Characteristics of electroencephalogram changes and correlation with seizures in hospitalised patients

Siti Nur Aisyah Satar[1], Shasi Mogan[1], Wan Putri Nursyuhada Jaafar[1], Sharveenraaj Maghalingam[1], Fadzil Afiq Ruslan Affendi[1], Chen Fei Ng, FRCP[1], Ching Soong Khoo, FRCP[1], Yong Chuan Chee, MRCP[3], Rozita Hod, PhD[2], Hui Jan Tan, FRCP[1]

[1]Department of Medicine, Faculty of Medicine, Universiti Kebangsaan Malaysia, Kuala Lumpur, Malaysia, [2]Department of Community Health, Faculty of Medicine, Universiti Kebangsaan Malaysia, Kuala Lumpur, Malaysia, [3]Department of Medicine, School of Medical Sciences, Universiti Sains Malaysia, Kota Bahru, Malaysia

ABSTRACT

Introduction: Electroencephalogram (EEG) is an important investigational tool that is widely used in the hospital settings for numerous indications. The aim was to determine factors associated with abnormal EEG and its clinical correlations in hospitalised patients.

Materials and Methods: Patients with at least one EEG recording were recruited. The EEG and clinical data were collated.

Results: Two hundred and fifty patients underwent EEG and 154 (61.6%) were found to have abnormal EEG. The abnormal changes consist of theta activity (79,31.6%), delta activity (20, 8%), focal discharges (41,16.4%) and generalised discharges (14, 5.6%). Older patients had 3.481 higher risk for EEG abnormalities, *p*=0.001. Patients who had focal seizures had 2.240 higher risk of having EEG abnormalities, *p*<0.001. Low protein level was a risk for EEG abnormalities, *p*=0.003.

Conclusion: This study emphasised that an abnormal EEG remains a useful tool in determining the likelihood for seizures in a hospital setting. The risk factors for EEG abnormality in hospitalised patients were age, focal seizures and low protein level. The EEG may have an important role as part of the workup in hospitalised patients to aid the clinician to tailor their management in a holistic manner.

KEYWORDS:
Electroencephalogram, hospital

INTRODUCTION

Electroencephalogram (EEG)is a safe and non-invasive investigation to record electrical cerebral activity[1] and plays an important diagnostic and therapeutic role in neurological diseases. The advent of EEG by Hans Berger in 1929 began when he recorded cortical oscillatory activity from the surface of the skull in humans.[2] Scalp electrodes record the electrical brain activity which reflects the summation of excitatory and inhibitory postsynaptic potentials in apical dendrites of pyramidal neurons in the more superficial layers of the cortex.[3]

The association between cortical frequency bands of delta (1–4 Hz), theta (4–7 Hz), alpha (8–12 Hz), beta (12–28 Hz) and gamma (>30 Hz) oscillations with different behavioural and disease states have been explored.

EEG enables the assessment of neural activity of the cerebral cortex in normal and disease states. It is widely used in hospital settings for numerous conditions such as epilepsy, delirium, encephalopathy,[4] drug toxicity, status epilepticus and treatment monitoring. Microstates in resting state EEG for diagnosis of neurological disorders such as schizophrenia, dementia, and depression have been studied by Khanna et al.[5] The diagnostic validation of brain death in France requires two EEG recordings that showed electrocerebral inactivity.[6] Long-term EEG monitoring aids in the detection of epileptiform activity in high-risk seizure-free individuals.[7]

Hospitalised patients are prone to various co-morbidities such as infection, malnutrition, altered mentation, and drug effects. The detection of cerebral dysfunction of hospitalised patients can be easily demonstrated by performing the EEG. Continuous EEG monitoring had been found to be associated with reduced in-hospital mortality.[8] Detection of non-convulsive seizures and non-convulsive status epilepticus using continuous EEG are important in critically ill patients.[9] Seizures are invariably associated with clinical[10], metabolic[11] and electrophysiological changes.[12]

Well-defined EEG patterns have been associated with specific conditions and outcomes in encephalopathic patients.[4,13] Pathologic EEG patterns have been identified in hospitalised patients with encephalopathy. Frontal intermittent rhythmic delta activity and triphasic waves were associated with past cerebrovascular accidents and liver or multiorgan failure, respectively.[4]

Many investigations are performed in hospital to determine the diagnosis and management of the patients. The role of EEG in hospitalised patients is still underutilised. There is still a paucity of data on the variable factors that affect EEG changes in hospitalised patients. The aim of this study was to determine the patterns and associations of abnormal EEG patterns in hospitalised patients in a tertiary hospital.

This article was accepted: 04 February 2023
Corresponding Author: Hui Jan Tan
Email: tanhuijan@ukm.edu.my

MATERIALS AND METHODS

This was a retrospective review carried out at the Neurology Laboratory, Universiti Kebangsaan Malaysia Medical Centre from October 2021to October 2022. The study was approved by the local Institution Research and Ethics Board (FF-2021-366). This study was carried out with written informed consent from all the subjects in accordance with the Declaration of Helsinki. We included hospitalised patients in the medical wards who had performed at least 1 EEG recording while patients with acute psychosis, nonepileptic seizures,critically ill patients, brain trauma and severe agitation were excluded. The patients were recruited by the purposive sampling method.

EEG was conducted in the awake state following application of surface electrodes according to the 10-20 system. Hyperventilation and photic stimulation activation procedures were carried out. The montages may be adjusted accordingly during the interpretation of the EEG. The EEG records were obtained using the filters of 1 Hz high-pass, 30 Hz low-pass and 60 Hz notch filters at a speed of 30 mm/s. Results from the routine scalp EEG recording were obtained through the EEG records reported by two neurologists and inter-rater agreement was determined. The report was classified as normal or abnormal. Normal EEG consists of 8–13 Hz alpha rhythm. Abnormal EEG findings include the following changes: 4–7 Hz theta activity, less than 3.5 Hz delta/slow activity, focal discharges or generalised discharges. The data of the patients were obtained from their records to determine the demographics, comorbidities, types of seizures, causes of seizures and investigations for seizures such as brain imaging such as computed tomogram or magnetic resonance imaging. The causes of seizures were classified into structural, infection, genetic, metabolic, immunologic and drugs. A structural cause refers to abnormalities visible on structural neuroimaging. A known infection cause refers to seizures which are a core symptom of the disorder. A genetic cause results directly from a known or presumed genetic disorder. A known or presumed metabolic disorder in which seizures are a core symptom of the disorder. An immune cause results directly from an immune disorder in which seizures are a core symptom of the disorder. A drug cause results directly from a known or presumed drug aetiology.

Statistical Analysis

Data were explored and analysed using SPSS software version 21.0. Numerical variables were presented using mean and standard deviation. The data were checked for normality. Categorical variables were presented as frequency and percentage. Distributions of continuous variables were compared using Student's t-tests; Pearson's chi-square tests or Fisher's exact tests were used for distributions of categorical variables. Statistical significance was defined by a p value of less than 0.05. Simple and multiple logistic regressions were used to determine the factors associated with electroencephalogram abnormalities. All odds ratios (ORs) are presented with 95% confidence intervals (CI).

RESULTS

Out of 250 patients, 131(52.4%) were male while females contributed 119 (47.6%). The highest group was contributed by patients in the age range between 61 and70 years(43,17.2%), followed by 31 to 40 years (39, 16.6%) and 71 to 80 years (38, 15.2%). The Malay ethnicity accounted for 122 (48.8%), followed by Chinese 93 (37.2%), Indian 29 (11.6%), and others 6 (2.4%). %). The main diagnoses for the patients were post-stroke seizures, meningoencephalitis, epilepsy with breakthrough seizures and sepsis. As for the distribution of electroencephalogram abnormalities, 154 (61.6%) were found to be abnormal readings while 96 (38.4%) had normal EEG. The distribution of EEG changes consists of normal (96,38.4%), theta activity (79,31.6%), delta activity (20, 8%), focal discharges (41,16.4%) and generalised discharges (14, 5.6%). The brain imaging findings consisted of cerebral atrophy, tumour, abscess, stroke, encephalomalacia, neurofibroma, meningeal enhancement and normal.

Table I shows the demographics of patients with and without EEG abnormalities. There was significant association between age, race, seizure type and brain imaging with EEG abnormalities.

Table II shows clinical parameters in patients with and without EEG abnormalities. There was no significant association between causes of seizure, laboratory parameters and EEG abnormalities. However, only protein level was significantly associated with EEG abnormalities (p<0.001).

The distribution of patient characteristics according to seizure types was as follows: no seizures (147, 58.8%), generalised seizures (73, 29.2%) and focal seizures (30, 12%). The proportion in the young age group (15–64) years were no seizures (83, 49.4%), generalised seizures (61,36.3%) and focal seizures (24, 14.3%). In comparison, the old age group (65–95) years were no seizures (64, 78%), generalised seizures (12, 14.6%) and focal seizures (6, 7.3%). Only age had significant association with seizure types (p<0.001). Both gender and race did not show any significant difference. The proportion of male to female in the group with no seizures were (81, 55.1%; 66, 44.9%), generalised seizures (37, 50.7%; 36, 49.3%), and focal seizures (13, 43.3%; 17, 56.7%). The proportion of Malay to non-Malay in the group with no seizures was(78, 53.1%; 69, 46.9%), generalised seizures (37, 50.7%; 36, 49.3%) and focal seizures (13, 43.3%; 17, 56.7%).

Table III shows the risk factors associated with EEG abnormalities. In simple logistic regression, the risk factors associated with EEG abnormalities were age, race, hypertension, brain imaging, focal seizures and protein level (p<0.005). Multiple logistic regression demonstrated that older patients had 3.481 higher risk than younger patients of having EEG abnormalities (adjusted OR=3.481; 95% CI 1.615, 7.500, p=0.001). Patients who had focal seizures had almost 2.240 higher risk of having EEG abnormalities (adjusted OR=2.240; 95% CI 1.425, 3.521, p<0.001). Low protein level has a significant risk with EEG abnormalities (adjusted OR=0.409; 95% CI 0.229, 0.731, p=0.003).

Table I: Demographics of patients with and without EEG abnormalities

	Without EEG abnormalities N=96		With EEG abnormalities N=154		*p* value
	Mean (SD)	n (%)	Mean (SD)	n (%)	
Sociodemographics					
Age (years)	46.31 (17.67)	96 (38.40)	56.47 (20.80)	154 (61.60)	<0.001 [a]
Young (15-64)		80 (83.3)		88 (57.1)	
Old (65-95)		16 (16.7)		66 (42.9)	
Gender					
Male		54 (56.20)		77 (50.0)	0.336 [b]
Female		42 (43.80)		77 (50.0)	
Race					
Malay		56 (58.3)		66 (42.9)	0.034 [b]
Chinese		32 (33.3)		61 (39.6)	
Indian		8 (8.3)		21 (13.6)	
Others		0 (0)		6 (3.9)	
Seizure type					
None		61 (63.5)		86 (55.8)	0.033 [b]
Generalized		30 (31.3)		43 (27.9)	
Focal		5 (5.2)		25 (16.2)	
Brain imaging					
Normal		37 (48.1)		40(51.9)	0.037 [b]
Abnormal		58 (34.1)		112 (65.9)	

[a]Student's t test
[b]Pearson's Chi-Square test

Table II: Clinical parameters in patients with and without electroencephalogram abnormalities

	Without EEG abnormalities N=96		With EEG Abnormalities N=154		*p* value
	Mean (SD)	n (%)	Mean (SD)	n (%)	
Causes of seizure					
Structural	46.31 (17.67)	96 (38.40)	56.47 (20.80)	154 (61.60)	<0.001 [a]
No		74 (39.6)		113 (60.4)	0.511 [a]
Yes		22 (34.9)		41 (65.1)	
Infection					
No		34 (31.2)		75 (68.8)	0.23 [a]
Yes		6 (20.0)		24 (80)	
Genetic					
No		36 (29.3)		87 (70.7)	1 [b]
Yes		4(25.0)		12 (75)	
Metabolic					
No		40 (29.9)		94 (70.1)	0.321 [b]
Yes		0 (0.00)		5 (100)	
Immunologic					
No		96(39.0)		150(61)	0.301 [b]
Yes		0 (0.00)		4 (100)	
Drugs					
No		96 (38.9)		151(61.1)	0.288 [b]
Yes		0 (0.00)		3(100)	
Laboratory parameters					
Haemoglobin	12.93 (2.67)		12.81 (8.38)		0.455 [c]
White cell count	11.43 (13.62)		11.53 (7.59)		0.779 [c]
Platelet	277.79 (107.86)		271.23 (123.26)		0.225 [c]
Urea	5.29 (4.40)		11.59 (47.78)		0.101 [c]
Creatinine	126.55 (202.69)		125.02 (146.98)		0.647 [c]
Protein	73.11 (8.41)		68.05 (10.84)		<0.001 [c]
Alanine transaminase	31.31 (28.49)		34.79 (33.15)		0.248 [c]

[a] Pearson's Chi Square Test
[b] Fisher's Exact Test
[c] Student's test

Table III: Risk factors associated with electroencephalogram abnormalities

Variables	Simple logistic regression			Multiple logistic regression		
	b	Crude OR (95% CI)	p	b	Adjustment OR (95% CI)	p
Age	1.322	3.750 (2.08–7.002)	<0.001	1.247	3.481(1.615–7.500)	0.001
Race	-0.624	0.536(0.320–0.898)	0.018	–0.244	0.784 (0.439–1.399)	0.410
Hypertension	-0.705	0.494(0.293–0.835)	0.008	0.339	1.404 (0.729–2.703)	0.310
Brain imaging	0.580	1.786(1.033–3.090)	0.038	0.000	1.000(0.973–1.029)	0.983
Focal seizures	1.266	3.547(1.286–9.783)	0.014	0.806	2.240 (1.425–3.521)	<0.001
Protein level	–1.836	0.159(0.055–0.466)	0.001	–0.893	0.409(0.229–0.731)	0.003

OR, odds ratio; b, regression coefficient; CI, confidence interval

Table IV: Distribution of patients according to seizure occurrence

	No seizures	Seizure	*p* value
	N=142	N=105	
	n (%)	n (%)	
Age (years)			
Young (15–64)	81 (57.0)	86 (81.9)	<0.001 [a]
Old (65–95)	61(43.0)	19 (18.1)	
Gender			
Male	79(54.5)	52(49.5)	0.438 [a]
Female	66(45.5)	53(50.5)	
Race			
Malay	76 (53.5)	51(48.6)	0.442 [a]
Non-Malay	66(46.5)	54(51.4))	
Hypertension			
No	59(41.5)	75(71.4)	<0.001 [a]
Yes	83 (58.5)	30 (28.6)	
Diabetes mellitus			
No	84(59.2)	89 (84.8)	<0.001 [a]
Yes	58(40.8)	16 (15.2)	

[a]Student's t test.

Table V: Risk factors associated with seizures occurrence

Variables	Simple logistic regression			Multiple logistic regression		
	b	Crude OR (95% CI)	*p*	b	Adjustment OR (95% CI)	*p*
Age	–1.226	0.293 (0.161–0.533)	<0.001	–0.775	0.461(0.190–0.750)	0.027
Hypertension	1.258	3.517 (2.051–6.030)	<0.001	–0.587	0.556(0.289–1.068)	0.078
Diabetes mellitus	1.346	3.841 (2.048–7.202)	<0.001	–0.973	0.378 (0.190–0.750)	0.005

OR, odds ratio; b regression coefficient; CI, confidence interval.

Table IV shows the distribution of patient characteristics according to seizure occurrence. Age, hypertension and diabetes mellitus have a significant association with developing seizures.

Table V presents the risk factors associated with seizure occurrence. Multiple logistic regression showed that risk factors included age and diabetes mellitus.

DISCUSSION

This study focussed on hospitalised patients in a tertiary hospital in Malaysia. We reported a prevalence of 61.6% EEG abnormalities in our cohort of patients. Another study from a tertiary centre from Karachi[14] obtained EEG records from consecutive patients from the neurology department quoted almost similar results with 60.2% of patients with abnormal EEG records. Another hospital-based setting studyfound abnormal EEG patterns in patients with altered mental status who were subdivided into structural causes (brain atrophy, white matter abnormalities, strokes) and non-structural causes (organ failures, intoxication, infections).[4] However, previous studies only conducted EEG on a selected group of patients such as intensive care patients,[15] epilepsy,[16] psychiatric[17] and encephalopathic[18] patients.

The type of EEG abnormalities found in this study was comparable to other studies. The proportion of theta activity (31.6%), delta activity (8%), focal discharges (16.4%) and generalised discharges (5.6%). Apart from theta and delta activity, Sutter et al[4] reported findings of triphasic waves 22% and frontal intermittent rhythmic delta activity (FIRDA) 17%. Younger patients were also more likely to have FIRDA and delta activity. The EEG changes obtained from a cohort of inpatients from a tertiary centre found diffuse neuronal dysfunction in 45.2% and mild neuronal dysfunction accounted for 33.5%.[14] A Nigerian based study had found 56% of patients with epileptiform activity[17] in a psychiatric-based hospital. A case–control study of EEG microstate analysis found a decreased in the microstate stability in the inpatient encephalopathy group.[19] Our study reported higher proportion of abnormal EEG as it included a heterogenous

pool of hospitalised patients who were admitted for various medical conditions.

The type of EEG abnormality has been shown to be associated with risk of seizures. In a multicentre cohort study of critically ill adult patients, EEG monitoring that showed lateralised periodic discharges, lateralised rhythmic delta activity, and generalised periodic discharges were associated with seizures.[18] On the contrary, generalised rhythmic delta activity had no association with seizures. Our study determined that focal seizures are invariably linked to the presence of EEG abnormalities. Focal-onset seizures originate from one hemisphere and may be discretely localised to a particular site. The patients who had focal seizures were found to have almost 2.240 higher risk of having EEG abnormalities. Similarly, another study by Manford et al found 75.9% had EEG abnormalities in focal seizures.[10]

Our findings emphasised that focal seizures had higher risk to develop EEG abnormalities. Temporal lobe epilepsy is the most common focal epilepsy, and therefore, interictal temporal spikes or sharp waves are commonly observed. Focal seizures are likely to have interictal epileptic discharges and lateralised ictal EEG changes.[20] The use of ictal EEG adequately localises in 72% of cases, largely in temporal epilepsy rather than extratemporal epilepsy. Localised ictal onsets were observed in 57% of seizures.[20] The presence of focal spikes and focal slow waves on EEG also predicts the likelihood of developing uncontrolled seizures.[21]

From our study, the age-related EEG abnormalities were more significant in older patients compared to younger patients. There is a progressive change in brain wave frequency, power, morphology and distribution during rest with ageing.[22] In a study of pathological brain on EEG changes, elderly people showed decrease in alpha oscillatory activity and alpha rhythm reactivity as well as slowing of the background activity, with an increase in delta or theta power diffusely or in posterior region rhythm abnormalities, which are linked to poor cognitive performance.[22] Jabes et al reported the resting-state brain activity of healthy older adults (65–75 years old) exhibited lower theta-band and alpha-band and absolute powers, and higher beta-band and gamma band relative powers were observed compared to healthy young adults (20–30 years old).[23] A study of ageing-related changes of EEG synchronisation revealed differences in old and young adults during working memory task.[24] It was observed that older adults had lower EEG synchronisation in alpha 1, alpha 2 and beta frequency bands which reflects the decline in cognitive function.[24] The study's findings concurred with previous epidemiological studies that showed that elderly population has a high incidence and prevalence of epilepsy.[25] The elderly population are prone to seizures due to the various comorbidity that includes stroke, brain tumours, infections, head trauma, dementia and metabolic-toxic syndromes. The utilisation of EEG to determine changes in the neuropsychological aspects has improved the understanding of diseases in the elderly.

The effects of nutrition on cognitive function have been well recognised. Our study has revealed that protein level was a risk factor for EEG abnormalities in hospitalised patients. Those who have a low protein level would have a greater chance of having an abnormal EEG finding. In a study of seizures and malnutrition, Stern et al[26] revealed that protein malnutrition could lead to enhanced seizure susceptibility. Protein energy malnutrition exhibited EEG abnormalities in childhood such asdevelopmental delay in alpha rhythm maturation and an insufficient decrease in beta activity.[27] In a study ofchildren with malnutrition, EEG abnormalities demonstrated the presence of slow and sharp waves in the frontal, parietal and temporal lobes.[28] Quantitative EEG analysis in protein energy malnutrition in children demonstrated an increase in theta activity, decrease in alpha 1 in fronto-central electrodes, increase in fast alpha in temporo-parietal electrodes and increase in beta activity in temporal leads.[29] However, most of these studies focussedon children and further studies are required to elucidate the effect of malnutrition on EEG changes in the adult population.

LIMITATIONS

This was a single-centre study being carried out, so the data may not be representative of the general population. As there were multiple comorbidities from the cohort, the subanalysis of each medical condition with the EEG abnormalities did not reach any statistical significance. Thus, a larger sample size may be required to study the effect of medical conditions on EEG abnormalities. Another limitation is that this work detailed only a single initial EEG in the patients. A repeated EEG may be useful to detect any evolving changes from the baseline EEG. As the EEG was analysed retrospectively, any abnormalities such as the presence of seizure activity may warrant urgent medical attention. However, the EEG records were reviewed by the neurologists who had commenced the appropriate treatment.

CONCLUSION

This study emphasised that an abnormal EEG remains a useful tool in determining the likelihood of seizures in a hospital setting. The risk factors for EEG abnormality in hospitalised patients were age, focal seizures and low protein level. The EEG does have an important role as part of the workup in hospitalised patients to aid the clinician tailor their management in a holistic manner.

ACKNOWLEDGEMENT

The authors would like to thank the staff in the neurology laboratory, Hospital Canselor Tuanku Muhriz, Universiti Kebangsaan Malaysia for their help.

CONFLICT OF INTEREST

We certify that there is no actual or potential conflict of interest in relation to this article.

REFERENCES

1. Fisher RS, Cross JH, French JA, Higurashi N, Hirsch E, Jansen FE, et al. Operational classification of seizure types by the International League Against Epilepsy: Position Paper of the ILAE Commission for Classification and Terminology. Epilepsia 2017; 58(4): 522-30.
2. Berger H. Über das Elektrenkephalogramm des Menschen. Arch Psychiatr 1929; 87: 527-70.
3. Smith SJ. MEEG in the diagnosis, classification, and management of patients with epilepsy. J NeurolNeurosurg Psych 2005; 76: ii2-ii7.
4. Sutter R, Stevens RD, Kaplan PW. Clinical and imaging correlates of EEG patterns in hospitalised patients with encephalopathy. J Neurol 2013; 260: 1087-98.
5. Khanna A, Pascual-Leone A, Michel CM, Farzan F. Microstates in resting-state EEG: current status and future directions. NeurosciBiobehav Rev 2015; 49: 105-13.
6. Szurhaj W, Lamblin MD, Kaminska A, Sediri H. EEG guidelines in the diagnosis of brain death. Neurophysiologie Clinique/Clinical Neurophysiol 2015; 45: 97-104.
7. Xinghua T, Lin L, Qinyi F, Yarong W, Zheng P, Zhenguo L. The clinical value of long - term electroencephalogram (EEG) in seizure - free populations: implications from a cross-sectional study. BMC Neurol 2020; 20(1): 88.
8. Hill CE, Blank LJ, Thibault D, Davis KA, Dahodwala N, Litt B, et al. Continuous EEG is associated with favorable hospitalization outcomes for critically ill patients. Neurology 2019;92(1):e9–e18.
9. Abend NS, Dlugos DJ, Hahn CD, Hirsch LJ, Herman ST. Use of EEG monitoring and management of non-convulsive seizures in critically ill patients: a survey of neurologists. Neurocrit Care 2010; 12(3): 382-9.
10. Manford M, Fish DR, Shorvon SD. An analysis of clinical seizure patterns and their localizing value in frontal and temporal lobe epilepsies. Brain 1996; 119: 17-40.
11. Chassoux F, Semah F, Bouilleret V, Landre E, Devaux B, Turak B, et al. Metabolic changes and electro-clinical patterns in mesio-temporal lobe epilepsy: a correlative study. Brain 2002; 127(1): 164-74.
12. Zhang ZJ, Koifman J, Shin DS, Ye H, Florez CM, Zhang L, et al. Transition to seizure: ictal discharge is preceded by exhausted presynaptic GABA release in the hippocampal CA3 region. J Neurosci 2012; 32(7): 2499-512.
13. Park KM, Shin KJ, Ha SY, Park JS, Kim SE, Kim HC, et al. Korean J Clin Neurophysiol 2014; 16: 15-20.
14. Mohammad D, Zaidi S, Fawad B, Qureshi M, Abubaker Z, Shaikh M, et al. Frequency of neurological disorders and related EEG finding in a Tertiary Care Hospital of Karachi. JBiosciMed 2019;7: 56-64.
15. Azabou E, Magalhaes E, Braconnier A, Yahiaoui L, Moneger G, Heming N, et al. Early standard electroencephalogram abnormalities predict mortality in septic intensive care unit patients. PLoS One 2015; 10(10): e0139969.
16. Owolabi LF, Reda AA, El Sayed R, Morsy DFM, Enwere OO, Mba UA, et al. Study of electroencephalography in people with generalized epilepsy in a Saudi population. J Community Hosp Intern Med Perspect 2020; 10(6): 549–54.
17. Aina OF, Malomo IO, Ladapo HT, Amoo IG. One year of EEG Unit at Psychiatric Hospital, Yaba, Lagos. Nigerian Postgraduate Med J 2004; 11: 212-4.
18. Rodriguez Ruiz A, Vlachy J, Lee JW, Gilmore EJ, Ayer T, Haider HA, et al. Critical Care EEG Monitoring Research Consortium. Association of Periodic and Rhythmic Electroencephalographic Patterns With Seizures in Critically Ill Patients. JAMA Neurol 2017; 74(2): 181-8.
19. Sarkis RA, Lee JW. Quantitative EEG in hospital encephalopathy: review and microstate analysis. J Clin Neurophysiol 2013; 30(5): 526-30.
20. Foldvary N, Klem G, Hammel J, Bingaman W, Najm I, Lüders H. The localizing value of ictal EEG in focal epilepsy Neurology 2001; 57(11): 2022-28.
21. Hughes JR, Fino JJ. Focal Seizures and EEG: prognostic considerations. ClinElectroencephalogr 2003; 34(4): 174-181.
22. Ishii R, Canuet L, Aoki Y, Hata M, Iwase M, Ikeda S, et al. Healthy and pathological brain aging: from the perspective of oscillations, functional connectivity, and signal complexity. Neuropsychobiology 2017; 75(4): 151-61.
23. Jabès A, Klencklen G, Ruggeri P, Antonietti JP, Banta Lavenex P, Lavenex P. Age-related differences in resting-state EEG and allocentric spatial working memory performance. Front AgeingNeurosci 2021; 13: 704362.
24. Teng C, Cheng Y, Wang C, Ren Y, Xu W, Xu J. Aging-related changes of EEG synchronization during a visual working memory task. CognNeurodyn 2018; 12(6): 561-568.
25. Cloyd J, Hauser W, Towne A, Ramsay R, Mattson R, Gilliam F, et al. Epidemiological and medical aspects of epilepsy in the elderly. Epilepsy Res 2006; 68 Suppl 1: S39-48.
26. Stern WC, Forbes WB, Resnick O, Morgane PJ. Seizure susceptibility and brain amine levels following protein malnutrition during development in the rat. Brain Res 1974; 79(3): 375-84.
27. Bosch-Bayard J, Razzaq FA, Lopez-Naranjo C, Wang Y, Li M, Galan-Garcia L, et al. Early protein energy malnutrition impacts life-long developmental trajectories of the sources of EEG rhythmic activity. Neuroimage 2022; 254: 119144.
28. Agarwal KN, Das D, Agarwal DK, Upadhyay SK, Mishra S. Soft neurological signs and EEG pattern in rural malnourished children. Acta PaediatrScand 1989; 78(6): 873-8.
29. Taboada-Crispi A, Bringas-Vega ML, Bosch-Bayard J, Galán-García L, Bryce C, Rabinowitz AG, et al. Quantitative EEG tomography of early childhood malnutrition. Front Neurosci 2018; 12: 595.