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Etiological Spectrum of Neonatal Seizures in Neonatal Intensive Care Unit in Tertiary Care Hospital

Muhammad Ihsan[1], Amjad Iqbal[1], Hasham Khan[1], Hizbullah Khan Burki[1], Akhlaq Uddin[1], Sahibzada Aziz Ullah[1]

[1]Department of Pediatrics, CMH Abbottabad, KP, Pakistan.

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Correspondence to: Muhammad Ihsan, Department of Pediatrics, CMH Abbottabad, KP, Pakistan.
Email: mihsancq786@gmail.com

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ABSTRACT

Background: Neonatal seizures rank as one of the most sensitive neurological emergent conditions presenting most often as foreboders of subbing pathologies that need immediate recognition, as well as treatment for prevention of late neurodevelopmental sequelae, particularly in resource-poor settings. **Objective:** To determine the frequency of the causes of neonatal seizures in neonatal intensive care unit in tertiary care hospital. **Study Design:** Descriptive cross-sectional study. **Duration and Place of Study:** The study was conducted from January to May 2025 in the Neonatal Intensive Care Unit, Department of Pediatrics, CMH Abbottabad. **Methodology:** A total of 110 neonates aged 1 to 28 days presenting with clinical seizure activity were enrolled. Seizure types were classified into subtle, clonic, tonic, and myoclonic based on observable features. Etiological factors including sepsis, hypoxic-ischemic encephalopathy (HIE), hypoglycemia, hypocalcemia, meconium aspiration syndrome (MAS), and polycythemia were investigated using defined clinical and laboratory criteria. **Results:** The most frequent seizure type was subtle (48.2%). Sepsis was the leading etiology (80.9%), followed by HIE (44.5%), MAS (24.5%), hypocalcemia (22.7%), hypoglycemia (17.3%), and polycythemia (14.5%). A statistically significant association was observed between age group and polycythemia (p=0.002), while other etiologies showed no significant association with clinical variables. **Conclusion:** Sepsis remains the predominant cause of neonatal seizures, with subtle seizures being the most common type.

INTRODUCTION

Neonatal seizures are the most common neurological emergency in the neonatal period, presenting in the first 28 days of life in most cases.[1] They indicate a causative cerebral insult and commonly present as the first sign of neurological illness in neonates.[2] Neonatal seizures distinctly differ from seizures in older children in that they present in the absence of gross obvious clinical or electrographic features of seizures, presenting as subtle clinical signs of eye deviation, lip smacking, apnea, or tonic posturing, instead of classical convulsions.[3] Early recognition is critical since prolonged or repeated seizures can lead to late-term neurodevelopmental disability.[4] Etiologies vary extensively, based on time of onset as well as perinatal circumstances, and it is therefore important to take a rigorous approach to diagnostically determine the cause in order to direct specific therapy.[5] Neonatal seizures most commonly result from hypoxic-ischemic encephalopathy (HIE), i.e., as a condition secondary to defective perinatal epoch cerebral oxygenation.[6] Frequently associated with birth asphyxia, prolonged labor, or placental insufficiency, the ensuing energy failure in the brain cells results in neuronal injury, culminating in seizures in the first 24 hours of life in most cases.[7] Severity of the seizures is typically associated with the severity of encephalopathy, and therapeutic hypothermia has been proven to be one of the cornerstone measures of lessening brain injury in presentations of moderate to severe severity.[6,7] Prognosis is largely dependent upon the severity of hypoxic insult as well as the timeliness of supportive care.

Sepsis continues to be one of the leading causes of neonatal seizures, most commonly in the developing world.[8] Neonatal bacterial infections, for instance, by the Escherichia coli, Group B Streptococcus, or Listeria monocytogenes, can lead to meningitis or encephalitis, consequently ensuing in seizures.[9] Inflammatory mediators as well as direct invasion of the microorganisms upset the neurons' stability, commonly leading to multifocal resistant seizure activity.[9] Early recognition of sepsis, facilitated by laboratory indicators as well as clinical evidence, is keys to the administration of antimicrobial therapy.[10] Hypoglycemia as well as hypocalcemia, common metabolic derangements in the neonate, also comprise vital correctable causes of the seizures.[11] Such disturbances commonly remain secondary to conditions such as intrauterine growth restriction, maternal diabetes, or prematurity as well as

characteristically manifest with subtle neurological findings that advance unless corrected in time.[11] Less frequently, the causes of seizure etiology are meconium aspiration syndrome (MAS) as well as neonatal polycythemia.[12] Hypoxia as well as subsequent respiratory insufficiency in MAS can cause insufficient delivery of oxygen to the brain with subsequent seizure activity.[12] Polycythemia increases blood viscosity, thereby impairing cerebral perfusion function and, consequently, bringing about ischemic injury.[13] In these situations, the neurological impact is caused by indirect mechanisms of cerebral damage, for which treatment of the systemic illness is essential.

A study conducted by Rastogi S. et al. reported that among neonates with seizures admitted to the neonatal intensive care unit, sepsis was the most frequent underlying condition (70%), followed by hypoxic-ischemic encephalopathy (48.3%), hypoglycemia (25%), hypocalcemia (21.6%), meconium aspiration syndrome (18.3%), and polycythemia (11.67%).[14]

Carrying out research on the etiological profile of neonatal seizures in Abbottabad is valuable since there is no region based information available and the neonatal health determinants can vary from the rest of the regions. Regional factors like the accessibility of healthcare, perinatal care practices, as well as the prevalence of infectious diseases, can modify the pattern of causes. Creating evidence for this population can facilitate the timely identification of the condition as well as specific interventions, ultimately enhancing neonatal care and lessening the risk of late neurological sequelae. This can also inform the creation of guidelines appropriate for the regional-specific context of neonates.

METHODOLOGY

This descriptive cross-sectional study was conducted over a period of five months, from January to May 2025, in the Neonatal Intensive Care Unit of the Department of Pediatrics, CMH Abbottabad. A total of 110 neonates presenting with seizure activity were included. The sample size was calculated using the WHO sample size calculator, applying a 95% confidence interval, 6% margin of error, and an expected proportion of polycythemia at 11.67% among neonates with seizures.[14]

Inclusion criteria comprised neonates aged 1 to 28 days of either sex who were admitted to the NICU and exhibited seizure activity identified through clinical signs. Seizures were identified as subtle if the neonate displayed repetitive movements such as chewing, blinking, or bicycling of limbs lasting at least 10 seconds. Rhythmic limb jerks occurring at 1–4 jerks per second for 10 seconds or more were labeled as clonic seizures. Sustained muscle tightening lasting 3 seconds or longer was considered tonic seizure activity. Sudden, brief muscular contractions lasting less than 100 milliseconds were classified as myoclonic seizures. Neonates were excluded if they had a history of conditions mimicking seizures such as jitteriness, documented congenital structural anomalies, benign sleep-related myoclonus, metabolic disorders diagnosed during the antenatal period, or major birth-related trauma. Approval was obtained from the institutional ethics committee, and informed consent was taken from parents or guardians.

To investigate the underlying causes, each neonate was assessed through clinical examination and relevant laboratory investigations. Sepsis was diagnosed if the neonate had a fever above 38°C along with laboratory evidence including total leukocyte count above 20%, micro ESR exceeding 55 mm, and gastric aspirate containing more than five polymorphs per high power field. Hypoxic-ischemic encephalopathy was considered when the neonate had an Apgar score of 0 to 3 persisting for more than five minutes along with neurological abnormalities such as reduced muscle tone and generalized convulsions lasting longer than one minute. Hypoglycemia was defined as a plasma glucose concentration below 45 mg/dL, while hypocalcemia was diagnosed if the total serum calcium level was less than 8 mg/dL. Meconium aspiration syndrome was suspected when coarse breath sounds were audible on chest auscultation or if chest X-ray revealed asymmetrical patchy lung opacities with pleural effusion. Polycythemia was identified when venous hematocrit exceeded 65% or hemoglobin levels surpassed 22 g/dL.

All findings were recorded by the researcher in a structured proforma. Data analysis was conducted using IBM SPSS version 26. Categorical variables were summarized as frequencies and percentages. Continuous variables were expressed as mean ± standard deviation or median with interquartile range based on the distribution assessed by the Shapiro-Wilk test. Stratification was done to examine relationships between the causes of seizures and demographical variables. Associations were tested using the chi-square test or Fisher's exact test where appropriate, with p-values ≤ 0.05 considered statistically significant.

RESULTS

The study examined 110 neonates with seizures, revealing a mean age of 9.05±7.42 days and mean weight of 2.88±0.93 kg, with females comprising 57.3% (n=63) and males 42.7% (n=47) of the cohort (Table-I). The majority were term infants (84.5%, n=93), while 11.8% (n=13) were preterm and 3.6% (n=4) were post-term. Regarding parental occupation, 40.9% (n=45) had jobs, 30.0% (n=33) were in business, and 29.1% (n=32) were jobless. Socioeconomic distribution showed 40.9% (n=45) middle class, 30.0% (n=33) wealthy, and 29.1% (n=32) poor families. Rural residence predominated at 60.9% (n=67) versus 39.1% (n=43) urban. Seizure types included subtle seizures as the most common at 48.2% (n=53), followed by clonic 24.5% (n=27), myoclonic 21.8% (n=24), and tonic 5.5% (n=6) (Table-I).

Table I

Patient Demographics

Demographics	Mean ± SD
Age (days)	9.05±7.42
Weight (kg)	2.88±0.93
Gender	
Male n (%)	47 (42.7%)
Female n (%)	63 (57.3%)
Gestational Age	
Preterm n (%)	13 (11.8%)
Term n (%)	93 (84.5%)
Post term n (%)	4 (3.6%)

Parents Profession	
Job n (%)	45 (40.9%)
Jobless n (%)	32 (29.1%)
Business n (%)	33 (30.0%)
Socioeconomic Status	
Poor n (%)	32 (29.1%)
Middle n (%)	45 (40.9%)
Rich n (%)	33 (30.0%)
Residential Status	
Rural n (%)	67 (60.9%)
Urban n (%)	43 (39.1%)
Type of Seizures	
Subtle n (%)	53 (48.2%)
Tonic n (%)	6 (5.5%)
Clonic n (%)	27 (24.5%)
Myoclonic n (%)	24 (21.8%)

Etiological analysis demonstrated sepsis as the leading cause affecting 80.90% (n=89) of patients with 19.10% (n=21) having no sepsis, followed by hypoxic-ischemic encephalopathy in 44.50% (n=49) with 55.50% (n=61) unaffected, meconium aspiration syndrome in 24.50% (n=27) with 75.50% (n=83) unaffected, hypocalcemia in 22.70% (n=25) with 77.30% (n=85) unaffected, hypoglycemia in 17.30% (n=19) with 82.70% (n=91) unaffected, and polycythemia in 14.50% (n=16) with 85.50% (n=94) unaffected (Table-II).

Table II

Frequency of Etiological Factors Among Patients Presenting with Neonatal Seizures

Etiological Factors	Frequency	% age
Sepsis		
Yes	89	80.90%
No	21	19.10%
Total	110	100%
Hypoxic-Ischemic Encephalopathy		
Yes	49	44.50%
No	61	55.50%
Total	110	100%
Meconium Aspiration Syndrome		
Yes	27	24.50%
No	83	75.50%
Total	110	100%
Hypocalcemia		
Yes	25	22.70%
No	85	77.30%
Total	110	100%
Hypoglycemia		
Yes	19	17.30%
No	91	82.70%
Total	110	100%
Polycythemia		
Yes	16	14.50%
No	94	85.50%
Total	110	100%

Demographic associations revealed age-stratified analysis showing sepsis in 64 patients (78.0%) aged ≤10 days versus 25 patients (89.3%) aged >10 days (p=0.268), HIE in 37 patients (45.1%) versus 12 patients (42.9%) respectively (p=0.835), hypoglycemia in 13 patients (15.9%) versus 6 patients (21.4%) respectively (p=0.565), hypocalcemia in 18 patients (22.0%) versus 7 patients (25.0%) respectively (p=0.74), MAS in 21 patients (25.6%) versus 6 patients (21.4%) respectively (p=0.657), and polycythemia in 7 patients (8.5%) versus 9 patients (32.1%) respectively with significant association (p=0.002). Gestational age analysis showed sepsis in 10 preterm patients (76.9%), 76 term patients (81.7%), and 3 post-term patients (75.0%) (p=0.879), HIE in 6 preterm patients (46.2%), 41 term patients (44.1%), and 2 post-term patients (50.0%) (p=1.000), hypoglycemia in 1 preterm patient (7.7%), 18 term patients (19.4%), and 0 post-term patients (0.0%) (p=0.439), hypocalcemia in 2 preterm patients (15.4%), 21 term patients (22.6%), and 2 post-term patients (50.0%) (p=0.344), MAS in 6 preterm patients (46.2%), 21 term patients (22.6%), and 0 post-term patients (0.0%) (p=0.104), and polycythemia in 2 preterm patients (15.4%), 14 term patients (15.1%), and 0 post-term patients (0.0%) (p=0.884). Weight-based analysis demonstrated sepsis in 24 patients ≤2kg (82.8%) versus 65 patients >2kg (80.2%) (p=0.795), HIE in 12 patients ≤2kg (41.4%) versus 37 patients >2kg (45.7%) (p=0.689), hypoglycemia in 3 patients ≤2kg (10.3%) versus 16 patients >2kg (19.8%) (p=0.277), hypocalcemia in 4 patients ≤2kg (13.8%) versus 21 patients >2kg (25.9%) (p=0.208), MAS in 11 patients ≤2kg (37.9%) versus 16 patients >2kg (19.8%) (p=0.051), and polycythemia in 6 patients ≤2kg (20.7%) versus 10 patients >2kg (12.3%) (p=0.274). Seizure type associations showed sepsis in 44 patients with subtle seizures (83.0%), 5 patients with tonic seizures (83.3%), 21 patients with clonic seizures (77.8%), and 19 patients with myoclonic seizures (79.2%) (p=0.938), HIE in 22 patients with subtle seizures (41.5%), 4 patients with tonic seizures (66.7%), 12 patients with clonic seizures (44.4%), and 11 patients with myoclonic seizures (45.8%) (p=0.710), hypoglycemia in 7 patients with subtle seizures (13.2%), 2 patients with tonic seizures (33.3%), 6 patients with clonic seizures (22.2%), and 4 patients with myoclonic seizures (16.7%) (p=0.535), hypocalcemia in 12 patients with subtle seizures (22.6%), 0 patients with tonic seizures (0.0%), 8 patients with clonic seizures (29.6%), and 5 patients with myoclonic seizures (20.8%) (p=0.496), MAS in 14 patients with subtle seizures (26.4%), 1 patient with tonic seizures (16.7%), 5 patients with clonic seizures (18.5%), and 7 patients with myoclonic seizures (29.2%) (p=0.776), and polycythemia in 8 patients with subtle seizures (15.1%), 0 patients with tonic seizures (0.0%), 4 patients with clonic seizures (14.8%), and 4 patients with myoclonic seizures (16.7%) (p=0.829) (Table-III).

Table III

Association of Etiological Factors with Demographic Variables

Demographic Factors		Sepsis	HIE	Hypoglycemia	Hypocalcemia	MAS	Polycythemia
Age (days)	≤10	64 (78.0%)	37 (45.1%)	13 (15.9%)	18 (22.0%)	21 (25.6%)	7 (8.5%)
	>10	25 (89.3%)	12 (42.9%)	6 (21.4%)	7 (25.0%)	6 (21.4%)	9 (32.1%)
p-value		0.268*	0.835	0.565	0.74	0.657	0.002
Gestational Age	Preterm	10 (76.9%)	6 (46.2%)	1 (7.7%)	2 (15.4%)	6 (46.2%)	2 (15.4%)
	Term	76 (81.7%)	41 (44.1%)	18 (19.4%)	21 (22.6%)	21 (22.6%)	14 (15.1%)
	Post-term	3 (75.0%)	2 (50.0%)	0 (0.0%)	2 (50.0%)	0 (0.0%)	0 (0.0%)
p-value		0.879*	1.000*	0.439*	0.344	0.104*	0.884*

Weight (kg)	≤2	24 (82.8%)	12 (41.4%)	3 (10.3%)	4 (13.8%)	11 (37.9%)	6 (20.7%)
	>2	65 (80.2%)	37 (45.7%)	16 (19.8%)	21 (25.9%)	16 (19.8%)	10 (12.3%)
p-value		0.795*	0.689	0.277*	0.208*	0.051	0.274
Type of Seizures	Subtle	44 (83.0%)	22 (41.5%)	7 (13.2%)	12 (22.6%)	14 (26.4%)	8 (15.1%)
	Tonic	5 (83.3%)	4 (66.7%)	2 (33.3%)	0 (0.0%)	1 (16.7%)	0 (0.0%)
	Clonic	21 (77.8%)	12 (44.4%)	6 (22.2%)	8 (29.6%)	5 (18.5%)	4 (14.8%)
	Myoclonic	19 (79.2%)	11 (45.8%)	4 (16.7%)	5 (20.8%)	7 (29.2%)	4 (16.7%)
p-value		0.938*	0.710*	0.535*	0.496*	0.776*	0.829*

***Fischer Exact Test**

DISCUSSION

The present study analyzed the etiologic continuum of neonatal seizures in 110 patients, revealing the most prominent causative factor as sepsis (80.9%), the second most common as hypoxic-ischemic encephalopathy (44.5%), in concordance with the multifactorial pathophysiologic setting of neonatal neurological deficits. Sepsis-induced seizures were extremely common due to the immature blood-brain barrier, allowing the entry of bacterial exotoxins as well as inflammatory mediators across the blood-brain barrier, as well as into the central nervous system, resulting in neuronal hyperexcitability, as well as the initiation of seizures. Sepsis-induced system inflammation further incites cytokine release, as well as metabolism disturbances, as well as cerebral hypoperfusion, further contributing to the causation of seizures. The high prevalence of hypoxic-ischemic encephalopathy further indicates the vulnerability of the neonatal brain to perinatal oxygen inadequacy, whereby impairment of energy causes excitotoxicity, calcium influx, ultimately with ensuing neuronal damage as seizures. Interesting finding that subtle seizures were responsible for nearly half of all the cases (48.2%) is in concordance with the developmental immaturity of the neonatal nervous system, whereby incomplete myelination as well as decreased cortical circuitry make the neonate nervous system less integrated in the propagation of seizures, as compared to older children as well as adults. Metabolic cause dominance of the type of hypocalcemia (22.7%), as well as hypoglycemia (17.3%), further reflects the crucial dependency of the neonatal brain's function upon the precise homeostasis of the metabolisms, whereby glucose is the preferred substrate for energy for maturing neurons, as well as calcium has essential roles in the release of neurotransmitters as well as membrane stability.

Our study results were consistent with several international findings while revealing some notable regional variations. The female predominance in our cohort (57.3%) contrasts with most previous studies where males typically outnumbered females, including Patel S, et al. [15] (56.67% males), Hassan Ali Hassan et al. [16] (54% males), Chesti et al. [17] (60% males), Almuqbil et al. [18] (57.4% males), and Shahzad et al. [19] (68% males). This gender distribution difference may reflect regional demographic patterns or sampling variations, though the clinical significance remains unclear as neonatal seizure susceptibility is not typically gender-dependent.

The gestational age distribution in our study showed a predominance of term infants (84.5%), which aligns closely with Chesti et al. [17] (73.7% term-AGA plus 15% term-SGA), Shahzad et al. [19] (90% term), and Hassan Ali Hassan et al. [16] (60% term), though our preterm percentage (11.8%) was lower than Patel S, et al. [15] (52.2% preterm). This variation likely reflects different referral patterns and inclusion criteria, as tertiary NICUs with higher preterm populations would naturally show different distributions. Regarding seizure types, our findings of subtle seizures being most common (48.2%) strongly correlate with Chesti et al. [17] (57.5% subtle) and Patel S, et al. [15] (44.4% subtle), supporting the established understanding that subtle seizures are frequently underrecognized and represent the most prevalent seizure type in neonates. However, this contrasts with Hassan Ali Hassan et al. [16] where myoclonic seizures predominated (36%), and Shahzad et al. [19] where generalized tonic-clonic seizures were most common (28%). These differences may reflect variations in seizure recognition training, EEG monitoring capabilities, or different population characteristics across study centers.

The etiological profile revealed sepsis as our leading cause (80.90%), which significantly exceeds all comparative studies. While Chesti et al. [17] reported sepsis with meningitis in 18.7% and Shahzad et al. [19] reported septicemia/meningitis in 29%, our substantially higher rate suggests either a different case definition, more sensitive diagnostic criteria, or potentially higher baseline infection rates in our population. This finding warrants careful interpretation as it may reflect regional epidemiological factors, healthcare delivery patterns, or diagnostic practices rather than true biological differences. Hypoxic-ischemic encephalopathy occurred in 44.50% of our patients, which closely matches Hassan Ali Hassan et al. [16] (40% HIE) and Almuqbil et al. [18] (42.6% HIE), but differs from studies where birth asphyxia was the predominant cause, such as Patel S, et al. [15] (41.1%), Chesti et al. [17] (57.5%), and Shahzad et al. [19] (46%). This consistency in HIE rates across geographically diverse populations suggests relatively uniform perinatal care standards and recognition criteria for hypoxic-ischemic injury.

Metabolic causes in our study, including hypocalcemia (22.70%) and hypoglycemia (17.30%), showed considerable variation from other studies. Our hypocalcemia rate was higher than Patel S, et al. [15] (12.2%) and Hassan Ali Hassan et al. [16] (12%), while hypoglycemia rates were comparable to Patel S, et al. [15] (17.8%). These metabolic variations likely reflect differences in maternal nutrition, feeding practices, and early neonatal care protocols across different healthcare systems and socioeconomic contexts. The demographic associations we observed, particularly the significant association between polycythemia and age >10 days (p=0.002), provide unique insights not extensively reported in the comparative literature. Most studies focused on onset timing rather than age-specific etiological associations, making our findings particularly valuable for understanding how

certain conditions manifest temporally in neonatal seizures.

These comparative results highlight regional and institutional factors contributing to the etiological profile of neonatal seizures. Although certain patterns persist in various populations, as in the case of the dominance of subtle seizures as well as the predominance of HIE as the most important cause, the marked difference in the rates of sepsis as well as metabolic disturbances indicates the necessity for regionally specific differential diagnosis as well as therapeutic strategies based on regional epidemiological profiles. Elucidating these differences is essential in the formulation of evidence-based guidelines that remain globally informed as well as locally applicable in the end, enhancing the outcomes for neonates presenting with seizures.

This research has several limitations, which must be appreciated. Due to the fact that it is a single-center, tertiary care hospital study, the findings would not transfer to the rest of the healthcare settings or populations with differential referral pattern and case mix. Also, the moderate size of the sample of 110 neonates, although adequate for descriptive analysis, would limit the power of the study for recognizing smaller relationships as well as rare etiological factors. Due to the fact that it is, per se, a hospital-based study, it is subject to the risk of selection bias towards more severe cases or cases with specific clinical presentations, subrepresenting mild seizure episodes treated at the primary or secondary care levels. Cross-sectional design of the study further limits the causal relationships as well as the long-term prognostic outcomes of the etiological factors observed.

CONCLUSION

Our study has established that the etiological spectrum of neonatal seizures is mostly defined by sepsis as the leading cause, in succession by hypoxic-ischemic encephalopathy, meconium aspiration syndrome, and metabolic disturbances in the form of hypocalcemia and hypoglycemia. Subtle seizures were the most common pattern of seizures, most frequently observed in term babies with the clear predominance of the female sex in the patient material. Strong association of polycythemia with the acute appearance of seizures after the tenth day of life confirms marked temporal patterns in the etiology of neonatal seizures.

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