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ORIGINAL ARTICLE

Factors Associated with Electroencephalographic Epileptiform Abnormalities in Epilepsy Patients: A Cross-sectional Study

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ABSTRAK

Elektroensefalogram (EEG) ialah salah satu alat diagnostik dalam penyakit epilepsi. Kami mengkaji faktor yang dikaitkan dengan keabnormalan EEG pada pesakit epilepsi. Ini ialah satu kajian rentas pusat tunggal yang dijalankan di Hospital Canselor Tuanku Muhriz. Populasi kajian terdiri daripada pesakit epilepsi tanpa penyakit neurodegeneratif, penyakit psikiatri, gangguan neurologi progresif, serta penyalahgunaan dadah atau keracunan. Data demografi mereka, jenis dan etiologi epilepsi, komorbiditi, ubat anti-kejang dan data EEG dikumpul. Seramai 183 pesakit yang didiagnos dengan epilepsi telah direkrut dalam kajian ini. Purata umur adalah 41.6 tahun (SD ± 17.8) dengan 94 (51.4%) lelaki dan 89 (48.6%) perempuan. Purata umur mula epilepsi adalah 30.4 tahun (SD ± 21.2). Etiologi yang paling biasa adalah keabnormalan struktur (134, 73.2%). Seramai 134 pesakit (73.2%) mempunyai keputusan EEG yang abnormal yang terdiri daripada epilepsi fokus (103, 57.3%) dan epilepsi umum (31, 16.8%). Faktor risiko yang dikaitkan dengan EEG abnormal adalah epilepsi fokus, sawan yang tidak terkawal, pengimejan otak yang abnormal, bilangan ubat anti-sawan, serta penggunaan carbamazepine dan levetiracetam. Penggunaan EEG dalam pesakit epilepsi telah membantu dalam aspek diagnosis, lokalisasi dan pengurusan. Kami melaporkan peratusan yang tinggi bagi EEG abnormal dalam kalangan pesakit epilepsi kami. Faktor risiko yang dikaitkan dengan EEG abnormal adalah setanding dengan literatur Barat. Data ini menekankan

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kepentingan pelaksanaan EEG sebagai sebahagian daripada siasatan rutin. Pengenalpastian faktor risiko EEG yang abnormal boleh membantu menentukan risiko kekambuhan, prognosis pesakit dan pengurusan seterusnya.
Kata kunci: *Elektroensephalogram; epilepsi; faktor risiko*

ABSTRACT

Electroencephalogram (EEG) is one of the diagnostic tools in epilepsy. We studied the factors associated with epileptiform abnormalities in epilepsy patients. This was a single-centre cross-sectional study conducted in Hospital Canselor Tuanku Muhriz, Malaysia. The study population was epilepsy patients without underlying neurodegenerative diseases, psychiatric illnesses, progressive neurological disorders and drug abuse or intoxication. Their demographic data, type and aetiology of epilepsy, comorbidities, anti-seizure medications and EEG data were collected. A total of 183 patients diagnosed with epilepsy were recruited in this study. The mean age was 41.6 years (SD ± 17.8) with 94 (51.4%) male and 89 (48.6%) female. The mean age of epilepsy onset was 30.4 years (SD ± 21.2). The most common aetiology was structural abnormalities (134, 73.2%). 134 patients (73.2%) had abnormal EEG results which consisted of focal epileptic discharges (103, 57.3%) and generalised epileptic discharges (31, 16.8%). The risk factors associated with abnormal EEG were focal epilepsy, uncontrolled seizures, abnormal brain imaging, the number of anti-seizure medications and the usage of carbamazepine and levetiracetam. The use of EEG in epilepsy patients has assisted in the diagnosis, localisation and management aspects. We reported a high proportion of abnormal EEG in our epilepsy patients. The risk factors associated with abnormal EEG were comparable to the Western literature. The data highlights the importance of performing the EEG as part of the routine investigations. The identification of the risk factors of abnormal EEG may help to determine the recurrence risk, prognosis of the patients and subsequent management.
Keywords: Electroencephalogram; epilepsy; risk factors

INTRODUCTION

Epilepsy is defined as a brain disorder which is characterised by an enduring predisposition to generate epileptic seizures (Fisher et al. 2014; Fisher et al. 2017). This may manifest as episodic disturbances in movement, consciousness or feeling. It is clinically defined by at least two unprovoked (or reflex) seizures occurring more than 24 hours apart; one unprovoked (or reflex) seizure and a probability of further seizures similar to the general recurrence risk (at least 60%) after two unprovoked seizures, occurring over the next 10 years or the diagnosis of an epilepsy syndrome. It is a common worldwide public health disease without any gender bias, affecting all ages with a bimodal distribution of incidence and an estimated 50 to 70 million people suffering worldwide, with numbers growing annually; approximately 2.4 million people diagnosed each year (Trinka et al. 2019).

Neurological disorders in general, ranked as the leading cause of disability-adjusted life years (DALYs) in 2015 (~250.7 million, comprising 10.2% of global DALYs) (Feigin 2017). Epilepsy represents the fourth most common neurological disorder in the United States after migraine, stroke and Alzheimer's

disease with a multitude of physical and economic implications (Hirtz et al. 2007). Closer to home (Fong et al. 2021), it is projected that 23 million people in Asia have epilepsy with its prevalence in this region standing at 1.5 to 14.0 in 1000 persons (Mac et al. 2007; Yemadje et al. 2011).

EEG is an electrophysiological method that measures voltage fluctuations resulting from ionic currents within the brain's neurons. Electroencephalogram (EEG) spectral analysis quantifies the amount of rhythmic (or oscillatory) activity of different frequencies in EEG (Zhang et al. 2023). Several methods have been formatted to analyse the EEG signals to determine the epileptic activity detection/ classification (Guo et al. 2010; Subasi 2007). The cerebral brain waves are divided based on their frequency into several sub-bands, being delta (< 4 Hz), theta (4-7 Hz), alpha (8-12 Hz), beta (13-30 Hz) and gamma (30-50 Hz).

EEG analysis can help in the diagnosis as it can analyse interictal EEG recordings (Liang et al. 2010). Bettus et al. (2008) found that during the interictal state in mesial temporal lobe epilepsy, there are decreased oscillations in the theta sub-band in the epileptogenic zone. EEG analysis also can correctly localise the ictal onset zone. EEG pattern analysis in epilepsy patients comprises characterisation and localisation of corticoelectrical activity. There is a lack of studies to determine the factors associated with abnormal EEG in Malaysian epilepsy patients. Thus, we embarked on this study to study the epilepsy population in a local tertiary teaching institution.

MATERIALS AND METHODS

Study Design and Study Population

This cross-sectional study was conducted between September 2021 and September 2023 in Hospital Canselor Tuanku Muhriz, Universiti Kebangsaan Malaysia (UKM) with approval by the UKM Research Ethics Committee (JEP-2021-693/FF-2021-424). The study population was recruited via universal sampling and patients had to fulfil the following criteria: Aged 18 years old and above with a diagnosis of epilepsy. The patients were excluded if they had underlying neurodegenerative diseases, psychiatric illnesses like schizophrenia, acute psychosis, progressive neurological disorder and drug abuse or intoxication.

Data Collection

The patients were selected based on specific inclusion criteria using purposive sampling. After obtaining informed consent, data were collected on their social demographics and epilepsy history. Epilepsy and seizure types were classified based on the International League Against Epilepsy (ILAE) classification in 2017.

Study Tools
- Electroencephalogram recording

The EEG was recorded on the Nicolet One Extension (V32 Amplifier, Natus Medical Incorporated, Middleton, USA) using 25 reusable gold electrodes affixed to the scalp according to the international 10-20 system. The abbreviations on the EEG were as follows: Fp- frontopolar, C-central, F-frontal, T-temporal, P-parietal, O-occipital. The interpretation of the EEG was performed by two neurologists who were blinded to the clinical and radiological data. Both the neurologists gave their independent report and if there were any discrepancies, the EEG record was reviewed again to determine a final report. The five main EEG frequency bands were determined during the EEG analysis. This consists of delta

(0.5-4 Hz), theta (4-8 Hz), alpha (8-13 Hz), beta (13-30 Hz), and gamma (30-100 Hz) across all channels for each subject.

Sample size

The sample size was calculated using the study by Baldin et al. (2014), in which the prevalence of EEG abnormalities among epilepsy patients was 12.0%. Using the formula below, with a confidence level of 95% and a maximum error permissible of 0.05, the sample size was 162 patients. Considering a 10.0% dropout, the total sample size required was 183 patients.

$$N = \frac{Z^2 \, p \, (1-p)}{d^2}$$

with,

Z = Degree of confidence 1.96 (95%)
p = prevalence (0.12)
d = Maximum error permissible (0.05)

Statistical Analysis

All the data collected was analysed using Statistical Package for Social Sciences version 26 (SPSS, IBM Corp, Armonk, NY). Descriptive statistical analyses were performed to analyse demographic and clinical characteristics, using frequencies and percentages to summarise categorical variables, medians and interquartile ranges for continuous variables. Inferential statistics were analysed using Kruskal Wallis for non-parametric continuous variables and Independent T-test for parametric continuous variables. The statistical significance was set at $p<0.05$.

RESULTS

We recruited a total of 183 patients diagnosed with epilepsy (Table 1). The mean age of patients in this study was 41.6 years (SD ± 17.8), comprising 94 males (51.4%) and 89 females (48.6%). When dividing the age in more detail, most of the epilepsy patients were in the 18-44 years old age group (112, 61.2%). The mean age of epilepsy onset was 30.4 years (SD ± 21.2). Most patients were Malay (103, 56.3%). There was nearly equivalent number of patients with focal epilepsy (90, 49.2%) and generalised epilepsy (93, 50.8%). About three-quarters of patients had epilepsy due to structural abnormalities (134, 73.2%), followed by genetic (12, 6.6%), infection (10, 5.5%), and autoimmune causes (3, 1.6%). 101 (55.2%) patients had abnormal brain imaging.

Table 2 showed anti-seizure medication usage and the EEG findings among study participants. Abnormal EEG was present in 134 (73.2%) patients while 49 patients (26.8%) had normal EEG. There were focal epileptic discharges in 103 (57.3%) and generalised epileptic discharges in 31 patients (16.8%). The median number of anti-seizure medications was 1 (1, 2). Almost all patients were on anti-seizure medication (75, 95.6%). The proportion of patients on one medication (102, 55.7%) followed by two medications (49, 26.8%), three medications (14, 7.7%), four medications (8, 4.4%) and five medications (2, 1.1%).

The most common comorbidity was hypertension (39, 21.3%), followed by diabetes mellitus (26, 14.2%), stroke (21, 11.5%), ischemic heart disease (5, 2.7%) and bronchial asthma (3, 1.6%). The median duration of epilepsy was 5 years (1, 19). The total duration of epilepsy among the participants was relatively equal. Slightly more than half of the patients were seizure-free (103, 56.3%), while the rest had recurrent seizures over the past year (80, 43.7%).

There was a significant association between the aetiology of epilepsy, type of epilepsy, brain imaging findings, seizure control for the past 1 year and the number of anti-seizure

TABLE 1: Demographic and clinical characteristics of patients in this study

Demographic/ Clinical characteristics	N=183	Normal EEG (n=49)	Abnormal EEG (n=134)
Age group, n (%)			
18 – 44	112 (61.2)	35 (71.4)	77 (57.5)
45 – 64	48 (26.2)	9 (18.4)	39 (29.1)
65 and more	23 (12.6)	5 (10.2)	18 (13.4)
Gender, n (%)			
Male	94 (51.4)	24 (49)	70 (52.2)
Female	89 (48.6)	25 (25)	64 (47.8)
Race, n (%)			
Malay	103 (56.3)	27 (55.1)	76 (56.7)
Chinese	56 (30.6)	14 (28.6)	42 (31.3)
Indian	21 (11.5)	6 (12.2)	15 (11.2)
Others	3 (1.6)	2 (4.1)	1 (0.7)
Type of epilepsy, n (%)			
Focal	90 (49.2)	19 (38.8)	71 (53.0)
Generalised	93 (50.8)	30 (61.2)	63 (47.0)
Aetiology of epilepsy, n (%)			
Genetic	12 (6.6)	1 (2.0)	11 (8.2)
Structural	134 (73.2)	31 (63.3)	103 (76.9)
Infection	10 (5.5)	4 (8.2)	6 (4.5)
Autoimmune	3 (1.6)	2 (4.1)	1 (0.7)
Others	24 (13.1)	11 (22.4)	13 (9.7)
Brain imaging findings, n (%)			
Normal	65 (35.5)	23 (46.9)	42 (31.3)
Abnormal	101 (55.2)	21 (42.9)	80 (59.7)
Not available	17 (9.3)	5 (10.2)	12 (9.0)
Medication status, n (%)			
No antiseizure medication	8 (4.4)	4 (8.2)	4 (3.0)
Single antiseizure medication	102 (55.7)	33 (67.3)	69 (51.5)
2 or more antiseizure medication	73 (39.9)	12 (24.5)	61 (45.5)
Concomitant diseases, n (%)			
Diabetes mellitus			
No	157 (85.8)	43 (87.8)	114 (85.1)
Yes	26 (14.2)	6 (12.2)	20 (14.9)
Hypertension			
No	144 (78.7)	40 (81.6)	104 (77.6)
Yes	39 (21.3)	9 (18.4)	30 (22.4)
Ischemic heart disease			
No	178 (97.3)	47 (95.9)	131 (97.8)
Yes	5 (2.7)	2 (4.1)	3 (2.2)
Stroke			
No	162 (88.5)	45 (91.8)	117 (87.3)
Yes	21 (11.5)	4 (8.2)	17 (12.7)
Bronchial asthma			
No	180 (98.4)	48 (98.0)	132 (98.5)
Yes	3 (1.6)	1 (2.0)	2 (1.5)
Epilepsy duration, n (%)			
≤ 5 years	93 (50.8)	26 (53.1)	67 (50.0)
> 5 years	90 (49.2)	23 (46.9)	67 (50.0)

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Seizure frequency/month			
No seizure	103 (56.3)	33 (67.3)	70 (52.2)
1-2 seizure	55 (30.1)	11 (22.4)	44 (32.8)
3-4 seizure	14 (7.7)	4 (8.2)	10 (7.5)
5 or more seizure	11 (6.0)	1 (2.0)	10 (7.5)
	Mean ± SD (95% CI)		
Age (years)	41.67 ± 17.84 (39.07 - 44.27)		
Age at epilepsy onset (years)	30.48 ± 21.28 (27.38 - 33.58)		
	Median (Percentile 25th, 75th)		
Number of anti-seizure medications	1 (1, 2)		
Seizure frequency per year	5 (1, 19)		
Duration of epilepsy (years)	0 (0, 12)		

EEG: Electroencephalogram; Continuous values were presented as mean ± standard deviation for normally distributed data and median (interquartile range) for non-normally distributed data

TABLE 2: Anti-seizure medications usage among study participants

Anti-seizure medications	No of participant N (%)	Normal EEG (n=49)	Abnormal EEG (n=134)
Clonazepam			
No	170 (92.9)	47 (95.9)	123 (91.8)
Yes	13 (7.1)	2 (4.1)	11 (8.2)
Clobazam			
No	179 (97.8)	49 (100)	130 (97.0)
Yes	4 (2.2)	0 (0)	4 (3.0)
Carbamazepine			
No	149 (81.4)	46 (93.9)	103 (76.9)
Yes	34 (18.6)	3 (6.1)	31 (23.1)
Diamox			
No	181 (98.9)	49 (100)	132 (98.5)
Yes	2 (1.1)	0 (0)	2 (1.5)
Gabapentin			
No	183 (100)	49 (100)	134 (100)
Yes	0 (0)	0 (0)	0 (0)
Levetiracetam			
No	75 (41.0)	32 (65.3)	43 (32.1)
Yes	108 (59.0)	17 (34.7)	91 (67.9)
Lamotrigine			
No	158 (86.3)	40 (81.6)	118 (88.1)
Yes	25 (13.7)	9 (18.4)	16 (11.9)
Perampanel			
No	176 (96.2)	49 (100.0)	127 (94.8)
Yes	7 (3.8)	0 (0)	7 (5.2)
Phenytoin			
No	151 (82.5)	40 (81.6)	111 (82.8)
Yes	32 (17.5)	9 (18.4)	23 (17.2)
Phenobarbitone			
No	179 (97.8)	48 (98.0)	131 (97.8)
Yes	4 (2.2)	1 (2.0)	3 (2.2)

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Topiramate			
No	177 (96.7)	45 (91.8)	132 (98.5)
Yes	6 (3.3)	4 (8.2)	2 (1.5)
Valproate			
No	136 (74.3)	35 (71.4)	101 (75.4)
Yes	47 (25.7)	14 (28.6)	33 (24.6)
Zonisamide			
No	181 (98.9)	49 (100.0)	132 (98.5)
Yes	2 (1.1)	0 (0)	2 (1.5)

EEG: Electroencephalogram

medications with EEG abnormality. As shown in Table 3, there was a statistically significant association between type of epilepsy and EEG abnormality (p=0.045), with more EEG abnormal findings in focal epilepsy. Epilepsy due to structural abnormalities had significantly higher EEG abnormalities as compared to those with non-structural causes. Those with poorly controlled seizures had more abnormal EEG as compared to those with good seizure control. Among the anti-seizure medications, carbamazepine and levetiracetam had a significant association with abnormal EEG. Topiramate, on the other hand, was associated with normal EEG. The following variables such as age, age of onset of epilepsy, gender, race, comorbidities and duration of epilepsy, have no association with EEG abnormality. Using multivariate analysis (Table 4), abnormal brain imaging, uncontrolled seizure, the usage of carbamazepine and levetiracetam and the number of anti-seizure medications had a positive correlation with EEG abnormality. Whereas the type of epilepsy and the usage

TABLE 3: Associations between sociodemographic, clinical factors and type of anti-seizure medications with EEG abnormality

Variables	EEG	Mean Rank	U	Z	p-value
Gender	Normal	89.82	3176.000	-0.390	0.348
	Abnormal	92.80			
Type of epilepsy	Normal	101.52	2816.500	-1.698	0.045
	Abnormal	88.52			
Brain imaging findings	Normal	81.55	2771.000	-1.947	0.026
	Abnormal	95.82			
Medication status	Normal	88.53	3113.000	-0.429	0.334
	Abnormal	93.27			
Diabetes mellitus	Normal	90.20	3195.000	-0.459	0.323
	Abnormal	92.66			
Hypertension	Normal	89.31	3117.500	-0.587	0.279
	Abnormal	92.99			
Ischemic heart disease	Normal	93.23	3222.500	-0.675	0.250
	Abnormal	91.55			
Stroke	Normal	88.97	3134.500	-0.848	0.198
	Abnormal	93.11			

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Bronchial asthma	Normal Abnormal	92.37 91.87	3265.000	-0.258	0.398
Duration range of epilepsy	Normal Abnormal	89.95 92.75	3182.500	-0.366	0.358
Seizure control for past 1 year	Normal Abnormal	81.88 95.70	2787.000	-1.819	0.034*
Clonazepam	Normal Abnormal	89.23 93.01	3147.500	-0.960	0.169
Clobazam	Normal Abnormal	90.00 92.73	3185.000	-1.220	0.111
Carbamazepine	Normal Abnormal	80.60 96.17	2724.500	-2.613	0.004*
Diamox	Normal Abnormal	91.00 92.37	3234.000	-0.858	0.196
Gabapentin	Normal Abnormal	92.00 92.00	3283.000	0.000	0.500
Levetiracetam	Normal Abnormal	69.74 100.14	2192.500	-4.035	<0.001*
Lamotrigine	Normal Abnormal	96.31 90.43	3072.000	-1.118	0.132
Perampanel	Normal Abnormal	88.50 93.28	3111.500	-1.627	0.052
Phenytoin	Normal Abnormal	92.81 91.71	3243.500	-0.189	0.425
Phenobarbitone	Normal Abnormal	91.87 92.05	3276.500	-0.081	0.468
Topiramate	Normal Abnormal	96.47 90.37	3064.000	-2.238	0.013*
Valproate	Normal Abnormal	94.64 91.03	3153.500	-0.539	0.295
Zonisamide	Normal Abnormal	91.00 92.37	3234.000	-0.858	0.196

Kruskal Wallis

	H	df	p
Age groups	3.002	2	0.223
Race	2.562	3	0.464
Etiology of epilepsy	10.702	4	0.030
Seizure frequency range	4.496	3	0.213
Number of anti-seizure medication	7.755	2	0.021

Independent t-Test

	t	df	p
Age (years)	-0.635	181	0.263
Age at epilepsy onset (years)	-0.09	181	0.464
Duration of epilepsy (years)	-0.743	181	0.229

*p-value significant at <0.05; EEG: Electroencephalogram; U: Mann-Whitney test; Z: Z value; t: t statistics; H: Kruskal Wallis test; df: degree of freedom

TABLE 4: Relationship between sociodemographic, clinical features and anti-seizure medication with EEG abnormality

	1	2	3	4	5	6	7
1. EEG abnormality							
2. Type of epilepsy	-0.126*						
3. Carbamazepine	0.194**	-0.148*					
4. Levetiracetam	0.299**	-0.086	-0.059				
5. Topiramate	-0.166*	-0.064	-0.088	-0.034			
6. Seizure control	0.135*	-0.059	0.230**	0.130*	0.085		
7. Imaging finding	0.144*	-0.205**	0.002	0.055	0.073	0.056	
8. Number of ASM	0.204**	-0.167*	0.330**	0.419**	0.161*	0.189**	0.136*

* Correlation is significant at the 0.05 level (2-tailed)
** Correlation is significant at the 0.01 level (2-tailed)

of topiramate have a negative correlation with EEG abnormality.

DISCUSSION

This study evaluated the relationship between the demographics and clinical characteristics of epilepsy patients with abnormal EEG. EEG plays an important role in detecting epilepsy by measuring the voltage changes between electrodes and providing temporal and spatial brain information (Pachori & Patidar 2014). Detection of EEG abnormalities often requires a thorough and significant amount of time and effort by the neurologist. It is a qualitative measure which can have subjective interobserver variability based on individual experience. The role of EEG in epilepsy patients is to determine the type, classification, aetiology of epilepsy, prognostication and presurgical evaluation. However, a normal EEG does not exclude epilepsy and may require repeated monitoring. The detection of interictal epileptiform abnormalities was 53% after the first EEG and 72% after the third EEG (Baldin et al. 2014). The presence of epileptiform abnormalities on EEG is related to seizure disorders and is an important factor in determining the risk of recurrence after a single unprovoked seizure (Debicki 2017). The number of seizures of all types at presentation, the presence of a neurological disorder, and an abnormal EEG were significant factors in indicating future seizures (Kim et al. 2006).

The proportion of abnormal EEG in our study population was 73.2%. Our reported figures were comparable to a hospital-based prevalence study of seizures among children in India, with abnormal EEG in 60% of patients (Das et al. 2022). A study from Somalia reported abnormal EEG study was seen in 77.45% of pediatric epilepsy patients (Elmi et al. 2024). Population studies in Rochester, Minnesota found epileptiform abnormalities on the first EEG recording in 52.7% (Baldin et al. 2014). Similarly, abnormal EEG findings were observed in 55.2 % of Ethiopia patients with epilepsy that consisted of interictal epileptiform discharges and focal/generalised slowing (Ayele et al. 2022). The variations in the reports may be attributed to the different aetiology, subtypes of epilepsy population and types of research methods.

The factors associated with abnormal EEG were further analysed in our study population. Among the epilepsy characteristics, focal

epilepsy and uncontrolled seizures were significant associated factors for abnormal EEG. Up to 90-95% of patients with epilepsy have abnormal EEG consisting of spikes in the midline, anterior temporal, mid-temporal and multifocal spikes (Ehle et al. 1981; Hughes 1985). The presence of focal sharp waves in the centrotemporal or occipital regions has a moderate association with clinically active epilepsy (Smith 2005). Focal spikes and focal slow waves are more often seen in uncontrolled epilepsy patients (Hughes & Fino 2003). Focal epilepsy is also more likely to develop drug-resistant epilepsy (DRE). DRE is defined as the failure of adequate trials of two tolerated, appropriately chosen, and used antiepileptic drug schedules (whether as monotherapies or in combination) to achieve sustained seizure freedom (Kwan et al. 2010). Focal epilepsy is more likely to be resistant to antiseizure therapy, particularly in structural such as cortical dysplasia, mesial temporal sclerosis or tuberous sclerosis (Fattorusso et al. 2021). Uncontrolled seizures may be attributed to several reasons, such as poor compliance, wrong medication, misdiagnosis and DRE (Asadi-Pooya et al. 2013). Our patients with uncontrolled seizures were likely to have abnormal EEG. This concurs with reported findings from a cross-sectional study in Africa that reported 58% of generalised epilepsy patients had abnormal EEG (Owolabi et al. 2018). The independent predictors of EEG abnormality include age, poor seizure control and high frequency of seizures (Owolabi et al. 2018). A previous study in hospitalised patients in Malaysia found that focal seizures had a 2.24 higher risk of having EEG abnormalities (Satar et al. 2023).

The relationship between brain imaging and EEG findings has been explored in research to determine their significance. Brain magnetic resonance imaging (MRI) is often performed in focal onset seizures to detect underlying structural lesions. T2-weighted imaging (T2WI) and diffusion-weighted imaging (DWI) signal abnormalities are often seen in cases with a cortico-subcortical distribution ipsilateral to the epileptogenic hemisphere. Our study highlighted abnormal brain imaging as an associated factor of abnormal EEG. This was similar to a study in Kenya, which found that inter-ictal epileptiform activity on EEG was associated with abnormal imaging findings (Samia et al. 2021). Velioğlu et al. (1997) observed a correlation between MRI-CT abnormalities and EEG findings. Conversely, some studies did not find any significant correlation between MRI and EEG findings (Elmi et al. 2024). This variable findings in the analysis may be limited by the availability of MRI imaging in resource-limited settings and the cause of the epilepsy.

The usefulness of EEG as a tool in managing seizure disorders remains significant. Studies in children with epilepsy found that persistent interictal epileptic activity has been associated with a higher risk of seizure relapse during discontinuation of antiseizure medications (Duncan 1987). Our data reported that an increased number of antiseizure medications and the use of carbamazepine and levetiracetam have been associated with abnormal EEG. Our findings were similar to Ethiopian epileptic patients on phenobarbitone, phenytoin, carbamazepine and those on two or more antiseizure medications have a higher proportion of abnormal EEG findings (Ayele et al. 2022). On the contrary, a double-blind study comparing carbamazepine and placebo found no significant change in the number of fits nor was there any apparent relation between the frequency of fits and EEG findings (Pryse-Phillips & Jeavons 1970).

Carbamazepine works by blocking the voltage gated sodium channel while

levetiracetam's mode of action involves the modulation of neurotransmitters released through binding to the synaptic vesicle glycoprotein 2A. Both are approved for the treatment of focal-onset epilepsy (Beydoun et al. 2020; Celdran de Castro et al. 2023). Both older and newer antiseizure drugs are likely to improve seizure control with the advantage of lesser side effects in the latter. Patients who are on multiple antiseizure drugs are more likely to be refractory and have abnormal EEG findings. A study on the effect of carbamazepine on background EEG showed that there was a significant increase in slow-wave activity (Marciani et al. 1993). A quantitative EEG analysis of neurological patients on carbamazepine 400 g twice daily for 35 days showed that the mean values of the total power and relative powers of the theta and delta bands were increased (Besser et al. 1992). The use of levetiracetam has been associated with the acceleration of background EEG frequencies (Cho et al. 2012). Bouma et al. (2016) reported that the presence of epileptiform discharges on routine EEG after a first unprovoked seizure has a 77% probability of having a second seizure. The presence of abnormal EEG helps in the management and prognosis of epilepsy as it may herald the likelihood of developing recurrent seizures.

Limitations

This study has some limitations as it does not have control arm to compare with epilepsy patients. Secondly, this study was conducted in a single tertiary centre, which may not reflect the underlying epilepsy population in the country. Finally, the EEG recording was only performed once. A repeated EEG may increase the yield of epileptiform abnormalities.

CONCLUSION

Abnormal EEG was present in 73.2% of epilepsy patients in this tertiary university hospital. The risk factors associated with abnormal EEG were focal epilepsy, uncontrolled seizures, abnormal brain imaging, the usage of carbamazepine and levetiracetam and the number of anti-seizure medications. The identification of the risk factors of abnormal EEG helps in the determination of the recurrence risk, prognosis and management of patients.

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