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Original Article

Electrical Activity of the Brain in Children with Developmental Delay: A Preliminary Study

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ABSTRACT

Background and Aim: Developmental disorders are often irreversible and debilitating for a child. Hypoxic ischemic insult during perinatal period is an important cause of global/ focal developmental delay in children and may trigger epileptogenesis. Thus, it is crucial to explore electrical activity of the brain in patients with developmental delay and identify early electrophysiological correlates that could be used as potential signatures for its diagnosis/ prognosis. The objectives of this study were to explore electrical activity of the brain in children with developmental delay, to assess occurrence of seizures in these children and to find predominant type of seizure pattern. **Methods:** In this study, 70 referred cases from the Pediatrics Department of AIIMS, Rishikesh with a history of global/specific developmental delay underwent video-EEG (Nicolet) for 30-45 min to assess electrical activity of the brain and presence of any abnormal discharge. Along with EEG, a brief clinical history including symptomatology was also obtained. **Results:** 69 out of 70 referred cases showed EEG changes. 16 had focal seizures (1 focal, 15 focal with secondary generalization), 40 generalized presentations (none had absence seizures), 3 abnormal patterns, 10 had hysparrhythmia and only 1 had a normal pattern on EEG. Attenuation of waves/ background suppression was also observed in 12 out of 70 (17.14%) patients. This pattern was found predominantly in peripheral electrodes such as parietal and temporal. **Conclusion:** EEG could be a useful electrophysiological tool to evaluate cases of developmental delay. Not only can it diagnose subclinical inter-ictal seizures in such cases, EEG signatures such as suppression pattern may be useful biomarkers to assess severity or for prognostic purposes. It would be worthwhile to understand the mechanisms why certain areas of the brain could be more sensitive to developmental defects due hypoxia or other etiologies. The functional deficits corresponding to these areas could be further explored.

Keywords: Hypoxic-ischemic encephalopathy, Seizures, EEG, Perinatal hypoxia, Background suppression, Developmental delay.

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INTRODUCTION

Child's development is a dynamic process that includes age-specific tasks that children attain at a certain age.[1] Developmental delay is often referred to as "delay in any gross motor, fine motor, speech and language, cognitive and social, hearing and vision domain in a young child's development compared to other children". When this occurs in one area it is said to be a "Focal Delay" while a delay in more than one area is called a "Global Developmental Delay." Epilepsy is linked with developmental delay in 0.7% of low-middle income countries as compared to 0.6% in high income countries.[2] The etiology for majority of cases of developmental delay is idiopathic. When known, etiology may include genetic, environmental, and/or psychosocial factors. Asphyxia during labor is the cause of almost 60% cases of moderate to severe encephalopathy in infants.[3] As a result of hypoxic injury to the brain, some affected newborns die in the postnatal period, while some others develop severe and irreversible neuropsychological manifestations such as mental retardation, epilepsy, visual, auditory, motor dysfunction, increased hyperactivity and cerebral palsy.[4] In children with cerebral palsy various causes of epilepsy and seizures are defined such as diffuse cortical malformations, injuries and perinatal arterial ischemic stroke.[5-8] The neuropathological effect of these etiological insults vary considerably with the gestational age of the infant, the nature of the insult, and the type of intervention.[9]

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During routine EEG evaluation, we observed that children with a history of perinatal hypoxia, delayed crying and developmental delay since birth had seizures and suppression patterns on certain electrodes. We henceforth hypothesized that seizures/suppression patterns might be associated to hypoxia and developmental delay. Thus, this cross-sectional qualitative study was undertaken to explore the association between developmental delay and the electrical activity of the human brain. This preliminary study has tremendous clinical implications as it intends to shed some light on the electrophysiological correlates of developmental delay in the brain of children. These insights can be of much use in the diagnosis, prognosis, evaluation, and treatment of patients affected with developmental delay due to various etiologies including Hypoxic Ischemic Encephalopathy (HIE).

MATERIALS AND METHODS

Study Setting

The study was conducted in the Electroencephalography (EEG) Laboratory, Department of Physiology, All India Institute of Medical Sciences, Rishikesh, India from 2017-2018.

Ethical Considerations

Ethical approval was obtained from the Institutional Ethical Committee of AIIMS Rishikesh, in accordance with the Declaration of Helsinki. Informed consent and assent (if applicable) were duly taken from the guardians/patients.

Study Population

A total of 70 referred cases from the Department of Pediatrics with a history of delayed milestones with or without suspected seizure on/ off anti-epileptics were selected for the study. Children up to 18 years of age were considered for this study.

Definitions

'Global' delay: Significant developmental delay in two/ more domains - motor (gross and fine), cognition, speech/ language, personal/ social or daily living activities.[10]

'Focal' / specific developmental delay: Delay occurring in any specific developmental domain.

Inclusion and Exclusion Criteria

Inclusion criteria: Children ≤18 years with delayed milestones, with or without suspected seizures.

Exclusion criteria: History of co-existing drug/substance use, psychiatric illness, major medical or surgical disease, endocrine disorders affecting mentation, or sleep disorders.

Data Collection

A brief history including symptoms was taken from the attendants/ patients. The patients underwent video-EEG (Nicolet) recording for 30-45 min to assess the electrical activity of the brain and presence of any abnormal discharge. EEG recording was done using 10-20 rule of electrode placement in a soundproof dark room using appropriate precautions. Attendants were advised to wash hair of patients beforehand. The patients were advised not to have any drugs 12 hr prior to the test and stay awake as much as possible before the test. As per age and requirement, photic stimulation, hyperventilation, sleep and sleep deprivation were triggers used to increase sensitivity of the test. Children were given adequate amount of sedation whenever needed (Triclophos). Patients were instructed not to talk or move during the test and the attendant was told to be present during the test.

Statistical Analysis of Data

Data were presented as percentage/prevalence. The Fisher Exact test was used to assess association. $p<0.05$ was considered statistically significant.

RESULTS

Background Characteristics

The study sample population consisted of 70 children. Mean age was 5.674±5.19 years. Geographical distribution of population was mainly from two states of India: 47 from Uttarakhand and 23 from Uttar Pradesh. Patients with history of delayed crying (n=33, 47%), delayed milestones (n=57, 81%), global developmental delay (n=11, 16%) and/or known HIE cases (n=1, 1.4%) were selected for the study. The types of delivery were as follows: normal vaginal (n=48, 69%), instrumental (forceps-n=0, 0%, vacuum- n=1, 1.4%) and caesarian section (n=14, 20%). The places of delivery were: home (n=15, 21.4%), institutional (n=46, 66%) and vehicle (n=1,1.4%). The ages of gestation were: full term (n=53, 76%), preterm (n=5, 7%) and advanced for gestational age (n=5, 7%). Some of the data was missing/unknown as attendants were unaware of birth history.

Electrical Activity of the Brain

Presence of seizures/abnormal patterns was found in 69 out of 70 (98.6%) cases of developmental delay (Figure 1). Out of 70 cases, 40 (57%) had generalized onset seizures, 16 (23%) had focal onset seizures (6% focal only, 94% focal with secondary generalization), 10 (14%) had hypsarrhythmia, 3 (4%) had abnormal patterns and only 1 (2%) had normal pattern on EEG (Figure 2). Those having focal seizures had abnormal waves at varied locations mainly on central, parietal and temporal areas. 6 (38%) were left sided, 8 (50%) right sided, 1(6%) had multiple foci and 1 (6%) had frontocentral focus.

Attenuation of background waves/suppression pattern was found in peripheral (parieto-temporal) electrodes in 12 (17%) out of 70 patients (Figure 1). However, suppression patterns were not found to be associated with age of patients, gender, type of delivery (normal/cesarean), place of delivery (institutional/

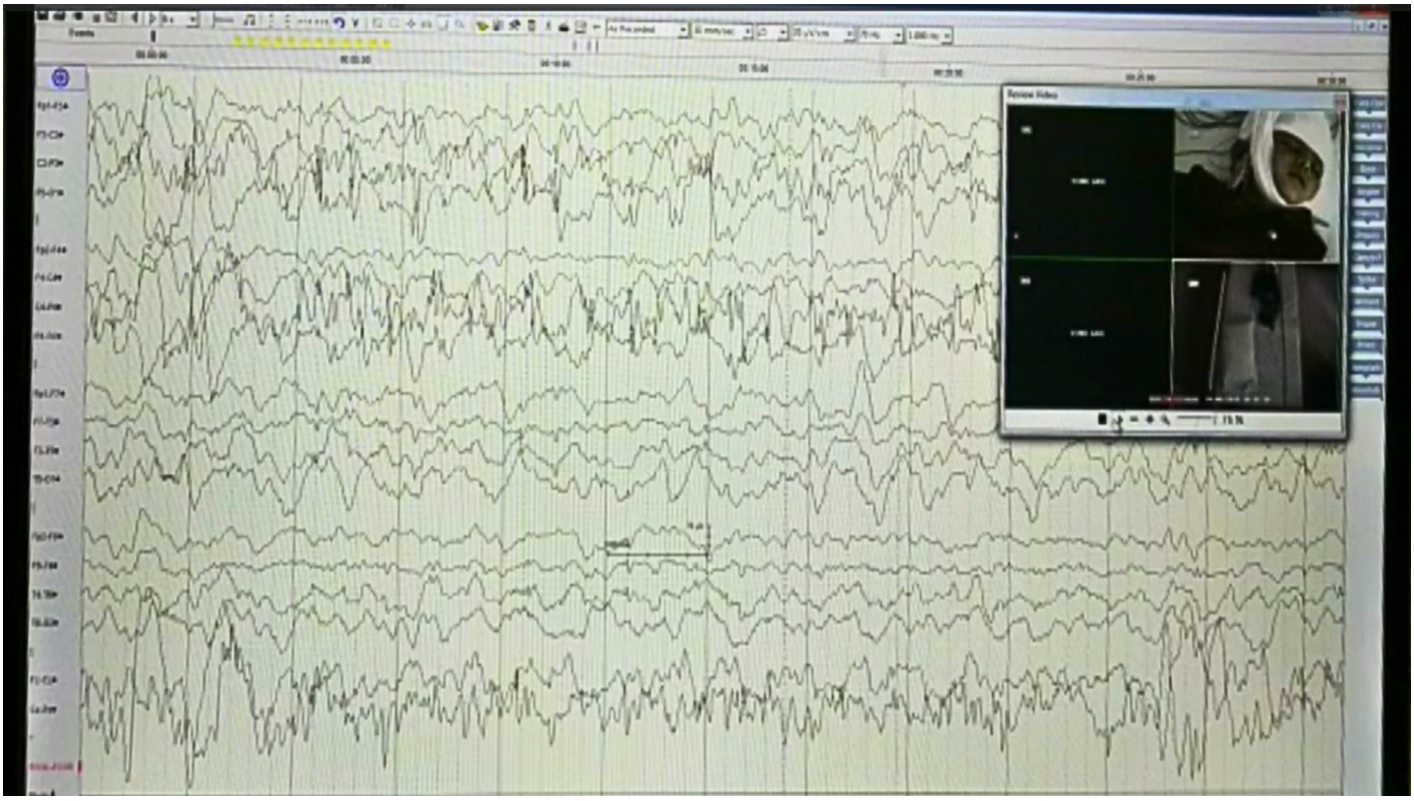

Figure 1: Seizures and burst suppression pattern seen in peripheral electrodes in a child of global developmental delay.

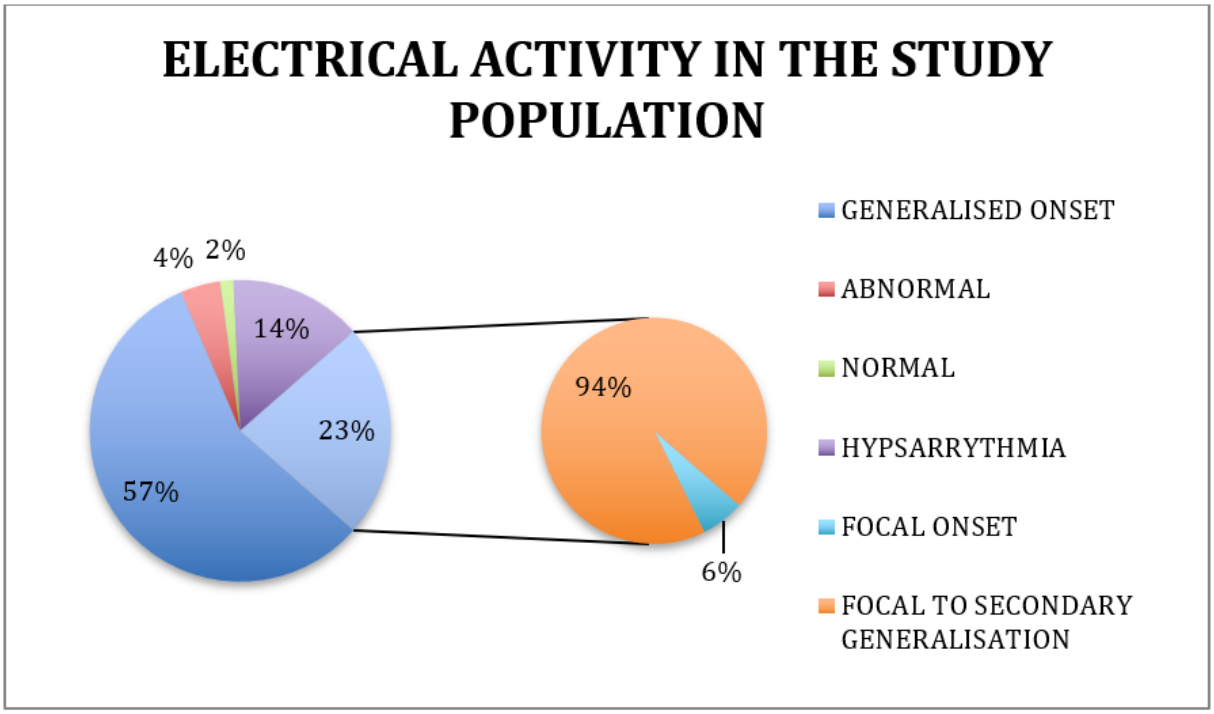

Figure 2: Electrical activity in the study population.

non-institutional) or period of gestation (preterm/full term/ AGA) with $p>0.05$.

DISCUSSION

The major cause of symptomatic epilepsy in developing children is perinatal insult, including hypoxic ischemic encephalopathy.[11] Though developmental delay could be attributed to various factors, there is considerable evidence on how hypoxia drives seizures.Undesirable perinatal events can lead to a state of hypoxia disrupting generation of energy inside the cells, thus affecting normal functioning of neuronal cells. Cellular depolarization pushes calcium, sodium and water inside the cells and potassium outside. [12] As a result of this, cellular activity drops severely leading to neuronal damage due to accumulation of excitatory amino acids extracellularly leading to seizures.[13,14] In our sample population, most cases of developmental delays with/ without evidence of perinatal asphyxia had symptomatic seizures.

The exact prevalence of GDD is unknown, but it is estimated to be 1%-3% in the literature.[15] Many studies in the past suggest an association between GDD and epilepsy.[15-17] In a study done by Masri et al, seizures were associated with developmental delay in 51% of cases.[18] In addition to the underlying pathology leading to developmental delay and the effect of drugs, the epileptic process itself may contribute to adverse impact on cognition and development. Epilepsy in GDD can have impact on developing brain structures and functions, such as brain volume reduction (especially white matter tissue, which is seen in patients with childhood-onset temporal lobe epilepsy). Brain reduction due to epileptic insult itself is associated with poor cognitive skills.[19-21] Many authors in the past have tried to evaluate EEG changes seen in children with cerebral palsy and developmental delays and demonstrated that it can be used for prognosis of these patients.[22] These abnormal interictal electroencephalographic recordings could be used as biomarkers to assess severity of brain insult with/without evidence of frank clinical seizures in children with poor development.[23]

The mean age of our study population was 5.674±5.19 years. This is comparable to a study that assessed EEG pattern among children with developmental delay and found mean age to be 4.8 years (±3.9).[24] In our study, history of delayed crying among cases was found to be 47%, which is comparable to the study among children with cerebral palsy done by Khan *et al.*[25] Among perinatal factors, normal vaginal delivery is found more commonly in our cohort (69%) which is comparable to study done by Thomaidis *et al.*, (61.26%).[26] But difference of incidence of hypoxia and developmental delay in normal vs assisted delivery was not found to be significant in our study (p value <0.05). This result is also comparable to study done by Thomaidis *et al*, Suchdeva *et al* and Tikaria *et al.*[26-28] This may be because most of the deliveries in our study were institutional only (66%). In our study, most of the children were born full term (76%). A study done by Yang *et al*. supports this finding. In their study preterm infants were more likely to have developmental delay and prematurity was found to be a significant risk factor for language development in the univariate analysis, but in the multivariable logistic regression model, after taking into account the effects of confounding factors, it was no longer statistically significant.[29] In our sample, occurrence of preterm deliveries was found in 7% of children and these preterm neonates also not found to be associated with higher risk of global developmental delay, abnormal EEG pattern or burst suppression pattern.

Around 98.6% of patients had seizures/ abnormal EEG activity in our study population. In a retrospective study, EEG abnormalities were found only in one-third of the children having developmental delay without overt seizures.[30] Higher proportion of seizures/abnormal EEG activity in our study might be because of convenient sampling as cases of developmental delay having high suspicion of seizures were referred to undergo EEG. 57% cases had generalized onset seizures, 23% had focal onset seizures (6% focal, 94% focal with secondary generalization), 14% had hypsarrhythmia, 4% had abnormal patterns and only 2% had normal pattern on EEG in this study. None of the individuals presented with absence seizures suggesting a completely different pathogenesis for this type of seizure. It should also be kept in mind that there may be difficulty in detecting the possible presence of absence attacks and subtle complex-partial seizures especially in very young subjects as absence seizures are commonly seen in an age group of 8-21 years. This could be the plausible reason that we were not able to find any EEG recording of absence seizures in our study.

In a study done by A Al-Sulaiman , overall EEG abnormalities in the seizure group of children with cerebral palsy was found to be 92.6% which is almost similar to our findings.[31] In the same study, EEG abnormalities were as follows- slow waves in 44% patients (generalized asynchronous in 91.7% and generalized synchronous in 8.3%); amplitude abnormalities in 2.5% (focal in 50%, generalized 50%); epileptiform activity (including isolated sharp waves, isolated spikes, and spike-wave and polyspike-wave complexes) in 81.5% (focal in 18%; generalized in 72.7% and multifocal in 9.1%). Hypsarrythmia was found in 5% and burst suppression in 1.2%. Only 7.4% recordings were normal.[31] In the study done by Gururaj *et al* over half of children with cerebral palsy presented with generalized tonic clonic seizures; the Electroencephalogram (EEG) showed focal epileptic discharges with or without secondary generalization in 39.3%.[32] Those having focal seizures in our study, had abnormal waves at varied locations mainly on central, parietal and temporal areas- 50% were right sided 38% were left sided, 6% had multiple foci and 6% had frontocentral focus. With respect to the localization of focal epileptiform activity, these abnormalities are different from those from previous studies where the left temporal and frontal lobes or

fronto/temporal regions were the commonest sites for focal EEG abnormalities.[24,33]

We also found suppression patterns/attenuation of waves in 17% patients which is similar to results found in study done by Shen *et al*.[34] Perinatal insult in the form of hypoxia is a significant cause for GDD. It has been proposed in the literature that though hypoxia may initially lead to hyper-excitability and seizures, progressive ongoing neuronal damage during hypoxia could further lead to blunting of electrographic activity. Neuronal degeneration caused by prolonged hypoxia and ischemia cause a progressive decrease in background EEG creating significant background suppression patterns leading to progressive decrease in the seizures.[35] In a study done by Madeleine, burst suppression pattern in neonates were associated with death or severe neuro-developmental disability in 93% patients in infancy/ childhood.[36] In the study done by Shen *et al*, burst suppression pattern were associated with poor outcome in Ohtahara syndrome, early myoclonic encephalopathy and HIE.[34] So, if a child with poor development comes with burst suppression pattern on EEG, thorough evaluation must be done in terms of good clinical history, physical examination, radiological investigation such as MRI and genetic studies if indicated.

Limitations and Future Directions

In the future, burst suppression pattern in children with poor developmental delay could be used as a biomarker to assess severity/ prognostic factor for children with neurodevelopmental delay with symptomatic or asymptomatic seizures. It was also interesting to see suppression pattern presenting only in the peripheral electrodes such as parietal and temporal in our study. Even focal seizures correspond to these areas in particular. This could be attributed to differential distribution of blood flow in the brain or higher sensitivity of certain brain tissues to hypoxia. Interestingly, Middle Cerebral Artery (MCA) that supplies blood to parieto-temporal areas, is known to be the most common artery involved with ischemic strokes.[37] The reasons why middle meningeal artery is the most affected by ischemic conditions could be speculated. While emboli could lodge easily in MCA as it is directly connected to Internal Carotid in Ischemic stroke, ischemia in perinatal hypoxia could result from high metabolic demand of excitatory neurons in this region, larger area supplied by one artery and susceptibility of watershed areas to hypoxia due to low perfusion pressure.

Our study had certain limitations, including the use of convenience sampling. Only cases of developmental delay with a high clinical suspicion of seizures were referred to the EEG laboratory and subsequently analyzed for the purpose of this study. Thus, the sample population estimates may not be generalizable to reflect actual occurrence of seizures in children of developmental delay. Most of our patients were already on anti-epileptics; the effect of medication was a necessary confounding factor that could not be eliminated. Further studies can be done in the future with detailed clinical data. Quantitative assessment of EEG using source localization can be done in correlation with radiological and genetic work up, to find out specific brain areas that are involved the evolution of seizures / neurodegeneration in cases of developmental delay.

As repeated seizures and epilepsy could further deteriorate for cognitive domain in these children and in general often go undiagnosed, these children should be periodically screened for GDD to enable early intervention and to maximize these patients' chances for academic, professional, and social success.[16,17,19,38] In addition, when managing a child with psychomotor delay, the contribution of epilepsy in its pathogenesis must be determined: Is it involved in the cognitive deficit? Is it spontaneous? Is it the consequence of an underlying neurological lesion? To understand it better, a series of clinical, electrophysiological and neuroradiology investigations as well as knowledge of the relation between age and the epileptic syndromes may be helpful.[39]

CONCLUSION

In our study population, seizures were common with generalized seizure being the predominant type in children with poor developmental delay. Suppression pattern was seen in 12 out of 70 patients (17.14%), commonly in temporal-parietal areas. EEG signatures such as suppression patterns could be early indicators of neuro-degeneration and serve as biomarkers to assess severity or as a prognostic indicator in developmental delay. Further, it would be worthwhile to explore if some areas of the brain are affected more than others and why.

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CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

ABBREVIATIONS

AGA: Advanced for Gestational Age; **EEG:** Electroencephalography; **GDD:** Global Developmental Delay; **HIE:** Hypoxic Ischemic Encephalopathy**; MCA:** Middle Cerebral Artery.

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