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The Role Of Intraventricular Hemorrhage In Mortality And Early Neurological Outcome Of Premature Infants

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ABSTRACT

Background: Intraventricular hemorrhage (IVH) remains a major cause of mortality and long-term neurological impairment in preterm neonates. Understanding the associated risk factors is critical for early diagnosis and improved outcomes.

Material and Methods: A prospective observational study was conducted at a tertiary care hospital including 100 preterm neonates. Clinical, demographic, prenatal, and perinatal data were collected and analyzed. Cranial ultrasonography was used for diagnosis, and neurodevelopmental outcomes were assessed at discharge. Statistical analyses were performed using SPSS version 25.0, with significance set at $p<0.05$.

Results: The incidence of IVH among preterm neonates was 20%. Significant neonatal risk factors included lower gestational age, lower birth weight, and lower Apgar scores at 1 and 5 minutes. Prenatal factors such as pregnancy-induced hypertension and premature rupture of membranes (PROM) were also associated with an increased risk of IVH. Neurological complications like seizures, hydrocephalus, and posthemorrhagic ventricular dilatation were common in neonates with IVH.

Conclusion: Lower gestational age, low birth weight, compromised Apgar scores, PIH, and PROM significantly increased the risk of IVH in preterm neonates. Early identification and preventive strategies are essential to reduce associated morbidity and mortality.

Keywords: *Preterm neonates, Intraventricular hemorrhage, Neurological outcomes*

1. INTRODUCTION

Intraventricular hemorrhage (IVH) remains a significant cause of morbidity and mortality among premature neonates, particularly those born before 32 weeks of gestation [1]. Despite advances in neonatal intensive care, the incidence of IVH continues to be considerable, ranging from 15% to 45% among extremely low birth weight infants [2]. The pathogenesis of IVH is multifactorial, with predisposing factors such as immaturity of the germinal matrix vasculature, fluctuations in cerebral blood flow, respiratory distress syndrome, and coagulation abnormalities playing a major role [3].

Mortality rates among neonates with severe IVH (grades III and IV) remain high, and survivors are at increased risk for a range of adverse neurological outcomes, including cerebral palsy, epilepsy, hydrocephalus requiring shunting, and cognitive impairment [4]. The severity of IVH is directly proportional to the risk of long-term neurodevelopmental disabilities, making early identification and management of risk factors critically important [5]. Emerging evidence suggests that antenatal corticosteroids, optimal management of respiratory distress, and stabilization of hemodynamics can reduce the incidence and severity of IVH [6]. However, the outcomes vary widely depending on factors

understand contemporary risk factors and outcomes to guide preventive strategies and postnatal interventions [9]. This study aims to evaluate the risk factors associated with IVH, its impact on mortality, and neurological outcomes in preterm neonates in a tertiary care setting, contributing valuable data to inform clinical practice and improve prognostication in this high-risk population [10].

Material and Methods

This prospective observational study was conducted at tertiary care hospital. A total of 100 preterm neonates (gestational age less than 37 weeks) diagnosed with intraventricular hemorrhage (IVH) were enrolled. Cranial ultrasonography was performed routinely within the first week of life and repeated as clinically indicated to detect IVH. Neonates with congenital brain malformations, chromosomal anomalies, or death within 12 hours from non-IVH causes were excluded.

The study protocol was reviewed and approved by the Institutional Ethics Committee of tertiary care hospital. Written informed consent was obtained from the parents or legal guardians of all participants prior to enrollment.

Demographic data including gestational age, birth weight, gender, and mode of delivery were recorded. Perinatal factors such as use of antenatal corticosteroids, presence of respiratory distress syndrome, sepsis, hypotension requiring treatment, and need for mechanical ventilation were documented. The severity of IVH was graded according to the Papile classification system. Follow-up assessments for neurological outcomes were performed at discharge and during outpatient visits using standardized neurodevelopmental evaluation tools.

Statistical analysis was carried out using SPSS version 25.0. Descriptive statistics were expressed as mean ± standard deviation for continuous variables and percentages for categorical variables. Associations between clinical factors and outcomes were assessed using the Chi-square test or Fisher’s exact test for categorical variables and Student's t-test for continuous variables. Logistic regression analysis was performed to identify independent predictors of mortality and adverse neurological outcomes. A p-value of <0.05 was considered statistically significant.

Results

Table 1 shows the demographic and clinical characteristics of preterm neonates enrolled in the study. The mean gestational age was 30.8 ± 2.9 weeks, and the mean birth weight was 1487.35 ± 322.48 grams. A higher proportion were males, and most neonates presented to the NICU within 24 hours of birth. Caesarean section was the predominant mode of delivery, and the majority of neonates had an Apgar score of ≤5 at 1 minute.

Table 2 presents the neonatal risk factors associated with the development of IVH. Lower gestational age (28–31 weeks), lower birth weight (500–1499 grams), and low Apgar scores at 1 and 5 minutes were significantly associated with a higher incidence of IVH, while gender and mode of delivery did not show a statistically significant association.

Table 3 describes the prenatal risk factors contributing to IVH. The presence of pregnancy-induced hypertension (PIH) and premature rupture of membranes (PROM) were significantly associated with an increased risk of IVH, while tocolysis use showed no significant association.

Table 4 outlines the clinical features observed among preterm neonates with IVH. Convulsions, irritability, pallor, and the need for respiratory support were significantly more common in the IVH group compared to those without IVH, whereas respiratory distress, apnea, and cyanosis did not show a significant difference between groups.

Table 5 highlights the neurological complications among neonates with IVH. Seizures were observed universally in affected neonates, while complications such as CNS infections, hydrocephalus, posthemorrhagic ventricular dilatation, and periventricular leukomalacia were also noted with varying frequencies.

Table 1: Demographics and clinical characteristic of the preterm neonates.

Parameters	**Preterm neonates (n=100) (%)**
Gestational age in weeks (mean ± SD)	30.8 ± 2.9
Birth weight in grams (mean ± SD)	1487.35 ± 322.48
Males	62%
Females	38%
Time at presentation to NICU	
<24 hours	76%
24–72 hours	18%
4–7 days	6%
Mode of delivery	
Caesarean section	83%
Vaginal delivery	17%
Apgar scores at 1 min	
≤5	79%
>5	21%
Apgar scores at 5 min	
≤5	52%
6–7	32%

>7	16%

Table 2: Neonatal risk factors associated with the development of IVH in preterm neonates.

Variables	IVH (n=20) (%)	No IVH (n=80) (%)	Total (n=100) (%)	P value
Gender				
Male	13 (20.9)	49 (79.1)	62 (100)	0.08 NS
Female	7 (18.4)	31 (81.6)	38 (100)	
Mode of delivery				
C-section	16 (19.3)	67 (80.7)	83 (100)	0.60 NS
Vaginal delivery	4 (23.5)	13 (76.5)	17 (100)	
Gestational age (weeks)				
28–31 weeks	15 (39.5)	23 (60.5)	38 (100)	0.001*
32–36 weeks	5 (7.9)	57 (92.1)	62 (100)	
Birth weight (grams)				
500–1499 g	17 (37)	29 (63)	46 (100)	0.01*
1500–2500 g	3 (6.8)	51 (93.2)	54 (100)	
Apgar score at 1 min				
≤5	17 (28.8)	42 (71.2)	59 (100)	0.001*
>5	3 (7.9)	35 (92.1)	38 (100)	
Apgar score at 5 min				
≤5	16 (34.8)	30 (65.2)	46 (100)	0.01*
6–7	3 (8.3)	33 (91.7)	36 (100)	

>7	1 (5.5)	17 (94.5)	18 (100)	

Table 3: Prenatal risk factors associated with the development of IVH in preterm neonates.

Variables	IVH (n=20) (%)	No IVH (n=80) (%)	Total (n=100) (%)	P value
Tocolysis use				
Yes	3 (30.0)	7 (70.0)	10 (100)	0.65 NS
No	17 (21.3)	63 (78.7)	80 (100)	
Pregnancy induced hypertension (PIH)				
Yes	7 (13.2)	46 (86.8)	53 (100)	0.004*
No	13 (48.1)	14 (51.9)	27 (100)	
Premature rupture of membranes (PROM)				
Yes	14 (51.8)	13 (48.2)	27 (100)	0.01*
No	6 (10.7)	67 (89.3)	73 (100)	

Table 4: Clinical features associated with IVH among the preterm neonates.

Clinical features	IVH (n=20) (%)	No IVH (n=80) (%)	Total (n=100) (%)	P value
Convulsions				
Yes	13 (100)	0 (0)	13 (100)	0.001*
No	7 (10.4)	80 (89.6)	87 (100)	
Irritability				
Yes	15 (71.4)	6 (28.6)	21 (100)	**0.01***
No	5 (8.6)	74 (91.4)	79 (100)	
Respiratory distress				
Yes	9 (22.5)	31 (77.5)	40 (100)	**0.76 NS**
No	11 (23.9)	35 (76.1)	46 (100)	
Apnea				
Yes	8 (42.1)	11 (57.9)	19 (100)	**0.09**

No	12 (20)	69 (80)	81 (100)	**NS**
Cyanosis				
Yes	3 (14.3)	18 (85.7)	21 (100)	**0.52**
No	17 (28.8)	42 (71.2)	59 (100)	**NS**
Pallor				
Yes	13 (65)	7 (35)	20 (100)	**0.01***
No	7 (11.3)	55 (88.7)	62 (100)	
Respiratory support				
Yes	13 (68.4)	6 (31.6)	19 (100)	**0.001***
No	7 (12.1)	74 (87.9)	81 (100)	

Table 5: Neurological complications among the IVH preterm neonates.

Neurological outcome	Premature neonates with IVH (n=20) (%)
CNS infection	6 (30%)
Seizure	20 (100%)
Hydrocephalus	5 (25%)
Posthemorrhagic ventricular dilatation	7 (35%)
Periventricular leukomalacia	4 (20%)

2. DISCUSSION

Intraventricular hemorrhage (IVH) continues to be a significant contributor to morbidity and mortality in preterm neonates, despite advances in neonatal care. In the present study, 20% of preterm neonates developed IVH, which is consistent with findings reported in contemporary neonatal literature [11]. Lower gestational age and birth weight were found to be significant risk factors for IVH, aligning with previous studies that emphasize the vulnerability of the immature germinal matrix vasculature in extremely preterm infants [12].

Apgar scores at 1 and 5 minutes were notably lower in neonates who developed IVH compared to those who did not. This correlation suggests that perinatal asphyxia and compromised neonatal adaptation play a substantial role in the pathogenesis

of IVH [13]. Prenatal factors, particularly pregnancy-induced hypertension (PIH) and premature rupture of membranes (PROM), were significantly associated with the development of IVH. PIH is thought to impair placental blood flow, leading to fetal hypoxia and vascular instability, predisposing neonates to cerebral hemorrhage [14].

Clinically, neonates with IVH exhibited a higher incidence of convulsions, irritability, pallor, and the requirement for respiratory support. Convulsions were found in all neonates with IVH, indicating that seizures may serve as an important early clinical indicator of cerebral injury in this population [15]. Neurological complications such as hydrocephalus, posthemorrhagic ventricular dilatation, CNS infections, and periventricular leukomalacia were observed at significant rates among neonates with IVH, highlighting the long-term neurological burden associated with this condition.

The study reinforces the importance of early identification and monitoring of at-risk neonates to mitigate the progression and complications of IVH. Strategies such as optimizing antenatal care, promoting the use of antenatal corticosteroids, minimizing fluctuations in cerebral blood flow, and maintaining stable hemodynamics are crucial preventive measures [16]. Future research should focus on enhancing early diagnostic tools, neuroprotective therapies, and long-term neurodevelopmental surveillance programs for affected neonates.

3. CONCLUSION

Intraventricular hemorrhage remains a significant challenge among preterm neonates, with low gestational age, low birth weight, low Apgar scores, and prenatal complications such as PIH and PROM identified as major risk factors. Early recognition of clinical features like seizures and irritability, combined with focused perinatal and neonatal management, is crucial to minimizing neurological sequelae and mortality. Preventive strategies including antenatal corticosteroids, vigilant hemodynamic monitoring, and neuroprotective interventions can further improve outcomes. Future research must emphasize long-term follow-up and neurodevelopmental rehabilitation for affected neonates.

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