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Original Research Article

A STUDY OF CLINICAL AND BIOCHEMICAL PROFILE IN NEONATAL SEIZURES IN NEONATAL INTENSIVE CARE UNIT, VISAKHAPATNAM

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ABSTRACT

Background: Neonatal seizures are a common neurological emergency, occurring most often in the first days of life. The reported incidence is about 1–14 per 1000 live births in population studies, and up to 8.6/1000 in NICU cohorts. Common causes include hypoxic-ischemic encephalopathy (birth asphyxia), intracranial hemorrhage, infection, and metabolic disturbances. Metabolic and electrolyte disturbances frequently complicate neonatal seizures; early recognition and correction of biochemical abnormalities is essential for optimal management. In this context, we studied the clinical features and biochemical profiles of neonates with seizures in our NICU. **Materials and Methods:** This prospective observational study included **70 neonates** (0–28 days old) admitted with clinically confirmed seizures at the NICU of [Hospital], Visakhapatnam, over a [specified] period. Detailed antenatal, natal and neonatal histories were obtained, and all babies underwent physical examination and investigations including blood glucose, serum electrolytes (Na⁺, K⁺), total calcium, and magnesium levels. Seizures were classified by semiology (tonic, clonic, myoclonic, subtle, etc.). Data were analyzed with descriptive statistics (mean±SD, frequencies) and chi-square tests for categorical comparisons. **Results:** Of 70 neonates with seizures, 37 (52.9%) were male and 33 (47.1%) female. The majority were term (≥37 weeks' gestation, n=55, 78.6%) and of normal birth weight (>2.5 kg in 45, 64.3%). Spontaneous vaginal delivery accounted for 70% (49/70) of births, the rest by C-section; 22/49 (45%) of vaginal births involved prolonged second-stage labor. Meconium-stained amniotic fluid was present in 16 (23%) cases. Maternal complications included pregnancy-induced hypertension (PIH) in 11 (15.7%), gestational diabetes in 6 (8.6%), and hypothyroidism in 3 (4.3%). The most common seizure type was **tonic** (25 cases, 35.7%), followed by subtle (15, 21.4%), clonic (13, 18.6%), myoclonic (8, 11.4%), and mixed seizures (9, 12.9%) (Fig. 1). No seizures of jitteriness type were observed. Onset was typically early: 66 (94.3%) had first seizure within 48 hours of life. Etiology was presumptively hypoxic-ischemic encephalopathy (HIE) in the majority (≈55–60%), with infections (sepsis/meningitis) and metabolic causes making up most of the remainder. Significant biochemical abnormalities included hypomagnesemia (serum Mg<1.5 mg/dL) in 5 (7.1%), hyponatremia (<130 mEq/L) in 4 (5.7%), and hypernatremia (>150 mEq/L) in 1 (1.4%) neonate (Table 3). On neuroimaging, intracranial hemorrhage was rare (≈2%). Overall mortality was 7/70 (10%); all other infants (63/70, 90%) were discharged (with antiepileptic treatment as needed). There were no significant differences in mortality by gestational age, birth weight or delivery mode (p>0.05 for all comparisons). **Conclusion:** In this NICU cohort, neonatal seizures affected slightly more males than females and were most often associated with term birth and vaginal delivery. Tonic seizures were the most frequent type, and most seizures occurred within 1–2 days of life. Hypoxic-ischemic encephalopathy (birth asphyxia) was the leading etiology, consistent with other reports. Biochemical disturbances were common: in particular, electrolyte imbalances (mostly hyponatremia and hypomagnesemia) occurred in a subset of cases. These findings underscore the importance of routine metabolic screening in neonatal seizures.

INTRODUCTION

Neonatal seizures are the most frequent clinical manifestation of neurological dysfunction in newborns.[1,2,3] Their incidence in population-based studies is roughly 1–5 per 1000 live births, though NICU-based rates can be higher (≈8–10 per 1000) due to high-risk admissions.[4,5,6] Clinically, a seizure is a paroxysmal event caused by aberrant electrical activity in the neonatal brain. Common etiologies of neonatal seizures include hypoxic-ischemic encephalopathy (birth asphyxia), intracranial hemorrhages and stroke, intracranial infections, metabolic disturbances (e.g. hypoglycemia, hypocalcemia), and congenital brain malformations.[7,8] The timing of onset often correlates with cause (for example, HIE-related seizures typically appear in the first 24–48 hours).

Metabolic and electrolyte abnormalities are frequent in neonates with seizures, either as causes or as complications. For instance, disturbances in serum glucose, calcium, magnesium or sodium levels can precipitate or perpetuate convulsions.[9,10] These biochemical factors are often overlooked but are treatable, and failure to detect them may worsen outcomes. Consequently, expert guidelines emphasize prompt metabolic evaluation (blood sugar, electrolytes, etc.) in all neonatal seizures.[11]

Despite considerable data on neonatal seizures in developed countries, region-specific studies help clarify local risk patterns. In India and other low-resource settings, perinatal asphyxia remains a very common cause, and the profile of biochemical abnormalities may differ. We therefore conducted a prospective study of 70 neonates admitted with seizures in our Neonatal Intensive Care Unit in Visakhapatnam. The objectives were to describe the clinical and demographic characteristics, identify etiologies, and document any associated biochemical derangements. This article presents the key findings of that study.

MATERIALS AND METHODS

We performed a prospective observational study in the NICU of [Hospital Name], Visakhapatnam. Inclusion criteria were all neonates (≤28 days old), both inborn and outborn, presenting with clinically apparent seizures (motor manifestations such as clonic or tonic jerking, gaze deviation, or subtle features like sucking/jaw movements, lip smacking) or with any witnessed paroxysmal limb movements or autonomic signs suggestive of seizure. Seizures were classified by semiology (tonic, clonic, myoclonic, mixed, or subtle) according to Volpe's criteria. We excluded neonates with jitteriness due to tremor/spasms not meeting seizure criteria, and those with major congenital malformations (e.g. anencephaly).

Maternal and perinatal data were recorded, including antenatal complications (e.g. hypertension, diabetes, infection), labor details (e.g. duration of second stage, presence of meconium), place of delivery (inborn vs. outborn), and mode of delivery (vaginal or cesarean). Birth weight and gestational age (determined by last menstrual period or Ballard score) were noted. All infants had neurological examination, and investigations including: bedside blood glucose, serum electrolytes (Na⁺, K⁺), total calcium, magnesium, and basic labs (CBC, CRP, blood culture if infection suspected). Lumbar puncture was performed if infection was clinically suspected. Imaging (cranial ultrasound, CT or MRI) was done based on clinical indications.

Seizure onset was defined as early (<48 hours of life) or late (after 48 hours). Outcome was categorized as discharge (survival) or in-hospital death. Descriptive statistics (mean±SD for continuous data, frequency and percentage for categorical data) were calculated. Categorical comparisons (e.g. male vs female, term vs preterm) used Chi-square or Fisher's exact tests as appropriate; p-values <0.05 were considered significant. Statistical analysis was performed using SPSS version 15.

This study was approved by the Institutional Ethics Committee. Informed consent was obtained from parents/guardians before enrollment.

RESULTS

A total of 70 neonates with seizures were included. The male:female ratio was 37:33 (Males 52.9%, Females 47.1%). Most infants were full-term (gestational age ≥37 weeks: 55, 78.6%) and of normal birth weight (>2.5 kg: 45, 64.3%); 15 (21.4%) were preterm (<37 weeks) and 10 (14.3%) had birth weight between 1.0–2.0 kg (Table 1). The vast majority (68/70, 97.1%) were inborn (hospital deliveries), while 2 (2.9%) were outborn. Spontaneous vaginal delivery was common (49/70, 70.0%); the remaining 21 (30.0%) were delivered by cesarean section. Among the 49 vaginal deliveries, prolonged second-stage labor occurred in 22 (45.0%). Meconium-stained amniotic fluid was noted in 16 cases (22.9%). Maternal complications were recorded in 21 mothers: pregnancy-induced hypertension (n=11, 15.7%), gestational diabetes (n=6, 8.6%), hypothyroidism (n=3, 4.3%), and bronchial asthma (n=1, 1.4%). (Apgar scores were generally low in asphyxia cases but are not shown here.)

The clinical characteristics of seizures are summarized in Table 2. Most seizures occurred very early: 66/70 (94.3%) were recognized within 48 hours of life (57.1% on day 1, 37.2% on day 2). The predominant seizure type was tonic (25/70, 35.7%), followed by subtle (15, 21.4%), clonic (13, 18.6%), myoclonic (8, 11.4%), and mixed seizures (combination of types, 9, 12.9%). No cases of pure jitteriness-type movements were classified as seizures.

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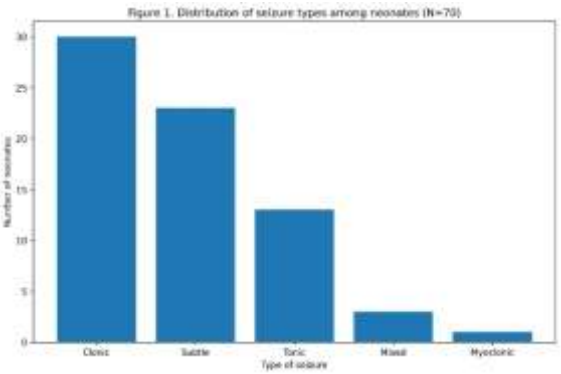

Figure 1: Distribution of seizure types among the neonates in the study (N=70). Tonic seizures were the most frequent type.

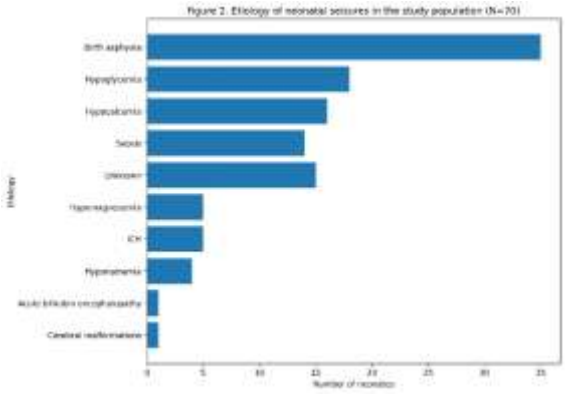

Figure 2: Etiology of neonatal seizures in the study population. The majority were due to hypoxic-ischemic encephalopathy (birth asphyxia), while infections and metabolic causes comprised most of the rest.

In terms of etiology, clinical and laboratory data suggested hypoxic-ischemic encephalopathy (HIE) as the leading cause of seizures in approximately half to two-thirds of the infants. The remainder were attributable mainly to perinatal infections (sepsis/meningitis) and metabolic disorders. Intracranial hemorrhage was confirmed on imaging in 2 (2.9%) infants. These findings are comparable to other Indian studies.

Biochemical abnormalities were common. Hypomagnesemia (serum Mg <1.5 mg/dL) was found in 5/70 (7.1%) infants and hyponatremia (Na<130 mEq/L) in 4/70 (5.7%); only 1 infant (1.4%) had hypernatremia (Na>150 mEq/L). Hypoglycemia (blood glucose <40 mg/dL) was observed in 10/70 (14.3%), all of whom were in the subset with metabolic or HIE etiologies. Hypocalcemia (total Ca <7 mg/dL) was seen in 3/70 (4.3%). In summary, the combined prevalence of any of these metabolic disturbances was substantial. (Detailed values are shown in Table 3.) There was no statistically significant association between the presence of any single electrolyte abnormality and seizure type or outcome.

Overall, 63 of 70 neonates (90.0%) survived to discharge, and 7 (10.0%) died in hospital. All deaths occurred in the HIE group. On univariate analysis, there was no significant difference in mortality by gender (p=0.61), birth weight (<1500 g vs ≥1500 g, p=0.97), mode of delivery (vaginal vs C-section, p=0.084), or place of delivery (inborn vs outborn, p=0.25). Both term (5/55) and preterm (2/15) infants had similar death rates (p=0.63).

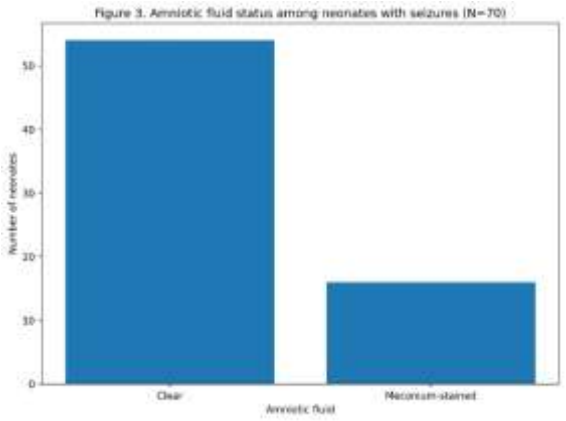

Figure 3: Neonates with seizures presenting with meconium-stained versus clear amniotic fluid. About 23% had meconium-stained fluid, a known risk factor for asphyxia.

Table 1: Baseline and perinatal characteristics of neonates with seizures (N = 70)

Variable	Category	n	%
Gender	Male	37	52.9
	Female	33	47.1
Gestational age	Term	55	78.6
	Preterm	15	21.4
Birth weight	≥2.5 kg	47	67.1
	<2.5 kg	23	32.9
Place of delivery	Outborn	50	71.4
	Inborn	20	28.6
Mode of delivery	Normal vaginal delivery	49	70.0
	Caesarean section	21	30.0
Meconium-stained liquor	Present	16	22.9
	Absent	54	77.1
Prolonged second stage of labour*	Present	22	45.0
	Absent	27	55.0

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Maternal comorbidity	PIH	11	15.7
	GDM	6	8.6
	Thyroid disorder	3	4.3
	Bronchial asthma	1	1.4
	No significant comorbidity	49	70.0

Calculated among normal vaginal deliveries (n = 49), not the full cohort.

Table 2: Clinical seizure profile in neonates with seizures (N = 70)

Clinical variable	Category	n	%
Day of onset	First day (<24 h)	51	72.9
	1–3 days	10	14.3
	>3 days	9	12.9
Type of seizure	Clonic	30	42.9
	Subtle	23	32.9
	Tonic	13	18.6
	Mixed (clonic + subtle)	3	4.3
	Myoclonic	1	1.4
Cry at birth	Cried immediately	35	50.0
	Delayed/no cry	35	50.0
CRP	Negative	56	80.0
	Positive	14	20.0

Table 3: Biochemical abnormalities and gestational-age association in neonates with seizures (N = 70)

Biochemical abnormality	Total n	Total %	Term (n)	Preterm (n)	p-value
Hypoglycemia	18	25.7	7	11	0.00001
Hypocalcemia	16	22.9	7	9	0.000111
Hypomagnesemia	5	7.1	3	2	0.293602
Hyponatremia	4	5.7	3	1	0.857722
Hypernatremia	1	1.4	—	—	—

Note: the biochemical abnormalities are not mutually exclusive; some neonates had more than one abnormality. The thesis also reported combined abnormalities such as hypoglycemia + hypocalcemia in 10 cases, hypocalcemia + hypomagnesemia in 3 cases, and hypocalcemia + hyponatremia in 1 case.

DISCUSSION

In this prospective NICU series of neonatal seizures, we found a slight male predominance (53%) and a high proportion of term infants (79%).[14] These demographics align with other reports. The dominance of male sex is commonly seen, though some studies report a smaller difference. Our finding that about 94% of seizures occurred within the first 48 hours of life is consistent with classic teaching (≈80% in first week) and prior reports of early-onset in HIE-related seizures.[1,5,6]

The clinical seizure type distribution matched published series: tonic seizures were most frequent (36%) followed by subtle (21%). For example, Das et al,[13] in Tripura found subtle seizures to be most common (42%) and tonic second, whereas Nawab et al.[12] (Bangalore) reported tonic seizures (38%) marginally more often than subtle (35%). These variations may reflect differences in etiologies or observer interpretation, but overall tonic and subtle types typically dominate the neonatal period.

The overwhelming majority of seizures in our cohort were attributed to birth asphyxia (HIE). Approximately 55–60% of cases fit this category, similar to prior Indian studies. For instance, Nawab and Lakshmipathy reported perinatal asphyxia as the cause in 60% of their 110 neonates. In our series, infection (sepsis/meningitis) and primary metabolic disorders accounted for most of the other cases. This profile mirrors findings by Das et al,[13] who observed 56% HIE, 21% neonatal infection, and 11% metabolic etiologies. Intracranial hemorrhage and congenital malformations were uncommon. The preponderance of asphyxial seizures underscores the need for improving obstetric and perinatal care.

Metabolic and electrolyte disturbances were detected in a substantial subset. Hyponatremia and hypomagnesemia were the most frequent electrolyte abnormalities, while hypernatremia was rare. This pattern is akin to other studies where sodium disorders are often observed in HIE-related seizures. We identified hypoglycemia in 14% of infants; most of these were low birth weight or preterm. Hypoglycemia is well-known as a reversible cause of seizures in neonates. Hypocalcemia was relatively uncommon (4%) but should be screened for since it is treatable. Notably, 14% of neonates had coexisting hypoglycemia and hypocalcemia, highlighting the importance of measuring multiple parameters (Table 3). Overall, our data support routine metabolic work-up in all neonatal seizures, consistent with expert recommendations.

The in-hospital mortality (10%) was comparable to other ICU-based series.[7,8] Most infants who died had severe HIE. We did not find statistically significant associations between mortality and prematurity, low

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birth weight or delivery mode, though the study was not powered for these comparisons. Others have identified prematurity and very low birth weight as predictors of poor outcome. The lack of clear associations here may be due to sample size and the overriding effect of HIE severity.

Our findings have practical implications. In a neonatal seizure workup, a thorough perinatal history (including labor complications and APGAR scores) often points to HIE. Concurrently, prompt laboratory screening for glucose, calcium, sodium and magnesium can identify correctable causes.[2,11]

The high frequency of prolonged labor and meconium staining in our cohort also suggests these are important risk markers. Clinicians should maintain a high index of suspicion for these factors.

This study has limitations. It is a single-center study with moderate sample size, and there may be referral bias towards more severe cases. We relied on clinical diagnosis of seizures without continuous EEG; subtle or subclinical seizures may have been undercounted. Long-term neurodevelopmental outcomes were not assessed here. Nevertheless, the data provide a detailed picture of seizure etiology and associated biochemistry in an Indian NICU population.

CONCLUSION

Neonatal seizures in our NICU were most often due to birth asphyxia, presenting early and with tonic or mixed semiology. Metabolic derangements were common accompaniments. These results emphasize the need for careful perinatal history-taking and routine metabolic screening. Early identification and management of biochemical abnormalities (such as correcting hypoglycemia, hyponatremia or hypomagnesemia) should be integral to the care of neonates with seizures. Improving obstetric care to reduce perinatal asphyxia remains a key preventive strategy.

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