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Clinical Spectrum of Childhood Epilepsy: A Prospective Tertiary-Care Study

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Abstract

Background: Epilepsy is among the most important chronic neurological disorders of childhood and is associated with considerable clinical, developmental, psychological, and social consequences. Its presentation varies with age at onset, underlying etiology, seizure type, associated co morbidities, electroencephalographic abnormalities, neuro imaging findings, treatment pattern, and seizure control. Characterization of these features is important for accurate classification and rational management of affected children.

Aim: To study the clinical profile of epilepsy in children attending a tertiary-care hospital.

Methodology: This single - centre prospective observational study was conducted in the Department of Pediatrics, Aditya Birla Memorial Hospital, Pune, over 24 months from November 2015 to October 2017. Thirty-two children with epilepsy fulfilling predefined eligibility criteria were enrolled after parental/guardian informed consent. Children older than one month and younger than 16 years with epilepsy confirmed by a pediatric neurologist, including previously diagnosed cases attending outpatient services, were eligible. Acute symptomatic seizures, febrile seizures, seizures associated with head injury, and no nepileptic seizure mimics were excluded. Demographic profile, seizure history, family and perinatal history, neurological findings, associated conditions, electroencephalography (EEG), magnetic resonance imaging (MRI), epilepsy classification according to ILAE 2010, antiepileptic drug therapy, seizure control, and factors associated with breakthrough seizures were evaluated.

Results: The mean age was 5.6 ± 3.8 years, with 50.0% of children aged 1–5 years; 56.3% were males. Idiopathic epilepsy was the predominant etiological category (59.4%), followed by West syndrome (12.5%) and remote symptomatic epilepsy (9.4%). Generalized epilepsy occurred in 68.8% and focal epilepsy in 31.2%; tonic-clonic seizures constituted 50.0% of seizure subtypes. EEG was abnormal in 87.5%. MRI was

abnormal in 37.5% overall, including 31.8% of generalized and 50.0% of focal epilepsy cases, without a significant difference between the groups (p=0.438). Co morbidity was present in 21.9%. Mono therapy was used in 81.3% and poly therapy in 18.7%. Breakthrough seizures occurred in 43.7%, with poor compliance, lack of drug revision, and fever described as common precipitating factors.

Conclusion: Childhood epilepsy demonstrated a heterogeneous clinical profile, although idiopathic etiology and generalized seizure types predominated. EEG abnormalities were frequent, while MRI, clinical classification, associated conditions, treatment pattern, and treatment adherence provided complementary information for comprehensive assessment and management.

Keywords: Childhood Epilepsy, Seizure Classification, Electroencephalography, Magnetic Resonance Imaging, Antiepileptic Drugs

Introduction

Epilepsy is a chronic neurological disorder characterized by an enduring predisposition to recurrent epileptic seizures and constitutes an important cause of neurological morbidity during childhood. A contemporary clinical definition includes at least two unprovoked or reflex seizures occurring more than 24 hours apart, or one unprovoked seizure accompanied by a sufficiently high probability of recurrence, or the diagnosis of an epilepsy syndrome.[1] Childhood epilepsy is clinically important not only because of the recurrent paroxysmal events themselves but also because poorly controlled seizures may interfere with learning, daily activities, psychosocial development, and quality of life. Children with epilepsy may experience additional psychiatric, developmental, and physical co morbidities, making the disorder considerably broader than an isolated tendency to have seizures.[2] Appropriate management therefore requires attention to seizure control as well as the wider developmental and psychosocial implications of the disease. Rasp all - Chaure et al. emphasized that medical management of childhood epilepsies requires integration of diagnostic classification, treatment selection, and practical considerations relevant to long-term care.[3] The clinical profile of epilepsy is heterogeneous because almost any significant cerebral pathology may produce seizures, while individual susceptibility reflects the interaction of genetically determined seizure threshold, underlying structural or metabolic abnormality, and precipitating factors. Neuro imaging has consequently become an important component of assessment in appropriately selected children. Magnetic resonance imaging offers superior soft-tissue contrast and anatomical resolution and has transformed the recognition of structural abnormalities associated with epilepsy. Bronen highlighted the role of MRI in epilepsy evaluation,[4] while Roy and Pandit reviewed its importance in defining underlying epileptogenic pathology.[5] Classification is equally fundamental because seizure type and epilepsy syndrome influence diagnostic interpretation, investigation, choice of antiepileptic medication, and prognosis. The study was initiated when the International League Against Epilepsy 2010 classification was in clinical use; accordingly, the thesis classified epilepsy using that framework. Berg et al. presented the revised terminology and conceptual organization of seizures and epilepsies that formed the basis of this classification.[6] Childhood epilepsy is also frequently accompanied by conditions such as developmental and behavioral abnormalities. Russ et al.

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demonstrated the broader burden of childhood epilepsy and seizure disorders, including increased developmental, mental-health, and physical co morbidity.[7] Electroencephalography remains an important adjunct to the clinical diagnosis because it may support seizure classification, help define an epilepsy syndrome, and contribute to therapeutic decision-making, although a normal interictal EEG does not exclude epilepsy. Smith emphasized the role of EEG in the diagnosis, classification, and management of epilepsy and its value when interpreted in conjunction with the clinical history.[8] Treatment is generally directed toward achieving seizure control with the least possible treatment burden. The thesis literature noted that a substantial proportion of children can achieve seizure control with appropriately selected mono therapy, and Unver et al. reported the effectiveness of single-drug treatment in a considerable proportion of pediatric epilepsy patients.[9] Nevertheless, breakthrough seizures remain clinically relevant because seizure recurrence despite ongoing treatment may arise from inadequate adherence, failure to revise therapy, inter current illness, or other precipitating factors. Kaddumukasa and Katabira identified treatment non-compliance and other clinical factors among important contributors to breakthrough seizures.[10] The clinical evaluation of a child with epilepsy must therefore extend from demographic characteristics and age at seizure onset to etiology, seizure classification, family and perinatal history, associated co morbidity, EEG and neuro imaging findings, therapeutic pattern, and seizure control. Advances in neuro imaging and evolving epilepsy classifications have progressively refined the understanding of childhood epilepsy, but the spectrum encountered in routine tertiary-care practice remains variable. A prospective characterization of these features can provide a clinically coherent representation of affected children and identify the relative contributions of idiopathic, syndromic, structural, and other etiological categories. Against this background, the present study prospectively evaluated children with epilepsy attending a tertiary-care hospital, with particular emphasis on demographic profile, causes of epilepsy, ILAE 2010 seizure classification, associated conditions, EEG and MRI findings, antiepileptic drug usage, seizure control, and factors associated with breakthrough seizures.

Aim

To study the clinical profile of epilepsy in children attending a tertiary-care hospital.

Methodology

This was a single-centre prospective observational study conducted in the Department of Pediatrics, including both outpatient and inpatient services, at Aditya Birla Memorial Hospital, Pune, over a period of 24 months from November 2015 to October 2017. The study population comprised children attending the institution with epilepsy who fulfilled the predetermined eligibility criteria, and a final sample of 32 children was included. The study was undertaken after approval from the institutional ethics committee, and every child was enrolled only after written informed consent had been obtained from the parent or legal guardian following provision of detailed information regarding the study. Children older than one month and younger than 16 years were eligible when they were admitted with a history of seizures or clinical features suggestive of epilepsy and the diagnosis was confirmed by a pediatric neurologist, or when they were known cases of epilepsy or an epilepsy syndrome attending the outpatient department. Children younger than one month or older

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than the eligible age range were excluded. Children presenting with acute symptomatic seizures, including febrile seizures, seizures associated with head injury, and events mimicking epilepsy such as breath-holding spells, reflex anoxic seizures, vasovagal syncope, and psychogenic seizures were also excluded. All children presenting to or admitted under the Department of Pediatrics were screened for a history suggestive of seizures and for known epilepsy or an epilepsy syndrome. Eligibility criteria were applied before enrolment, after which information was recorded using the standardized data-information form appended to the thesis. The assessment included demographic characteristics; a detailed clinical history of epilepsy; family history of epilepsy; clinical examination findings; neurological assessment; and relevant laboratory investigations undertaken according to the advice of the primary treating physician or pediatric neurologist. EEG findings were evaluated by the pediatric neurologist, while MRI findings were documented whenever neuro imaging had been performed as part of standard clinical care. Any surgical intervention was also recorded. Epilepsy was categorized according to the ILAE 2010 classification, which was the classification in use when the study commenced. The treatment profile included the spectrum of antiepileptic drug therapy and whether the child was receiving mono therapy or poly therapy. Clinical history was also specifically evaluated for seizure control and factors considered responsible for breakthrough seizures. Outcome assessment incorporated compliance with drug therapy, control of seizures, the presence of any residual neurological deficit identified clinically through sensory and motor assessment, and associated co morbidities. Particular attention was given to relevant coexisting conditions, with attention deficit hyperactivity disorder and autistic spectrum disorder among the conditions identified in the study population. Data were collected only after the inclusion and exclusion criteria, ethical requirements, and informed-consent requirements had been satisfied. As the investigation was observational, no investigation or procedure was performed solely for research purposes; investigations and procedures undertaken in the children were based on standard clinical indications and routine management. The analytical approach was designed to address the study objectives by describing demographic characteristics, identifying causes of epilepsy, classifying seizure type, documenting associated conditions and neurological status, examining EEG and MRI findings, characterizing antiepileptic treatment, and assessing seizure control and breakthrough seizures.

Statistical Analysis

The collected data were entered into Microsoft Excel and subsequently analysed using Statistical Package for the Social Sciences (SPSS), version 21.0. Categorical variables were expressed as frequencies and percentages, whereas continuous variables were summarized as mean ± standard deviation (SD). Intergroup comparisons of categorical variables were performed using the Chi-square test or Fisher's exact probability test, as appropriate. The thesis also specified the use of the Student's t-test and Z-test wherever applicable. All statistical hypotheses were evaluated using two-tailed tests, and a p-value <0.05 was considered statistically significant.

Ethical Considerations

The study was conducted after obtaining approval from the Institutional Ethics Committee. Children were enrolled only after the study objectives and procedures

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had been explained to the parent or legal guardian and written informed consent had been obtained. As this was an observational study, no investigation or intervention was performed solely for research purposes; all investigations and procedures were undertaken according to standard clinical indications and routine patient care.

Results

Thirty-two children with epilepsy were evaluated. The mean age was 5.6 ± 3.8 years, ranging from 0.25 to 15.0 years. One child (3.1%) was aged <1 year, 16 (50.0%) were 1–5 years, 13 (40.6%) were 6–10 years, and 2 (6.3%) were 11–15 years. There were 18 males (56.3%) and 14 females (43.7%), yielding a male: female ratio of 1.29:1. Age at seizure onset was neonatal in 4 (12.5%), infancy in 8 (25.0%), 2–12 years in 19 (59.3%), and adolescence in 1 (3.1%). Perinatal asphyxia was documented in 6 (18.7%), while a positive family history of epilepsy was present in 5 (15.6%).

Etiologically, 19 children (59.4%) had idiopathic epilepsy, 4 (12.5%) West syndrome, and 3 (9.4%) remote symptomatic epilepsy. Niemann-Pick disease, perinatal insult, poren cephalic cyst, post-vaccination epilepsy, generalized epilepsy with febrile seizures plus, and symptomatic epilepsy each accounted for 1 case (3.1%). Although perinatal insult was present in six children, it was identified as the direct cause of epilepsy in one child. Generalized epilepsy occurred in 22 (68.8%) and focal epilepsy in 10 (31.2%). Tonic-clonic seizures constituted 16 (50.0%), simple focal seizures 8 (25.0%), absence and myoclonic seizures 3 (9.4%) each, and complex focal seizures 2 (6.3%).

EEG was abnormal in 28/32 (87.5%) and normal in 4/32 (12.5%). MRI was normal in 20 (62.5%) and abnormal in 12 (37.5%). Among generalized epilepsy, MRI was normal in 15/22 (68.2%) and abnormal in 7/22 (31.8%); among focal epilepsy, MRI was normal in 5/10 (50.0%) and abnormal in 5/10 (50.0%). The MRI distribution did not differ significantly between seizure types (p=0.438).

Co morbidity was present in 7 (21.9%), including autistic spectrum disorder in three children and attention deficit hyperactivity disorder in two; other reported conditions included patent duct us arteriosus and Niemann-Pick disease. Mono therapy was used in 26 (81.3%), whereas 6 (18.7%) received poly therapy. Poly therapy was used in 13.6% of generalized and 30.0% of focal epilepsy cases; the difference was not statistically significant (p=0.346). Breakthrough seizures occurred in 14 (43.7%), whereas 18 (56.3%) had no breakthrough seizures. Poor compliance, absence of drug revision, and fever were described as common factors associated with breakthrough seizures. Residual neurological deficit was documented in 1 child (3.1%), described as cognitive delay.

Table 1: The age distribution of children with epilepsy

Age Group (years)	No. of cases	% of cases
<1 year	1	3.1
1 – 5 years	16	50.0
6 – 10 years	13	40.6
11 – 15 years	2	6.3
Total	32	100.0

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Table 2: The distribution of children with epilepsy according to cause of epilepsy

Cause of Epilepsy	No. of cases	% of cases
Idiopathic	19	59.4
West Syndrome	4	12.5
Remote Symptomatic	3	9.4
Niemann Pick Disease	1	3.1
Perinatal Insult	1	3.1
Porencephalic Cyst	1	3.1
Post Vaccination	1	3.1
Generalized epilepsy with febrile seizure plus (GEFS+)	1	3.1
Symptomatic	1	3.1
Total	32	100.0

Table 3: The distribution of type of epilepsy

Type of Epilepsy	No. of cases	% of cases
Generalized	22	68.8
Focal	10	31.2
Total	32	100.0

Table 4: Distribution of MRI Findings according to type of epilepsy

Type of Epilepsy	Normal n	Normal %	Abnormal n	Abnormal %	Total %	P-value
Generalized (n=22)	15	68.2	7	31.8	100.0	0.438 NS
Focal (n=10)	5	50.0	5	50.0	100.0	
Total (n=32)	20	62.5	12	37.5		

The distribution of MRI findings did not differ significantly between the two types of epilepsy in the study group ($p>0.05$).

Discussion

The demographic findings represented in Table 1 demonstrate that childhood epilepsy in the present cohort was concentrated predominantly in the younger age groups. Half of the children were between 1 and 5 years, another 40.6% were 6–10 years, and the mean age was 5.6 ± 3.8 years. The predominance of younger children is clinically plausible because childhood epilepsy frequently begins early in life, although the spectrum varies according to epilepsy syndrome and referral pattern. Amirsalari et al. evaluated 200 children aged 1–15 years and reported a mean age of 7.7 ± 4 years, with a male: female ratio of approximately 1.3, which was closely comparable to the male: female ratio of 1.29:1 in the present cohort.[11] Duggan also observed a slight male predominance, with a male: female ratio of 1.08, supporting the modest male preponderance found here.[12] The age-related pattern is consistent with the observations of Cam field et al., who demonstrated that epilepsy incidence varies markedly throughout child hood and is particularly high during the earlier years.[13] Hauser et al. likewise described an age - dependent

incidence of epilepsy and unprovoked seizures, reinforcing the importance of age at onset when characterizing pediatric epilepsy.[14] In the present series, seizure onset occurred during the neonatal period in 12.5%, infancy in 25.0%, childhood in 59.3%, and adolescence in only 3.1%. Together, these observations indicate that the demographic profile in this tertiary-care cohort was weighted toward early and middle childhood rather than adolescence. The presence of perinatal asphyxia in 18.7% and a positive family history in 15.6% additionally underlines the importance of carefully documenting antenatal, perinatal, developmental, and familial information when assessing a child with recurrent seizures.

The etiological spectrum presented in Table 2 was dominated by idiopathic epilepsy, accounting for 59.4%, whereas West syndrome accounted for 12.5%, remote symptomatic epilepsy for 9.4%, and several individually uncommon etiologies each contributed 3.1%. This finding emphasizes the heterogeneity of childhood epilepsy: a substantial proportion may lack an identifiable structural cause, while a smaller but clinically important group has syndromic, structural, metabolic, or acquired etiologies. Dura-Trave et al. reported a different etiological distribution in their pediatric epilepsy cohort, with a substantial symptomatic component and age-related variation in epilepsy syndromes, demonstrating that etiological proportions can vary materially between study populations.[15] De Bitten court et al. highlighted the influence of environmental, socioeconomic, infectious, and other acquired factors on epilepsy etiology, illustrating why the distribution of symptomatic and idiopathic epilepsy may differ according to the population being studied.[16] Chinchure and Kesavadas emphasized the role of structural and infective cerebral abnormalities in the etiological evaluation of epilepsy and the importance of neuro imaging in demonstrating potentially epileptogenic lesions.[17]

Senanayake and Roman similarly described substantial epidemiological and etiological heterogeneity in epilepsy, particularly where perinatal, infectious, traumatic, and other acquired factors contribute to disease burden.[18] Against this background, the 59.4% idiopathic proportion in the present study should be interpreted as the clinical pattern of this particular tertiary-care sample rather than a universal etiological distribution. The finding that six children had a history of perinatal asphyxia but perinatal insult was considered the principal cause of epilepsy in only one case is also important: a historical risk factor is not necessarily synonymous with the established etiology of epilepsy. This distinction supports a structured etiological assessment combining clinical history, neurological examination, EEG, neuro imaging, and syndrome recognition.

The seizure classification shown in Table 3 demonstrated a clear predominance of generalized epilepsy, which occurred in 22 children (68.8%), while focal epilepsy occurred in 10 (31.2%). Analysis of seizure subtypes further showed that tonic-clonic seizures were the largest group, accounting for 50.0%, followed by simple focal seizures at 25.0%; absence and myoclonic seizures each accounted for 9.4%, while complex focal seizures accounted for 6.3%. Verma et al. reported generalized tonic-clonic followed by generalized tonic or clonic seizures as the most common seizure patterns, together accounting for approximately 62.9%, while localized or partial seizures and those with secondary generalization represented approximately

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30% of their study population.[19] This pattern closely resembles the generalized predominance observed in the present study. Sridharan and Murthy also described a predominance of generalized seizure patterns within their epidemiological assessment, and their findings were cited in the thesis as being in agreement with the observed seizure distribution.[20] Mac et al., in their systematic assessment of epilepsy epidemiology, etiology, and clinical management, demonstrated that seizure-type distributions vary between pediatric populations but include substantial contributions from both generalized and focal epilepsies.[21] Sebit and Mielke also described the sociodemographic, etiological, diagnostic, and EEG characteristics of epilepsy and were cited in the thesis among reports supporting the predominance of generalized epilepsy in comparable clinical settings.[22] The current distribution therefore corresponds with several published observations, although other cohorts have reported a greater proportion of focal epilepsies. Such variation is expected because classification is influenced by age, underlying pathology, referral characteristics, diagnostic resources, and the epilepsy classification system employed. Since the study began when the ILAE 2010 classification was in use, application of that system maintained methodological consistency throughout data collection.

The neuroimaging relationship in Table 4 adds an important structural dimension to the clinical profile. Overall, MRI was abnormal in 12/32 children (37.5%) and normal in 20/32 (62.5%). Abnormal MRI findings occurred in 7/22 (31.8%) children with generalized epilepsy and 5/10 (50.0%) with focal epilepsy. Although the proportion was numerically higher among focal cases, the difference was not statistically significant (p=0.438). Wongladarom et al. reviewed brain MRI findings in pediatric epilepsy and demonstrated the clinical value of MRI for identifying structural abnormalities that may underlie epileptic seizures.[23] Bano et al. similarly emphasized neuro imaging as an important component of epilepsy evaluation because structural abnormalities may materially affect etiological classification and subsequent management.[24] Hsieh et al. examined the role of neuro imaging in infants with new-onset a febrile seizures and supported imaging in appropriately selected pediatric patients, particularly when clinical features increase suspicion of structural pathology.[25] Bradley and Shey also described MRI evaluation of seizures and its contribution to identifying epileptogenic structural lesions.[26] The present observation of a 37.5% MRI abnormality rate therefore reinforces the usefulness of neuro imaging, while the non-significant difference between generalized and focal epilepsy cautions against assuming that structural abnormalities occur exclusively in focal disease. The thesis also showed an exceptionally high proportion of abnormal EEGs, 87.5%, compared with 12.5% normal studies. These diagnostic findings are complementary: EEG contributes primarily to electrophysiological classification, whereas MRI provides anatomical information, and neither should be interpreted in isolation from the clinical syndrome.

The remaining objectives further broadened this profile. Associated co morbidity was identified in 21.9%, most notably autistic spectrum disorder and attention deficit hyperactivity disorder. Treatment was predominantly with mono therapy (81.3%), while 18.7% received poly therapy. The relative use of poly therapy was greater in focal than generalized epilepsy (30.0% vs 13.6%), although the difference was not statistically significant. This high mono therapy proportion is consistent with the

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treatment principle reflected by Unver et al., who reported seizure control with monotherapy in a substantial proportion of children.[9] Breakthrough seizures were nevertheless documented in 43.7% of the present cohort. Poor compliance, failure to revise medication, and fever were identified as common precipitating factors, corresponding with the emphasis by Kaddumukasa and Katabira on medication non-compliance and clinical precipitating factors in breakthrough seizure occurrence.[10] Only one child had a residual neurological deficit, described as cognitive delay. Collectively, the findings show that clinical profiling in childhood epilepsy requires more than seizure labeling alone. Age, etiology, seizure type, EEG, MRI, neurodevelopmental and behavioral co morbidity, treatment pattern, adherence, and breakthrough events collectively determine the clinical picture and should be considered together when planning continuing pediatric epilepsy care.

Conclusion

Childhood epilepsy in this prospective cohort was characterized predominantly by younger age, idiopathic etiology, generalized seizure types, frequent EEG abnormalities, and largely normal MRI findings, although clinically relevant structural abnormalities occurred in more than one-third of children. Monotherapy was the principal treatment strategy, while breakthrough seizures remained frequent, emphasizing the importance of accurate classification, appropriate EEG and MRI evaluation, systematic recognition of co morbidities, treatment adherence, and regular therapeutic review.

Limitations

The study was limited by its small sample size, relatively short study duration, and single tertiary-care setting. Consequently, representation of all socioeconomic sections of the population may have been restricted, and the observed clinical, etiological, diagnostic, and therapeutic distributions may not be generalizable to the wider pediatric population.

References

1. Fisher RS, et al. A practical clinical definition of epilepsy. Epilepsia. 2014;55(4):475-482.
2. Jones JE, Austin JK, Caplan R, Dunn D, Plioplys S, Salpekar JA. Psychiatric disorders in children and adolescents who have epilepsy. Pediatr Rev. 2008; 29 (2):e9-e14.
3. Rasp all-Chaure M, Neville BG, Scott RC. The medical management of epilepsies in children: conceptual and practical considerations. Lancet Neurol. 2008; 7(1):57-69.
4. Bronen RA. Epilepsy: the role of MR imaging. AJR.1992;159:1165-1174.
5. Roy T, Pandit A. Neuro imaging in epilepsy. Ann Indian A cad Neurol. 2011;14(2):78-80.
6. Berg AT, Berkovic SF, et al. Revised terminology and concepts for organization of seizures and epilepsies: report of the ILAE Commission on Classification and Terminology, 2005-2009. Epilepsia. 2010;51(4):676-685.
7. Russ S, Larson K, Half on N. A national profile of childhood epilepsy and seizure disorder. Pediatrics.2012; 129:256-264.
8. Smith SJ. EEG in the diagnosis, classification, and management of patients with epilepsy. J Neurol Neurosurg Psychiatry. 2005;76(Suppl 2):ii2-ii7.
9. Unver O, Keskin SP, Uysal S, Unver A. J Child Neurol. 2015;30(6):698-702.

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10. Kaddumukasa M, Katabira E. The frequency and precipitating factors for breakthrough seizures among patients with epilepsy. BMC Neurol.2013;13:182.

11. Amirsalari S, Saburi A, Hadi R, Toraman M. Magnetic resonance imaging findings in epileptic children and its relation to clinical and demographic findings. Acta Med Iran. 2012;50(1):37-42.

12. Duggan MB. Epilepsy in rural Ugandan children: seizure pattern, age of onset and associated findings. Afr Health Sci. 2010;10(3):218-225.

13. Camfield CS, Cam field PR, Gordon K, Wirrell E, Dooley JM. Incidence of epilepsy in childhood and adolescence: a population-based study from 1977 to 1985. Epilepsia. 1996;37(1):19-23.

14. Hauser WA, Annegers JF, Kurland LT. Incidence of epilepsy and unprovoked seizures: 1935-1984. Epilepsia.1993;34:453-468.

15. Dura-Trave T, Eugenia M, Petri Y, Gallina-Victoria F. Incidence of epilepsies and epileptic syndromes among children: 2002 through 2005. J Child Neurol.2008;23:878-882.

16. De Bitten court PR, Adamolekum B, Bharucha N, Carpio A, Cossío OH, Danesi MA, et al. Epilepsy in the tropics: I. Epidemiology, socioeconomic risk factors, and etiology. Epilepsia. 1996; 37(11):1121-1127.

17. Chinchure S, Kesavadas C. Neuro imaging in epilepsy in tropics. Neuro imaging Clin N Am. 2011; 21 (4): 867-877.

18. Senanayake N, Roman GC. Epidemiology of epilepsy in developing countries. Bull World Health Organ.1993;71:247-258.

19. Verma SR, Sardana V, Gupta PK, Verma SC, Munshi A, Suryavanshi A. Evaluation of non-febrile seizure disorder on MRI with correlation with seizure type and EEG records in a tertiary care teaching hospital. Internet J Third World Med. 2013;11(1).

20. Sridharan R, Murthy BN. Prevalence and pattern of epilepsy in India. Epilepsia. 1999;40(5):631-636.

21. Mac TL, Tran DS, Quet F, Odermatt P, Preux PM, Tin-Tan C. Epidemiology, a etiology, and clinical management of epilepsy in Asia: a systematic review. Lancet Neurol.2007; 6:533-543.

22. Sebit MB, Mielke J. Epilepsy in sub-Saharan Africa: its socio-demography, a etiology, diagnosis and EEG characteristics in Harare, Zimbabwe. East Afr Med J. 2005;82(3):128-137.

23. Wongladarom S, Laothamatas J, Visudtibhan A, Sawatsut P. Magnetic resonance imaging of the brain in epileptic pediatric patients: review of the experience in Ramathibodi Hospital. J Med Assoc Thai. 2004;87(9):1092-1099.

24. Bano S, Yadav SN, Chaudhary V, Garga UC. Neuro imaging in epilepsy. J Pediatr Neurosci. 2011;6(1):19-26.

25. Hsieh DT, Chang T, Tsuchida TN, Vezina LG, Vanderver A, Siedel J, et al. New-onset a febrile seizures in infants: role of neuro imaging. Neurology. 2010; 74 (2):150-156.

26. Bradley WG, Shey RB. MR imaging evaluation of seizures. Radiology.2000; 214:651-656.

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