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Original Research Article

Neurodevelopmental outcome in neonates with central nervous system insult: an observational study

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ABSTRACT

Background: Neonatal central nervous system (CNS) insults are a significant cause of morbidity and long-term neurodevelopmental problems. Common etiologies include hypoxic-ischemic encephalopathy (HIE), meningitis, intraventricular hemorrhage (IVH), and bilirubin encephalopathy. The severity of these conditions influences neurodevelopmental outcomes, making early assessment and intervention crucial for improving prognosis and long-term support.
Methods: This observational study was conducted at the Department of Pediatrics, Vivekananda Polyclinic & Institute of Medical Sciences, Lucknow, over 18 months. A total of 70 neonates with confirmed CNS insults admitted to the NICU were enrolled. The Hammersmith Infant Neurological Examination (HINE) was used for neurodevelopmental assessment at discharge, and at 3, 6-, 9-, 12-, and 18-months post-discharge. The severity of insults was classified based on clinical and imaging findings. Therapeutic interventions ranged from supportive care for mild cases to multidisciplinary rehabilitation for severe cases.
Results: Meningitis was the most common CNS insult (44.29%), followed by HIE (34.29%), bilirubin encephalopathy (12.86%) and IVH (8.57%). Mild cases, such as stage 1 HIE and low Bind scores in bilirubin encephalopathy, showed favorable neurodevelopmental recovery, whereas severe cases, such as stage 3 HIE and Grade 3 IVH, had poorer outcomes. Patients with meningitis consistently exhibited normal neurodevelopmental outcomes. Preterm birth, low birth weight, and perinatal complications were identified as significant risk factors for poor prognosis
Conclusions: This study underscores the critical role of early detection, consistent monitoring, and targeted interventions in optimizing neurodevelopmental outcomes in neonates with CNS injuries. Although mild cases demonstrate excellent recovery potential, severe cases require long-term support. These findings highlight the importance of neonatal care strategies in mitigating long-term developmental deficits.

Keywords: Neonatal CNS insults, Hypoxic-ischemic encephalopathy, Meningitis, Intraventricular hemorrhage, Bilirubin encephalopathy, Neurodevelopmental outcome, HINE score

INTRODUCTION

Neonatal central nervous system (CNS) insults occurring within the first 28 days of life contribute significantly to neonatal mortality, morbidity, and long-term neurodevelopmental challenges. Common causes include hypoxic-ischemic encephalopathy (HIE), infections, intracranial hemorrhage, and prematurity-related complications. These insults often result in cognitive, motor, sensory, and behavioral impairments that affect survivors' quality of life and development. Early assessment of neurodevelopmental outcomes in neonates with CNS insults is crucial for timely intervention, improved prognostication, and long-term support

planning to mitigate adverse outcomes and optimize developmental potential.[1]

HIE occurs due to insufficient oxygen and/or blood flow to the newborn brain. It is also known as "asphyxia," "birth asphyxia," or "perinatal asphyxia." and perinatal asphyxia. Although it often occurs before or during birth, it can develop immediately after birth. Apart from the brain, HIE can affect the lungs, liver, heart, intestine, and kidneys. HIE was classified as mild, moderate, or severe. Moderate-to-severe cases can lead to permanent disability and, in some cases, death.[1]

At birth, neonates transition from placental dependence to independent respiration (IR). Problems affecting glucose and oxygen exchange can arise at any gestational age before or during birth, although most deliveries are normal. Approximately half of all cerebral palsy cases are associated with neonatal encephalopathy. According to the Western Australian Cerebral Palsy Register, 15% of these cases are full-term births with acute encephalopathy at birth.[1] In developed countries, approximately 2–3 per 1000 newborns suffer from HIE, making it the single largest contributor to global disability, accounting for one-tenth of all disability-adjusted life years (DALYs).[2,3]

Newborn infections can be caused by bacteria, viruses, or fungi and often lead to similar outcomes. For instance, sepsis, a bloodstream infection, can result in brain damage and irreversible complications.[4] Meningitis, which is an infection of the brain and spinal cord membranes, is particularly dangerous in neonates. While various pathogens can cause meningitis, *Escherichia coli* (E. coli) and *Group B Streptococcus* (GBS) are the most common pathogens in the United States. Since neonatal meningitis is life-threatening, physicians initiate treatment before a formal diagnosis is made.[5] If treated promptly and effectively, neonates can fully recover. However, delayed or inadequate treatment can lead to severe deficits. Bacteria are the primary causes of neonatal meningitis.[5] GBS accounts for half of all GBS cases in the U.S., followed by E. coli (20%) and Listeria monocytogenes (5–10%).[6]

GBS is prevalent in approximately one in four pregnant women, making vertical transmission a major concern. GBS increases the risk of neonatal meningitis if undiagnosed and untreated. Consequently, the CDC recommends GBS screening in the third trimester, with antibiotics administered during labor to prevent transmission.[7]

Intraventricular hemorrhage (IVH) affects approximately 12,000 preterm neonates annually in the U.S.[8] The incidence has dropped from 40% to 50% in the early 1980s to 20% in the late 1980s, but rates have remained unchanged over the past 20 years. Nearly 45% of neonates born weighing 500–750 g develops IVH, making it a persistent challenge in modern neonatal critical care.[9]

Severe jaundice or hyperbilirubinemia can lead to bilirubin encephalopathy, which is a rare but serious neurological condition. Hyperbilirubinemia is common in the first few days and weeks of life. If progressive indirect hyperbilirubinemia (BIE) is not promptly diagnosed and treated, it can cause CNS injury and deficits in verbal, auditory, visual, dental, and neuromotor skills.[10] The severity of hyperbilirubinemia-related brain damage depends on the duration and timing of therapeutic interventions such as phototherapy and exchange transfusion. Determining neurodevelopmental outcomes in high-risk neonates requires long-term follow-up, especially as research shifts focus from major disabilities to high-prevalence, low-severity dysfunction.

This study is significant as it addresses the urgent need to understand the neurodevelopmental consequences in neonates with CNS insults, a leading cause of long-term disabilities in children. Early detection of neurodevelopmental delays enables timely intervention to improv the quality of life of affected neonates and their families. In addition, this study has the potential to influence clinical practice and policy decisions by bridging research gaps, particularly in diverse and underserved populations. These findings can help guide healthcare providers in predicting outcomes, optimizing resource allocation, and designing neonatal follow-up programs to improve long-term pediatric care.

METHODS

This observational study was conducted at a tertiary care hospital of north India, over 18 months after institutional ethical committee approval (Ref. No.-VPIMS/ME/NBEMS/Th,Proto/2022). Study involved 70 neonates with confirmed CNS insults admitted to the NICU. This study aimed to assess the neurodevelopmental outcomes of these neonates post-discharge through regular follow-up. Ethical clearance was obtained, and informed consent was obtained from parents or guardians before enrollment. The inclusion criteria were neonates diagnosed with HIE, meningitis, IVH, and bilirubin encephalopathy, whereas those with syndromic disorders, congenital anomalies, metabolic disorders, or inborn errors of metabolism were excluded. The Hammersmith Infant Neurological Examination (HINE) was used as the primary neurological assessment tool to evaluate cranial nerve function, posture, movements, tone, and reflexes at 3, 6, 9, 12, and 18 months. Each neonate was assigned a risk score, classifying them as low (≤5), moderate (6-9), or high-risk (>9) for neurodevelopmental disorders (NDDs).

Comprehensive therapeutic management was implemented based on the severity of the CNS injury. Mild cases are primarily managed with supportive care, including adequate nutrition, thermal regulation, infection prevention, and early stimulation therapies to enhance neurodevelopmental outcomes. Moderate cases required pharmacological interventions such as anticonvulsants for

seizure control, pain management, and antibiotics in cases of infections. Additionally, physical and occupational therapy were initiated early to improve motor function, reflexes, and cognitive abilities. Severe cases, such as stage 3 HIE or Grade 3 IVH, necessitate a multidisciplinary approach involving neurologists, developmental pediatricians, neonatologists, and rehabilitation specialists.

These neonates often require long-term medical supervision, including intensive physiotherapy, speech therapy, and occupational therapy, to optimize functional recovery and mitigate long-term impairment.

Regular follow-ups at 3, 6, 9, 12, and 18 months ensured continuous monitoring of neurodevelopmental progress, enabling the early identification of deficits and timely intervention. This study emphasized the importance of early detection, individualized treatment, and long-term rehabilitation in optimizing the developmental potential of neonates affected by CNS insults. These findings reinforce the critical role of neonatal care strategies in improving the outcomes and quality of life of the affected infants (Figure 1).

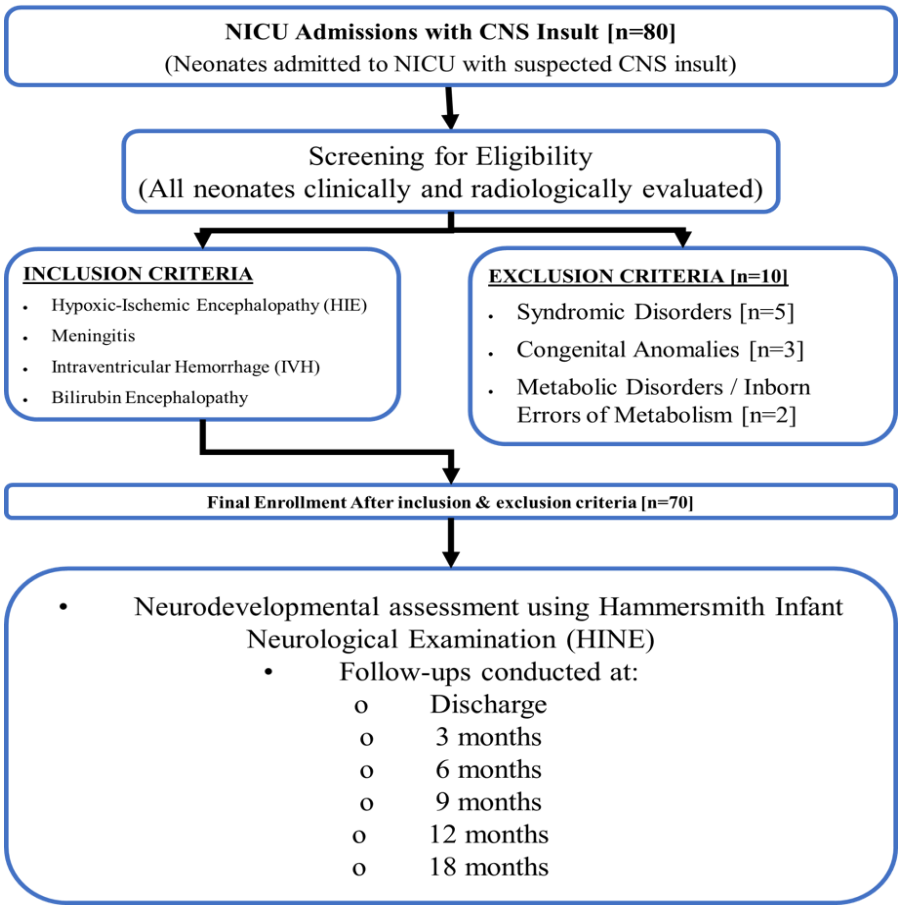

Figure 1: Flow chart of methodology.

RESULTS

Maternal demographic analysis revealed that the majority of mothers were aged 25-29 years (57.14%, 40), followed by 20-24 years (31.43%, 22) and 30-34 years (11.43%, 8), with a mean maternal age of 26.09±2.94 years. Primi-gravida accounted for 41.43% (29) of the cases, while multi-gravida cases included gravida 2 (24.29%, 17), gravida 3 (27.14%, 19), and gravida 4 (7.14%, 5). Regarding parity, primipara comprised 41.43% (29) of cases, while multipara included parity 2 (37.14%, 26) and parity 3 (7.14%, 5), with nullipara accounting for 14.29% (10). The mean gestational age of neonates at birth was 36.00±2.93 weeks. The most common maternal blood groups were B+ (27.14%, n=19), A+ (24.29%, n=17), and O+ (18.57%, n=13), whereas negative blood groups were less frequent. Maternal illnesses included fever (4.29%, 3), rash/fever (2.86%, 2), and UTI (1.43%, 1), whereas pregnancy complications included PIH (4.29%, 3), APH (10.00%, 7), and GDM (7.14%, 5) (Table 1). The mean birth weight was 2331.59±658.02 grams, birth length 49.87±2.33 cm, head circumference 30.86±1.31 cm, and chest circumference 29.47±1.48 cm. The most common neonatal blood groups were B+ (41.43%, n=29), A+ (25.71%, n=18), and O+ (14.29%, n=10). The mean APGAR score at 1 minute was 5.47±2.05, improving to 7.27±1.71 at 5 minutes (Table 2).

Table 1: Maternal and obstetric profile of the study population.

		Number	Percentage
Mother's age (in years)			
20-24		22	31.43
25-29		40	57.14
30-34		8	11.43
Mean±SD		26.09±2.94	
Gravida			
Primi-gravida		29	41.43
Multi-gravida	2	17	24.29
	3	19	27.14
	4	5	7.14
Parity			
Nullipara		10	14.29
Primi para		29	41.43
Multi-para	2	26	37.14
	3	5	7.14
Gestational weeks			
Mean±SD		36.00±2.93	
Blood group			
A-		1	1.43
A+		17	24.29
AB-		1	1.43
AB+		15	21.43
B-		2	2.86
B+		19	27.14
O-		2	2.86
O+		13	18.57
Illness			
Fever		3	4.29
Rash /Fever		2	2.86
UTI		1	1.43
Chief complaints			
PIH		3	4.29
APH		7	10.00
GDM		5	7.14

Table 2: Neonatal anthropometry and clinical parameters.

	Number	Percentage
Anthropometry parameters		
Birth weight	2331.59±658.02	
Birth length	49.87±2.33	
Head circumference	30.86±1.31	
Chest circumference	29.47±1.48	
Baby blood group		
A-	2	2.86
A+	18	25.71
AB-	1	1.43
AB+	8	11.43
B-	2	2.86
B+	29	41.43
O+	10	14.29
APGAR score at 1 min		
Mean±SD	5.47±2.05	
APGAR score at 5 min		
Mean±SD	7.27±1.71	

Among CNS insults, meningitis was the most prevalent (44.29%, 31 cases), followed by HIE (34.29%, 24 cases), bilirubin encephalopathy (12.86%, 9 cases), and IVH (8.57%, 6 cases) (Table 3). In bilirubin encephalopathy, three (33.33%) had a BIND score of 0, five (55.56%) had a BIND score of 1, and one (11.11%) had a BIND score of 2, reflecting varying severity. In HIE cases, stage 2 was the most common (50.00%, 12 cases), followed by stage 3 (29.17%, 7 cases) and stage 1 (20.83%, 5 cases), with increasing cord base excess and decreasing pH correlating with severity. (Figure 2) IVH cases were mostly preterm (83.33%, 5 out of 6), with grade 2 (4.29%, 3 cases) being the most frequent. Among meningitis cases, preterm births accounted for 80.65% (25 out of 31 cases), with prolonged leakage per vaginum observed in 64.52% (20 cases).

CSF analysis in meningitis cases showed mean sugar levels of 48.45 mg/dl±26.97, protein levels of 169.81 mg/dl±67.10, and cell counts of 43.00 cells/mm³±13.33. Blood cultures identified BYLC in 25.81% (8 cases), *Streptococcus hemolyticus* in 19.35% (6 cases), and *E. coli* and *Klebsiella* in 9.68% (3 cases each), while 29.03% (9 cases) had sterile blood cultures (Figure 3).

Table 3: Type of CNS insult in the enrolled patients.

Type of CNS insult	N	%
Bilirubin encephalopathy	9	12.86
HIE	24	34.29
IVH	6	8.57
Meningitis	31	44.29
Grand Total	70	100.00

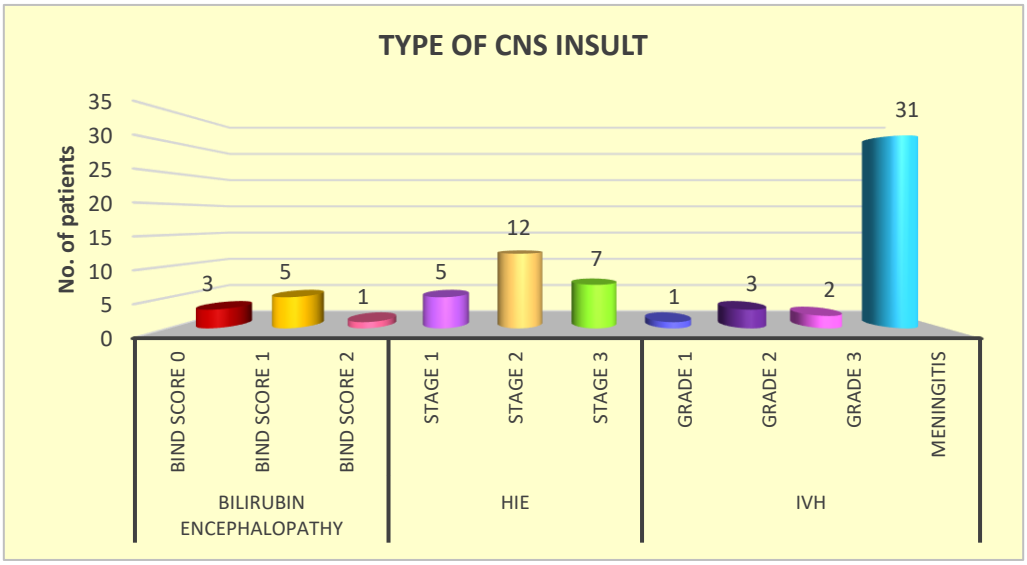

Figure 2: Graphical representation of the type of CNS insult as per severity.

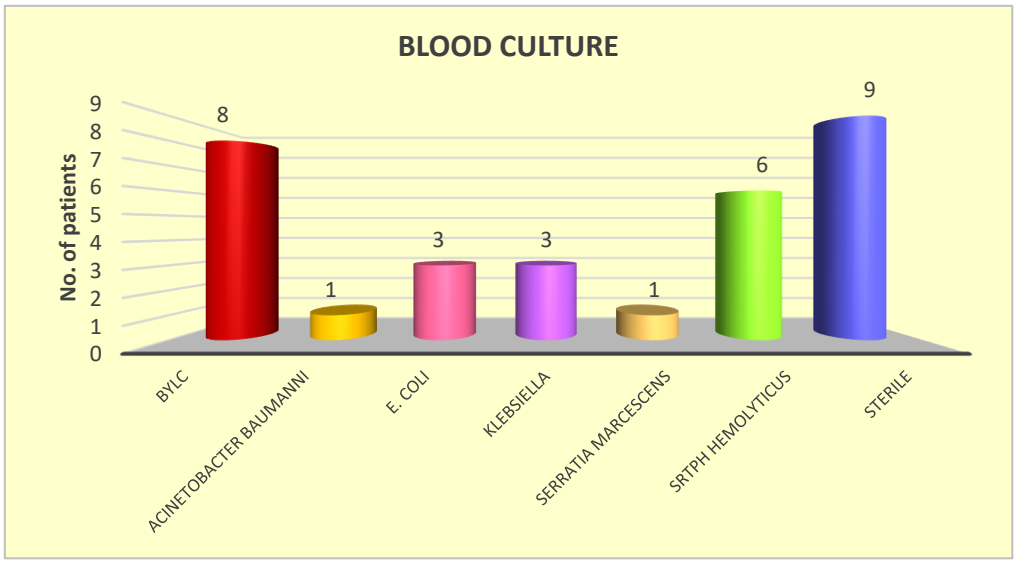

Figure 3: Graphical representation of the blood culture findings in the patients with meningitis CNS insult.

IVH was more prevalent in female neonates, with four out of six cases (66.67%), compared to two in males (33.33%). A majority, 5 neonates (83.33%) were born preterm, while only one (16.67%) was term. Regarding the mode of delivery, four neonates (66.67%) were delivered via lower segment cesarean section (LSCS), while two (33.33%) were born through normal vaginal delivery (NVD). Birth weight was another significant factor, as five neonates (83.33%) weighed less than 2500 grams, compared to only one neonate (16.67%) with a birth weight of 2500 grams or more. Additionally, ventilator support was required for 2 neonates (33.33%), whereas 4 neonates (66.67%) did not require ventilator support (Figure 4).

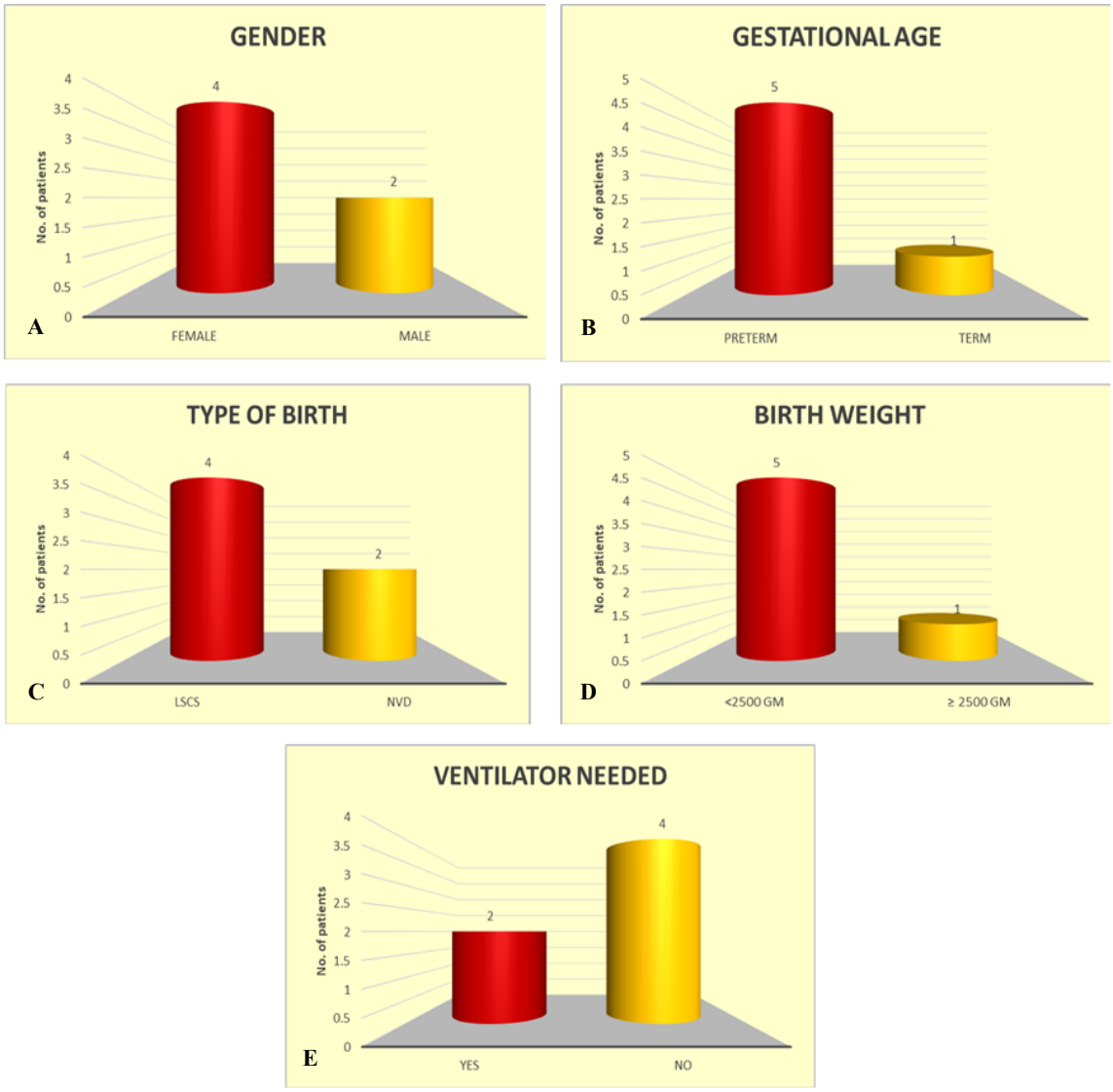

Figure 4 (A-E): Graphical representation of the clinico-etiological profile of the patients with IVH CNS insult.

Neurodevelopmental assessment using HINE scores revealed that all Stage 1 HIE neonates (100.00%) had normal outcomes, whereas Stage 2 cases had 75.00% normal and 25.00% abnormal outcomes. In Stage 3, 85.71% of neonates had abnormal outcomes. IVH Grade 1 and 2 cases had 100.00% normal outcomes, whereas grade 3 cases had 50.00% abnormal outcomes. All 31 neonates with meningitis (100.00%) had normal outcomes at all follow-up points. For bilirubin encephalopathy, neonates with a Bind score of 0 showed an improvement from 60.00±6.53 at discharge to 64.00±3.27 at 18 months (F=10.00, p<0.0001), while those with a Bind score of 1 improved from 64.80±5.88 67.20±3.92 (F=3.848, p=0.0105). For HIE, scores increased significantly in Stage 1 from 67.40±2.80 to 70.20±1.60 (F=25.13, p<0.0001) and in Stage 2 from 64.50±4.59 to 67.00±4.08 (F=5.524, p=0.0012), while Stage 3 showed modest improvement from 56.00±1.77 to 58.14±2.59 (F=15.51, p<0.0001). For IVH, Grade 1 improved from 65.00±2.94 to 67.00±3.56 (F=6.255, p=0.0004), while Grade 2 (68.00±1.00) and Grade 3 (69.00±0.00) remained stable. In meningitis, scores remained consistent from 70.26±1.61 at discharge to 70.32±1.55 at 18 months (F=0.02403, p=0.9949) (Table 4).

Table 4: Neurodevelopmental outcome by HINE score across CNS insults at various follow-ups.

Type of CNS insult		Neurodevelopment outcome (HINE score)								P value
		At discharge		3 months		6 months		18 months		
		Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd	
Bilirubin Encephalopathy	Bind score 0	60.00	6.53	60.00	6.53	64.00	3.27	64.00	3.27	F=10.00 p<0.0001*
	Bind score 1	64.80	5.88	64.80	5.88	67.20	3.92	67.20	3.92	F=3.848 p=0.0105*
	Bind score 2	67.00	0.00	67.00	0.00	67.00	0.00	67.00	0.00	--
HIE	Stage 1	67.40	2.80	67.40	2.80	70.20	1.60	70.20	1.60	F=25.13 p<0.0001*
	Stage 2	64.50	4.59	64.50	4.59	67.00	4.08	67.00	4.08	F=5.524 p=0.0012*
	Stage 3	56.00	1.77	56.00	1.77	58.14	2.59	58.14	2.59	F=15.51 p<0.0001*
IVH	Grade 1	65.00	2.94	65.00	2.94	67.00	3.56	67.00	3.56	F=6.255 p=0.0004*
	Grade 2	68.00	1.00	68.00	1.00	68.00	1.00	68.00	1.00	F=1.000 p=0.3940
	Grade 3	69.00	0.00	69.00	0.00	69.00	0.00	69.00	0.00	--
Meningitis		70.26	1.61	70.26	1.61	70.32	1.55	70.32	1.55	F=0.02403 p=0.9949

*Statistically significant.

DISCUSSION

Our study primarily involved young mothers, with 57.14% (40) aged 25–29 years and a mean age of 26.09±2.94 years. Among them, 41.43% (29) were primigravida, with a similar trend in parity. The most common maternal blood group was B+ (27.14%, 19), followed by A+ (24.29%, 17), and O+ (18.57%, 13). The mean gestational age was 36.00±2.93 weeks. The key maternal complications included APH (10.00%, 7), GDM (7.14%, 5), and PIH (4.29%, 3). Compared to Bas et al who studied adolescent mothers with lower gravidity (1.07) and parity (1.04), our study had better prenatal care access but lower rates of maternal anemia (33.6% in Bas EK et al and smoking.[11] Differences in obstetric complications were also evident, with our study reporting GDM (7.14%), PIH (4.29%), and APH (10%), whereas Bas et al focused on preeclampsia (5.8%).[11] Higher CS rates in their cohort (44.8%) were linked to fetal distress (28.7%) and cephalopelvic disproportion (18.5%).

Neonatal anthropometric data showed a mean birth weight of 2331.59±658.02 g, head circumference of 30.86±1.31 cm, and chest circumference of 29.47±1.48 cm. The most prevalent neonatal blood group was B+ (41.43%, 29). The mean APGAR scores at one and five minutes were 5.47±2.05 and 7.27±1.71, respectively. Compared to Bas EK et al our cohort had a lower birth weight (2331.59±658.02 g vs. 3070±566 g), smaller head circumference (30.86±1.31 cm vs. 33.6±1.8 cm), and lower APGAR scores (5.47 vs. 7.6 at one minute, 7.27 vs. 8.7 at five minutes), indicating higher neonatal distress and a greater proportion of preterm births.[11] Meningitis was the most common CNS insult (44.29%, 31), followed by HIE (34.29%, 24), bilirubin encephalopathy (12.86%, 9) and IVH (8.57%, 6). The HIE cases were categorized as stage 1 (7.14%), stage 2 (17.14%), and stage 3 (10.00%). The BIND scores showed increasing severity, with 33.33% scoring 0, 55.56% scoring 1, and 11.11% scoring 2. The IVH cases included grade 1 (1.43%), grade 2 (4.29%), and grade 3 (2.86%).

Male neonates predominated (79.17%, 19), whereas females accounted for 20.83% (5). All neonates were term (100.00%), with 41.67% delivered via NVD and 58.33% via LSCS. Antenatal findings included oligohydramnios in 25.00% (6) and reduced fetal movements in 12.50% (3). Birth weight analysis showed that 91.67% weighed over 2500 g, while 8.33% weighed below this threshold. The APGAR scores were low, with 95.83% scoring <7 at one minute and 70.83% at five minutes, reflecting significant neonatal distress.

Compared with Santosh et al our study had a higher male predominance (79.17% vs 59.5%) and fewer low-birth-weight neonates (<2.5 kg vs 8.33% vs. 40.5%). The LSCS rates were higher in our study (58.33% vs. 36.5% vaginal deliveries in Santosh et al.[14] Antenatal complications, such as oligohydramnios, were more frequent in our study (25.00%) than in Santosh et al where 62.2% of neonates were delivered outside hospitals, influencing neonatal distress levels.[12] While Santosh et al reported early onset seizures and low APGAR scores as poor prognostic factors, the higher LSCS rates and distress levels in our study likely stem from institutional and maternal differences, underscoring the impact of perinatal care on neurodevelopmental outcomes.[12]

In our study, 66% of newborns with bilirubin encephalopathy were preterm, whereas 33.33% were term. Birth weight analysis showed that 33.33% weighed <2500 g and 66.67% weighed >2500 g. The most common cause of hyperbilirubinemia was ABO incompatibility (33.33%) followed by Rh incompatibility (11.11%). These findings emphasize the importance of managing neonatal hyperbilirubinemia based on gestational age and hematological factors. Compared to Singh et al our study had a similar prevalence of ABO incompatibility, but differed in birth weight distribution and delivery mode, with more cesarean deliveries in our cohort.[13]

Meningitis was more prevalent in preterm neonates (80.65%) than term neonates (19.35%). Maternal factors such as fever during pregnancy (9.68%), urinary tract infections (16.13%), and prolonged leakage per vaginum (64.52%) were significant contributors. These findings align with those of Boskabadi et al who also reported a strong association between prolonged leakage per vaginum and neonatal meningitis.[14] Similar correlations were noted by Silva et al in which 45.4% of the neonates with meningitis had maternal UTIs.[15] Khalessi et al further highlighted the role of maternal infections, underscoring the need for timely diagnosis and treatment during pregnancy to reduce neonatal complications.[16]

HIE was most common in stage 2 (50%), followed by stage 3 (29.17%) and stage 1 (20.83%). This differs from the findings of Ansari et al in which stage 3 was predominant (43.3%). Differences in study populations, including a higher proportion of term and late preterm neonates in the study by Ansari et al may explain these variations.[17] Both studies reported near-equal sex distributions.[17]

HIE severity correlated with cord gas values, with increasing acidosis and base excess as the severity worsened. Stage 1 had the highest pH (7.31±0.09) and lowest base excess (5.20±0.40), while Stage 3 had the lowest pH (7.13±0.15) and highest base excess (12.57±1.40). These results are consistent with those of Blecharczyk et al emphasizing the importance of early detection and intervention, including therapeutic hypothermia, to improve neonatal outcomes.[11] Our findings highlight the necessity of close monitoring of cord gas values for early identification and management of HIE in newborns.

Our study's CSF analysis of neonatal meningitis showed a mean glucose level of 48.45 mg/dL and elevated protein levels (169.81 mg/dl), indicating blood-brain barrier disruption. Pleocytosis was observed with a mean CSF cell count of 43.00 cells/mm³, although all 50 CSF cultures were sterile, suggesting viral or nonbacterial meningitis or prior antibiotic treatment. Compared to Singh et al bacterial meningitis had lower CSF glucose (16.7±6.08 mg/dl) and higher protein levels (349.45±113.73 mg/dl).[19] Similarly, Moghtaderi et al reported higher CSF leukocyte counts in bacterial meningitis.[20] The absence of microbial growth in our study contrasts with the findings of Singh et al and Kaur et al highlighting the differences in infection types or prior antibiotic use.[21]

IVH was more common in female neonates (66.67%) and preterm infants (83.33%), with most cases (83.33%) weighing <2500 g. These findings align with those of Szpecht et al who associated IVH risk with prematurity, hypoxia, acidosis, and a lack of antenatal steroids.[22] Our study found higher IVH rates in cesarean births (66.67%) than Szpecht et al who found no significant difference.[22] MRI results showed severe IVH cases with periventricular edema (33.33%) and vascular abnormalities (16.67%), similar to those reported by Klebermass et al and Pinto et al linking severe IVH to poor neurodevelopmental outcomes.[23,24]

Neonates with bilirubin encephalopathy were predominantly preterm (66.67%), with ABO incompatibility (33.33%) as the leading cause, which is consistent with Singh et al.[15] Compared with Singh et al our study had more cesarean deliveries and fewer cases of G-6PD deficiency.[13] Newborn meningitis was more prevalent in preterms (80.65%), with maternal UTIs (16.13%) and prolonged leakage per vaginum (64.52%) as major risk factors, similar to Boskabadi et al and Silva et al.[14,15]

HIE was most frequent in stage 2 (50%), followed by stage 3 (29.17%), and stage 1 (20.83%), in contrast to Ansari et al where stage 3 was predominant. HIE severity correlated with worsening acidosis, with Stage 3 having the lowest pH (7.13±0.15) and highest base excess (12.57±1.40), consistent with Blecharczyk et al emphasizing early cord gas monitoring for timely intervention.[17,18]

Neurodevelopmental outcomes varied according to condition severity. Bilirubin encephalopathy cases with lower BIND scores showed significant improvement in developmental scores, similar to the findings of Kumar et al who linked early-stage ABE to better outcomes.[25] HIE cases showed better recovery in mild cases, while stage 3 HIE had persistent impairments, consistent with the findings of Haataja et al and Rutherford et al.[26,27] Low-grade IVH (grade 1–2) neonates had normal outcomes, while grade 3 IVH had mixed results, consistent with the findings of Yuan et al.[28] MRI findings supported these observations, linking severe IVH to worse neurodevelopment, as reported by Gilard et al.[29]

Overall, our study underscores the importance of early detection, therapeutic interventions, and advanced imaging techniques, such as DTI and MRI, in improving outcomes for neonates with CNS insults. These findings highlight the need for individualized management strategies to optimize neurodevelopmental prognoses.

CONCLUSION

In conclusion, this study highlights the significant impact of CNS insults on neurodevelopmental outcomes in neonates, with severity playing a crucial role in the prognosis. Meningitis demonstrated consistently positive outcomes, whereas severe HIE and IVH were associated with poorer recovery. Maternal and neonatal factors, including preterm birth, low birth weight, and perinatal complications, are significant contributors to CNS insults. Early detection, timely intervention, and continuous monitoring are essential for improving outcomes, particularly in high-risk neonates.

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