as a useful adjunct to clinical evaluation, especially for CHD. In the developing country, clinical evaluation and pulse oximetry after birth has increased the chances of detection of CHD.[3] An abnormal screening warrants prompt echocardiography. The major strengths of our study are the large number of babies prospectively screened for CHD.

Thus, this study concluded that the pulse oximetry screening is a simple, noninvasive test use in clinically normal newborns, for early detection of CCHD. Also, a detail physical examination along with pulse oximetry screening in the neonates will provide clues to the diagnosis of CHD.

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