A STUDY OF MECONIUM ASPIRATION SYNDROME AS A RISK FACTOR FOR PERSISTENT PULMONARY HYPERTENSION IN TERTIARY CARE CENTRE

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Abstract

When the typical fetal-to-neonatal circulatory transition fails as soon as the infant is delivered, persistent pulmonary hypertension of the newborn (PPHN) develops. PPHN, or persistent pulmonary hypertension of the newborn, is also known as the syndrome of inadequate circulatory acclimation. This has a significant impact on newborn morbidity and mortality. The prevalence is 2/1000 live births of infants. Most often, neonatal pulmonary diseases such meconium aspiration syndrome, congenital diaphragmatic hernia, and respiratory distress syndrome cause the pulmonary vasculature to relax slowly or inadequately, which leads to PPHN
Objective: to describe meconium aspiration syndrome leading to PPHN.[1]
Method: This study, which involved a 20-bed level III neonatal intensive care unit at a tertiary care institution at the DR. D.Y. Patil Medical College, Hospital and Research Center, Pimpri, Pune, was prospective and cross-sectional in nature. Term newborns with respiratory distress were assessed for PPHN in 160 cases out of the 1562 total admissions to the NICU over the course of two years. 90 instances did not progress to PPHN, five did not consent, and five patients had cyanotic heart disease. 60 patients in total were therefore enrolled. Perinatal information was gathered using a pre-filled proforma after receiving consent. Moreover, participants with gestational ages greater than 36 weeks plus 6 days, congenital heart disease, and other conditions were disqualified from the study.
Conclusion: A key risk factor for PPHN is meconium aspiration syndrome. Hence, reducing the likelihood of meconium-stained amniotic fluid, which can result in meconium aspiration syndrome, can be achieved by avoiding perinatal foetal distress and encouraging vaginal birth.

Keywords: meconium aspiration of fluid, respiratory distress, perinatal foetal distress,PPHN

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1. Introduction

PPHN is a syndrome characterised by high PVR that results in extrapulmonary right-to-left blood shunting across the ductus arteriosus and/or foramen ovale. It is linked to a variety of neonatal cardiac and pulmonary diseases. (2). Gersony et al. (1969), who described two babies with clear lung fields who had catastrophic hypoxemia coupled with severe PH and right-to-left shunting across the foramen ovale and ductus arteriosus, are credited with providing the first precise description of the pathogenesis of PPHN. While it is typically observed in term and near-term infants, certain premature infants with respiratory distress or bronchopulmonary dysplasia might be diagnosed with it. Most often, neonatal pulmonary diseases such meconium aspiration syndrome, congenital diaphragmatic hernia, and respiratory distress syndrome cause the pulmonary vasculature to relax slowly or inadequately, which leads to PPHN (1). Nelson 1157: Persistent pulmonary hypertension of the newborn (PPHN) occurs mostly in in term and post term infants. Predisposing factors include birth asphyxia, MAS, early-onset sepsis, RDS, hypoglycemia, polycythemia, maternal use of nonsteroidal antiinflammatory drugs with in utero constriction of the ductus arteriosus, maternal late trimester use of selective serotonin reuptake inhibitors, and pulmonary hypoplasia caused by diaphragmatic hernia, amniotic fluid leak, oligohydramnios, pleural effusions. The first newborn's excrement, or stool, is called meconium. Meconium aspiration syndrome happens when a newborn breaths amniotic fluid and meconium into the lungs shortly after birth. A significant cause of serious illness and infant death, meconium aspiration syndrome, affects 5 to 10 percent of newborns. It often happens when the foetus is under stress during birth, particularly if the baby is delivered past the due date. Low plasma arginine and NO metabolite concentrations in some PPHN patients as well as gene polymorphisms for carbamoyl phosphate synthase raise the possibility of a minor impairment in NO synthesis. The incidence varies greatly between clinical sites and ranges from 1 in 500 to 1500 live births. (4). PPHN is associated with a high mortality risk and risk of neurodevelopmental disabilities. Contemporary neonatal intensive care, including ventilator management, surfactant therapy, high-frequency ventilation, inhaled nitric oxide (iNO), and ECMO are the treatment modalities(5).

2. Method

This present study was a prospective and cross sectional study, involving 20 bed level - III neonatal intensive care unit at tertiary care center, DR. D.Y.Patil Medical College, Hospital and Research centre,Pimpri,Pune. Total admissions in NICU were 1562 in a period of 2 years out which 160 term neonates with respiratory distress were clinically evaluated by neonatologist with an experience of 20 years. The neonates had pre ductal and post ductal saturation difference of >5% on pulse oximeter, Philips Goldway[Shenzhen] with a power of 100V-240V, 50Hz/60Hz 115VA. The echocardiography done with Sonosite 2D echo machine by neonatologist, pediatric PPHN features such as TR JET, dilated right ventricle, leftward deviation of the interventricular septum, right to left shunt, and pulmonary artery hypertension >25 mm Hg were present. Mild PPHN was further classified as PAH of 45–48 mm Hg, moderate PPHN was classified as PAH of 55–60 mm Hg, and severe PPHN was classified as PAH of 65–70 mm Hg. Out of 160 cases, 90 did not progress to PPHN, 5 refused to provide their permission, and 5 patients had cyanotic heart disease (TGA, 1-Pentalogy of the Heart, 2-TOF, 1-TAPVC). 60 patients in total were enrolled as a result. After obtaining consent, perinatal data was gathered on a pre-filled proforma. Research participants with gestational ages 36 weeks + 6 days, congenital heart disease, other congenital defects such diaphragmatic hernia, and study participants who refused to provide permission were also removed. neonates. After obtaining consent, information about the mother, antenatal risk factors, perinatal risk factors, type of delivery, Downes score, type of ventilation, and saturation difference (preductal and post ductal), echocardiographic features, treatment, and response to treatment, as well as the diagnosis of PPHN, was collected using a pre-formatted proforma.

3. Result

- This study is a single center prospective, cross sectional study done at Dr DY Patil Medical College and Research Centre, Pune, a tertiary care center. We studied on 60 term neonates.
- Incidence of PPHN was found 3.8 /1000 live births.
- Total 3381 deliveries happened in 2 years from October 2020-October 2022. Term neonates with PPHN were 60, 46[76.7%] were born by LSCS and 14[23.3%] by normal vaginal deliveries.
- antenatal risk factors were oligohydramnios(13.3%) , Single loop of cord around neck(10%)
- Out of 27 [45%] meconium aspiration of fluid[MSAF] neonates , 18[30%] developed meconium aspiration syndrome[MAS].

4. Discussion

Persistent Pulmonary Hypertension of the newly borns (PPHN) occurs when the normal fetal-to-neonatal circulatory transition fails. This present study was a prospective and cross sectional study, involving 20 bed level -III neonatal intensive care unit at tertiary care centre, DR. D.Y.Patil Medical College,Hospital and Research Centre,Pimpri,Pune. Total admissions in NICU over 2 years were 1562, out of which term neonates with respiratory distress were screened for PPHN and 60 were enrolled. According to a 2017(98) study by Niralee Babooa et al., caesarean deliveries have a higher incidence of PPHN than normal deliveries. Infants who undergo a caesarean section are simultaneously exposed to an increase in endothelin-1 levels and respiratory distress syndrome. Due to hypoxia, acidosis, and delayed transition, PPHN results. As prenatal lung fluid clearance starts before labour, neonates delivered by caesarean section without any contractions may struggle to discharge the fluid from their lungs. This could result in ventilation blockage. PPHN is another outcome of this. In our research, we also discovered a significant increase in PPHN instances following caesarean procedures as compared to routine deliveries. According to a study by Ahmed, T. et al. (7), out of 400 patients who were tested, 66% had been born through LSCS. In this investigation, a higher incidence of 50–63.5% was discovered. Moreover, they noted that among PPHN patients, a larger percentage of newborns were delivered via Caesarean section (50%–63.5%). Out of 60 term neonates with PPHN in our study, 8 (13.3%) had oligohydramnios, 3 (5%), and 6 (10%] had a single loop of chord around their necks. A study by Olivia Williams et al. (8) found that after a protracted period of oligohydramnios, some of the children acquired pulmonary hypoplasia. Among the patients in my study, 8 (13.3%) had this condition. The pulmonary vascular bed is smaller due to pulmonary hypoplasia, which is caused by oligohydramnios, which also reduces the size of the intrathoracic cavity and impairs lung growth. A decrease in amniotic fluid caused by the onset of pulmonary hypoplasia in PROM causes an increase in chest compression, a reduction in foetal respiratory movements, and the evacuation of lung fluid from the foetus. These infants' decreased lung volumes exhibit PPHN. In my study, 10% had single loop of cord around in ANC scan. Babies with a tight nuchal cord have the possibility of developing signs and symptoms like hypovolemia, hypotension, decreased perfusion and weak respiratory distressIn our study we found that 27[45%] had MSAF. In their study have stated that meconium aspiration is the main cause of PPHN compared to other indications. At the time of delivery, the meconium may cause decrease in lung compliance and hypoxia which will cause respiratory distress. Small airways that are mechanically blocked may retain air, leading to chemical pneumonitis and surfactant inactivation. PPHN and pulmonary vasoconstriction may result from this. The primary contributor to PPHN in our study was likewise MSAF during delivery. Out of the 211 instances of neonates with MAS, Nakwan, N. et alstudy(9) .'s found that the prevalence of MAS was 7.7 per 1000 live births. 36 (17.1%) of the live births they examined suffered from PPHN, and the mortality rate was 2.4%. (5/211). In a different study, Olicker AL et al. (10) came to the conclusion that only 4% of children who received MSAF eventually had MAS. In a different study, 367 pregnant women who had previously undergone one LSCS and were in the reproductive age range were looked at by Gupta, P. et al. 52 ladies who failed the TOL (trial of labour) had to have a repeat section under emergency circumstances. They reported that 52 women, or 40.62% of the population, required LSCS.Satyan Lakshminrusimha and colleagues conducted a study (12). In 303 neonates with HIE, 67 (22%) of whom had PPHN, there was 3% culture-confirmed sepsis. 10% of neonates in the current study who were diagnosed with PPHN also had HIE, and 11.6% developed sepsis. Abdel Hakeem et al.(13) examined 640 infants in a different research. According to their research, diabetes mellitus was linked to an increased chance for 42 infants to experience PPHN, meconium aspiration, birth asphyxia, neonatal septicemia, and caesarean section.

5. Conclusion

In our study which was conducted among 60 term neonates showed that PPHN was more common in neonates born by lower segment caesearean section [LSCS]delivery and meconium stained amniotic fluid[MSAF] being the most common etiological factor.

6. References

Nair J, Lakshminrusimha S. Update on PPHN: Mechanisms and treatment. Seminars in Perinatology. 2014 March; 38(2): 78-91.

Gleason C, Juul S. Avery's Disease of the Newborn. 10th ed. Gleason C, DevaskarS, editors.: Elsevier.

Gersony WM DGSJ. PFC syndrome (persistence of the fetal circulation).. 1969;40:111. Circulation. 1969; 40: 111.

Kliegman R, Geme J. Nelson Text Book of Paediatrics. 2007th ed. Kliegman RM, editor.:

Elsevier/Saunders,; 2011.
Eichenwald G, Hansen A, Martin C, Stark A. Cloherty and Stark's Manual of Neonatal Care. South Asian Edition ed. Jain N, editor.: Wolters Kulwer; 2021.
Babooa N, Shi W, Chen C. Factors relating caesarean section to persistent pulmonary hypertension of the newborn. World J Pediatr. 2017 Dec; 13(6): 517- 527.
Ahmed T, Abqari S, Shahab T, Manazir S, Firdaus U. Prevalence of pulmonary arterial hypertension on echocardiography in newborns with maternal risk factors. International Journal of Pregnancy & Child Birth. 2017 Nov; I(3): 15.
Williams O, Hutchings G, Hubinont C, Debauche C, Greenough A. Pulmonary Effects of Prolonged Oligohydramnios following Mid-Trimester Rupture of the Membranes – Antenatal and Postnatal Management. Neonatology. 2012; 101: 83- 90.
Nakwan N, Chitrapatima C. Risk Factor Analysis of Persistent Pulmonary Hypertension of the Newborn in Meconium Aspiration Syndrome in Thai Neonates J Clin Neonatol 2020;9:242-8. 2020; 9: 242-8.
Olicker A, Raffay T, Ryan R. Neonatal Respiratory Distress Secondary to Meconium Aspiration Syndrome. 23;8(3). Children (Basel).. 2021 Mar ; 23(8(3)): 246.
Gupta P, Jahan I, Jograjiya G. Is vaginal delivery safe after previous lower segment caesarean section in developing country?. Niger Med J.. 2014 May; 55(3): 260-5.
Lakshminrusimha S, Shankaran S, Laptook A, McDonald S, Keszler M, Van Meurs Kea. Pulmonary Hypertension Associated with Hypoxic-Ischemic Encephalopathy-Antecedent Characteristics and Comorbiditie. J Pediatr. 2018 May; 196: 45-51.
Abdel MAA. Risk factors and outcomes of persistent pulmonary hypertension of the newborn in neonatal intensive care unit of Al-minya university hospital in egypt. Apr;2(2):78-82. J Clin Neonatol.. 2013 Apr; 2(2): 78-82.