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Research Article

Pulse oximetry as a simple diagnostic test for persistent pulmonary hypertension of newborn in limited resource settings

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ABSTRACT

Background: Persistent Pulmonary Hypertension of newborn (PPHN), results from the disruption in the normal perinatal fetal- neonatal circulatory transition. The condition remains a difficult neonatal emergency to manage which is mainly attributed to the delay in diagnosis and stabilisation. Although the management of PPHN involves advanced diagnostic and therapeutic interventions, our study highlights that PPHN can be diagnosed even in limited resource settings, with simple bedside evaluation of pre-ductal and post-ductal saturation (Spo_2) difference.
Methods: A retrospective study of neonates with PPHN, admitted in NICU, of a government hospital in a limited resource setting, over a period of 6 months.
Results: Out of the 592 neonates admitted during the 6 months period in NICU, PPHN was diagnosed in 26 neonates (4.4%).The incidence was higher for male (57.7%),term gestation (84.6%), and appropriate for gestational age- AGA (88.5%). Meconium aspiration (53.8%) followed by perinatal asphyxia- clear liquor (30.8%) were the most common underlying etiology for PPHN in our study. Around 61.5% were diagnosed with PPHN based on pulse oximetry alone before the first dose of sildenafil, which was confirmed by ECHO later in 75% of the cases. PPHN improved in 92.3% of the study population (24/26 cases), out of which 62.5% were diagnosed based on pulse oximetry alone before intervention.
Conclusions: Our study emphasises that even in peripheral health centres with limited resources, PPHN can be diagnosed by pulse oximetry (pre and post ductal Spo_2 difference of >10%) along with clinical assessment. Such critically ill neonates can be initiated on pulmonary vasodilators like sildenafil and stabilised at the earliest that is crucial before referral to a tertiary care centre.

Keywords: Meconium aspiration, PPHN, Pre and post ductal saturation, Sildenafil

INTRODUCTION

Persistent Pulmonary Hypertension of Newborn (PPHN) is a syndrome characterised by increased pulmonary vascular resistance (PVR), right to left shunt and severe hypoxemia. The reported incidence is 0.43-6.8/1000 live births and the mortality rate is around 10-20%.[1] Early stabilisation is crucial for reversal of hypoxemia and to maintain end-organ perfusion. Our study highlights that knowledge of the clinical setting of PPHN is important in resource-poor settings with limited diagnostic modalities, where the condition can be diagnosed with documentation of pre and postductal saturation difference (Spo_2). This helps in initiating PVR reducing agents like sildenafil, even when 24hours ECHO evaluation is not available, which in turn leads to early stabilisation of such neonates.

METHODS

This data was retrospectively collected from the neonatal intensive care unit (NICU) of a government hospital over a period of 6 months. All neonates diagnosed with PPHN were included in the study. Neonates with multiple congenital anomalies and those with antenatally diagnosed congenital heart diseases were excluded from the study. Basic blood investigations and sepsis screening was done for all neonates. The gestational age, sex, mode of delivery, birth weight and the underlying etiology were noted.

Diagnosis of PPHN

Being a resource limited setting, our diagnosis of PPHN was based on suggestive clinical history, clinical examination and pre/post ductal Spo_2 difference with pulse oximetry with or without echocardiogram.

History: PPHN was considered in the background setting of meconium aspiration, perinatal asphyxia with clear liquor, intrauterine growth retardation (IUGR), congenital diaphragmatic hernia (CDH), pneumonia, sepsis etc.

Clinical assessment

The following clinical findings were suggestive of PPHN.

- Neonates with respiratory distress (tachypnea, retractions, grunt), while on any form of oxygen support .
- Labile oxygenation, increased Fio_2 requirements on CPAP and mechanical ventilation
- Prominent RV impulse, single S2, tricuspid regurgitation
- Absence of the features which favour cyanotic heart disease like grade3+ murmur, weak pulses, active precordium, cardiomegaly in chest X-ray.

Pulse oximetry

4 limb saturation was monitored in such neonates when they presented with unexplained desaturation while on any form of oxygen support, provided other conditions were ruled out. Arnold in his study considers PPHN, when the infant's clinical condition worsens despite optimal available management. This is also stressed by Pankaj, et al who mention that DOPE-SIPP (Displacement, Obstruction, Pneumothorax, Equipment failure, Shock, Infection, PDA, PPHN); should be ruled out when an infant desaturates on mechanical ventilation.[2]

Spo_2 recording was done with Massimo pulse oximetry in all 4 limbs concentrating more on preductal (right hand), and either foot (post ductal).The findings were noted 2 minutes after stable signals were observed. PPHN was diagnosed when there was a gradient of 10% or more between pre and post ductal Spo_2.[3] Such neonates with abnormal readings were subjected to 2 observations separated by 20 minutes.

Echocardiogram

This is done to document hemodynamic shunting, evaluate ventricular function and exclude congenital heart disease. Being a limited resource setting, ECHO was available only during morning hours and in week days.

Successful response

Is characterised by improved oxygenation, namely a ≥10% increase in Spo_2 with a reduced differential between pre- and postductal values, ability to wean FiO_2, a repeat ECHO documenting normal pressures 48 hours after stable oxygenation, done on day 5 or 7 whichever is later.

Other factors assessed

The age of presentation of PPHN was divided as

A. At birth,
B. Within 3 days after birth,
C. >3 days after birth based on the study done by Abdel Mohsen, et al.[4] The diagnostic modality before the first dose of sildenafil was divided as a) 4 limb Spo_2 alone b) 4 limb Spo_2 + ECHO. The outcome of PPHN and the overall outcome of the neonate was also noted.

RESULTS

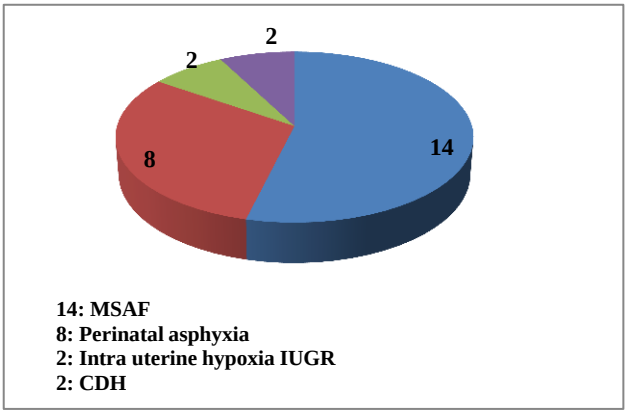

Figure 1: Distribution of etiological factors in the study.

Around 592 neonates were admitted in our NICU during the study period of 6 months. Retrospective data revealed around 26 neonates to have been diagnosed with PPHN; which constituted 4.4% of the total NICU admissions of that period. It was higher for male (57.7%), term gestation (84.6%), and appropriate for gestational age-AGA (88.5%). The most common underlying etiology for PPHN in our study was meconium aspiration (53.8%), perinatal asphyxia- clear liquor (30.8%). Intrauterine

hypoxia/IUGR and diaphragmatic hernia constituted 7.7% each (Figure 1). Majority of the neonates (69.2%) developed PPHN within 3 days after birth.

The mode of diagnosis before administration of first dose of sildenafil was assessed. Majority (61.5%) were diagnosed with PPHN based on suggestive history, clinical examination and pulse oximetry documentation of pre and post ductal Spo_2 difference. This group included neonates who were delivered or deteriorated at odd times and those who were referred from peripheral centres in critical stage, and started on sildenafil for stabilisation as round the clock ECHO evaluation was not available. The same group was subjected to ECHO at the earliest available time, which confirmed PPHN in75% of the cases. Around 38.5% of cases were diagnosed with pulse oximetry and simultaneous ECHO. PPHN improved in 92.3% of the study population (24 out of 26 cases), out of which 62.5% were diagnosed based on pulse oximetry alone before intervention with pulmonary vasodilators like sildenafil (Figure 2).

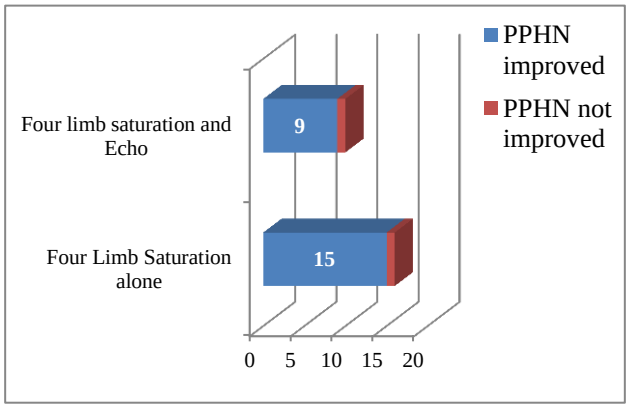

Figure 2: Outcome of PPHN in the study.

DISCUSSION

PPHN results from failure of the neonate to make a postnatal transition from a high resistance fetal pulmonary circulatory state to a low resistance pulmonary circulation. The etiology of PPHN can be due to:[5]

1. Pulmonary vasoconstriction in the presence of a normally developed pulmonary vascular bed as seen in alveolar hypoxia- meconium aspiration, birth asphyxia, infections, polycythemia, hypoglycaemia, hypocalcemia etc.
2. Pulmonary vascular smooth muscle hypertrophy as seen in chronic intrauterine asphyxia.
3. Decreased cross-sectional area of the pulmonary vascular bed as seen in congenital diaphragmatic hernia, primary pulmonary hypoplasia.

PPHN caused by the first group is relatively easy to reverse, and that caused by the second group is more difficult to reverse than the first one. Pulmonary hypertension caused by the third group is most difficult or impossible to reverse. Although various clinical conditions lead to PPHN, meconium aspiration, intrauterine or perinatal asphyxia remain the most common etiologies. The most common underlying cause for PPHN in our study was meconium aspiration (53.8%), which was also supported by Abdel Mohsen, et al (50%).[4] Perinatal asphyxia- clear liquor (30.8%) was the second common cause for PPHN in our study.

The management of PPHN involves advanced diagnostic and therapeutic modalities like ECHO, blood gas analysis (ABG), ECMO, nitric oxide etc. Such facilities may not be available in limited resource settings. It should also be remembered that PPHN is a medical emergency where immediate appropriate intervention is critical to reverse hypoxemia. Birth asphyxia still remains an important cause of neonatal mortality despite advances in antenatal and perinatal care, and PPHN most commonly complicates the setting of perinatal asphyxia/ meconium aspiration. Early identification of PPHN in the background of such a clinical setting and stabilisation with initiation of a PVR reducing agent improves outcome. A simple bedside evaluation by recording the 4 limb Spo_2 using a pulse oximeter aids in the diagnosis, where a difference of >10% in the preductal and postductal Spo_2 is suggestive of PPHN. The role of ECHO in PPHN is to exclude a structural cyanotic heart disease, which may not be readily available in peripheral health centres with limited resources. Here clinical assessment, pulse oximetry difference with a suggestive history is sufficient to initiate management in such a medical emergency where delaying management for the sake of ECHO/ABG confirmation complicates the course of the illness. Such neonates diagnosed can be started on oral sildenafil, a pulmonary vasodilator given through an NG tube, in a dose of 0.5-2mg/kg/dose 6^{th} hourly.

Our centre with limited resources, does not have 24hours ECHO. Hence neonates with PPHN had to be started on sildenafil based on pulse oximetry alone. In our study 61.5% of neonates with PPHN were diagnosed with pulse oximetry alone before the first dose of sildenafil. ECHO was done at the earliest available time for the same group which was confirmatory in 75% of the cases. The remaining 25% had normal ECHO findings which can be explained by the fact that PPHN could have improved at the time of ECHO evaluation, as the response time with sildenafil varies between 20minutes to 3 hours.[1] We also observed that PPHN improved in 92.3% of our study population, out of which 62.5% were started on sildenafil for early stabilisation with pulse oximetry findings even without a simultaneous ECHO confirmation. This was supported in the study done by Engelbrecht in Worcester hospital, where PPHN was diagnosed only with clinical assessment, pulse oximetry and ABG, as ECHO was not available in their hospital.[1] The neonates were thereafter started on sildenafil, and the study stresses the importance of early diagnosis and stabilisation of PPHN in peripheral centres before referral to tertiary care. In

another observational case study by Daga et al which was also conducted in a resource-limited setting, a decrease in the pulmonary arterial pressures of non-ventilated premature neonates with PPHN was demonstrated by pre- and post-sildenafil echocardiography.[6]

Pulse oximetry - a useful screening tool

Zhao et al in his study concluded that pulse oximetry plus clinical assessment is feasible and reliable for the screening of major congenital heart disease in new-borns, where ECHO is not easily available.[7] Mathur et al in their study observed that the sensitivity of pulse oximetry to detect critical congenital heart disease and PPHN was 97.5% with negative predictive value of 99.5%.[8] They also concluded that the high negative predictive value is useful to reliably rule out critical congenital heart disease or PPHN among sick neonates, thus avoiding need for an urgent echocardiography.

Limitations of pre and post ductal Spo_2 difference

- Neonates with PPHN document a Spo2 difference only in the presence of a ductus arteriosus right to left hemodynamic shunt. A subset of infants with PPHN, with shunt at the foramen ovale do not have the saturation difference.[3]
- There are certain structural cardiovascular abnormalities associated with right to left ductal or atrial shunting. Some of them are infracardiac TAPVC, critical/supravalvular aortic stenosis, coarctation of aorta, interrupted aortic arch etc.[3] Such conditions can be ruled out in most cases with associated other clinical findings.

Benefits outweigh risks- no untoward effects

There are certain structural congenital heart diseases (CHD) which can be medically managed to a certain extent such as:

A. Prostaglandin E1 infusion for duct dependent lesions.
B. Ibuprofen/Indomethacin for pharmacological closure of symptomatic PDA.

Here a definitive ECHO diagnosis is essential before initiating treatment, as closure of PDA can have catastrophic effects in duct dependent conditions.

Although pre and post ductal Spo_2 analysis has its own limitations, a good clinical evaluation helps to delineate a structural heart disease and PPHN. We do not start sildenafil when there are more clinical pointers towards cyanotic heart disease. Yet certain cases with CHD may not have obvious clinical findings and in such cases, even if sildenafil is initiated with an incidental pre and post ductal Spo_2 difference, it does not have any untoward effects in the basic pathophysiology of the underlying condition. Despite the need for ECHO evidence of PPHN, treatment with sildenafil can be initiated in such settings as benefits outweigh the risks.

CONCLUSION

We conclude that PPHN, a neonatal emergency can be diagnosed in limited resource settings based on history, clinical assessment and pulse oximetry. This knowledge helps in early stabilization of such critically ill neonates before referral to a tertiary care centre.

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REFERENCES

1. Engelbrecht AL. Sildenafil in the management of neonates with PPHN: A rural regional hospital experience. South Afr J Child Health. 2008;2(4). Available from: http://www.ajol.info/index.php/sajchh/article/view/50513
2. AK Dutta AS. Advances in Pediatrics. 2nd ed. Jaypee. 2012;105:106.
3. Cloherty ECEJP. Manual of Neonatal Care. 7th ed. Wolters Kluwer. 2012:435-8.
4. Abdel Mohsen AH, Amin AS. Risk Factors and Outcomes of Persistent Pulmonary Hypertension of the Newborn in Neonatal Intensive Care Unit of Al-Minya University Hospital in Egypt. J Clin Neonatol. 2013;2(2):78-82.
5. Myung KP. Pediatric Cardiology for Practitioners. 5th ed. Elsevier. 2011:299-300.
6. Daga V. Internet Scientific Publications. Available from: http://ispub.com/IJPN/8/1/3701
7. Zhao Q, Ma X, Ge X, Liu F, Yan W, Wu L. Pulse oximetry with clinical assessment to screen for congenital heart disease in neonates in China: a prospective study. Lancet Lond Engl. 2014;384(9945):747-54.
8. Mathur NB, Gupta A, Kurien S. Pulse Oximetry Screening to Detect Cyanotic Congenital Heart Disease in Sick Neonates in a Neonatal Intensive Care Unit. Indian Pediatr. 2015;52(9):769-72.

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