MJMR, Vol. 35, No. 2, 2024, pages (163-169).

Open Access
ISSN:2682-4558

Research Article

Electrocardiographic Changes in Children with Idiopathic Epilepsy and Epilepsy Secondary to Structural Brain Lesions

Marwa Waly Eldin Ali[1], Arwa Osama Mohemad Said[1], Mohamed Abdelmaaboud[1], and Moustafa M. Abdel Raheem[1]

[1] Department of Pediatrics, Faculty of Medicine, Minia University, Egypt.

DOI: 10.21608/MJMR.2023.227098.1490

Abstract

Background: Several changes in autonomic functions were associated with epileptic seizures, which may contribute immediately or soon after the ictal event to cardiovascular, pulmonary, gastrointestinal, and urinary manifestations. This study aimed to assess Electrocardiographic (ECG) changes after an epileptic seizure in children with idiopathic epilepsy and epilepsy secondary to structural brain lesions (SBL). **Subject and Methods:** This was a case control study, it was conducted on epileptic children admitted to Pediatric Inpatient Department of Minia University Hospital for Obstetrics & Gynecology and Pediatrics. This studied included 60 epileptic children and 30 healthy children as controls. They were divided into three groups, group I: idiopathic epileptic children, group II: epileptic children secondary to structural brain lesions and group III: healthy children. CBC, LFT, electrolytes (Na, K, Ca), ECG, EEG were done for all patients. **Results:** In this study there were no significant difference in the ECG rhythm, ST segment, T wave, QRS complex among all the studied groups. On the other hand, there was a significant higher heart rate in group I (idiopathic epilepsy) and group II (epilepsy secondary to SBL) than group III (healthy controls). **Conclusion:** idiopathic epilepsy and epilepsy secondary due to SBL can cause myocardial strain and significant tachycardia

Key words: ECG, Tachycardia, convulsion

Introduction

Many changes in autonomic functions is associated with epileptic seizures , which may contribute to immediate or soon after cardiovascular, pulmonary, gastrointestinal, cutaneous, and urinary manifestations .**[1]**

One of the most dangerous changes occurs due to epileptic seizures are cardiovascular changes, which may result in sudden unexpected death in epileptic patients. Moreover, seizure leads to disturbance in cardiac functions and resulting in syncopal events.[2]

Notably generalized convulsive seizures, put the cardiovascular system on strain, which may promote cardiac complications such as cardiac arrhythmias, cardiomyopathy, and myocardial infarction.[3]

In the context of neurological diseases, wide variety of changes in the electrocardiogram (ECG) are seen. Arrhythmias and repolarization changes are the two major categories of change were noticed . [4]

It is likely that the increased tendency for life threatening arrhythmias in patients with acute neurological disease is a result of the repolarization change, which increases the vulnerable period during which an extrasystole would be likely to result in ventricular tachycardia and/or ventricular fibrillation.

Therefore, the essential and potentially most lethal features of the ECG, which are known to change in the context of neurological disease, are the ST segment and T wave, which reflect abnormalities in repolarization.[4]

Subjects and methods

This was a case control study conducted on children recruited from Pediatric Inpatient Department of Minia University Hospital for Obstetrics & Gynecology and Pediatrics in the period from April 2022 to April 2023.

This studied included 60 epileptic children diagnose according to ILAE criteria. [5] They were divided into group I: included idiopathic epileptic children, group II: included epileptic children secondary to structural brain lesions and 30 healthy children as controls represented group III.
Epileptic children age between 1 year old to 12 years old and who do not have any cardiological diseases were included in this study.

Ethical approval

For any patient enrolled into the study, the purpose and design were explained to the parents in details. A written consent was obtained from parents of each patient. We avoided utilizing deceptive practices by obtaining parents' informed consents prior to participant enrollment, which gave participants the option to withdraw from the study at any time. The ethical committee of the faculty of Medicine, Minia University approved this study (Approval No.337:5/202).

All patients were subjected to:

Full History taking: including; (Age, Sex, Family history, Prenatal, natal, and postnatal history, history of seizures (onset, duration, frequency, and type of seizures), (Diagnosis of epilepsy type done according to international league against epilepsy (ILAE) 2017 classification), developmental milestones, medications taken).Clinical examination including; systemic examination, anthropo-metric measures: (weight, length, height, head circumference and BMI) and complete neurological examination.

- **Laboratory investigations including:** Liver function tests (ALT, AST and serum albumin), renal function tests (blood urea and serum creatinine) and complete blood count to exclude other system affection.

- **Magnetic Resonance Imaging studies of the brain (Brain MRI)** were done to differentiate between idiopathic epilepsy and epilepsy secondary due to structural brain lesions (SBL).

- **Digital Electro-Encephalogram (EEG) for all patients in the study:** Nihon Koden digital EEG machine was used; the EEG was carried out on 16 channels.

- **Electrocardiogram (ECG);** Inter-ictal 12-lead ECG in the first 24h after the seizures was performed for all patients.

Results

This study showed no significant difference among all studied groups (children with idiopathic epilepsy, epilepsy secondary due to structural brain lesions (SBL) and control group) as regarding their demographic data. **(Table 1)**

No significant difference between group I (idiopathic epilepsy) and group II (epilepsy secondary to SBL) regarding the frequency of seizure per year, type of epilepsy, duration of seizure, duration of postictal drowsiness, family history of convulsion, and EEG changes, while there is a significant higher frequency of fever associated with seizure in group I (idiopathic epilepsy) than group II (epilepsy secondary to SBL) (p value <0.0001). There is also a significant higher frequency of motor and mental developmental delay in group II than group I (p value <0.0001) for each of them. Moreover, there is a significant higher age of seizure onset in epileptic children secondary due to SBL than in group I idiopathic epilepsy children (p value <0.027). **(Table 2)**

No significant differences in ECG rhythm, ST segment, T wave, and QRS complex among all studied groups. There is a significant higher heart rate (tachycardia regarding the cut point

for age) in idiopathic epilepsy children and children with epilepsy secondary to SBLthan controls (p value >0.001 & <0.0001) respectively. **(Table 3)**

There is no significant difference in liver function, renal function and Na, K, Ca results among all studied groups.
(Table 4)

Table 1: Socio demographic & clinical data of the studied groups

		Group I idiopathic epilepsy No.= 30	Group II Epilepsy secondary to SBL No.= 30	Group III Controls No.= 30	p value
Age (years)	**Median (IQR)**	3.8 (1.9 – 6.0)	4.0 (2.5 – 5.6)	5.8 (3.0 – 8.3)	0.07
Sex Male Female	**No. (%)**	17 (56.7%) 13 (43.3%)	19 (63.3%) 11 (36.7%)	17 (56.7%) 13 (43.3%)	0.832
Weight (Kg)	**Median (IQR)**	15.5 (11.6 – 20.0)	15.3 (11.4 – 19.0)	18.8 (14.9 – 23.0)	0.111
Height (Cm.)	**Median (IQR)**	99.5 (84.5 – 117)	104.0 (80.8 – 115.3)	108.5 (95.0 – 125.5)	0.174
BMI	**Median (IQR)**	15.8 (15.4 – 16.6)	15.9 (14.8 – 17.6)	15.9 (15.4 – 16.6)	0.925

SBL: Structural Brain Lesion IQR: interquartile range Kg: kilogram
Cm: Centimeter BMI: Body mass index p value is significant <0.05

Table 2: Comparison of clinical data, EEG finding and imaging studies between group I and group II

	Group I idiopathic epilepsy No.=30	Group II Epilepsy secondary to SBL No.= 30	p value
Age of seizures onset (years) Median (IQR)	2.3 (1.0 – 4.3)	3.3 (1.9 – 6.0)	**0.027***
Frequency of seizures /year Median (IQR)	2.0 (1.0 – 3.0)	2.0 (1.0 – 2.0)	0.912
Type of epilepsy:			
Generalized	24 (80%)	20 (66.6%)	
Focal	6 (20%)	8 (26.7%)	0.115
Combined Focal and generalized	0	2 (6.7%)	
Duration of seizure:			
<5 min	21 (70%)	22 (73.3%)	
5-30 min	5 (16.7%)	6 (20%)	0.561
>30 min	4 (13.3%)	2 (6.7%)	
Duration of postictal drowsiness:			
Conscious immediate after seizure.	4 (13.3%)	0	
<30 min	17 (56.7%)	19 (63.3%)	0.762
3o mint – 1 hour	2 (6.7%)	8 (26.7%)	
1h – 24 hours	7 (23.3%)	3 (10%)	
Family history of convulsions:			0.132
Positive	6 (20%)	2 (6.7%)	
Negative	24 (80%)	28 (93.3%)	
EEG:			0.16
-Normal	15 (50%)	16 (53.3%)	
-Focal epileptiform	14(46.7%)	4 (13.3%)	
-Generalized epileptiform	0	7 (23.3%)	
-Generalized slowness (encepalopthic)	1 (3.3%)	3 (10%)	
Imaging (structural brain lesion in radiology):			
-Normal	30 (100%)	0	
-Atrophy	0	14 (46.7%)	
-Hemorrhage	0	2 (6.7%)	
-Ischemic insult	0	8 (26.7%)	
- congenital brain malformation	0	6 (19.9%)	

SBL: Structural Brain Lesion EEG: Electro-encephalogram IQR: interquartile range
p value is significant <0.05

Table 3: ECG changes among studied groups

			Group I idiopathic epilepsy No.= 30	Group II (epilepsy secondary to SBL) No.= 30	Group III Controls No.= 30	p value		
ECG	HR Normal Tachycardia bradycardia	Median (IQR) (According to cut limited age)	95.5 (87.0-140.0) 20(66.7%) 7(23.3%) 3(10%)	92.0 (83.3–140.0) 18(60%) 10(33.3%) 2(6.7%)	92.5 (83.0 – 105.3) 30 (100%)	<**0.0001**		
						I VS 11	I VS 111	II VS III
						0.0717	**<0.001**	**<0.0001**
	Rhythm: Normal Abnormal	No. (%)	27 (90%) 3 (10%)	29 (96.7%) 1 (3.3%)	30 (100%) 0	0.062		
	St segment Normal Elevated	No. (%)	29(96.7%) 1 (3.3%)	30 (100%) 0	30 (100%) 0	0.221		
	T wave: Normal Tall	No. (%)	29(96.7%) 1 (3.3%)	30 (100%) 0	30 (100%) 0	0.221		
	QRS: Normal	No. (%)	30 (100%)	30 (100%)	30 (100%)	>0.99		

SBL: Structural Brain Lesion ECG: Digital electrocardiogram IQR: interquartile range
HR: Heart rate p value is significant<0.05

Table 4: Laboratory investigation of all the studied groups

	Group I idiopathic epilepsy No.= 30	Group II epilepsy secondary to SBL No.= 30	Group III Controls No.= 30	p value		
	Median (IQR)	Median (IQR)	Median (IQR)	I vs II	I vs III	II vs III
ALT (U/L)	20.5 (16.0 – 40.0)	29.5 (21.8 – 34.0)	18.5 (12.8 – 23.0)	0.124		
				0.153	0.24	0.89
AST (U/L)	30.0 (15.8 – 43.8)	28.0 (23.0 – 36.0)	18.0 (14.0 – 22.3)	0.23		
				0.894	0.876	0.96
Urea (mg/dl)	28.0 (20.0 – 35.8)	28.5 (24.8 – 32.0)	26.5 (17.0 – 36.0)	0.745		
Creatinine (mg/dl)	0.5 (0.4 – 0.7)	0.6 (0.4 – 0.8)	0.7 (0.5 – 0.8)	0.354		
Na (mmol/L)	139.0 (136.0 – 143.3)	139.0 (135.0 – 143.0)	140.0 (137.8 – 143.0)	0.58		
K (mmol/L)	3.9 (3.6 – 4.4)	4.0 (3.7 – 4.5)	4.0 (3.9 – 4.0)	0.761		
Ca (mg/dl)	1.1 (1.1 – 1.13)	1.1 (1.0 – 1.1)	1.1 (1.0 – 1.1)	0.04		
				0.108	0.13	0.39

ALT: alanine transaminase AST: aspartate transaminase Na: sodium K: potassium
Ca: calcium IQR: interquartile range p value is significant <0.05

Discussion

This study showed that the age of onset of seizure in idiopathic epilepsy children was significantly younger than children with epilepsy secondary to SBL, while there was no significant difference between them regards frequency of seizures per year, type of epilepsy, duration of seizures, postictal durations or EEG changes (P value 0.027)

This was in agreement with Camfield &Camfield and Falco-Walter who noticed that the incidence of idiopathic epilepsy is increased in the first year of life and declines to adult levels by the end of 10 years of age.[6] [7]

Beghi stated that idiopathic epilepsy and epilepsy secondary to SBL can occur at any age and there is no specific age for them, as secondary epilepsy may occur due to structural brain lesions, intracranial hemorrhage, ischemic insult in the brain or congenital brain malformation and tumors which could happen at any age and this is against our results. [8]

In this study, there was no significant changes in ECG rhythm, ST segment, T wave and QRS complex among idiopathic epileptic children & epileptic children secondary to SBL and control group.

Similar results were obtained from Ufongene, El Atrache who reported no changes in ECG in epileptic children early during postictal period. [9]

While Gigli, Sala stated that there is a higher incidence of ECG abnormalities in epileptic patients close to the seizure time and the epileptic patients have more ECG abnormalities in comparison to their baseline ECG. [10] Also Chan, Dervan stated that ECG changes were common in pediatric epileptic patients. Nonspecific ST segment and T-wave changes represented the most common alterations with the prevalence of 19% and 30%, respectively, this is against our results. [11]

This study reported that heart rate increases significantly in group I (idiopathic epilepsy) & group II (epilepsy secondary to SBL) when compared to group III (controls) (P value <0.001 &,0.0001) respectively.

This was in agreement with Moseley, Nickels, Chouchou, Bouet, Myers, Sivathamboo and Schomer, Baljak who reported that postictal tachycardia occurred in 61% of the studied epileptic patients and they emphasizes the role of tachycardia as the most frequent cardiac complication of seizures. [12],[13],[14],[15]

This study also found a significant positive correlation between heart rate (HR) and duration of seizure in group I (idiopathic epilepsy) & group II (epilepsy secondary to SB) (R=0.49, P=0.006 and R=0.69, P<0.0001) respectively.

Ali, Bubolz and Brewster, Marzec reported that patients who had status epilepticus (who suffered from seizure which last for 5 minutes or more) are associated with higher incidence of having postictal ECG abnormalities that reflect altered ventricular depolarization–repolarization in children without prior seizure history and in children with epilepsy, this was in agreement with our results. [16],[17]

Nass, Motloch and Ibrahim, Megahed reported that patients who had suffered prolonged seizure duration or continuous epileptic EEG discharges propagated to the central autonomic network, altering or disrupting the normal autonomic regulation of essential cardiac functions and causing sub-endocardial ischemia which lead to increase cardiac enzymes level and ECG changes and cause increase in heart rate, this is in agreement with our results. [18],[19]

Conclusions: Epilepsy increase tendency for life threatening arrhythmias mainly secondary epilepsy due to SBL .

References

1. Devinsky, O.J.E.c., Effects of seizures on autonomic and cardiovascular function. 2004. 4(2): p. 43-46.
2. Leutmezer, F., et al., Electrocardiographic changes at the onset of epileptic seizures. 2003. 44(3): p. 348-354.
3. Lee, G.P. and C.L.J.T.o.C.N. Clason, 23 Classification of seizure disorders and syndromes, and neuropsychological impairment in adults with epilepsy.2016:p. 437.

4. Samuels, M.A.J.C., The brain–heart connection. 2007. 116(1): p. 77-84.
5. Fisher, R. S., Acevedo, C., Arzimanoglou, A., Bogacz, A., Cross, J. H., Elger, C. E., ... & Wiebe, S. (2014). ILAE official report: a practical clinical definition of epilepsy. *Epilepsia*, *55*(4), 475-482.
6. Camfield, P. and C. Camfield, Incidence, prevalence and aetiology of seizures and epilepsy in children. Epileptic disorders, 2015. 17(2): p. 117-123.
7. Falco-Walter, J. Epilepsy—definition, classification, pathophysiology, and epidemiology. in Seminars in neurology. 2020. Thieme Medical Publishers, Inc.
8. Beghi, E.J.N., The epidemiology of epilepsy. 2020. 54(2): p. 185-191.
9. Ufongene, C., et al., Electrocardiographic changes associated with epilepsy beyond heart rate and their utilization in future seizure detection and forecasting methods. 2020. 131(4): p. 866-879.
10. Gigli, L., et al., Electrocardiogram Changes in the Postictal Phase of Epileptic Seizure: Results from a Prospective Study. 2023. 12(12): p. 4098.
11. Chan, S.W., et al., Epilepsy duration is an independent factor for electrocardiographic changes in pediatric epilepsy. 2021. 6(3): p. 588-596.
12. Moseley, B.D., et al., How common is ictal hypoxemia and bradycardia in children with partial complex and generalized convulsive seizures? 2010. 51(7): p. 1219-1224.
13. Chouchou, F., et al., The neural bases of ictal tachycardia in temporal lobe seizures. 2017. 128(9): p. 1810-1819.
14. Myers, K.A., S. Sivathamboo, and P.J.E. Perucca, Heart rate variability measurement in epilepsy: how can we move from research to clinical practice? 2018. 59(12): p. 2169-2178.
15. Schomer, A.C., et al., Measuring vagal activity in postictal bradycardia. 2023. 141: p. 109148.
16. Ali, W., et al., Epilepsy is associated with ventricular alterations following convulsive status epilepticus in children. 2017. 2(4): p. 432-440.
17. Brewster, A.L., et al., Early cardiac electrographic and molecular remodeling in a model of status epilepticus and acquired epilepsy. Epilepsia, 2016. 57(11): p. 1907-1915.
18. Nass, R.D., et al., Blood markers of cardiac stress after generalized convulsive seizures. 2019. 60(2): p. 201-210.
19. Ibrahim, D.E. and W.K.H.J.A.-N.J.o.S. Alhashemi, Study of Some Biochemical Parameters in Epileptic Patients. 2019. 22(4): p. 8-12.