Electroencephalographic (EEG) correlation of clinical Seizure among Bangladeshi Patients

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Abstract

To detect the changes in electro encephalogram (EEG) and correlate the findings with clinical seizure events among epilepsy patients. This retrospective chart review was carried out in the electrophysiology laboratory of Dhaka Medical College Hospital from January 2011 to December 2013, which included 1154 patients. EEG was obtained through scalp electrodes following international 10/20 system. Information regarding patients was collected from the laboratory register with the help of a checklist. The EEG findings and clinical seizure events were then compared. Among the 1154 patients, age varied from birth to 75 years. The mean age at presentation was 17±11.4 years and most of the patients were less than 10 years old (44.4%). The male (59.2%) female ratio was 3:2 in our study. Clinically diagnosed seizure was present in 970 patients (84.1%), among which Generalized tonic clonic seizure (GTCS) was the most common clinical type of seizure, followed by secondary generalized seizure in 19.4% (n=224) and focal seizure in 2% (n=30) patients and 6.8% (n=79) patients had pseudoseizure. Among the abnormal EEG (59%), generalized epileptiform discharge was found in 29%, whereas focal and secondary generalized discharge was found in 30%. The most common site of origin of epileptiform discharge was temporal lobe (28.3%). There was a negative correlation between EEG and history of seizure events (pearson correlation significance 0.33). Our study brings out the fact that EEG has a negative correlation with clinical seizure events.

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Introduction

An epileptic crisis was first described in 3000 b.c. Hans Berger, a German psychiatrist in 1929 invented the human electroencephalogram (EEG). As Gibbs and his colleagues[1] discovered the pattern of epileptic discharges in 1935, its potential applications in epilepsy rapidly became clear. EEG has been playing a central role in diagnosing and managing epilepsy, as it is a convenient and relatively inexpensive way to demonstrate the physiological manifestations of abnormal cortical events that underlie epilepsy. Though there is debate with regards to definition of epilepsy, it is often defined as the tendency to have recurrent unprovoked seizure, whilst a seizure is any clinical event caused by abnormal electrical discharge in the brain[2]. There is wide variation in incidence of epilepsy worldwide[3] and the prevalence being higher in developing countries[4-6]. The life time prevalence of epilepsy also varies from 1.5 to 14 per 1000 population among different countries[7-15]. More than half of the people with epilepsy worldwide live in Asian countries. According to World Health Organization (WHO) there are 1.5-2 million people suffering

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from epilepsy in Bangladesh[16]. The scalp EEG may have a number of limitations, but it can help to determine the seizure types and epilepsy syndromes among patients with epilepsy. There is a significant association between the interpreted presence of epileptiform discharges (EDs) and a subsequent seizure[17,18]. The frequency of reported EEG abnormalities in patients with seizures varies widely between studies (29-69%)[19-21] and interestingly enough when EEG are repeated in the same subject, the results are often inconsistent[22,23]. The objective of our study was to determine the correlation of EEG findings with clinical seizure events among Bangladeshi patients.

Material and methods

This retrospective chart review was carried out in Electrophysiology Laboratory in Neurology Department of Dhaka Medical College Hospital (DMCH) from January 2011 to December 2013. Our study population included 1154 epilepsy patients. Consultant neurologists took meticulous history along with proper physical examination in the lab. All the information was recorded in black and white. Our selection criteria included all the cases with convincing history of seizure events, which were sent for EEG and those with complete set of desired information in hospital recored. They were advised to wash hair with shampoo and not to sleep overnight. EEG was done within one to six months of seizure event. Each recording of EEG were obtained through digital equipments with minimal duration of 20-30 minutes and electrode positioned on scalp according to international 10.20 system. Recording was done in both awake and sleep state, except those who didn't sleep, only awakened state recording was taken. Provocative stimuli like hyperventilation, photic stimulation were given for three minutes each. For standardization, the background activity was classified as normal (organized and symmetrical) or abnormal (disorganized and/or asymmetrical). The EEG was interpretated by consultant neurologist, trained and experienced in electro-physiologic studies. The EEG was examined for specific epileptiform abnormality, the interictal spike or sharp wave. The abnormal electroencephalographic activity was also classified as generalized or focal. The presence and topography of bursts of slow waves and epileptiform paroxysms were evaluated. The latter were classified as spike-wave, sharp-wave and polyspike. EEG was done within one month of last seizure and mostly after a single event. Though a total of 1187 patients were initially included, 33 patients with marked artifacts on EEG were excluded from this study.

Results

In our study, the patient's age ranged from birth to 75 years. The mean age at presentation to the EEG room was 17±11.4 years. About three fourth of the patients (72.9%) were aged less than 20 years old. Only 3.7% were older than 50 years (Table-1).

Table-1: Distribution of patients by age and sex (n=1154)

Age Group (in years)	Number	Percentage
<10	512	44.4
11-20	239	28.5
21-30	171	14.8
31-40	65	5.6
41-50	34	2.9
51-60	23	2
>60	20	1.7
Sex		
Male	683	59.2
Female	471	40.8

The male predominated in our study (59.2%) with a male to female ratio of 3:2. Clinically diagnosed seizure was present in 970 patients (84.1%). Generalized tonic clonic seizure (GTCS) was the most common clinical type of seizure encountered at the electrophysiology lab, followed by secondary generalized seizure in 19.4% (n=224) and focal seizure in 2% (n=30) patients. About 6.8% (n=79) patients had pseudoseizure (Table-2).

Table-2: Clinical seizure events (n=1154)

Parameter		Number	Percentage
Seizure Event			
	Present	970	84.1
	Absent	184	15.9
Seizure Types			
	Focal	30	2.6
	Secondary Generalized	224	19.4
	Generalized	641	55.5
	Pseudo seizure	75	6.7

EEG was found to be normal in 473 patients (41%). Among the abnormal EEG (59%), generalized epileptiform discharge was found in 29%, whereas focal and secondary generalized discharge was found in 30%. The most common site of origin of epileptiform discharge was temporal lobe (28.3%). The origin was hemispheric in 26.5%, paracentric in 17.6% and Frontal in 9.24%. We could not identify the site of origin in 63 patients (18.2%) (Table-3).

Table-3: Findings in EEG (n=1154)

Findings		Number	Percentage
Normal		473	41
Abnormal			
	GE	333	28.9
	LRE	346	30
	Site of epileptogenesis of LRE		
	Temporal	98	28.3
	Frontal	32	9.24
	Paracentral	61	17.63
	Hemispheric	92	26.5
	Un determined	63	18.2

The EEG report and history of seizure was negatively correlated with pearson correlation significance at two tail of 0.331 (Table-4).

Table-4: Correlation between seizure and EEG (n=1154)

		Seizure	EEG
Seizure			
	Pearson Correlation	1	-.331
	Significance (2 tailed)		<.01
EEG			
	Pearson Correlation	-.331	1
	Significance (2 tailed)		<.01

Discussion

Through there are many published reports regarding sensitivity and specificity of EEG, the direct analysis of correlation between EEG and clinical seizure events are lacking. Patients having epileptiform discharges on an interictal EEG are significantly more likely to have another seizure than those with normal results on EEG. This denotes the fact that, there is a significant association between the presence of epileptiform discharges and a subsequent seizure[24,25]. But, the frequency of reported EEG abnormalities in patients with seizures varies widely between studies. The sensitivities of EEG range from 29 to 69%[26-29]. In addition, when EEGs are repeated in the same subject, the results are often inconsistent[30]. Tests with wide interobserver or intersubject variability may yield inaccurate or uninterpretable results[31]. This variation from both the patients and observer side made us conducting the study in our own population.

The incidence of epilepsy varies in different age groups. A number of studies reported the incidence of epilepsy in specific age groups. These include studies of children,[32-38] adults,[39-41] and the elderly[42,43.] . In contrast to the common text book learning that "epilepsy is a disease with onset at the extremes of life", we found a higher proportion of patients less than 10 years of age and the elderly being very small in number. This finding is also in consistence with other studies, where age-specific incidence is consistently high in the youngest age groups and with highest incidence occurring during the first few months of life. Incidence falls dramatically after the first year of life, seems relatively stable through the first decade of life, and falls again during adolescence[44-46]. Male are more prone to have seizure. In most total population studies, incidence of epilepsy or of unprovoked seizures is higher in males than in females. This seems true even after the higher incidence in males of definitive risk factors for epilepsy (i.e., head injury, stroke, central nervous system infection) is taken into account[6-12,47,48]. For most but not all incidence studies, sex-specific differences in incidence are not statistically significant. The consistency of the male-to-female difference across studies suggests that males are at higher risk than females for unprovoked seizures and epilepsy.

Seizure-specific incidence or proportions of cases with a specific seizure type based on the International Classification of Epileptic Seizures are provided in several contemporary incidence studies[49-53]. The frequency of both partial seizure and GTCS varied among different studies, which might account for the difference in methodological and geographical perspective. Granieri et al[49], Olafsson et al[51] and Tekle-Haimanot et al[53] found a higher proportion of HTCS like our study. The probability that an IED indicates epilepsy relates to the location of the discharge. Only about 40% of individuals with central-mid-temporal spikes and 50% of those with occipital spikes have seizures [54-57]. A photoparoxysmal response and generalized spike–wave discharges are often found in asymptomatic individuals, who may carry the genetic trait for spike–wave discharges, but lack the susceptibility of seizure, which might be the explanation of these negative EEG. The photoparoxysmal response accounts for 63% of IEDs found in subjects without epilepsy, who are not apt to develop seizures later in life[54,58-60]. In contrast, people with midline, anterior temporal, midtemporal, and multifocal spikes usually have epilepsy; up to 90–95% of these individuals have epilepsy[54,58]. Studies on adult epilepsies showed that the chance of detecting interictal epileptiform discharges (IEDs) from the first EEG varies between 29% and 55%[59-61]. A Repeat EEG may ultimately demonstrated the IEDs in 80%-90% of the patients[61,63]. We had a similar yield of abnormal report in EEG. There is wide variation in sensitivity (23%-77%) and specificity (24%-99%) of reported EEG in general population[64-73]. Donald L et al[74] concluded three important findings related to EEG. "The first is that the sensitivity and specificity of EEG varies widely among published studies. The second finding is that a large proportion of this variance appears to be accounted for by the interpretation threshold of the neurologist readers. The third finding is that there is an uneven trade-off between specificity and sensitivity in EEG interpretations. Higher specificity readers had greater diagnostic accuracy in predicting seizure recurrence than did higher sensitivity readers". Due to this wide variation we wanted to draw some conclusion on the basis of direct correlation analysis between EEG and reported seizure events. We found a negative correlation between EEG and seizure event. This establishes the role of clinical judgment over the mere investigation report of EEG. As far as diagnosis is concerned, no investigation can replace the meticulous history from an eye witness of a seizure event.

Conclusion

Diagnosis of epilepsy is clinical and based mostly on the acquisition of correct history of seizure event. Though, the EEG is often regarded as a key supportive investigation with a reasonable sensitivity and specificity, we found a negative correlation between EEG and seizure events. Another interesting observation in our study was that in cases of secondary epilepsies, temporal lobe was the most common site of epileptogenesis.

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