

Utility of pulse-oximetry screening in newborns with nonductus-dependent cyanotic congenital heart defects: A reason to alarm?

Balaji Arvind¹, Anita Saxena^{1,2}, Sivasubramanian Ramakrishnan¹

¹Department of Cardiology, All India Institute of Medical Sciences, New Delhi, India, ²Pt BD Sharma University of Health Sciences, Rohtak, Haryana, India

ABSTRACT

- Objectives** : We aimed to compare the performance of pulse-oximetry screening in detecting nonductus-dependent cyanotic congenital heart defects (CCHDs).
- Methods** : In a prospective cross-sectional study, we recorded post ductal saturation of neonates (<48 h old) born at a community hospital in northern India. Subsequently, all underwent clinical examination and echocardiogram by a trained cardiologist. A saturation <95% was considered a “failed” screen.
- Results** : Ten neonates were identified to have nonductus-dependent CCHD on echocardiogram, five of whom had passed pulse-oximetry screen. This translated to a sensitivity of 50% (95% confidence interval [CI] 23.7%–76.3%) and a positive predictive value of 0.08 (95% CI 0.03–0.2), both of which were significantly less compared to that in ductus-dependent congenital heart defect.
- Conclusions** : Up to half of the nonductus-dependent CCHD may be missed if screened only using pulse oximetry. Parents should not be reassured regarding the absence of CCHD only based on a “pass” in pulse-oximetry screening.
- Keywords** : Cyanotic congenital heart disease, ductus-dependent cyanotic congenital heart diseases, nonductus dependent cyanotic congenital heart diseases, pulse-oximeter screening

INTRODUCTION

Newborn pulse-oximetry screening is an important intervention proposed for the earlier detection of cyanotic congenital heart defects (CCHDs). As a screening tool, it has been shown to have high specificity for detecting CCHD, however, the sensitivity is <80% which is still modest.^[1] It is likely that many serious CCHD that are considered within the scope of “targetable lesions”^[2] and which require early intervention may be missed

when pulse-oximetry is relied upon as the lone screening test, thereby providing a false sense of assurance to the family. Whether there is any difference in the performance of pulse-oximetry screening for detecting ductus-dependent CCHD and nonductus-dependent CCHD has not been studied. We studied the role of pulse-oximetry in detecting nonductus-dependent CCHD in a large cohort of neonates.

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

For reprints contact: WKHLRPMedknow_reprints@wolterskluwer.com

How to cite this article: Arvind B, Saxena A, Ramakrishnan S. Utility of pulse-oximetry screening in newborns with nonductus-dependent cyanotic congenital heart defects: A reason to alarm? *Ann Pediatr Card* 2022;15:41-3.

Access this article online

Quick Response Code:



Website:

www.annalspc.com

DOI:

10.4103/apc.apc_9_22

Address for correspondence: Dr. Anita Saxena, Vice Chancellor, Pt. BD Sharma University of Health Sciences, Rohtak, Haryana - 124 001, India.

E-mail: anitasaxena@hotmail.com

Submitted: 18-Jan-2022

Revised: 10-Mar-2022

Accepted: 01-Apr-2022

Published: 14-Jun-2022

METHODS

In a prospective cross-sectional model, we performed pulse-oximetry screening on neonates who were born during a particular 8-h period of each day at a community hospital in northern India. Over a period of 3 years, 19,009 neonates were enrolled. Postductal arterial oxygen saturation using pulse oximetry was obtained from either foot in all these neonates within 48 h of delivery. The test was carried out by the field investigator who was trained in using the Mindray PM-60 handheld pulse oximeter. Neonates with postductal saturation <95% were labeled to have “failed” the pulse-oximetry screen. Subsequently, all the neonates underwent a clinical and echocardiographic examination by a trained cardiologist. Clinical examination was considered abnormal if the child was detected to have cyanosis, murmur, or respiratory distress. The detailed methodology has been published elsewhere.^[3] In this brief communication, we aimed to characterize the nonductus-dependent CCHD that went undetected on pulse-oximetry screen (POS) in our cohort. The study protocol was approved by the institute ethics committee *a priori*.

RESULTS AND DISCUSSION

Significant congenital heart defects (CHDs) categorized as lesions targetable by pulse-oximetry screening, was confirmed by echocardiography in 33 neonates (prevalence: 1.7 (95% confidence interval [CI] 1.1–2.3)/1000 live births).^[2] Of which, ductus-dependent CCHD was identified in 23 neonates, the characteristics of which have been alluded to in our previous publication.^[3] The remaining 10 neonates were diagnosed to have nonductus-dependent CCHD, the details of which are enlisted in Table 1. Five of these 10 neonates passed the pulse-oximetry screen, which translated to a pulse-oximetry sensitivity of 50% (95% CI 23.7%–76.3%) for detecting nonductus-dependent CCHD. This is significantly less compared to the sensitivity for detecting ductus dependent CCHD of 91.3% (95% CI 73.2%–97.6%) ($P < 0.01$). The positive predictive value of a failed pulse-oximetry screening for detecting nonductus-dependent CCHD was 0.08 (95% CI 0.03–0.2) compared to 0.3 (95% CI 0.2–0.5) for detecting ductus dependent CCHD ($P < 0.01$) [Figure 1]. There was no difference in other performance parameters such as specificity (ductus-dependent CCHD: 68.3 [67.6–68.9] vs. nonductus-dependent CHD: 68.0 [67.4–68.7]) or negative predictive value (ductus-dependent CCHD: 99.9 [99.9–100] vs. nonductus-dependent CCHD: 99.9 [99.9–100]). When pulse-oximetry was combined with findings of an abnormal clinical examination identified by a trained physician, the sensitivity for detecting nonductus-dependent CCHD improved to

Table 1: Description of all neonates detected to have nonductus-dependent cyanotic congenital heart defects

Lesion	SpO ₂ (%)	Clinical examination result	Result (SpO ₂ <95% considered failed POS)
Single ventricle, No PS (SV physiology with severe PAH)	93	Normal	True positive
Tetralogy of Fallot	97	Abnormal	False negative
Tetralogy of Fallot	94	Normal	True positive
CC-TGA, very large VSD, and mild PS (SV physiology with severe PAH)	96	Normal	False negative
Tetralogy of Fallot	95	Abnormal	False negative
Persistent truncus arteriosus	93	Abnormal	True positive
Nonobstructive TAPVC-mixed type	90	Normal	True positive
Complete unbalanced AVSD, No PS, and hypoplastic arch (SV physiology with severe PAH)	97	Abnormal	False negative
Nonobstructive TAPVC- Coronary sinus type	96	Normal	False negative
Persistent truncus arteriosus	93	Abnormal	True positive

AVSD: Atrioventricular septal defect, CC-TGA: Congenitally corrected transposition of great arteries, PAH: Pulmonary artery hypertension, POS: Pulse-oximeter screen, PS: Pulmonic stenosis, SV: Single ventricle, TAPVC: Total pulmonary venous connection, VSD: Ventricular septal defect

80% (95% CI 49%–94.3%) which was still lesser than that for ductus-dependent CCHD, although the difference was not statistically significant.

In our cohort, two of the three neonates who had single-ventricle physiology with severe pulmonary artery hypertension, two of the three neonates with tetralogy of Fallot, one of the two neonates with nonobstructive total anomalous pulmonary venous connection went undetected on POS (false negative). These CCHDs are known to have a very high attrition rate in the 1st year of life if untreated.^[4] Although not immediately fatal, early diagnosis and intervention are crucial in all these lesions as well. All these missed lesions were considered within the scope of “target” lesions that were supposed to be detected on pulse oximetry as per the statement issued by the US Health and Human services’ advisory committee.^[2]

Almost all major studies published from high-income countries refer to the high false-positive rates of neonatal pulse-oximetry screening as the most important fallacy.^[1,5] According to the authors of these studies, neonates without CHD who falsely fail the POS are subjected to further investigations, thereby imposing additional burden to the health system. However, none of the studies emphasize the problems due to false-negative POS results. That is when

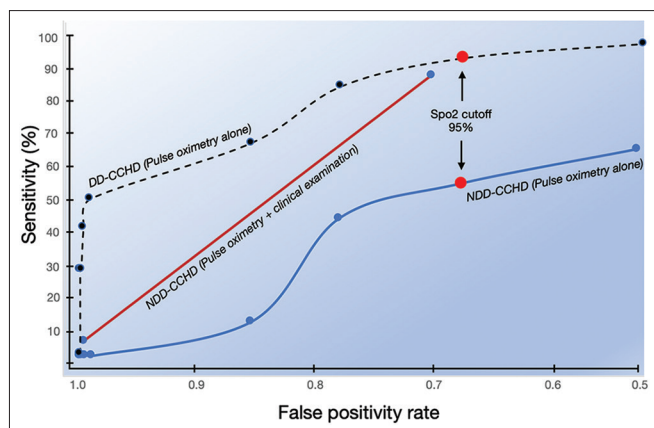


Figure 1: Receiver operating characteristic curves showing the performance of pulse-oximeter screening in the detection of ductus-dependent (dashed black line) and nonductus-dependent CCHDs (blue line). The area under the curve for the detection of nonductus-dependent CCHD is better if clinical examination is combined along with pulse-oximetry (red line)

a neonate with CHD passes the POS. This is an equally serious concern since it leads to a false reassurance to the family of a newborn with CHD. This is of great significance, especially in low-and middle-income countries where newborns are often discharged within 24–48 h without a detailed clinical examination. Delayed diagnosis of CHD is a common scenario in low-and middle-income countries and assurance of a normal heart may further delay the treatment.^[6] In our entire cohort, seven neonates with ductus-dependent CCHD and five with nonductus-dependent CCHD) passed the POS, resulting in a false-negative rate of 0.04%. This is higher compared to what has been reported in the previous pulse-oximetry screening studies.^[5,7] However, false-negative cases in the previous studies were identified only by review of clinical follow-up records, local CHD registries, or mortality databases rather than echocardiographic confirmation.^[1,5,7] This introduces an element of differential verification bias into all these studies, meaning that many “real” false negatives could have possibly been misclassified as true negatives, thereby underestimating the false negativity rate and at the same time overestimating the sensitivity of pulse-oximetry. Chances for the occurrence of this bias are negligible in our study since all neonates screened in our study had an echocardiographic confirmation of their underlying cardiac anatomy. Hence, the false-negative rates observed in our study could be the ideal estimate of the performance of pulse-oximetry as a screening tool. Despite the limitation of using a noncontemporary screening protocol in our study, reanalysis of our data has brought out a very important caveat associated with the utility of pulse oximetry as a screening tool for the detection of nonductus-dependent CCHD.

CONCLUSIONS

Up to half of the nonductus-dependent CCHD that require early intervention may be missed if pulse oximetry screening is used in isolation. Hence, pulse-oximetry should only be used as a tool to identify neonates needing further evaluation for the confirmation of an underlying CCHD. A pass in POS should not be used to reassure the parent regarding the absence of a CCHD in their neonate.

Financial support and sponsorship

The study was funded by a restricted grant from the Indian Council of Medical Research, New Delhi, India. The grant ID was 5/4/1-15/08-NCD-II, and it was used only for conducting the study.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Plana MN, Zamora J, Suresh G, Fernandez-Pineda L, Thangaratinam S, Ewer AK. Pulse oximetry screening for critical congenital heart defects. *Cochrane Database Syst Rev* 2018;3:CD011912.
2. Mahle WT, Martin GR, Beekman RH 3rd, Morrow WR, Section on Cardiology and Cardiac Surgery Executive Committee. Endorsement of Health and Human Services recommendation for pulse oximetry screening for critical congenital heart disease. *Pediatrics* 2012;129:190-2.
3. Saxena A, Mehta A, Ramakrishnan S, Sharma M, Salhan S, Kalaivani M, *et al.* Pulse oximetry as a screening tool for detecting major congenital heart defects in Indian newborns. *Arch Dis Child Fetal Neonatal Ed* 2015;100:F416-21.
4. Delisle G, Ando M, Calder AL, Zuberbuhler JR, Rochenmacher S, Alday LE, *et al.* Total anomalous pulmonary venous connection: Report of 93 autopsied cases with emphasis on diagnostic and surgical considerations. *Am Heart J* 1976;91:99-122.
5. Ewer AK, Middleton LJ, Furnston AT, Bhojar A, Daniels JP, Thangaratinam S, *et al.* Pulse oximetry screening for congenital heart defects in newborn infants (PulseOx): A test accuracy study. *Lancet* 2011;378:785-94.
6. Rashid U, Qureshi AU, Hyder SN, Sadiq M. Pattern of congenital heart disease in a developing country tertiary care center: Factors associated with delayed diagnosis. *Ann Pediatr Cardiol* 2016;9:210-5.
7. Diller CL, Kelleman MS, Kupke KG, Quarry SC, Kochilas LK, Oster ME. A modified algorithm for critical congenital heart disease screening using pulse oximetry. *Pediatrics* 2018;141:e20174065.