Electroencephalographic profile and its association with seizure recurrence in children with a first unprovoked seizure: a prospective observational study from Eastern India

Sourav Mandal, Dr Arijit Chattopadhyay, Dr Kaushik Maulik, Dr Soma Mandal,

Dr Viswajeet Pratap
Apollo Multi-speciality Hospital, Kolkata
Corresponding
Sourav Mandal
Apollo Multi-speciality Hospital, Kolkata
souravich1@gmail.com

Abstract
Background: A first unprovoked seizure is a common neurological presentation in childhood, and the electroencephalogram (EEG) is recommended as a key investigation in its evaluation. Data on the EEG profile of such children from Eastern India and its relationship with early seizure recurrence remain limited. This study aimed to evaluate the seizure semiology and EEG findings in neurologically normal children presenting with a first unprovoked seizure and to determine their association with recurrence within three months.
Methods: This single-centre, prospective, observational study enrolled 176 neurologically and developmentally normal children aged 1–18 years presenting with a first unprovoked seizure to a tertiary care hospital in Kolkata between May 2022 and April 2023. All children underwent EEG within 24 hours of the seizure and magnetic resonance imaging (MRI) of the brain. Participants were followed up for three months to detect seizure recurrence. Associations were tested using the chi-square test, with $p<0.05$ considered significant.
Results: The mean age was 6.8 (±4.9) years with a male preponderance (60.2%; M:F 1.4:1). Generalized seizures occurred in 61.4% and focal seizures in 38.6% of children. An abnormal EEG was found in 58.0%, of which 79.4% showed epileptiform discharges. Among the 160 children (90.9%) who completed follow-up, seizure recurred in 45.0%. Recurrence was significantly higher in children with an abnormal EEG (58.5% vs 25.8%; $p<0.001$) and with focal seizure semiology (54.8% vs 38.8%; $p=0.047$).
Conclusion: An abnormal EEG was present in more than half of children after a first unprovoked seizure and was the strongest predictor of early recurrence. EEG, particularly when combining awake and sleep recordings, is a valuable tool for risk stratification in this population.
Keywords: Electroencephalography; First unprovoked seizure; Children; Seizure recurrence; Epileptiform discharge; Seizure semiology
How to cite this article: Mandal S, Chattopadhyay A, Maulik K, Mandal S, Pratap V. Electroencephalographic profile and its association with seizure recurrence in children with a first unprovoked seizure: a prospective observational study from Eastern India. Int J Drug Deliv Technol. 2026;16(64s):992-998. DOI: 10.25258/ijddt.16.64s.96

Introduction

A seizure is defined as the paroxysmal, involuntary discharge of cortical neurons that may manifest clinically as impairment or loss of consciousness, abnormal motor activity, behavioural or emotional disturbance, sensory phenomena, or autonomic dysfunction.[1] Seizures are the most frequent neurological problem in children, affecting around 10% of children and accounting for nearly 5% of paediatric emergency visits.[2] In India, the overall prevalence of epilepsy is estimated at 5.59 per 1,000 population, with a substantial burden in the paediatric age group.[3]

A seizure is considered unprovoked when it occurs in a child older than one month, in the absence of an acute precipitating factor such as fever, head injury, metabolic derangement, toxin exposure, stroke, or intracranial haemorrhage.[4] The incidence of a single unprovoked seizure ranges from 23 to 64 per 100,000 person-years.[4] Distinguishing an epileptic seizure from a non-epileptic paroxysm is not always straightforward in children, and although the diagnosis is largely clinical, ancillary investigations frequently improve diagnostic confidence.

Electroencephalography (EEG) is one of the principal investigations in the evaluation of a first unprovoked seizure. It helps to characterise the seizure type, supports the diagnosis of specific epilepsy syndromes, and assists in differentiating epileptic seizures from non-epileptic events such as syncope, movement disorders, transient ischaemic attack, or psychogenic episodes.[5,6] The interictal EEG may, however, be

normal, and the diagnostic yield is increased by recording during both wakefulness and sleep.

The risk of a subsequent unprovoked seizure after the first episode is reported to be between 40% and 80% in children, and is highest within the first one to two years.[7,8,9] Age, seizure semiology, family history, comorbidities, neuroimaging findings, and EEG abnormalities have all been proposed as predictors of recurrence. The American Academy of Neurology and the Indian Academy of Paediatrics recommend EEG as part of the evaluation of all children with an unprovoked seizure.[10,11] Because of the higher prevalence of focal structural lesions in this region, Indian guidelines additionally advise neuroimaging in children with an unprovoked seizure.[11]

Despite these recommendations, region-specific data describing the EEG profile of children with a first unprovoked seizure, and its relationship with early recurrence, are limited from Eastern India. The present study was therefore designed to evaluate the seizure semiology and EEG findings in neurologically normal children presenting with a first unprovoked seizure to a tertiary care hospital, and to determine the association of these findings with seizure recurrence within three months.

Materials and Methods

Study design and setting. This was a single-centre, prospective, cross-sectional, observational and analytical study conducted in the Department of Paediatrics of a tertiary care hospital in Kolkata, Eastern India. Children were recruited from both the outpatient and inpatient departments between May 2022 and April 2023, and each was followed up for three months from the first seizure.

Study population. Children aged 1–18 years presenting with a first episode of an unprovoked seizure, who were neurologically and developmentally normal, were eligible. A cluster of seizures occurring within 24 hours was regarded as a single event, and children with status epilepticus (a seizure lasting more than five minutes, or recurrent seizures without recovery of consciousness between them) were included. Children younger than one year; those with febrile seizures; a previously diagnosed seizure disorder, cerebral palsy or other neurological illness; meningitis; electrolyte disturbance, hypoglycaemia or hypothyroidism; non-epileptic paroxysmal events; head injury; drug overdose or intoxication; stroke or brain tumour on imaging; or an immunocompromised state were excluded.

Sample size. The sample size was calculated for the primary objective (the proportion of abnormal EEGs after a first unprovoked seizure), assuming an expected proportion of 42% based on an earlier study by Shinnar et al.,[12] a confidence level of 80% and a margin of error of 5%. The estimated requirement was 159; allowing for a 10% attrition, 176 children were recruited.

EEG recording. After written informed consent, EEG was performed within 24 hours of the seizure using a 16-channel machine with 21 scalp electrodes placed according to the international 10–20 system. Each recording lasted at least 20–30 minutes and was obtained during wakefulness and/or sleep according to feasibility; for sleep recordings, sleep was induced with oral triclofos sodium (50 mg/kg) or intravenous lorazepam (0.1 mg/kg). Hyperventilation and photic stimulation were used as activation procedures where appropriate, and recordings were analysed by a paediatric neurologist.

Other investigations and follow-up. In accordance with Indian guidelines, MRI of the brain was performed in all children to exclude stroke, tumour, space-occupying lesions and focal structural abnormalities. Demographic data, seizure semiology, seizure duration, the presence of aura, post-ictal features, and family history were recorded on a structured proforma. Children were reviewed at three months (or earlier if required); recurrence was defined as any further unprovoked seizure occurring more than 24 hours after the index event.

Statistical analysis. Data were summarised as frequencies, percentages, mean ± standard deviation and median. Associations between categorical variables were tested using the chi-square test, with a p-value <0.05 considered statistically significant. Ethical clearance was obtained from the institutional ethics committee before commencement of the study.

Results

A total of 176 children with a first unprovoked seizure were analysed. The mean age was 6.8 (±4.9) years (median 5 years 11 months; range 1 year 2 months to 17 years 6 months). The largest age group was 6–12 years (34.7%), and there was a clear male preponderance (60.2%; male-to-female ratio 1.4:1) (Figure 1; Table 1).

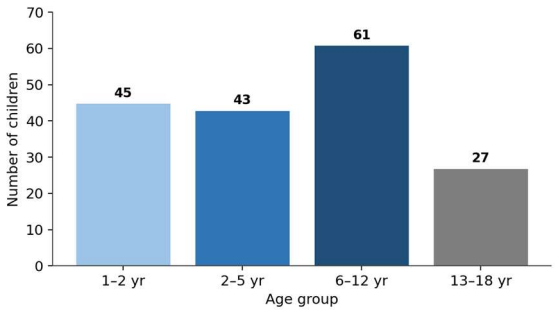

Figure 1. Distribution of children according to age group (N=176).

Table 1. Demographic and baseline clinical characteristics of the study population (N=176).

Variable	Category	n	%

Age group	1–2 years	45	25.6
	2–5 years	43	24.4
	6–12 years	61	34.7
	13–18 years	27	15.3
Sex	Male	106	60.2
	Female	70	39.8
Seizure duration	<1 minute	27	15.3
	1–5 minutes	112	63.6
>5 minutes (status epilepticus)		37	21.0
Preceding aura	Present	45	25.6
Post-ictal confusion/drowsiness	Present	96	54.5
Family history of seizure	Present	52	29.5
Type of EEG recorded	Sleep	64	36.4
	Awake	60	34.1
	Sleep + awake	52	29.5
MRI brain	Normal	160	90.9
	Cerebral atrophy	12	6.8
	White-matter hyperintensity	4	2.3

Seizure semiology. Generalized seizures predominated (61.4%), with generalized tonic–clonic seizures (GTCS) being the commonest subtype (67.6% of generalized seizures), followed by atypical absence seizures (20.4%). Focal seizures accounted for 38.6% of cases, of which focal awake seizures were most frequent (51.5%), followed by focal seizures with impaired awareness (38.2%) (Figure 2; Table 2). The distribution of seizure type varied significantly across age groups (p=0.004): generalized seizures predominated in the younger groups, whereas focal seizures became the more common type in adolescents aged 13–18 years (63.0%). There was no significant difference in seizure type between the sexes (p=0.335).

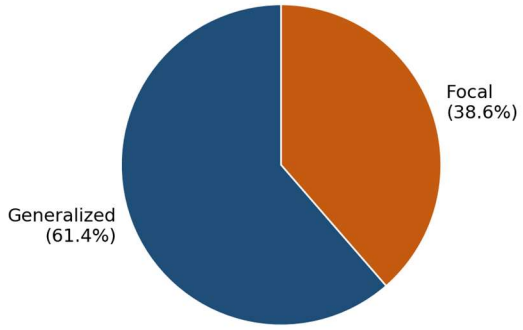

Figure 2. Distribution of children according to type of seizure (N=176).

Table 2. Distribution of seizure semiology (N=176).

Seizure type	n	%
Generalized seizure	**108**	**61.4**
Generalized tonic–clonic seizure	73	67.6*
Atypical absence seizure	22	20.4*
Myoclonic seizure	6	5.6*
Atonic seizure	6	5.6*
Tonic seizure	1	0.9*
Focal seizure	**68**	**38.6**
Focal awake seizure	35	51.5†
Focal seizure with impaired awareness	26	38.2†
Focal with secondary generalization	7	10.3†

*Percentage of generalized seizures; †percentage of focal seizures.

EEG findings. An abnormal EEG was recorded in 102 children (58.0%) (Figure 3). Among these, epileptiform discharges were the predominant abnormality, present in 81 records (79.4%), while waveform slowing accounted for the remaining 21 records (20.6%). The most frequent specific patterns were generalized spike-and-sharp-wave discharges (16.7%), <3 Hz spike-and-slow-wave complexes (13.7%), focal temporal spikes (11.8%) and frontal sharp waves (9.8%) (Figure 4; Table 3). The proportion of abnormal EEGs was highest in the 6–12

year group (67.2%), but the association between age and an abnormal EEG was not statistically significant (p=0.253), nor was there any difference between the sexes (57.5% in males vs 58.6% in females; p=0.893).

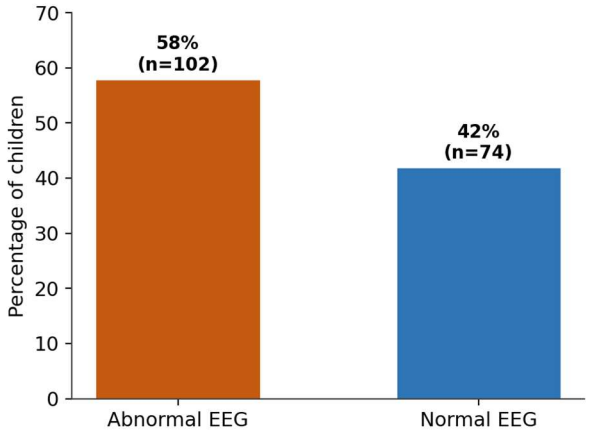

Figure 3. Distribution of children according to EEG findings (N=176).

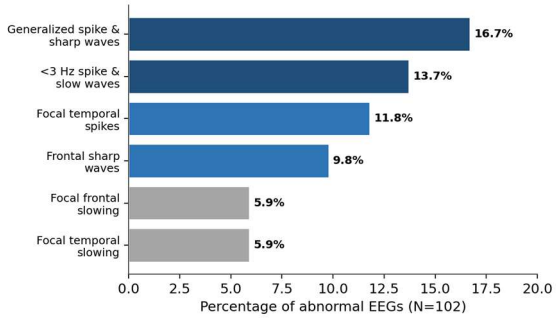

Figure 4. Distribution of the commonest abnormal EEG patterns among children with an abnormal record (N=102).

Table 3. Pattern of abnormalities among children with an abnormal EEG (N=102).

EEG abnormality	n	%
Epileptiform discharges	**81**	**79.4**
Generalized spike & sharp waves	17	16.7
<3 Hz spike & slow waves	14	13.7
Focal temporal spikes	12	11.8
Frontal sharp waves	10	9.8
Centrotemporal spikes	9	8.8
Other focal sharp waves/spikes	19	18.6
Waveform slowing	**21**	**20.6**
Focal frontal slowing	6	5.9
Focal temporal slowing	6	5.9
Hemispheric / generalized / other slowing	9	8.8

Other investigations and treatment. MRI of the brain was normal in 90.9% of children; cerebral atrophy (6.8%) and bilateral white-matter hyperintensity (2.3%) were the only abnormalities detected. Levetiracetam was the most commonly prescribed anticonvulsant (75.6%), followed by sodium valproate (15.3%), with dual therapy used in a minority of complicated cases.

Seizure recurrence. Of the 176 children, 160 (90.9%) completed the three-month follow-up. Seizure recurrence was observed in 72 children (45.0%). Recurrence showed no significant association with age (p=0.649) or sex (46.8% in males vs 42.4% in females; p=0.583). However, recurrence was significantly higher among children whose first seizure was focal (54.8%) than among those with a generalized seizure (38.8%) (p=0.047). The strongest association was with the EEG: recurrence occurred in 58.5% of children with an abnormal EEG compared with only 25.8% of those with a normal EEG (p<0.001) (Figure 5; Table 4).

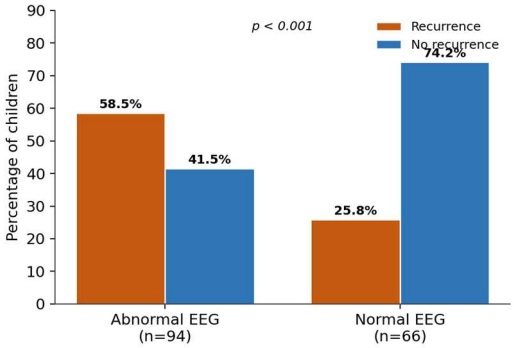

Figure 5. Seizure recurrence at three months according to EEG findings (N=160).

Table 4. Factors associated with seizure recurrence at three months (N=160).

Factor	Category	Recurrence (%)	p-value
Age group	<2 years	47.6	0.649
	2–5 years	48.6	
	6–12 years	45.6	
	13–18 years	33.3	
Sex	Male	46.8	0.583
	Female	42.4	
Seizure type	Generalized	38.8	0.047
	Focal	54.8	

EEG findings	Normal	25.8	<0.001
	Abnormal	58.5	

Discussion

This prospective study examined the seizure semiology, EEG profile and early recurrence pattern in 176 neurologically normal children presenting with a first unprovoked seizure to a tertiary centre in Eastern India. The two principal findings were that an abnormal EEG was present in well over half of the children, and that both an abnormal EEG and a focal first seizure were significantly associated with seizure recurrence within three months.

The demographic profile of our cohort is consistent with previous reports. The mean age of 6.8 years and the predominance of the 6–12 year age group mirror the findings of Shinnar et al., who reported a mean onset age of 6.8 years,[12] and of Vieira et al., whose patients had a mean age of 4.2 years.[13] The male preponderance we observed (M:F 1.4:1) is in keeping with Dudipala et al. (1.75:1)[14] and other paediatric series.[15] Status epilepticus as the first presentation occurred in 21% of our children, somewhat higher than the 9–10% reported by Dudipala et al. and Gupta et al.,[14,16] likely reflecting the referral pattern of a tertiary centre. A positive family history of seizures was present in 29.5%, comparable with the 21–22% reported in other cohorts.[14,17]

Generalized seizures predominated in our study (61.4%), with GTCS the commonest subtype, in agreement with Shinnar et al. (62% generalized)[12] and several Indian series.[18] In contrast, Vieira et al. found a slight predominance of focal seizures.[13] An important and statistically significant observation was the shift in seizure type with age: generalized seizures dominated in younger children whereas focal seizures became more frequent in adolescents (p=0.004). A similar age-related pattern, with generalized seizures more common in children under two years, was reported by Dedeoglu and Ardicli.[17]

An abnormal EEG was found in 58.0% of our children, with epileptiform discharges accounting for nearly four-fifths of abnormalities. This yield is higher than the 42% reported by Shinnar et al.[12] and the 33.8% reported by Dedeoglu and Ardicli,[17] but is broadly consistent with Pohlmann-Eden and Newton, who found epileptiform abnormalities in about 59% of children after a first non-febrile seizure,[19] and with Dudipala et al. (76%)[14] and Das et al.[20] The relatively high yield in our cohort is likely attributable to the routine performance of EEG within 24 hours of the seizure and the frequent use of combined awake and sleep recordings, a strategy shown by Shinnar et al. to substantially increase the detection of abnormalities.[12] Generalized spike-and-sharp-wave discharges and temporal focal spikes were the commonest patterns, consistent with the abnormality profile described in other paediatric studies.[12]

The central clinical message of this study concerns recurrence. Overall, 45% of children experienced a further seizure within three months, which falls within the wide range reported across studies with varying follow-up.[26] Crucially, an abnormal EEG more than doubled the recurrence rate (58.5% vs 25.8%; p<0.001). This closely replicates the findings of Shinnar et al., who reported recurrence in 54% of children with an abnormal EEG versus 25% with a normal EEG,[12] and is supported by Boulloche et al.,[15] Stroink et al.,[21] and Maia et al.,[22] all of whom identified epileptiform EEG abnormalities as a key risk factor for recurrence. Pohlmann-Eden and Newton similarly concluded that an early abnormal EEG showing focal epileptiform activity is a highly accurate predictor of recurrence,[19] and Khan and Baheerathan noted that an abnormal EEG roughly doubles the likelihood of a further seizure.[23] Not all studies agree, however; Dedeoglu and Ardicli found no significant association between EEG abnormalities and recurrence,[17] underscoring that the predictive value of EEG, while substantial, is not absolute.

Seizure semiology was the second significant predictor of recurrence in our study, with focal seizures recurring more often than generalized ones (54.8% vs 38.8%; p=0.047). The same direction of effect has been reported by Dedeoglu and Ardicli[17] and Akter et al., the latter identifying focal seizures, status epilepticus, sleep-related seizures and an abnormal EEG as significant risk factors for recurrence.[24] In contrast, age and sex were not significantly associated with recurrence in our cohort, consistent with Maia et al.[22] These observations support the use of EEG and careful seizure characterisation for risk stratification, which is clinically relevant because identifying a high-risk group may inform the often-debated decision about when to commence antiseizure medication after a first seizure.[24,25] Recurrence rates have historically been used to argue against routine treatment of an isolated first seizure,[25,26] and our data reinforce that this decision should be individualised according to EEG and semiology.

MRI of the brain was abnormal in only 9.1% of our neurologically normal children, with non-specific findings, echoing the low yield (8–11%) reported by Shwetha et al. and Sharma et al. in low-risk children.[27,28] This supports current guidance that emergent neuroimaging is most useful in children with focal deficits or those failing to return to baseline, while EEG remains a more informative first-line investigation for risk stratification in the neurologically normal child.[28,29] Comparative work has likewise found EEG and MRI to have

complementary roles as initial investigations after a first afebrile seizure.[30]

The strengths of this study include its prospective design, the standardised performance of EEG within 24 hours, the routine use of awake and sleep recordings, and complete neuroimaging. Several limitations should be acknowledged. The study was conducted at a single tertiary centre, which may limit generalisability and may have enriched the cohort for more severe presentations such as status epilepticus. The follow-up was limited to three months, so later recurrences were not captured. Infants under one year, children with typical absence epilepsy, and those with neurological impairment or developmental delay were excluded, so the findings apply specifically to neurologically normal children aged 1–18 years.

Conclusion

In neurologically normal children presenting with a first unprovoked seizure, an abnormal EEG was found in more than half, with epileptiform discharges predominating. An abnormal EEG was the strongest predictor of seizure recurrence within three months, more than doubling the recurrence risk, and a focal first seizure was also significantly associated with recurrence. EEG performed early after the seizure, ideally combining awake and sleep recordings, is therefore a valuable tool for risk stratification and can help guide individualised decisions about follow-up and the initiation of antiseizure medication. Larger multicentric studies with longer follow-up are warranted to confirm these associations.

References

1. Haslam RHA. Nonfebrile seizures. Pediatr Rev. 1997;18(2):39–49.
2. Saini N, Baghel A. Neuroimaging abnormalities in children with first afebrile seizure. J Dent Med Sci. 2013;5:21–4.
3. Arora NK, Nair MKC, Gulati S, Deshmukh V, Mohapatra A, Mishra D, et al. Neurodevelopmental disorders in children aged 2–9 years: population-based burden estimates across five regions in India. PLoS Med. 2018;15(7):e1002615.
4. Beghi E, Carpio A, Forsgren L, Hesdorffer DC, Malmgren K, Sander JW, et al. Recommendation for a definition of acute symptomatic seizure. Epilepsia. 2010;51(4):671–5.
5. Russ SA, Larson K, Halfon N. A national profile of childhood epilepsy and seizure disorder. Pediatrics. 2012;129(2):256–64.
6. Koutroumanidis M, Arzimanoglou A, Caraballo R, Goyal S, Kaminska A, Laoprasert P, et al. The role of EEG in the diagnosis and classification of the epilepsy syndromes: a tool for clinical practice by the ILAE Neurophysiology Task Force (Part 1). Epileptic Disord. 2017;19(3):233–98.
7. Chan D, Phuah HK, Ng YL, Choong CT, Lim KW, Goh WH. Pediatric epilepsy and first afebrile seizure in Singapore: epidemiology and investigation yield at presentation. J Child Neurol. 2010;25(10):1216–22.
8. Daoud AS, Ajloni S, El-Salem K, Horani K, Otoom S, Daradkeh T. Risk of seizure recurrence after a first unprovoked seizure: a prospective study among Jordanian children. Seizure. 2004;13(2):99–103.
9. Lizana JR, Garciá EC, Marina LC, López MV, Martín González M, Hoyos AM. Seizure recurrence after a first unprovoked seizure in childhood: a prospective study. Epilepsia. 2000;41(8):1005–13.
10. Expert Committee on Pediatric Epilepsy, Indian Academy of Pediatrics. Guidelines for diagnosis and management of childhood epilepsy. Indian Pediatr. 2009;46(8):681–98.
11. Gaillard WD, Chiron C, Cross JH, Harvey AS, Kuzniecky R, Hertz-Pannier L, et al. Guidelines for imaging infants and children with recent-onset epilepsy. Epilepsia. 2009;50(9):2147–53.
12. Shinnar S, Kang H, Berg AT, Goldensohn ES, Hauser WA, Moshe SL. EEG abnormalities in children with a first unprovoked seizure. Epilepsia. 1994;35(3):471–6.
13. Vieira SC, Liberalesso PBN, Spinosa MJ, Ortega AB, Olmos ASF, Löhr Júnior A. First unprovoked seizure: clinical and electrographic aspects. J Epilepsy Clin Neurophysiol. 2006;12(2):69–72.
14. Dudipala MP, Shankar SR, Reddy RS, Kumar VN. A prospective hospital-based study on the clinico-etiological profile of the first episode of a seizure in children. Cureus. 2022;14(11):e31748.
15. Boulloche J, Leloup P, Mallet E, Parain D, Tron P. Risk of recurrence after a single, unprovoked, generalized tonic–clonic seizure. Dev Med Child Neurol. 1989;31(5):626–32.
16. Gupta A, Solanki R, Roy S, Chakrabarty S. Clinico-etiological profile of “first seizure” in children: experience from an armed forces tertiary care hospital in Eastern India. Acta Med Int. 2022;9(1):49–53.
17. Dedeoglu O, Ardicli D. Evaluation and management of the first unprovoked seizure in children: single-centre experience. Ann Med Res. 2022;29(11):1263–7.
18. Singh RD, Suryavanshi S. A hospital-based study on clinicoetiological profile of seizures in children — a Kanpur experience. Int J Contemp Med Res. 2016;3(10):3003–7.
19. Pohlmann-Eden B, Newton M. First seizure: EEG and neuroimaging following an epileptic seizure. Epilepsia. 2008;49(Suppl 1):19–25.

20. Das R, Mondal A, Islam MA, Roy S, Sarkar P, Mondal S. Study of correlation between neuroimaging and EEG changes in seizure disorder in children (six months to twelve years). J Evid Based Med Healthc. 2020;7(26):1228–33.
21. Stroink H, Brouwer OF, Arts WF, Geerts AT, Peters AB, van Donselaar CA. The first unprovoked, untreated seizure in childhood: a hospital-based study of the accuracy of the diagnosis, rate of recurrence, and long-term outcome after recurrence. J Neurol Neurosurg Psychiatry. 1998;64(5):595–600.
22. Maia C, Moreira AR, Lopes T, Martins C. Risk of recurrence after a first unprovoked seizure in children. J Pediatr (Rio J). 2017;93(3):281–6.
23. Khan A, Baheerathan A. Electroencephalogram after first unprovoked seizure in children: routine, unnecessary or case specific. J Pediatr Neurosci. 2013;8(1):1–4.
24. Akter N, Rahman MM, Akhter S, Fatema K, Billah SMB. Predictors of recurrence after a first unprovoked seizure in childhood: a prospective study. Eur J Clin Med. 2021;2(3):1–6.
25. Beghi E, Giordano L, Marson A. Treatment for first epileptic seizure (Protocol). Cochrane Database Syst Rev. 2008;(2):CD007144.
26. Hauser WA, Rich SS, Annegers JF, Anderson VE. Seizure recurrence after a first unprovoked seizure: an extended follow-up. Neurology. 1990;40(8):1163–70.
27. Shwetha G, Bhat S, Soans ST. Role of electroencephalogram and neuroimaging in first afebrile seizure in children: a retrospective study. Asian J Clin Pediatr Neonatol. 2019;7(1):51–4.
28. Sharma S, Riviello JJ, Harper MB, Baskin MN. The role of emergent neuroimaging in children with new-onset afebrile seizures. Pediatrics. 2003;111(1):1–5.
29. Hirtz D, Ashwal S, Berg A, Bettis D, Camfield C, Camfield P, et al. Practice parameter: evaluating a first nonfebrile seizure in children. Neurology. 2000;55(5):616–23.
30. Bagla J, Kaur H, Singhal A, Mishra D, Kumari S, Dubey AP, et al. Electroencephalogram versus magnetic resonance imaging brain as the initial investigation of choice in neurologically normal children with first afebrile seizure in India. J Epilepsy Res. 2021;11(1):58–64.