

ORIGINAL ARTICLE

Frequency of hypoglycemia among neonates presenting with Fits in pediatric ward Bahawal Victoria Hospital Bahawalpur.

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ABSTRACT... Objective: To determine the frequency of hypoglycemia among neonates presenting with clinical seizures. **Study Design:** Cross Sectional study. **Setting:** Department of Pediatrics-II, Bahawal Victoria Hospital, Bahawalpur. **Period:** July 2025 to January 2026. **Methods:** Fifty-nine neonates exhibiting clinical fits were enrolled via consecutive sampling. Cases with known metabolic diseases, congenital malformations, or tetany were excluded. Point of care blood glucose was measured immediately upon seizure recognition. Hypoglycemia was operationally defined as <30 mg/dL in term or <20 mg/dL in preterm infants within 72 hours of life, and <40 mg/dL thereafter. Statistical analysis employed SPSS version 25.0. **Results:** The cohort comprised 57.6% males with mean birth weight 2.8 ± 0.7 kg. Hypoglycemia was documented in 23 participants (39.0%). Preterm neonates demonstrated a higher prevalence (52.9%) compared to term infants (33.3%), though statistically non-significant ($p=0.18$). Hypoglycemic infants experienced significantly greater seizure frequency (3.6 ± 1.4 vs 2.3 ± 1.3 episodes; $p=0.002$) and predominantly exhibited subtle semiology ($p=0.04$). Prolonged hospitalization exceeding seven days correlated significantly with hypoglycemia (47.8% vs 19.4%; $p=0.03$). Mortality was confined to the hypoglycemic subgroup (13.0% vs 0.0%; $p=0.06$). **Conclusion:** Hypoglycemia affects approximately two-fifths of neonates presenting with seizures, significantly associating with elevated seizure frequency, subtle clinical manifestations, and extended hospital stays. Mandatory immediate glucose assessment is crucial for optimizing acute neurological management.

Key words: Blood Glucose, Hypoglycemia, Infant, Newborn, Point of Care Systems, Seizures.

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INTRODUCTION

Neonatal seizures are defined as paroxysmal events characterized by abnormal, excessive, and synchronous neuronal activity in the brain.¹ These are common neurological emergencies, with various underlying etiologies including metabolic disorders, hypoxic-ischemic encephalopathy, and infections. Neonatal seizures are classified into various types, including subtle seizures (such as eye blinking or sucking movements), clonic seizures (rhythmic jerking movements), tonic seizures (muscle stiffness), and myoclonic seizures (sudden, brief muscle jerks).^{2,3}

Neonatal hypoglycemia is the most common form of a metabolic disturbance in newborns, while seizures are the most common symptom of neurological dysfunction. The incidence of neonatal seizure is high: 10-25% in neonatal intensive care units

(NICU). Hypoxic-ischemic encephalopathy (HIE) is found in ~50% of the patients with neonatal hypoglycemia. HIE may potentiate the permanent brain damage caused by hypoglycemia.^{4,5} Delay in therapy often results in poor neurological outcome. Prolonged and repeated episodes of hypoglycemia are associated with poor neurologic outcomes.⁶ Hypoglycemic brain injury is a recognized insult that triggers seizures and is typically associated with both focal and generalized epilepsy.⁷ Hypoglycemia has a significant prevalence in at-risk infants, with that of 47% in large-for-gestational age (LGA) infants, 52% in small-for-gestational age (SGA) infants, 48% in neonates of diabetic mothers, and 54% in late preterm infants. In infants born before 33 weeks, the prevalence of hypoglycemia is nearly 34%.⁸

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A study was conducted in 2003 on 59 neonates to determine the biochemical disturbances in neonatal seizure. Biochemical abnormalities were detected in 29 (49.15%) of cases. Primary metabolic abnormalities occurred in 10(16.94%) cases of neonatal seizures, the most common being hypocalcemia followed by hypoglycemia. Biochemical abnormalities were seen in 19(38.77%) cases of non-metabolic seizure in neonates. Associated metabolic abnormalities were observed more often with Hypoxic-ischemic-encephalopathy (11 out of 19) cases and hypoglycemia was the most common.⁹ Another study was conducted in 2015 to assess the etiology of neonatal seizures. The study found that the most common type of neonatal seizure was subtle 95 (63.33%). Most common cause of neonatal seizures was perinatal asphyxia 80 (53%) followed by metabolic causes 24 (16%). Hypoglycemia was the most common metabolic disorder (4%).¹⁰

Despite the clinical importance of hypoglycemia in neonates, its frequency among neonates presenting with fits has not been well-studied. This study aims to determine the frequency of hypoglycemia among neonates presenting with fits. Understanding the frequency of hypoglycemia in this specific population is crucial for implementing appropriate screening and management strategies. Existing literature primarily focuses on the etiology, clinical presentation, and management of neonatal seizures. And limited studies specifically investigate the frequency of hypoglycemia among neonates presenting with fits. This study aims to bridge this gap by providing valuable insights into the prevalence of hypoglycemia in the local population.

OBJECTIVE

To determine the frequency of hypoglycemia among neonates presenting with fits in the pediatric ward of Bahawal Victoria Hospital, Bahawalpur.

METHODS

This cross sectional study was conducted at the Department of Pediatrics-II, Bahawal Victoria Hospital, Bahawalpur from July 2025 to January 2026 after taking approval from Institutional Ethical Review Committee (Reference No. 2615 /DME/ QAMC, BAHAWALPUR Dated: 21- 06- 2024).

Sample size of 59 neonates was determined at 95% confidence level and 4%^{9,10} anticipated frequency of hypoglycemia. Participants were enrolled using non-probability consecutive sampling. The study included neonates of either sex presenting with clinical seizures, whether at admission or developing during hospitalization, while excluding those with known metabolic disorders, congenital anomalies, or tetanic spasms. For the purpose of this study, a neonate was operationally defined as an infant within 28 days of life for term births and up to 44 weeks post-conception for preterm infants.

Following ethical approval from the institutional review board data collection commenced after obtaining written informed consent from parents or legal guardian and ensuring strict participant confidentiality. Clinical histories were recorded using a standardized proforma, with particular emphasis on the onset, duration, frequency, and semiology of seizures. Random blood glucose was measured urgently using calibrated glucometer. Hypoglycemia was operationally defined as a blood glucose level <30 mg/dL in term infants and <20 mg/dL in preterm infants within the first 72 hours of life, or <40 mg/dL thereafter. Data were analyzed using SPSS version 25.0. Categorical variables, including gender, seizure type, hypoglycemia status, and clinical outcomes, were summarized as frequencies and percentages. Continuous variables, such as age, weight, seizure duration and frequency, blood glucose levels, and time from seizure onset to glucose measurement, were expressed as mean $\pm$ standard deviation. Between-group comparisons for quantitative and qualitative data were performed using the independent samples t-test and Chi-square test, respectively, with statistical significance set at $p \leq 0.05$.

RESULTS

A total of 59 neonates presenting with clinical seizures were enrolled. The mean postnatal age at presentation was 5.2 ± 3.8 days, and mean birth weight was 2.8 ± 0.7 kg. Male infants comprised 57.6% (n=34) of the cohort. Term neonates accounted for 71.2% (n=42), while 28.8% (n=17) were born preterm. Seizures were evident at admission in 64.4% (n=38) of cases; the remainder developed during hospitalization. The most frequent

seizure semiology was generalized tonic-clonic activity (42.4%), followed by focal motor seizures (37.3%) and subtle manifestations such as eye deviation or apneic episodes (20.3%) (Table-I).

Mean duration of individual seizure episodes was 4.2 ± 2.1 minutes, with a median of 3 episodes (IQR: 2–4) recorded within the initial 24 hours. Random blood glucose was measured at mean interval of 28 ± 15 minutes following seizure recognition. Overall, hypoglycemia meeting operational criteria was identified in 23 neonates (39.0%) (Table-II).

Hypoglycemia was more frequent among preterm infants (52.9%) compared to term counterparts (33.3%), though this difference did not reach statistical significance ($p=0.18$). Neonates with hypoglycemia demonstrated a significantly greater frequency of seizures within the first day (mean 3.6 ± 1.4 vs. 2.3 ± 1.3 ; $p=0.002$) and were more likely to exhibit subtle seizure types ($p=0.04$). Mean blood glucose concentration was markedly lower in the hypoglycemic subgroup (26.4 ± 5.2 mg/dL) versus the normoglycemic group (62.1 ± 12.3 mg/dL; $p<0.001$) (Table-III).

Outcomes favored the normoglycemic group, 83.3% achieved seizure control with standard therapy and were discharged without neurological deficits, compared to 65.2% in the hypoglycemic group ($p=0.09$). However, prolonged hospitalization (>7 days) was significantly associated with

hypoglycemia (47.8% vs. 19.4%; $p=0.03$). Three neonates (5.1%) expired during the study period; all had documented hypoglycemia and refractory seizures. No adverse events related to glucose monitoring were observed (Table-IV). Seizure frequency exceeding three episodes within 24 hours ($p=0.01$) and presentation with subtle seizure semiology ($p=0.04$) were significantly associated with hypoglycemia. Preterm birth and low birth weight (<2.5 kg) showed non-significant trends toward higher hypoglycemia prevalence ($p=0.18$ and $p=0.12$, respectively) (Table-V).

DISCUSSION

The present study identified hypoglycemia frequency of 39.0% among neonates presenting with clinical seizures, underscoring the substantial metabolic contribution to early neurological dysfunction in this population. This proportion aligns with regional data indicating that nearly two-fifths of neonatal seizure group exhibit concurrent glucose derangements, though it remains higher than historical estimates from tertiary centers in South Asia.¹¹ The observed trend toward greater hypoglycemia burden in preterm infants (52.9% versus 33.3% in term neonates) did not achieve statistical significance, likely reflecting the modest sample size; nonetheless, it corroborates established physiological vulnerabilities related to depleted glycogen stores, immature gluconeogenesis, and heightened cerebral metabolic demand in premature neonates.¹²

TABLE-I

Baseline demographic and clinical characteristics of neonates with seizures (n=59)

Variable	Category	Frequency (n)	Percentage (%)	Mean $\pm$ SD
Postnatal age at presentation (days)	—	—	—	5.2 ± 3.8
Birth weight (kg)	—	—	—	2.8 ± 0.7
Gender	Male	34	57.6	—
	Female	25	42.4	—
Gestational age	Term (≥ 37 weeks)	42	71.2	—
	Preterm (<37 weeks)	17	28.8	—
Presentation timing	At admission	38	64.4	—
	During hospitalization	21	35.6	—
Seizure semiology	Generalized tonic-clonic	25	42.4	—
	Focal motor	22	37.3	—
	Subtle (apnea, eye deviation)	12	20.3	—

TABLE-II

Seizure characteristics and timing of glucose assessment (n=59)

Variable	Category	Frequency (n)	Percentage (%)	Mean $\pm$ SD
Time of first seizure onset	<24 hours of life	28	47.5	—
	24–72 hours	19	32.2	—
	>72 hours	12	20.3	—
Seizure duration per episode (minutes)	—	—	—	4.2 $\pm$ 2.1
Number of seizures in first 24h	—	—	—	2.8 $\pm$ 1.5
Time from seizure onset to glucose check (minutes)	—	—	—	28 $\pm$ 15
Random blood glucose (mg/dL)	Overall	—	—	48.3 $\pm$ 18.6
	Hypoglycemic subgroup (n=23)	—	—	26.4 $\pm$ 5.2
	Normoglycemic subgroup (n=36)	—	—	62.1 $\pm$ 12.3

TABLE-III

Frequency of hypoglycemia stratified by gestational age and timing of assessment

Stratification Variable	Subgroup	Total n	Hypoglycemia n (%)	P-Values
Gestational age	Term infants	42	14 (33.3)	0.18
	Preterm infants	17	9 (52.9)	
Postnatal age at assessment	$\leq$ 72 hours	47	18 (38.3)	0.72
	>72 hours	12	5 (41.7)	
Seizure onset timing	<24 hours	28	13 (46.4)	0.21
	$\geq$ 24 hours	31	10 (32.3)	

TABLE-IV

Clinical outcomes according to hypoglycemia status

Outcome Parameter	Hypoglycemia Present (n=23)	Hypoglycemia Absent (n=36)	P-Values
Seizure control with first-line therapy	18 (78.3)	33 (91.7)	0.14
Prolonged hospitalization (>7 days)	11 (47.8)	7 (19.4)	0.03
Neurological sequelae at discharge	4 (17.4)	2 (5.6)	0.16
Mortality	3 (13.0)	0 (0.0)	0.06
Favorable composite outcome	15 (65.2)	30 (83.3)	0.09

TABLE-V

Bivariate analysis of factors associated with hypoglycemia in neonates with seizures

Factor	Category	Hypoglycemia n (%)	Normoglycemia n (%)	P-Values
Preterm birth	Yes	9 (52.9)	8 (47.1)	0.18
	No	14 (33.3)	28 (66.7)	
Seizure frequency >3/24h	Yes	14 (60.9)	10 (27.8)	0.01
	No	9 (39.1)	26 (72.2)	
Subtle seizure type	Yes	8 (66.7)	4 (33.3)	0.04
	No	15 (31.9)	32 (68.1)	
Birth weight <2.5 kg	Yes	10 (55.6)	8 (44.4)	0.12
	No	13 (31.7)	28 (68.3)	

Clinical semiology demonstrated a distinct pattern in the hypoglycemic subgroup, with these infants exhibiting a significantly higher seizure burden (mean 3.6 versus 2.3 episodes within 24 hours; $p=0.002$) and a predominance of subtle manifestations such as ocular deviation and apneic spells ($p=0.04$). Such findings reinforce the notion that metabolic seizures frequently present with atypical or fragmented motor activity rather than overt clonic or tonic patterns, potentially delaying recognition in the absence of routine biochemical screening.¹³

The marked disparity in random blood glucose concentrations between hypoglycemic and normoglycemic cohorts (26.4 ± 5.2 mg/dL versus 62.1 ± 12.3 mg/dL; $p<0.001$) further highlights the critical window during which glucose deprivation triggers neuronal hyperexcitability and subsequent clinical manifestations.¹⁴ Prompt point of care assessment, as implemented in this protocol, remains indispensable for timely intervention and mitigates the risk of prolonged cerebral energy failure.¹⁵

Clinical outcomes were notably less favorable in neonates with documented hypoglycemia. Prolonged hospitalization exceeding seven days was significantly more frequent in this group (47.8% versus 19.4%; $p=0.03$), reflecting the complexity of managing refractory seizures, correcting metabolic instability, and monitoring for secondary complications. Although mortality (13.0% versus 0.0%; $p=0.06$) and unfavorable composite outcomes (34.8% versus 16.7%; $p=0.09$) did not reach conventional statistical thresholds, the consistent directional trend aligns with contemporary evidence linking sustained neonatal hypoglycemia to extended neurocritical care requirements and increased vulnerability to long-term neurological impairment.¹⁶ The pathophysiological cascade involving excitotoxic neurotransmitter release, oxidative stress, and apoptotic neuronal loss provides a plausible mechanism for the observed clinical trajectory, particularly when glucose correction is delayed beyond the initial seizure episode.¹⁷ Recent multicenter analyses emphasize that seizure semiology, particularly subtle or multifocal patterns, serves as an early clinical predictor of underlying metabolic etiologies, advocating for

integrated neuro-metabolic evaluation protocols in neonatal wards.¹⁸ Long-term neurodevelopmental surveillance remains essential, as emerging South Asian cohorts demonstrate that even transient neonatal hypoglycemia may correlate with subtle cognitive and motor deficits detectable at 12 months of age.¹⁹ Quality improvement initiatives targeting standardized dextrose administration pathways and continuous glucose monitoring in high-risk preterm and small-for-gestational-age infants have shown promise in reducing metabolic-related seizure recurrence and improving acute stabilization metrics.²⁰ Consensus guidelines published in recent years further advocate for age-specific glucose thresholds and immediate therapeutic intervention to prevent irreversible cortical injury.²¹

The strengths of this investigation include its cross sectional design, strict operational definitions for both gestational age categories and hypoglycemia thresholds and the implementation of rapid point of care glucose testing within 30 minutes of seizure recognition. The single center setting and small sample size ($n=59$) may limit the generalizability of findings. The cross sectional design limits causal inference regarding hypoglycemia as a primary seizure trigger versus a secondary metabolic consequence. Future research should prioritize multicenter prospective design with larger sample size to validate the association between subtle seizure semiology and metabolic derangements. Standardized institutional protocols mandating immediate glucose screening in all seizing neonates, coupled with structured neuroimaging and metabolic panels, are recommended to refine diagnostic accuracy.

CONCLUSION

Hypoglycemia affects nearly two-fifths of neonates presenting with clinical seizures and is significantly associated with higher seizure frequency, subtle semiology and prolonged hospitalization.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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