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Managing Newborns with Persistent Pulmonary Hypertension: An Evaluation of the Available Options

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Introduction

Persistent Pulmonary Hypertension in the New-Born (PPHN) is a challenging neonatal condition that is not rare. PPHN is a life-threatening condition that affects 1.9 out of every 1000 lives newborns. Hypoxia, infection, or pulmonary hypoplasia can all cause PPHN. To enhance cardiac output in newborns with PPHN, a robust treatment of systemic hemodynamic with volume and cardio tonic medicine is first used. Increased systemic arterial pressure improves oxygenation by reducing right-to-left extra pulmonary shunting, a hallmark of PPHN physiology.

The goals of ventilation methods are to reduce acidity while avoiding barotrauma and ensuring adequate oxygenation. Alkalinisation with sodium bicarbonate or promethazine may be required to restore normal arterial pH. High-frequency breathing or oscillator therapy may be required to achieve these goals. Inhaled Nitric Oxide (iNO) and Extracorporeal Membrane Oxygenation (ECMO) are used when standard treatment fails. Surfactant therapy may be beneficial in the treatment of lung parenchymal disease. Unfortunately, in our part of the world, high-frequency ventilation, ECMO, and iNO delivery require dedicated, expensive facilities that may not be available in every New-Born Critical Care Unit (NICU).

Pulmonary hypertension

The frequency of neonatal refractory hypoxemia and/or (PPHN) has been observed to range between 0.43 and 6.8 per 1000 live births in term or near-term newborns. Although the treatment for PPHN has improved over the last 10 to 15 years, neonates with the disease still die at a rate of 10% to 20%. The study included 40 newborns that were 40 weeks old at the time of the study. Male newborns with a higher mortality rate had a higher SPAP than female newborns with a lower mortality rate. In contrast to previous research, Hernandez-Dias and colleagues discovered that male gender was a risk factor for PPHN, with males having a higher mortality rate than females.

We discovered that diabetic mothers' newborns, meconium aspiration syndrome, and maternal use of non-steroidal anti-inflammatory drugs were the most common PPHN risk factors. This is in line with recent stories. Preeclampsia and caesarean birth were revealed to be less important risk factors for PPHN in our study. Contrary to popular perception, Araujo and colleagues discovered that infants born via caesarean section had a fivefold increased risk of PPHN (p=0.027), a risk factor backed up by several additional studies.

This could be related to huge sample size differences, as the number of patients in our series was significantly fewer than in previous studies. The condition of our PPHN group was improved by a variety of therapeutic options. There was a statistically significant decrease in SPAP after treatment strategies, notably sildenafil medication with satisfactory systolic blood pressure records.

Traditional therapy strategies have been shown to be effective in the treatment of PPHN by some researches. Many other authors have said that sildenafil is safe, effective, and well tolerated in the treatment of baby pulmonary hypertension; albeit further multicenter trials are required before it can be recommended. The neonates who received inotropic support spent less time in the NICU in the current study than those who did not (p0.004). Other studies, on the other hand, consistently associated inotropes to an increased risk of hospitalization and death.

The differences in inotropic support beginning times between our groups and the others could be seen as a trend toward an early decision for this therapeutic strategy. After PPHN therapy, SaO2 saturation, as measured non-invasively by pulse oximetry, improved significantly, as did all blood gas parameters such as pH, PCO2, and HCO3. These results are similar to those seen in other studies, especially when oral sildenafil is utilized.

In terms of total mortality, 20% of patients died in this study, which is comparable to death rates seen in previous studies using conventional therapy. Male gender and a high PIP>25 cm H2O were shown to be associated with a higher risk of death. This is consistent with the Bellettato study, which revealed that hypomania mixed with a high PIP increased the risk of death.

The addition of nasogastric sildenafil did not reduce mechanical ventilation duration (p=0.3), which is in line with a recent study that compared oral sildenafil to placebo and found no significant difference in mechanical ventilation duration between the two groups. In a recent trial, oral sildenafil was found to be more effective than magnesium sulphate in lowering mechanical breathing duration in patients with PPHN. Oral sildenafil, in contrast to surfactant therapy, significantly lowers overall mortality.

Surfactant therapy for PPHN in meconium aspiration syndrome is still contentious; some authors found comparable results to ours, while others showed that surfactant therapy reduced mortality significantly (especially in low birth weight newborns).

The group that received sildenafil as adjuvant medication had a mortality rate of 7.6%, which is comparable to previous recent studies (6.5%). Finally, when used as an adjuvant therapy in newborns with PPHN, oral sildenafil reduced the length of time spent in the NICU. This discovery was not made in any other previously published study. The limited sample size, absence of randomization, and lack of a control group were all flaws in this study.

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