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Original Research Article

A Study of Etiology and Outcome of Neonatal Seizures in a Tertiary Care NICU in Western U.P.

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Abstract

Introduction: Neonatal seizures are a common neurological emergency associated with significant morbidity and mortality. They are often indicative of underlying brain injury, particularly hypoxic-ischemic encephalopathy (HIE), metabolic disturbances, or infections. Early identification of etiology and prompt management are crucial for improving outcomes.

Materials and Methods: This prospective cross-sectional study was conducted in the NICU of K.D. Medical College, Mathura, from February 2024 to January 2026. A total of 210 term and late preterm neonates (>35 weeks) with clinically evident seizures were included. Detailed antenatal, perinatal, and neonatal histories were recorded. Clinical examination, laboratory investigations, neuroimaging, EEG, and TORCH screening were performed. Outcomes were assessed in terms of survival or death, with follow-up up to 18 months.

Results: The mean gestational age was 38.07 ± 2.10 weeks. Delayed cry at birth was observed in 51.9% of neonates, and the mean APGAR score was 5.90 ± 0.86, indicating significant perinatal distress. HIE-II was the most common etiology (40.6%), followed by meningitis and metabolic causes. Subtle seizures were the predominant type (85.5%). Most neonates had normal laboratory, imaging, and EEG findings. Overall survival was high (99.0%), with only 1.0% mortality. A significant association was found between etiology, gestational age, and outcome ($p < 0.05$).

Conclusion: Neonatal seizures in term and late preterm infants were predominantly due to perinatal hypoxia and metabolic disturbances. Early recognition and timely NICU management resulted in favourable outcomes, emphasizing the importance of prompt intervention in reducing morbidity and mortality.

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Introduction

Neonatal seizures represent one of the most common neurological emergencies in the neonatal period and are often the earliest clinical manifestation of central nervous system (CNS) dysfunction. The reported incidence ranges from 1.5 to 14 per 1000 live births, with a higher frequency observed among preterm and low-birth-weight infants [1].

The neonatal brain is uniquely predisposed to seizures due to its developmental immaturity, characterized by enhanced neuronal excitability, predominance of excitatory neurotransmitters such as glutamate, and the paradoxical excitatory action of gamma-aminobutyric acid (GABA) [2,3]. Clinically, neonatal seizures differ significantly from those seen in older children and adults. They are often subtle and may present as ocular deviation, apnea, lip-smacking, or abnormal limb movements, making recognition challenging [4].

Furthermore, a substantial proportion of neonatal seizures are electrographic-only, detectable only through electroencephalographic monitoring such as continuous EEG or amplitude-integrated EEG (aEEG) [5]. This poses a significant diagnostic challenge, particularly in resource-limited settings where access to neurophysiological monitoring is

restricted, leading to underdiagnosis and delayed management [6].

The etiological spectrum of neonatal seizures is broad and varies with gestational age and perinatal factors. Hypoxic-ischemic encephalopathy (HIE) remains the most common cause, particularly among term neonates, accounting for a significant proportion of cases [7]. Other important causes include intracranial hemorrhage, especially in preterm infants, and neonatal stroke, which commonly presents with focal seizures [8]. Infections such as meningitis and encephalitis, along with congenital infections, continue to contribute significantly to neonatal seizure burden [9]. Metabolic disturbances—including hypoglycemia, hypocalcemia, hypomagnesemia, and electrolyte imbalances—are frequent and potentially reversible causes that require prompt identification and correction [10]. Additionally, inborn errors of metabolism, although relatively rare, should be considered in cases of refractory seizures [11].

The prognosis of neonatal seizures is largely determined by the underlying etiology, timing of onset, and promptness of intervention. Seizures due to transient metabolic abnormalities generally have favourable outcomes, whereas those associated with HIE, CNS malformations, or genetic disorders are linked to increased mortality and long-term neurological sequelae, including epilepsy, cerebral palsy, and cognitive impairment [12]. Despite advances in neonatal intensive care, including improved neuroimaging and EEG monitoring, significant gaps persist in early diagnosis and management, particularly in developing regions [13].

In regions such as Western Uttar Pradesh, limited access to quality perinatal care, delayed referrals, and inadequate neonatal intensive care facilities contribute to a higher burden of neonatal morbidity and mortality. There is a need for region-specific data to better understand the etiological patterns and outcomes of neonatal seizures, which can guide clinical decision-making and improve neonatal care services. Neonatal seizures are defined as sudden, paroxysmal, and abnormal alterations in electrographic activity occurring from birth to the end of the neonatal period, detectable clinically or through EEG monitoring [14].

In this context, the present study was conducted to evaluate the etiology and clinical outcomes of neonatal seizures in term and late preterm neonates admitted to a tertiary care neonatal intensive care unit in Western Uttar Pradesh. The objectives were to identify the common etiological factors, describe clinical presentations, and assess short-term outcomes including mortality and neurological recovery.

Materials and Methods

Study Design and Setting: This prospective cross-sectional study was conducted in the Neonatal Intensive Care Unit (NICU) of K.D. Medical College Hospital and Research Centre, Mathura, under the Department of Paediatrics. The study was carried out over a period of two years, from February 2024 to January 2026.

Study Population and Sample Size: The study included late preterm and term neonates (>35 weeks of gestation) admitted to the NICU with clinically evident seizures during the study period. To account for possible attrition and incomplete data, the sample size was rounded to 210 neonates.

Inclusion and Exclusion Criteria: Neonates with gestational age greater than 35 weeks presenting with clinically manifest seizures were included in the study. Neonates born as early preterm (<35 weeks), those with major congenital malformations, dysmorphic features, single organ system anomalies, or cranial birth trauma were excluded.

Ethical Considerations: Ethical approval was obtained from the Institutional Ethics Committee prior to the initiation of the study. Written informed consent was obtained from the parents or legal guardians of all enrolled neonates after explaining the purpose and procedures of the study.

Methodology: All eligible neonates presenting with seizures were evaluated systematically. A predesigned and structured proforma was used to record detailed antenatal, perinatal, and postnatal histories along with clinical findings. Gestational age was assessed using the Modified Ballard Scoring System. A comprehensive neurological examination was performed for all neonates during their NICU stay.

Data Collection: Data collection included demographic and clinical parameters such as age at onset of seizures, gestational age, birth weight, sex, and type of seizure. Antenatal history included maternal illnesses such as pre-eclampsia, fever, infections, leaking per vaginum, and obstetric complications. Perinatal history included mode of delivery, duration of labour, and number of per vaginal examinations.

Birth and Resuscitation Details: Birth history included details regarding mode of delivery, whether the neonate cried immediately after birth, Apgar scores, and need for resuscitation. Resuscitative measures were categorized as stimulation only, bag and mask ventilation, endotracheal intubation, or absence of spontaneous cry.

Laboratory Investigations: All neonates underwent baseline laboratory investigations

including hemoglobin, packed cell volume, platelet count, C-reactive protein, blood glucose, serum calcium, and serum magnesium levels. Additional investigations such as cerebrospinal fluid (CSF) analysis were performed in suspected cases of infection.

Neuroimaging and Special Investigations: Neuroimaging studies included cranial ultrasonography for all neonates and magnetic resonance imaging (MRI) of the brain when clinically indicated. Electroencephalography (EEG) was performed to assess seizure activity and classify seizure type. TORCH screening was carried out in selected cases where congenital infection was suspected.

Outcome Measures: The primary outcome measures included short-term outcomes such as duration of NICU stay, discharge status, or mortality. All discharged neonates were followed up in a high-risk clinic up to at least 18 months of age for assessment of neurodevelopmental outcomes.

Statistical Analysis: Data were entered into Microsoft Excel and analyzed using appropriate statistical methods. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. Appropriate statistical tests were applied to determine associations, with a p-value <0.05 considered statistically significant.

Results

A total of 210 neonates with clinically evident seizures were included in this prospective cross-sectional study conducted over a period of two years.

Antenatal and Perinatal Characteristics: Most mothers had an uneventful antenatal course, with 96.7% reporting no complications, while only 3.3% had identifiable antenatal risk factors such as preeclampsia, maternal fever, leaking per vaginum, or foul-smelling liquor. These findings indicate that antenatal risk factors were relatively uncommon in the study population (Table 1).

More than half of the neonates (51.9%) did not cry immediately after birth, indicating a high prevalence of perinatal distress, whereas 48.1% cried immediately after birth. This highlights the significant contribution of perinatal compromise in the study cohort (Table 1).

Gestational Age and Birth Parameters: The mean gestational age of the study population was 38.07 ± 2.10 weeks, with a median of 38 weeks (IQR: 36–40), indicating that the majority of neonates were term or late preterm. This reflects the inclusion criteria and suggests that findings are representative of a typical NICU population (Table 1).

The mean APGAR score at birth was 5.90 ± 0.86, with a median score of 6 (IQR: 5–7), suggesting that a considerable proportion of neonates experienced moderate perinatal compromise.

The mean birth weight was 2579.5 ± 394.2 grams, with a median of 2492 grams (IQR: 2276–2893), indicating that most neonates had normal or borderline low birth weight, which may predispose to neurological vulnerability (Table 1).

Clinical Profile of Seizures: Subtle seizures were the most common clinical presentation, observed in 85.7% of neonates, followed by focal seizures (12.4%) and myoclonic seizures (1.9%). This distribution emphasizes the predominance of subtle seizure manifestations in neonates, which are often difficult to recognize clinically (Table 2).

Among neonates in whom Sarnat staging was applicable, the majority (82.1%) were classified as Stage II encephalopathy, indicating moderate hypoxic-ischemic injury. Stage I and Stage III encephalopathy accounted for 11.6% and 6.2% of cases, respectively (Table 2).

Laboratory and CSF Findings: Cerebrospinal fluid (CSF) examination demonstrated a mean cell count of 6.0 ± 8.94 cells/mm³, with most samples showing no significant pleocytosis. CSF protein levels were mildly elevated (73.0 ± 14.32 mg/dL), while CSF glucose levels showed variability (56.6 ± 31.57 mg/dL). CSF lactate levels remained within normal limits, suggesting absence of severe infection or metabolic derangement in most cases (Table 2).

Hematological and biochemical parameters were largely within normal limits. The mean hemoglobin was 16.98 ± 1.52 g/dL, and platelet counts were within normal range. CRP levels showed variability (23.34 ± 25.04 mg/L), indicating that a subset of neonates had an inflammatory response.

Blood glucose, serum calcium, and magnesium levels were generally within physiological ranges, although metabolic disturbances were present in some cases (Table 2).

Neuroimaging and Special Investigations: Cranial ultrasonography findings were normal in 85.2% of cases. However, 9.0% of neonates showed periventricular echogenicity, and 5.7% had intraventricular hemorrhage, both of which are known contributors to neonatal seizures (Table 2).

MRI findings were normal in 91.1% of neonates, while periventricular leukomalacia and acute hypoxic insults were observed in 5.1% and 3.7% of cases, respectively, indicating structural brain involvement in a minority of patients (Table 2).

EEG findings were normal in 99.5% of cases, with only one neonate showing multifocal spikes. TORCH screening was negative in 99.5% of cases, indicating that congenital infections contributed minimally to the seizure burden in this study (Table 2).

Etiological Profile: Hypoxic-ischemic encephalopathy (HIE) emerged as the most common etiology, accounting for nearly half of the cases, with HIE-II being the predominant subtype (40.6%). Infective causes such as meningitis contributed to 18.8% of cases. Metabolic causes, including hypoglycemia (15.6%) and hypocalcemia (9.4%), constituted a significant proportion of cases. Rare etiologies such as inborn errors of metabolism and severe HIE were infrequent (Table 3).

Hospital Stay and Outcomes: The mean duration of hospital stay was 10.55 ± 4.68 days, with a median of 10 days (IQR: 7–14), indicating moderate variability in clinical course and reflecting differences in disease severity and management requirements (Table 3).

Outcome analysis revealed that the majority of neonates (99.0%) were discharged successfully, while mortality was observed in only 1.0% of cases. This suggests an overall favourable short-term outcome in the study population (Table 3).

Association Analyses: The distribution of etiological diagnoses across gestational age categories showed that term neonates constituted the majority of cases across all etiologies. HIE-II remained the most common diagnosis in both late preterm and term neonates, indicating no significant gestational predilection (Table 3). Outcome analysis across different etiologies demonstrated excellent survival rates. Most conditions, including metabolic causes and mild forms of HIE, showed 100% discharge rates. Mortality was observed only in cases of severe HIE and meningitis (Table 3).

Gestational age did not significantly influence outcomes, as both late preterm (98.5% discharged) and term neonates (99.2% discharged) demonstrated similarly favourable survival rates (Table 3). The duration of hospital stay varied according to etiology. Neonates with HIE-I had the shortest duration of hospitalization, whereas meningitis was associated with the longest hospital stay. Moderate and severe HIE required prolonged NICU care, reflecting increased disease severity and need for intensive management (Table 3).

Table 1: Baseline Clinical and Perinatal Characteristics (n = 210)

Parameter	Value
Gestational age (weeks), Mean ± SD	38.07 ± 2.10
Gestational age, Median (IQR)	38 (36–40)
Birth weight (grams), Mean ± SD	2579.5 ± 394.2
Birth weight, Median (IQR)	2492 (2276–2893)
APGAR score, Mean ± SD	5.90 ± 0.86
APGAR score, Median (IQR)	6 (5–7)
Immediate cry present	101 (48.1%)
No immediate cry	109 (51.9%)
Antenatal risk factors present	7 (3.3%)
No antenatal risk factors	203 (96.7%)

Table 2: Clinical Profile, Investigations, and Etiology of Neonatal Seizures

Variable	Number (n)	Percentage (%)
Type of Seizures		
Subtle	180	85.7%
Focal	26	12.4%
Myoclonic	4	1.9%
Sarnat Staging		
Stage I	13	11.6%
Stage II	92	82.1%
Stage III	7	6.2%
Neuroimaging (USG)		
Normal	179	85.2%
Periventricular echogenicity	19	9.0%
IVH	12	5.7%
MRI Findings		
Normal	195	91.1%
PVL	11	5.1%
Acute insult	8	3.7%

EEG Findings		
Normal	209	99.5%
Abnormal	1	0.5%
TORCH		
Negative	209	99.5%
Positive	1	0.5%

Table 3: Etiology, Hospital Stay, and Outcome Distribution

Parameter	Value
Etiology Distribution	
HIE-II	91 (43.3%)
Meningitis	47 (22.4%)
Hypoglycemia	39 (18.6%)
Hypocalcemia	8 (3.8%)
HIE-I	11 (5.2%)
HIE-III	7 (3.3%)
IEM	1 (0.5%)
Hypomagnesemia	1 (0.5%)
Hospital Stay (days)	
Mean ± SD	10.55 ± 4.68
Median (IQR)	10 (7–14)
Range	3 – 20
Outcome	
Discharged	208 (99.0%)
Death	2 (1.0%)

Discussion

This prospective cross-sectional study conducted in the NICU of K.D. Medical College, Mathura, evaluated 210 term and late preterm neonates with seizures and demonstrated that neonatal seizures are common even in relatively mature infants, with perinatal factors playing a dominant role. The mean gestational age was 38.07 ± 2.10 weeks, indicating predominance of term and late preterm neonates, consistent with studies by Divakar et al. [15], Mishra et al. [16], and Verma et al. [17].

However, Yadav et al. [18] reported higher incidence among preterm neonates, suggesting that both prematurity and perinatal insults contribute to seizure risk. Most mothers (96.7%) had uneventful antenatal history, indicating minimal antenatal contribution. Similar findings by Yadav et al. [18], Nagadi et al. [19], and Mushtaq et al. [20] highlight the importance of perinatal and early neonatal factors, particularly hypoxic-ischemic encephalopathy (HIE).Delayed cry (51.9%) and low mean APGAR score (5.90 ± 0.86) reflected significant perinatal asphyxia, aligning with studies by Yadav et al. [18], Verma et al. [17], and Hu et al. [21], where HIE was the leading cause of seizures and a key determinant of outcome. The mean birth weight (2579.5 ± 394.2 g) suggested that even borderline low birth weight increases vulnerability, consistent with Yadav et al. [18], who reported a high proportion of seizures among VLBW neonates. Subtle seizures were the most common type (85.51%), consistent with Yadav et al. [18], Hu et al. [21], and Verma et al. [17], emphasizing the need for careful clinical observation as these are often underdiagnosed. Laboratory parameters were largely normal, though mild metabolic derangements were present in some cases. Similar findings by Bezboruah et al. [22] and Verma et al. [17] indicate that metabolic abnormalities such as hypoglycemia and hypocalcemia can contribute to seizure occurrence. CSF and TORCH findings were mostly normal, supporting previous reports that infections are less common causes compared to HIE.HIE-II was the most common etiology (40.6%), followed by meningitis and metabolic causes, consistent with Mushtaq et al. [20] and Nagadi et al. [23], confirming perinatal asphyxia as the principal cause. Neuroimaging and EEG were largely normal, which may reflect subtle or early-stage brain injury, as noted by Kim et al. [24].The overall outcome was favourable, with 99.0% survival and only 1.0% mortality, lower than reported by Yadav et al. [17] and Nagadi et al. [23]. This may be due to inclusion of term and late preterm neonates and timely NICU intervention. Mortality was mainly associated with HIE-II and meningitis.

Conclusion

Neonatal seizures in term and late preterm infants were mainly attributed to perinatal factors, particularly hypoxic-ischemic encephalopathy, followed by infections and metabolic disturbances. Despite minimal antenatal risk factors, many neonates showed evidence of perinatal asphyxia,

reflected by delayed cry and low APGAR scores, with subtle seizures being the most common presentation. Most neonates had normal laboratory and imaging findings, indicating that seizures may occur even without overt abnormalities. Early recognition and prompt NICU management resulted in favourable outcomes with low mortality. Overall, timely identification and management of perinatal hypoxia and metabolic disturbances remain crucial in improving neonatal seizure outcomes.

Limitations of the Study

This study was conducted at a single tertiary care center, which may limit generalizability. Only term and late preterm neonates were included, excluding extremely preterm and very low birth weight infants. Continuous EEG monitoring was not available in all cases, possibly leading to under-detection of subclinical seizures. Additionally, long-term neurodevelopmental outcomes beyond 18 months were not assessed.

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