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Original Article

STUDY OF THE CLINICO-ETIOLOGICAL PROFILE AND IMMEDIATE OUTCOME OF NEONATAL SEIZURES

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ABSTRACT

Background: Neonatal seizures are a common neurological emergency and are often associated with significant morbidity and mortality. The present study aimed to determine the distribution of clinical seizure types, the spectrum of causative etiologies, and the relationship between etiology and the timing of seizure onset in term neonates.

Methods: This prospective observational study was conducted in the NICU of a tertiary care hospital over 24 months (June 2023–June 2025). A total of 238 term neonates presenting with seizures within the first 28 days of life were enrolled after informed consent. Detailed clinical evaluation was done and immediate outcomes were assessed in terms of survival, treatment response, and duration of hospital stay.

Results: Most seizures occurred within 24–72 hours of life (50%), followed by >72 hours (25.63%) and <24 hours (24.36%). Subtle seizures were the most common type (56.72%). Metabolic disturbances were the leading etiology (40.75%), followed by sepsis/meningitis (33.19%) and hypoxic-ischemic encephalopathy (24.36%). Hypoglycemia (31.9%) and hypocalcemia (29.9%) were the most frequent biochemical abnormalities. HIE predominated in early-onset seizures (<24 hours), while metabolic and infectious causes were more common after 24 hours. Abnormal neurosonogram findings were observed in 21.85% of neonates, most commonly diffuse parenchymal echoes. Escherichia coli was the most frequently isolated organism in culture-positive cases (36.7%). Phenobarbitone monotherapy was effective in 71% of neonates. Overall survival was 90.75%, with a mortality rate of 9.24%.

Conclusion: Metabolic abnormalities and infections were the predominant causes of neonatal seizures. Early recognition, timely correction of biochemical derangements, and etiology-specific management are crucial to improving outcomes and reducing mortality.

Keywords: *Neonatal seizures, Etiology, Metabolic disturbances, Hypoglycemia, Sepsis, Term neonates.*

Introduction

Neonatal seizures are the most common neurological emergency in the neonatal period and serve as an important indicator of underlying cerebral dysfunction[1]. They are defined as sudden alterations in neurological function—sensory, motor, or autonomic—resulting from abnormal synchronous cortical neuronal discharge. The incidence ranges from 1.1–4 per 1,000 live births and is higher in preterm and low-birth-weight neonates due to neurological immaturity and increased susceptibility to perinatal complications[2]. Neonatal seizures occur within the first 28 days of life and may present as clinical, subclinical, or electrographic-only events[3]. Unlike seizures in older children, neonatal seizures are often subtle and non-convulsive, making clinical recognition challenging[4].

Identification of the underlying etiology is essential for appropriate management and prognostication. Hypoxic-ischemic encephalopathy (HIE) is the most common cause, particularly in term neonates, and typically presents within the first 24 hours of life[4,5]. Other significant causes include metabolic disturbances such as hypoglycemia, hypocalcemia, hypomagnesemia, and hyponatremia[6]. The timing of seizure onset often provides diagnostic clues: seizures within 24 hours are frequently associated with HIE or vascular events; those occurring between 24–72 hours are commonly related to metabolic abnormalities or infections; and seizures after 72 hours may be due to structural brain malformations, inborn errors of metabolism, or viral infections such as herpes simplex virus[7,8].

Clinically, neonatal seizures are classified as subtle, tonic, clonic, or myoclonic according to Volpe's classification, with subtle seizures being the most frequent[9,10]. Due to their often subtle presentation, electroencephalographic (EEG) monitoring plays a crucial role in accurate diagnosis[11]. Immediate outcomes depend on etiology, timing, seizure burden, and response to treatment. While seizures secondary to transient metabolic disturbances generally have favorable outcomes, those associated with HIE or structural abnormalities carry higher risks of morbidity and adverse neurodevelopmental sequelae[12]. Despite advancements in neonatal care, neonatal seizures continue to pose diagnostic and therapeutic challenges, particularly in low- and middle-income countries where regional data remain limited[5,13].

The present study was undertaken to evaluate the clinical profile, etiological spectrum, and immediate outcomes of neonatal seizures in term neonates.

MATERIALS AND METHODS

This prospective observational study was conducted in the Neonatal Intensive Care Unit (NICU) of the Department of Pediatrics at a tertiary care hospital over a period of 24 months (June 2023–June 2025), after obtaining approval from the Institutional Ethics Committee. A total of 238 term neonates presenting with seizures within the first 28 days of life were enrolled after written informed consent from parents or legal guardians.

Preterm neonates, very low birth weight infants, neonates aged more than 28 days, those with inborn errors of metabolism, major congenital malformations, prior exposure to antiepileptic drugs, recurrent seizures, or lack of consent were excluded. A detailed clinical evaluation was performed using a structured Case Record Form. Seizure characteristics, including timing of onset (<24 hours, 24–72 hours, >72 hours) and type (subtle, clonic, tonic, myoclonic, multifocal), were documented. Antenatal, perinatal, and birth histories were recorded. Relevant investigations included complete blood count, CRP, blood glucose, serum electrolytes (calcium, magnesium), CSF analysis, neurosonography, EEG, CT scan (if indicated), and metabolic screening. Immediate outcomes were assessed in terms of survival, response to antiepileptic therapy, and duration of hospital stay.

Data were entered in Microsoft Excel and analyzed using SPSS version 22.0. Continuous variables were expressed as mean ± SD and categorical variables as frequencies and percentages.

OBSERVATIONS AND RESULTS

Table 1: Perinatal and Birth Characteristics of Term Neonates with Seizures (n = 238)

Variables		No. of patients	Percentage (%)
Mode of Delivery	Normal vaginal	131	55.0%
	LSCS	99	41.6%
	Forceps	08	3.4%
Baby Cried Soon After Birth	Yes	180	75.6%
	No	58	24.4%
Timing of crying of neonates	Cried immediately after birth	180	75.63%
	After stimulation & suction	20	8.40%
	Resuscitation with BMV	18	7.56%
	Intubation with ET tube	20	8.40%
Apgar Score at 1 min	0–3	20	8.40%
	4–6	38	15.96%
	7–10	180	75.63%
Apgar Score at 5 min	0–3	20	8.40%
	4–6	36	15.12%
	7–10	182	76.47%
Gender of babies	Male	143	60.1%
	Female	95	39.9%
Birth Weight	2.5–3.5 kg	205	86.1%
	>3.5 kg	33	13.9%

Table 2: Clinical Profile of Seizures in Term Neonates (n = 238)

Variables		No. of patients	Percentage (%)
Time of Onset of Seizures	<24 hours	58	24.36%
	24–72 hours	119	50.00%
	>72 hours	61	25.63%
Types of Seizures	Subtle	135	56.72%
	Focal Clonic	47	19.74%
	Multifocal	26	10.92%
	Tonic	28	11.76%
	Myoclonic	02	0.84%
Etiological Profile of Seizures	HIE	58	24.36%
	Metabolic Disturbances	97	40.75%
	Sepsis / Meningitis	79	33.19%
	Intracranial Hemorrhage	02	0.84%
	Unknown	02	0.84%

Table 3: Etiology vs. Time of Onset of Seizures

Etiology / Onset Time	<24 hrs	24–72 hrs	>72 hrs	Total
HIE	32	17	09	58
Metabolic	13	49	35	97
Sepsis / Meningitis	12	51	16	79
Intracranial Hemorrhage	01	01	00	02
Unknow	00	01	01	02
Total	58	119	61	238

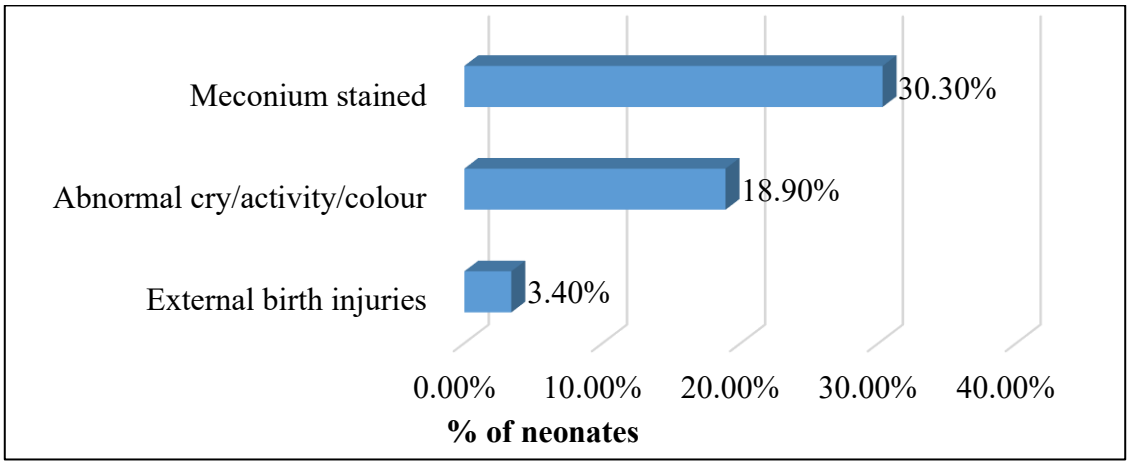

Figure 1: General Examination in Term Neonates with Seizures

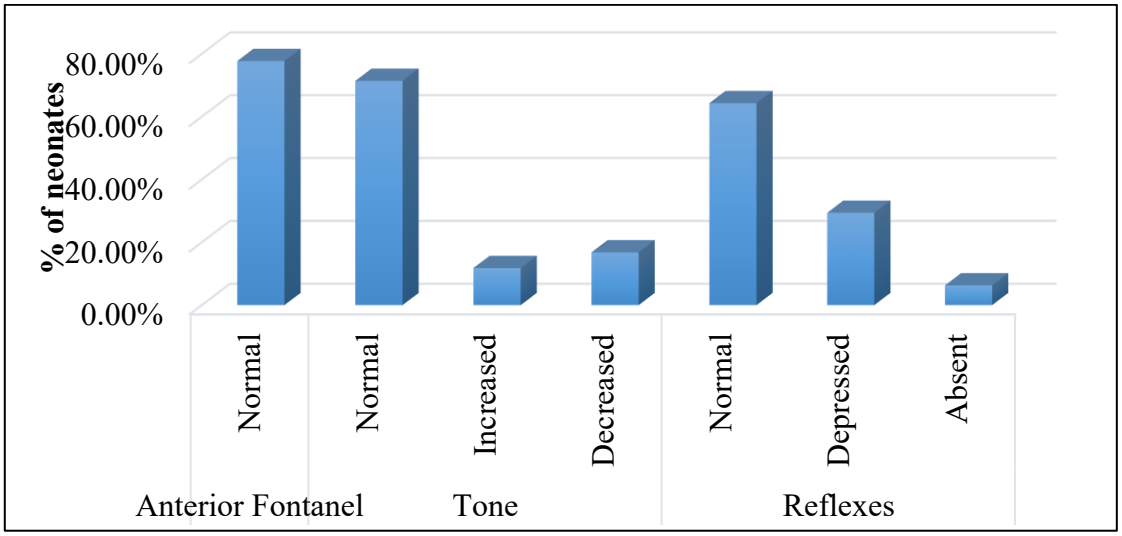

Figure 2: Central Nervous System (CNS) Examination Findings

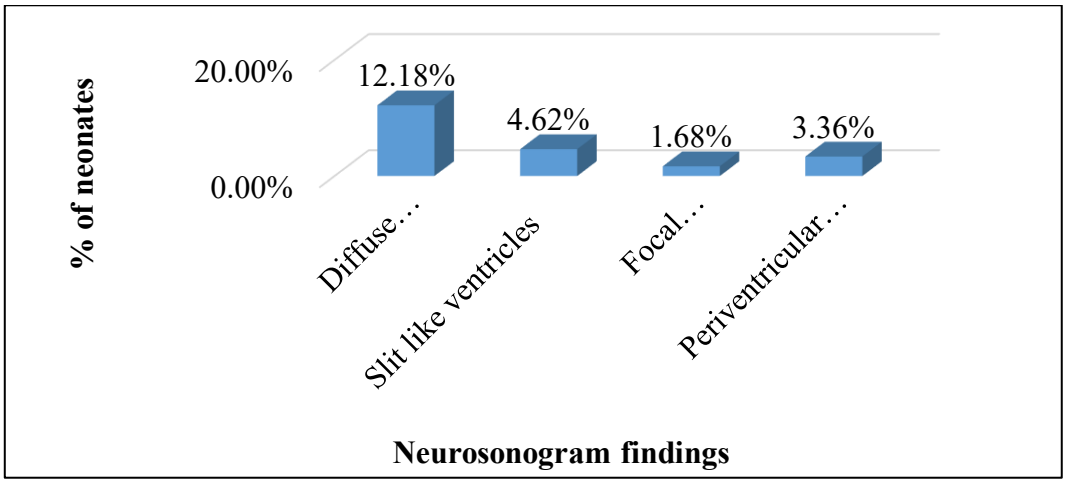

Figure 3: Study of neurosonogram abnormality in neonates with HIE

Table 4: Laboratory Investigations

Investigation		Mean ± SD	Reference Range (Term Neonates)
Hemoglobin (Hb)		15.8 ± 2.2 g/dL	14–20 g/dL
Packed Cell Volume (PCV)		46.5 ± 4.8 %	40–60%
Differential Count (DC)	Neutrophils	58 ± 9	50–60%
	Lymphocytes	35 ± 7	30–40%
Platelets		245 ± 62 x10⁹/L	150–450 x10⁹/L
C-Reactive Protein (CRP)		14.6 ± 8.3 mg/L	Often elevated in infections/seizures
Blood Glucose		68.2 ± 18.6 mg/dL	45–125 mg/dL
Serum Calcium		7.3 ± 0.7 mg/dL	8–10.5 mg/dL
Serum Magnesium		1.85 ± 0.25 mg/dL	1.6–2.3 mg/dL

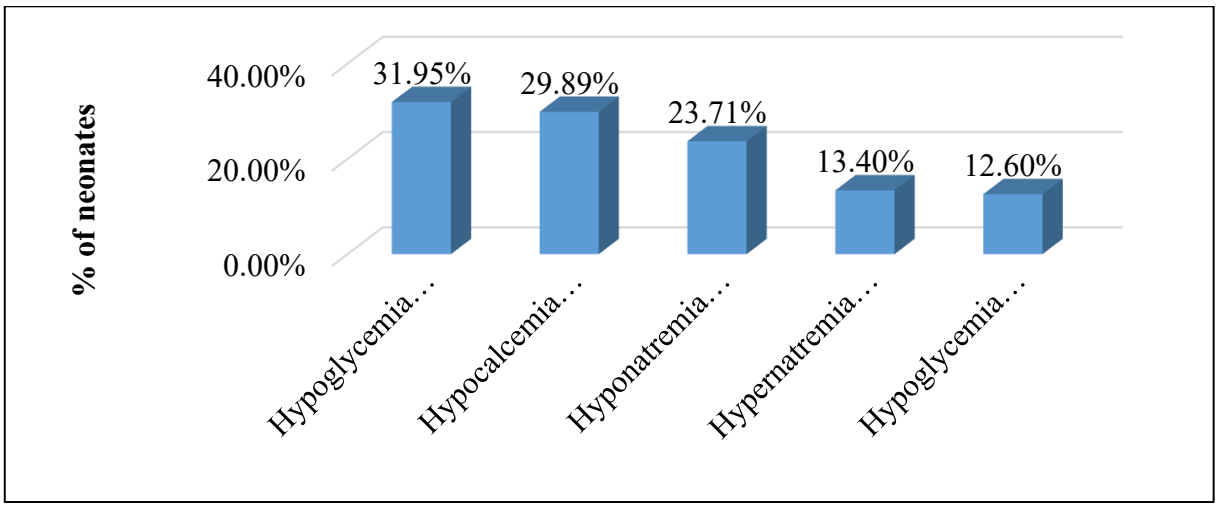

Figure 4: Various biochemical abnormalities in study population (n=97)

Table 5: Distribution of various microorganisms implicated in neonatal seizures

Microorganisms	No. of patients	Percentages
E. coli	29	36.70%
Klebsiella	25	31.64%
S.aureus	18	22.78%
Group B streptococcus	07	8.86%
Total	79	100.0%

Table 6: Response to AED/Treatment

AED/Treatment	No. of patients	Percentages
Phenobarbitone	169	71.00%

Pheno + Phenyt	47	19.74%
Pheno + Phenyt + Levit	08	3.36%
Dextrose + anticonvulsant	07	2.94%
Calcium	30	12.60%
Total	238	100.0%

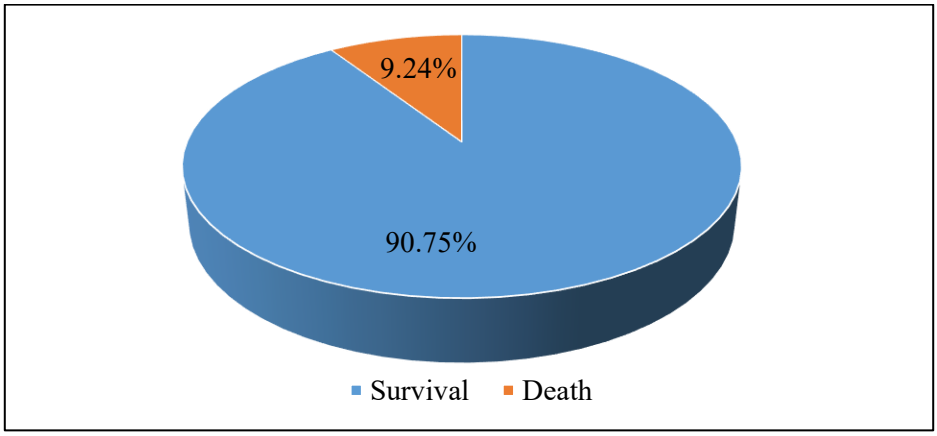

Figure 5: Immediate Outcome of Neonatal Seizures

A total of 238 term neonates with seizures were included in the study. Normal vaginal delivery was the most common mode of delivery (55.0%), followed by lower segment cesarean section (41.6%). Nearly one-fourth (24.4%) required resuscitation at birth. Male neonates predominated (60.1%), and most had a birth weight between 2.5–3.5 kg (86.1%).

Seizures most frequently occurred between 24–72 hours of life (50.0%), followed by >72 hours (25.63%) and <24 hours (24.36%). Subtle seizures were the predominant type (56.72%), followed by focal clonic (19.74%), tonic (11.76%), and multifocal seizures (10.92%); myoclonic seizures were rare (0.84%).

Metabolic disturbances were the leading etiology (40.75%), followed by sepsis/meningitis (33.19%) and hypoxic-ischemic encephalopathy (24.36%). HIE was the major cause of seizures within the first 24 hours, whereas metabolic and infectious causes predominated after 24 hours.

Abnormal neurosonogram findings were observed in 21.85% of neonates, most commonly diffuse parenchymal echoes. Laboratory evaluation revealed elevated CRP levels and biochemical abnormalities suggestive of metabolic derangements. Among metabolic causes, hypoglycemia (31.95%) and hypocalcemia (29.89%) were most frequent.

Among culture-positive cases, *Escherichia coli* (36.70%) was the most commonly isolated organism. Phenobarbitone monotherapy controlled seizures in 71.0% of neonates. Overall survival was 90.75%, with a mortality rate of 9.24%. Most neonates required hospitalization for 11–15 days.

DISCUSSION

Neonatal seizures remain the most frequent neurological emergency in the neonatal period and are important indicators of underlying cerebral dysfunction[1–4]. In the present study, normal vaginal delivery was the most common mode of delivery, and nearly one-fourth of neonates required resuscitation at birth, suggesting a significant contribution of perinatal asphyxia. Similar findings have been reported in previous studies[14–16]. Male predominance observed in our study is also consistent with earlier reports[14–17].

Most seizures occurred within the first 72 hours of life, particularly between 24–72 hours, emphasizing the vulnerability of the early neonatal period. Subtle seizures were the most common type, reflecting the known difficulty in clinical recognition of neonatal seizures[9,10]. Metabolic disturbances emerged as the leading etiology, followed by sepsis/meningitis and hypoxic-ischemic encephalopathy (HIE), comparable to findings reported by Chaudhary et al[14], Paul et al[15], Patel and Mehta[16], and Nair et al[17]. HIE was the predominant cause of seizures within the first 24 hours, while metabolic and infectious causes were more common after 24 hours, in agreement with other studies[18,19].

Clinical examination revealed abnormalities in tone and reflexes in a significant proportion of neonates, indicating varying degrees of neurological dysfunction. Similar observations have been documented by Soul et al[20], Tekgul et al[21], Kumar et al[22], and Sahana et al[23]. Abnormal neurosonogram findings in approximately one-fifth of neonates further supported structural or hypoxic brain injury, consistent with previous literature[17,22].

Laboratory findings highlighted the major role of metabolic abnormalities. Hypoglycemia and hypocalcemia were the most common biochemical disturbances, aligning with earlier studies[14],[16],[22],[25]. Elevated CRP levels supported an infectious etiology in a substantial subset, similar to findings by Bhatt et al[24]. Among culture-positive cases, *E. coli* was the most common organism isolated, consistent with previous reports[25].

Phenobarbitone was effective as first-line therapy in the majority of neonates, confirming its continued role as the drug of choice for neonatal seizures[8],[26]. Combination therapy was required in refractory cases, indicating higher seizure burden or severe underlying pathology. The overall survival rate of 90.75% in the present study is comparable with earlier studies[14],[17], though variations in mortality across studies may reflect differences in case severity and availability of neonatal intensive care facilities[16].

Despite improvements in neonatal care, neonatal seizures remain associated with significant morbidity and mortality. Early identification of etiology, prompt correction of metabolic derangements, and timely initiation of appropriate antiepileptic therapy are crucial to improving immediate outcomes and preventing long-term neurological sequelae[11–13].

CONCLUSION

In conclusion, present study demonstrates that metabolic abnormalities, particularly hypoglycemia and hypocalcemia, were the most common causes of neonatal seizures, followed by sepsis or meningitis. Seizures most frequently occurred between 24–72 hours of life, showed a male predominance, and were predominantly subtle in nature, emphasizing the need for careful clinical observation. E. coli was the most commonly isolated pathogen in culture-positive cases, and neurosonography proved to be a valuable tool in assessing neurological involvement and predicting outcomes. Phenobarbitone monotherapy was effective in most neonates, though some required combination therapy or correction of underlying metabolic disturbances. Despite advances in neonatal care, seizures were associated with significant morbidity, prolonged hospitalization, and a mortality rate of 9.2%. Early detection, prompt correction of biochemical abnormalities, and etiology-specific management are essential to improve outcomes and reduce long-term neurological sequelae, highlighting the need for continuous video-EEG monitoring and larger, long-term studies for more definitive conclusions.

REFERENCES

1. Cloherty And Stark`s Manual Of Neonatal Care, 2017:812-818.
2. Pisani F, Piccolo B, Cantalupo G, et al. Neonatal seizures and postneonatal epilepsy: a 7-y follow-up study. Pediatr Res. 2012;72(2):186–193.
3. Shellhaas RA, Clancy RR. Characterization of neonatal seizures by conventional EEG and single-channel EEG. Clin Neurophysiol. 2007 Oct;118(10):2156-61.
4. Jensen FE. Neonatal seizures: an update on mechanisms and management. Clin Perinatol. 2009 Dec;36(4):881-900.
5. Kim EH, Shin J, Lee BK. Neonatal seizures: diagnostic updates based on new definition and classification. Clin Exp Pediatr. 2022 Aug;65(8):387-397.
6. Almannai M, Al Mahmoud RA, Mekki M, El-Hattab AW. Metabolic Seizures. Front Neurol. 2021 Jul 6;12:640371.
7. Spagnoli C, Pisani F. Acute symptomatic seizures in newborns: a narrative review. Acta Epileptol. 2024;6:5.
8. Rennie JM, Boylan GB. Neonatal seizures and their treatment. Curr Opin Neurol. 2003 Apr;16(2):177-81.
9. Chesti MS, Shahzad N, Chaman S, Gazala S. Clinical profile, etiology, type and outcome of neonatal seizures: a hospital-based study. Int J Contemp Pediatr 2022;9:104-8.
10. Panayiotopoulos CP. The Epilepsies: Seizures, Syndromes and Management. Oxfordshire (UK): Bladon Medical Publishing; 2005. Chapter 5, Neonatal Seizures and Neonatal Syndromes. Available from: https://www.ncbi.nlm.nih.gov/books/NBK2599/
11. Spenard S, Ivan Salazar Cerda C, Çizmeci MN. Neonatal Seizures in Low- and Middle-Income Countries: A Review of the Literature and Recommendations for the Management. Turk Arch Pediatr. 2024 Jan;59(1):13-22.
12. McBride MC, Laroia N, Guillet R. Electrographic seizures in neonates correlate with poor neurodevelopmental outcome. Neurology. 2000 Aug 22;55(4):506-13.
13. Vegda H, Krishnan V, Variane G, Bagayi V, Ivain P, Pressler RM. Neonatal Seizures-Perspective in Low-and Middle-Income Countries. Indian J Pediatr. 2022 Mar;89(3):245-253.
14. Chaudhary N, Paudel A, Chaudhary D, Gupta BK. Clinico-Etiological Profile and Outcome of Neonatal Seizures - A Hospital-Based Cross-Sectional Study in Western Nepal. J Univ Coll Med Sci. 2023;11(1):2–6.
15. Paul V, Kumar R, Gupta NP. Clinico-Etiological Profile of Neonatal Seizures in Term Neonates. Int J Pharm Clin Res. 2024;16(4):1436–1439.
16. Patel S, Mehta N. Incidence of Neonatal Seizures and its Clinico-Etiological Profile in a Tertiary Care Hospital in Western India. Natl J Med Res. 2023;13(1):7-12.
17. Nair BK, Sharma J, Chaudhary S. Clinicoetiological profile of neonatal seizure in a newborn care unit of a tertiary care teaching hospital in Northern India. J Clin Neonatol 2020;9:27-31.
18. Yadav S, Agrawa lP, Sharma VK, Rekha, Yadav SL. Study of etiological factors and immediate outcomes of neonatal seizure among preterm and term neonate in a tertiary health centre of northern India. Int J Contemp Pediatr 2023; 10:1406-11.

19. Shah B, Gosai D, Singh A. A Study of Clinico-Etiological Profile and Early Outcome of Neonatal Seizure. Ann. Int. Med. Den. Res. 2018; 4(5):PE07-PE10.
20. Soul JS. Acute symptomatic seizures in term neonates: Etiologies and treatments. Semin Fetal Neonatal Med 2018; 23:183-90.
21. Tekgul H, Gauvreau K, Soul J, et al. The current etiologic profile and neurodevelopmental outcome of seizures in term newborn infants. Pediatrics 2006;117:1270 –1280.
22. Kumar R, Kumari N, Singh BK, Ansari MA. Clinico-etiological profile of neonatal seizures in a tertiary care hospital of Northern India: a prospective cohort study. International Journal of Health and Clinical Research, 2021;4(11):259-264.
23. Sahana G, Anjaiah B. Clinical profile of neonatal seizures. Int J Med Applied Sci. 2014;3(1):21-7.
24. Bhatt S, Raju N, Phanse S, Patel SV, Madan G, Mehta S, et al. Clinico-etiological and EEG profile of neonatal seizures. Ind J Clin Pract. 2013;24(1):69-75.
25. Sood A, Grover N, Sharma R. Biochemical abnormalities in neonatal seizures. Indian Journal of Paed. 2003;70(3):221-4.
26. Venkatesh G, V Prakash, Sajjid M, Elango. Etiological and Clinical Profile of Seizures in Late Preterm and Term Neonates - A Retrospective Study in an Intramural Tertiary Care Centre. Asian J. Clin. Pediatr. Neonatol.2020;8(1):69-73.
27. Kulkarni SK, Shukla DA. Study of clinico-etiological profile of neonatal seizures in term neonates at a tertiary care hospital. MedPulse International Journal of Pediatrics. August 2021; 19(2): 38-41.