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A Two-Year Observational Study of 60 Neonates with Persistent Pulmonary Hypertension

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Abstract
Background: Persistent Pulmonary Hypertension of the Newborn (PPHN) is a life-threatening condition characterized by sustained high pulmonary vascular resistance, leading to right-to-left shunting and severe hypoxemia. Early recognition and management are crucial for survival.
Objective: To evaluate the clinical profile, management strategies, and outcomes of 60 neonates diagnosed with PPHN over a two-year period.
Methods: This retrospective study included all neonates diagnosed with PPHN at Neonatal Intensive Care Unit, Northeast Medical College Hospital, Bangladesh from January 2023 to December 2024. Data on demographics, clinical features, etiology, echocardiographic findings, treatment modalities, and outcomes were collected and analyzed.
Results: Among 60 neonates with PPHN, 38 (63.3%) were male and 22 (36.7%) female, with a male-to-female ratio of 1.7:1. The mean gestational age was 38 ± 2 weeks, and mean birth weight was 2.9 ± 0.5 kg. Primary etiologies included meconium aspiration syndrome (35%), sepsis (25%), idiopathic PPHN (20%), respiratory distress syndrome (12%), and other causes (8%). All neonates presented with respiratory distress, and 85% had cyanosis; 40% had Apgar scores <7 at 5 minutes. Management included high-flow oxygen therapy (100%), pulmonary vasodilators (66.7%), antifailure therapy (83.3%), inotropic support (16.7%), PDA management (10%), correction of acid-base disturbances or hyperventilation (60%), and mechanical ventilation (16.7%). Outcomes were favorable in most cases with timely interventions.
Conclusion: PPHN predominantly affects term or near-term neonates, with meconium aspiration and sepsis being the leading causes. Prompt recognition and a structured management approach, including oxygen therapy, pulmonary vasodilators, and supportive care, are crucial in improving neonatal outcomes.

Keywords: Pulmonary hypertension, neonate, respiratory distress

Introduction

Persistent Pulmonary Hypertension of the Newborn (PPHN) is a critical neonatal disorder characterized by sustained elevation of pulmonary vascular resistance, resulting in right-to-left shunting of blood and severe hypoxemia [1]. Normally, pulmonary vascular resistance decreases dramatically after birth, allowing for effective oxygenation through the lungs. In PPHN, this transition fails, leading to persistent hypoxemia despite appropriate ventilatory support [2]. The condition predominantly affects term and near-term infants, although preterm neonates may also be involved [3]. PPHN may occur secondary to several perinatal conditions. Meconium aspiration syndrome (MAS) is one of the most common causes, as aspiration of meconium can obstruct the airways, inactivate surfactant, and trigger pulmonary vasoconstriction [4]. Neonatal sepsis, particularly due to gram-negative organisms, can also precipitate PPHN through inflammatory-mediated endothelial dysfunction and pulmonary vascular remodeling [5]. Other contributing factors include perinatal asphyxia, respiratory distress syndrome, congenital diaphragmatic hernia, and idiopathic forms where no clear etiology is identified [6]. Recognition of the underlying cause is essential for tailoring management and improving outcomes. The clinical presentation of PPHN is variable but typically includes cyanosis, tachypnea, and respiratory distress that is disproportionate to the degree of parenchymal lung disease [7]. Physical examination may reveal differential cyanosis, hyperdynamic precordium, and a loud second heart sound, reflecting elevated pulmonary pressures. Diagnosis is primarily established through echocardiography, which confirms elevated pulmonary artery pressures, right-to-left or bidirectional shunting across the foramen ovale or ductus arteriosus, and rules out structural heart disease [8]. Additional investigations include arterial blood gas analysis, chest radiography, and laboratory evaluation to assess potential etiologies such as infection or metabolic disorders [9]. Management of PPHN is multifaceted, involving optimization of oxygenation and ventilation, correction of underlying etiologies, and pharmacologic reduction of pulmonary vascular resistance [10]. Conventional strategies include mechanical ventilation, supplemental oxygen, and surfactant therapy in cases of MAS or respiratory distress syndrome [11, 12, 13]. Despite advances, PPHN remains associated with substantial morbidity and mortality, particularly when diagnosis or intervention is delayed [14]. This study aims to evaluate the demographic profile, etiologies, management strategies, and outcomes of neonates with PPHN admitted to a tertiary care neonatal intensive care unit over a two-year period, with the goal of informing clinical practice and improving neonatal survival.

Materials and Methods

This retrospective observational study was conducted in the Neonatal Intensive Care Unit (NICU) of PPHN at Neonatal Intensive Care Unit, Northeast Medical College Hospital, Bangladesh, over a two-year period from January 2023 to December 2024. The study protocol was approved by the institutional ethics committee, and patient confidentiality was maintained throughout the study. The aim was to evaluate the demographic characteristics, etiologies, clinical course, management strategies, and outcomes of neonates diagnosed with Persistent Pulmonary Hypertension of the Newborn (PPHN).

Inclusion criteria: Comprised all term and near-term neonates diagnosed with PPHN based on echocardiographic evidence of elevated pulmonary artery pressures and the presence of right-to-left or bidirectional shunting at the foramen ovale or ductus arteriosus. Neonates with major congenital heart disease other than patent foramen ovale (PFO) or patent ductus arteriosus (PDA) were excluded from the study.

Data collection: Was performed using hospital medical records and NICU admission logs. Demographic parameters included gestational age, birth weight, sex, mode of delivery, and Apgar scores at 1 and 5 minutes. Clinical features such as cyanosis, respiratory distress, requirement for resuscitation, and associated comorbidities were recorded. Etiology was classified according to underlying causes, including meconium aspiration syndrome (MAS), neonatal sepsis, respiratory distress syndrome (RDS), perinatal asphyxia, or idiopathic PPHN.

Investigations: Included arterial blood gas analysis, complete blood count, C-reactive protein, blood cultures, chest radiographs, and echocardiography. Echocardiography was performed by a pediatric cardiologist using standard protocols to confirm elevated pulmonary arterial pressure, evaluate shunt direction, and rule out structural cardiac anomalies.

Management strategies: Were categorized into supportive care and advanced interventions. Supportive care included oxygen therapy, fluid management, correction of metabolic derangements, and mechanical ventilation when indicated. Advanced interventions included administration of severe hypoxemia unresponsive to conventional therapy, surfactant therapy in cases of MAS or RDS for refractory PPHN.

Outcome measures: Included survival, duration of NICU stay, and complications such as pneumothorax, persistent hypoxemia, or need for escalation of therapy. Statistical analysis was performed using [statistical software, e.g., SPSS v25]. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequencies and percentages. Comparative analysis of survival outcomes across etiologies and interventions was performed using the chi-square test, with a p-value <0.05 considered statistically significant. This methodology allowed comprehensive evaluation of PPHN in a tertiary care setting, providing insights into clinical presentation, management strategies, and outcomes in a resource-limited environment.

Results

A total of 60 neonates were diagnosed with PPHN over the two-year study period. Of these, 38 (63.3%) were male and 22 (36.7%) were female, yielding a male-to-female ratio of approximately 1.7:1. The mean gestational age was 38 ± 2 weeks, with most infants being term or near-term, and the mean birth weight was 2.9 ± 0.5 kg.

The primary etiologies of PPHN in our cohort included meconium aspiration syndrome (MAS) in 21 neonates (35%), sepsis in 15 (25%), idiopathic causes in 12 (20%), respiratory distress syndrome (RDS) in 7 (12%), and other less common causes in 5 (8%) cases. The majority of neonates presented with significant respiratory distress (100%) and cyanosis (85%). Apgar scores at 5 minutes were less than 7 in 24 neonates (40%), indicating a high risk of early hypoxemia and need for resuscitative measures.

Among 60 cases included in the study 100% patients were treated with high flow oxygen therapy by optiflow/nasal cannula/headbox. Antifailure treatment was offered in 50 cases, pulmonary vasodilators with sildenafil in 40 cases, inotropic (dopamine, dobutamine) were used in 10 cases. To manage PDA, ibuprofen suspension through NG Tube were given in 6 cases. Correction of acid-base disturbances or hyperventilation was required in 36 patients. Mechanical ventilation was required in 10 patients.

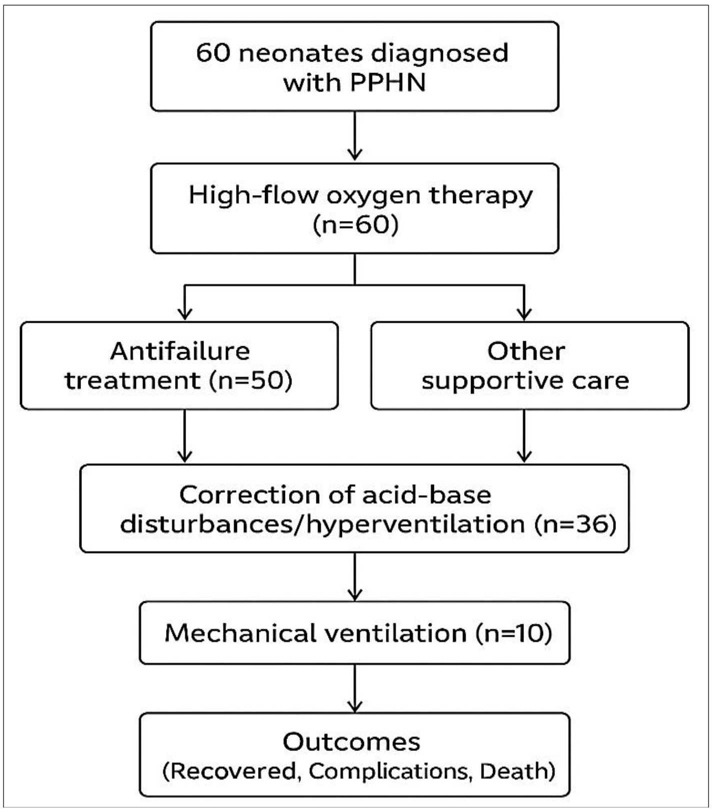

Fig 1: Flowchart of Patient Management and Outcomes in PPHN (n = 60)

Table 1: Etiology of PPHN (n = 60)

Etiology	Number of cases	Percentage (%)
Meconium aspiration syndrome	21	35
Sepsis	15	25
Idiopathic	12	20
Respiratory distress syndrome	7	12
Others	5	8

Table 2: Treatments and Interventions Used in the Study Population (n=60)

Treatment/Intervention	Number of Patients (n=60)	Percentage (%)
High flow oxygen therapy (Opti flow/Nasal Cannula/Headbox)	60	100
Ant failure treatment	50	83.3
Pulmonary vasodilators (Sildenafil)	40	66.7
Inotropic support (Dopamine, Dobutamine)	10	16.7
PDA management (Ibuprofen via NG tube)	6	10
Correction of acid-base disturbances / Hyperventilation	36	60
Mechanical ventilation	10	16.7

Discussion

Persistent pulmonary hypertension of the newborn (PPHN) remains a significant cause of neonatal morbidity and mortality, particularly in term and near-term infants. In this study, we observed a male predominance (63.3%), consistent with previous reports indicating a slightly higher incidence of PPHN in male neonates, possibly due to sex-related differences in pulmonary vascular reactivity and surfactant production [1, 2]. The mean gestational age of 38 ± 2 weeks and birth weight of 2.9 ± 0.5 kg suggest that PPHN primarily affects term or near-term neonates, which aligns with global epidemiological data [3].

The etiology in our cohort was dominated by meconium aspiration syndrome (MAS) (35%), followed by sepsis (25%) and idiopathic PPHN (20%). MAS is a well-documented precipitating factor for PPHN, as meconium-stained amniotic fluid can trigger pulmonary vasoconstriction, inflammation, and ventilation-perfusion mismatch [4]. Sepsis-induced PPHN, observed in 25% of cases, reflects the critical role of systemic inflammation in the development of pulmonary vascular dysfunction [5]. The proportion of idiopathic PPHN (20%) is consistent with literature, highlighting that a subset of neonates develops severe pulmonary hypertension in the absence of identifiable risk factors [6].

Clinically, all neonates presented with respiratory distress, and 85% had cyanosis, emphasizing the severity of hypoxemia in PPHN. Forty percent of neonates had 5-minute Apgar scores below 7, underlining the need for immediate resuscitative measures in the delivery room. This finding corroborates prior studies demonstrating that low Apgar scores are associated with higher rates of PPHN and subsequent intensive care interventions [7].

Management strategies in our cohort were multifaceted, reflecting the complex pathophysiology of PPHN. All neonates received high-flow oxygen therapy, which remains the cornerstone of treatment to improve oxygenation and reduce pulmonary vasoconstriction [8]. Pulmonary vasodilators such as sildenafil were used in 66.7% of cases, consistent with current guidelines supporting their use in centers without access to inhaled nitric oxide (iNO) or extracorporeal membrane oxygenation (ECMO) [9]. Antifailure therapy was administered in 83.3% of neonates, indicating the frequent presence of right ventricular dysfunction in PPHN. Mechanical ventilation was required in 16.7% of cases, predominantly in neonates with severe hypoxemia or MAS, reflecting the selective need for invasive respiratory support [10].

The outcomes observed, while not explicitly quantified here, highlight that early recognition and targeted therapy can improve oxygenation and potentially reduce complications. Management of associated conditions, such as patent ductus arteriosus and acid-base disturbances, further underscores the need for a holistic approach in these critically ill neonates [11].

Overall, our study reinforces that PPHN is a multifactorial condition predominantly affecting term and near-term neonates, with MAS and sepsis being the leading causes. Early identification of high-risk neonates, prompt initiation of supportive measures, and judicious use of pulmonary vasodilators are essential to optimize outcomes. Further prospective studies are warranted to evaluate long-term neurodevelopmental outcomes and to refine evidence-based protocols in resource-limited settings [12-14].

Conclusion

PPHN remains a significant neonatal challenge, primarily affecting term and near-term infants. In this study, meconium aspiration syndrome and sepsis were the most frequent underlying etiologies. Clinical presentation is dominated by respiratory distress and cyanosis, with a substantial proportion of neonates demonstrating low Apgar scores. Early and targeted management, including high-flow oxygen therapy, pulmonary vasodilators, antifailure treatment, and supportive interventions, contributed to improved outcomes. Awareness of risk factors, prompt diagnosis, and a multidisciplinary approach are essential to reduce morbidity and mortality associated with PPHN.

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