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Original Research Article

Clinical presentation and early diagnostic features of persistent pulmonary hypertension in newborns: insights from a tertiary care hospital

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ABSTRACT

Background: Persistent pulmonary hypertension of the newborn (PPHN) is a severe condition with significant mortality. Data on its clinical profile and early diagnostic features from low- and middle-income countries, particularly South Asia, are scarce. This study aimed to describe the clinical presentation and early echocardiographic diagnostic features of newborns with PPHN in a tertiary care hospital in Bangladesh.
Methods: This cross-sectional observational study was conducted over six months in a tertiary care hospital in Bangladesh. Data from 50 neonates with echocardiographically confirmed PPHN were retrospectively analyzed. Demographics, clinical presentation, underlying etiologies, and detailed echocardiographic findings, including associated cardiac shunts and disease severity, were recorded.
Results: The majority of neonates were male (70%) and diagnosed within the first week of life (78%). The most common presenting features were tachypnea (78%) and cyanosis (72%). Echocardiography revealed that 74% of cases had an associated cardiac shunt, with an atrial septal defect being the most frequent finding (present in 42% of the total cohort). Severe PPHN was the most common classification at presentation (48%).
Conclusions: PPHN in this setting presents early and is frequently associated with cardiac shunts, particularly ASDs. These findings highlight the critical role of early and comprehensive echocardiographic evaluation for accurate diagnosis and potential risk stratification in managing this vulnerable population.

Keywords: Persistent pulmonary hypertension of the newborn, Neonatology, Echocardiography

INTRODUCTION

Persistent pulmonary hypertension of the newborn (PPHN) is a critical clinical syndrome defined by the failure of the normal circulatory transition at birth, resulting in sustained elevation of pulmonary vascular resistance (PVR) and a subsequent right-to-left shunt across the ductus arteriosus and/or foramen ovale.[1] This pathophysiological state leads to severe hypoxemia and remains a significant source of neonatal mortality and morbidity worldwide, posing a considerable challenge in perinatal and neonatal care.[1] The global burden of this condition is substantial, with recent scoping reviews indicating a persistent and complex epidemiological landscape from 1993 to 2023, underscoring its continued clinical relevance.[2] The reported incidence in high-income countries ranges from 1.9 to 6.8 per 1,000 live births, with associated mortality rates varying widely from 4% to 33%,

depending on underlying etiologies and available therapeutic resources.[2] The core pathophysiology involves a maladaptive vascular response where the pulmonary circulation fails to adapt to the extrauterine environment, maintaining high PVR and perpetuating the fetal circulatory pattern.[3] This failure is often triggered by a diverse range of predisposing factors, with a comprehensive meta-analysis confirming that conditions such as meconium aspiration syndrome (MAS), respiratory distress syndrome (RDS), perinatal asphyxia, and early-onset sepsis are significant risk factors.[4] Furthermore, specific maternal factors, including late-trimester exposure to selective serotonin reuptake inhibitors, have been shown through systematic review and meta-analysis to significantly increase the risk of PPHN, highlighting the multifactorial nature of its etiology.[5]

Clinically, PPHN should be suspected in any newborn, particularly term or near-term infants, presenting with cyanosis that is disproportionate to the level of respiratory distress.[1] The classic presentation includes tachypnea, grunting, nasal flaring, and chest retractions, but the primary diagnostic challenge lies in differentiating PPHN from structural congenital heart disease (CHD), which can manifest with similar severe hypoxemic states.[6] In this context, early and accurate diagnosis is paramount for initiating appropriate management strategies and improving survival outcomes. Echocardiography has emerged as the cornerstone for definitive diagnosis, serving as the most sensitive and specific non-invasive tool available.[7] A targeted neonatal echocardiographic examination allows for the direct assessment of pulmonary artery pressures, visualization of the direction of intracardiac and ductal shunting, and the crucial exclusion of complex structural CHD, thereby confirming the diagnosis of PPHN.[7] Recent advances in understanding the pathophysiology have further refined diagnostic approaches, emphasizing the need for rapid identification to facilitate timely intervention.[6]

Despite these diagnostic and therapeutic advances, a significant knowledge gap exists in the understanding of PPHN within low- and middle-income countries (LMICs). A comprehensive state-of-the-art review highlights that the challenges in these regions are vastly different, encompassing limitations in diagnostic equipment, scarcity of trained personnel, and delayed patient presentation, which collectively contribute to poorer outcomes.[8] Consequently, the clinical profiles, early diagnostic features, and management outcomes documented in high-resource settings may not be generalizable to populations in South Asian nations like Bangladesh.[8] This issue is particularly acute, as robust, region-specific data are sparse. While a notable analysis of 181 cases over one year in Bangladesh has provided foundational insights into the local epidemiology and outcomes of PPHN, and a retrospective study from Eastern India has further profiled the disease in a similar tertiary care setting, more focused research is needed.[9,10] These studies underscore the unique regional burden but also highlight the variability in presentation and associated factors that warrant further investigation.

Therefore, given the substantial global burden of PPHN, the critical importance of early echocardiographic diagnosis, the distinct challenges in managing this condition within LMICs, and the limited yet valuable data emerging from South Asia, this study was conducted to systematically describe the clinical presentation and early echocardiographic diagnostic features of newborns diagnosed with persistent pulmonary hypertension in a tertiary care hospital in Bangladesh. The findings aim to provide crucial, context-specific insights that can inform clinical practice and improve outcomes for vulnerable neonates in similar resource-intensive settings.

METHODS

This cross-sectional observational study was conducted in the Department of Pediatric Cardiology at Combined Military Hospital (CMH), Dhaka, a tertiary care center in Bangladesh, from January 2019 to June 2019. During the study period, 50 neonates diagnosed with PPHN were purposively recruited. The inclusion criteria were all neonates within the first 28 days of life who were clinically suspected of PPHN and subsequently confirmed by echocardiography. Patients were excluded if they had other major congenital structural heart diseases (excluding atrial septal defect and patent ductus arteriosus), were beyond the neonatal period, or had received significant treatment for PPHN at another institution prior to admission. Data were collected using a pre-designed structured case report form through a retrospective review of patient medical records, including admission notes, investigation reports, treatment charts, and echocardiographic reports.

Key variables extracted included demographic data, clinical presentation at admission (such as tachypnea, cyanosis, chest retractions, nasal flaring, and grunting), and detailed echocardiographic findings. For the purpose of standardization, PPHN severity was classified based on estimated pulmonary artery pressure as mild (30-50 mm Hg), moderate (>50-60 mm Hg), and severe (>60 mm Hg). The presence and type of associated cardiac shunts, including atrial septal defect (ASD), patent ductus arteriosus (PDA), and patent foramen ovale (PFO), were meticulously recorded. All collected data were analyzed using statistical package for the social sciences (SPSS) version 25.0, with descriptive statistics such as frequencies, percentages, means, and standard deviations used to summarize the findings.

The study protocol was reviewed and approved by the institutional ethics committee, and written informed consent was obtained from the parents or guardians of all participants prior to their inclusion, ensuring adherence to ethical guidelines for research involving human subjects.

RESULTS

Table 1 shows the baseline demographics and presenting clinical features of the 50 neonates diagnosed with PPHN. The majority (78%) were diagnosed within the first week of life. Male infants accounted for 70% of the cases. The most common presenting signs were tachypnea (78%), cyanosis (72%), and chest retraction (66%). Nasal flaring was observed in 50% and grunting in 32% of the neonates.

Table 1: Baseline demographics and clinical characteristics of the study population (n=50).

Characteristic	N	%
Age at diagnosis (weeks)		
≤1	39	78
2	7	14
3-4	4	8
Sex		
Male	35	70
Female	15	30
Primary presenting features		
Tachypnea	39	78
Cyanosis	36	72
Chest retraction	33	66
Nasal flaring	25	50
Grunting	16	32

Table 2 outlines the underlying etiologies associated with PPHN in the study population. Respiratory distress syndrome (RDS) was the most frequent cause, identified in 42% of cases. Meconium aspiration syndrome (MAS) accounted for 22%, while pneumonia and congenital diaphragmatic hernia were each present in 18% of cases.

Table 2: Underlying etiologies associated with PPHN (n=50).

Underlying etiology	Number of cases (N)	Percentage (%)
Respiratory distress syndrome (RDS)	21	42
Meconium aspiration syndrome (MAS)	11	22
Pneumonia	9	18
Diaphragmatic hernia	9	18

Table 3 summarizes the echocardiographic diagnoses recorded at initial assessment. Isolated PPHN was present in 26% of neonates. The remaining 74% had PPHN associated with structural cardiac anomalies: ASD (28%), PDA (18%), PFO (8%), ASD with PDA (14%), or PDA with PFO (6%).

Table 4 presents the severity of pulmonary hypertension based on echocardiographic estimation of pulmonary artery pressure. Severe PPHN (>60 mm Hg) was the most common, affecting 48% of cases. Moderate PPHN (>50–60 mm Hg) was seen in 34%, while 18% had mild disease (30–50 mm Hg).

Table 3: Echocardiographic diagnostic findings in PPHN patients (n=50).

Echocardiographic diagnosis	Number of cases (N)	Percentage (%)
Isolated PPHN	13	26
PPHN with atrial septal defect (ASD)	14	28
PPHN with patent ductus arteriosus (PDA)	9	18
PPHN with patent foramen ovale (PFO)	4	8
PPHN with ASD and PDA	7	14
PPHN with PDA and PFO	3	6
Total	50	100

Table 4: Severity of pulmonary hypertension assessed by echocardiography (n=50).

Severity of PPHN	Pulmonary artery pressure (mm Hg)	Number of cases (N)	Percentage (%)
Mild	30-50	9	18
Moderate	>50-60	17	34
Severe	>60	24	48

Figure 1 shows the proportional distribution of underlying etiologies identified among neonates with PPHN. Respiratory distress syndrome (RDS) accounted for the largest proportion at 42%, followed by meconium aspiration syndrome (MAS) at 22%. Pneumonia and congenital diaphragmatic hernia each contributed 18% of cases.

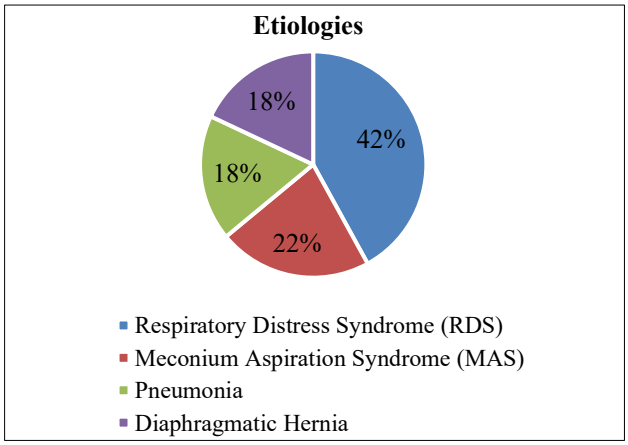

Figure 1: Proportion of underlying etiologies for PPHN.

Figure 2 illustrates the distribution of PPHN severity as assessed by echocardiography. Among the 50 neonates, severe pulmonary hypertension (>60 mm Hg) was observed in 24 cases (48%), while 17 cases (34%) were categorized as moderate (>50–60 mm Hg) and 9 cases (18%) as mild (30–50 mm Hg).

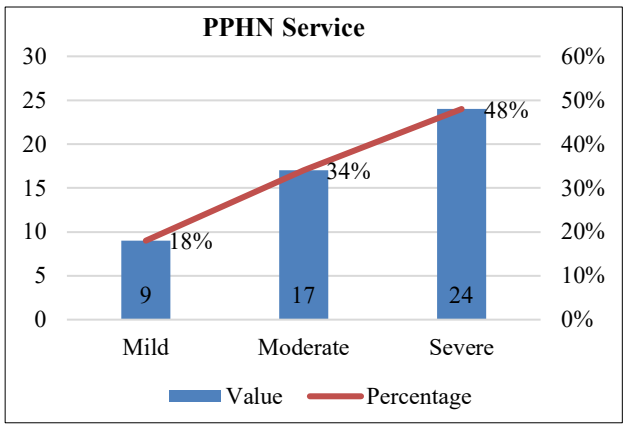

Figure 2: Distribution of PPHN severity based on echocardiographic findings.

DISCUSSION

This study provides a detailed profile of the clinical presentation and early diagnostic features of PPHN in a tertiary care setting in Bangladesh, revealing a pattern of early, severe disease with a high prevalence of associated cardiac shunts. The finding that the majority of neonates (78%) were diagnosed within the first week of life, with a clear male predominance (70%), aligns with established global epidemiological trends.[2] This demographic and temporal pattern is strongly corroborated by data from neighboring South Asian regions; a study from Eastern India reported a similar male preponderance (66%) and a comparable early presentation, while research from Pakistan also noted that 72% of their cohort presented within the first 72 hours of life.[10,11] The clinical presentation in our cohort was dominated by classic signs of respiratory distress, with tachypnea (78%) and cyanosis (72%) being the most frequent features, which is consistent with the pathophysiological understanding that these signs are cardinal triggers for urgent diagnostic evaluation.[3]

The distribution of underlying etiologies in our study further reinforces the current understanding of secondary PPHN. RDS was the most frequent cause (42%), followed by MAS (22%), pneumonia (18%), and congenital diaphragmatic hernia (18%), a profile that mirrors established causes listed in pragmatic reviews.[12] The 22% association with MAS is particularly significant, given literature suggesting that up to 40% of infants with MAS may develop PPHN, highlighting this as a high-risk group requiring vigilant monitoring.[13] Similarly, the high prevalence of RDS is consistent with its role as a leading cause of neonatal respiratory distress in the region, which often precedes the development of PPHN.[14]

Perhaps the most significant finding of our study was the high prevalence of associated cardiac shunts, identified in 74% of the neonates via echocardiography. Notably, an ASD was present in 42% of our total cohort, either alone or in combination. This may be more than a simple association; evidence suggests that ASDs act as accelerants for the development of pulmonary hypertension, leading to an earlier and more severe clinical presentation.[15] It is plausible that the presence of an ASD in our neonates exacerbated the underlying PPHN, contributing to their early diagnosis within the first week. Furthermore, the persistence of a PDA in 38% and a patent foramen ovale (PFO) in 14% of cases can be understood as a consequence of the PPHN itself, as elevated pulmonary pressures are known to prevent the normal postnatal closure of these fetal shunts.[16] This reframes these lesions not just as associated defects but as markers of the severity of the pulmonary hypertensive state.

The high disease severity at initial presentation, with nearly half (48%) of neonates classified as having severe PPHN, is a concerning finding that likely reflects the tertiary nature of our center, which receives the most critical referrals. However, this severity may also be intrinsically linked to the high prevalence of associated shunts. The hemodynamic burden of a left-to-right shunt, particularly an ASD, can exacerbate the underlying PPHN, potentially contributing to the high proportion of severe cases we observed.[15] In conclusion, our study confirms that the clinical presentation of PPHN in Bangladesh is consistent with regional patterns but highlights a notably high rate of associated cardiac shunts, especially ASDs. This finding underscores the critical importance of meticulous echocardiographic evaluation not only for diagnosis but also for risk stratification, as the presence of these shunts may influence disease severity and early presentation. These insights are crucial for clinicians in similar resource-intensive settings to anticipate the clinical course and optimize management strategies for this vulnerable population.

Limitations

This study has several limitations. The retrospective, single-center design with a small sample size (n=50) conducted over a short six-month period may limit the generalizability of our findings to the broader population of Bangladesh or other settings. The cohort, being from a tertiary care hospital, likely represents more severe cases, introducing a potential referral bias. Furthermore, the short duration precludes the assessment of long-term neurodevelopmental outcomes in these infants.

CONCLUSION

This study successfully characterized the clinical and early diagnostic profile of PPHN in a tertiary care setting in

Bangladesh. Our findings confirm an early onset of severe disease, with a notably high prevalence of associated cardiac shunts particularly atrial septal defects identified via echocardiography. These results underscore that PPHN in this context is rarely an isolated phenomenon and that the presence of these shunts may be a key factor influencing disease severity and early presentation. This highlights the critical importance of meticulous echocardiographic evaluation not only for diagnosis but also for risk stratification. The insights from this study are crucial for clinicians in similar resource-intensive settings to anticipate the clinical course and optimize management strategies for this vulnerable population.

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REFERENCES

1. Lakshminrusimha S, Keszler M. Persistent Pulmonary Hypertension of the Newborn. Neoreviews. 2015;16(12):e680-92.
2. Huang Y, Yang T, Liang X, Chen Y, Zhou P, Yu Z, et al. Global, regional and national trends in the burden of persistent pulmonary hypertension of the newborn and essentials of its management from 1993 to 2023: a scoping review. Front Pediatr. 2025;13:1502385.
3. Singh Y, Lakshminrusimha S. Pathophysiology and Management of Persistent Pulmonary Hypertension of the Newborn. Clin Perinatol. 2021;48(3):595-618.
4. Zhou R, Zheng YN, Zhang XY, Cheng YY. A Meta-Analysis of the Risk Factors of Persistent Pulmonary Hypertension in Newborns. Front Pediatr. 2021;9:659137.
5. Grigoriadis S, VonderPorten EH, Mamisashvili L, Tomlinson G, Dennis CL, Koren G, et al. Prenatal exposure to antidepressants and persistent pulmonary hypertension of the newborn: systematic review and meta-analysis. BMJ. 2014;348:f6932.
6. Cabral JEB, Belik J. Persistent pulmonary hypertension of the newborn: recent advances in pathophysiology and treatment. J Pediatr (Rio J). 2013;89(3):226-42.
7. Mertens L, Seri I, Marek J, Arlettaz R, Barker P, McNamara P, et al. Targeted Neonatal Echocardiography in the Neonatal Intensive Care Unit: practice guidelines and recommendations for training. Writing Group of the American Society of Echocardiography (ASE) in collaboration with the European Association of Echocardiography (EAE) and the Association for European Pediatric Cardiologists (AEPC). J Am Soc Echocardiogr. 2011;24(10):1057-78.
8. Hasan B, Hansmann G, Budts W, Heath A, Hoodbhoy Z, Jing ZC, et al. Challenges and Special Aspects of Pulmonary Hypertension in Middle- to Low-Income Regions: JACC State-of-the-Art Review. J Am Coll Cardiol. 2020;75(19):2463-77.
9. Fatema NN. Persistent Pulmonary Hypertension of the Newborn: Analysis of 181 cases over one Year. Cardiovasc J. 2018;11(1):17-22.
10. Sardar S, Pal S, Mishra R. A Retrospective Study on the Profile of Persistent Pulmonary Hypertension of Newborn in a Tertiary Care Unit of Eastern India. J Clin Neonatol. 2020;9(1):18.
11. Razzaq A, Iqbal Quddusi A, Nizami N. Risk factors and mortality among newborns with persistent pulmonary hypertension. Pak J Med Sci. 2013;29(5):1099-104.
12. Chojnacka K, Singh Y, Gahlaut S, Blaz W, Jerzak A, Szczapa T. Persistent Pulmonary Hypertension of the Newborn: A Pragmatic Review of Pathophysiology, Diagnosis, and Advances in Management. Biomedicines. 2025;13(10):2332.
13. Osman A, Halling C, Crume M, Al Tabosh H, Odackal N, Ball MK. Meconium aspiration syndrome: a comprehensive review. J Perinatol. 2023;43(10):1211-21.
14. Zaman S, Goheer L, Riaz H. Prevalence and etiology of respiratory distress in newborns. Pak Armed Forces Med J. 2013;63(1):22-5.
15. Vyas-Read S, Guglani L, Shankar P, Travers C, Kanaan U. Atrial Septal Defects Accelerate Pulmonary Hypertension Diagnoses in Premature Infants. Front Pediatr. 2018;6:342.
16. Yuan Z, Zhang LZ, Li B, Chung HT, Jiang JX, Chiang JY, et al. Investigation of echocardiographic characteristics and predictors for persistent defects of patent foramen ovale or patent ductus arteriosus in Chinese newborns. Biomed J. 2021;44(2):209-16.

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