Corrected QT interval and QT dispersion in temporal lobe epilepsy in children and adolescent

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ABSTRACT

Aim: The purpose of this research was to contrast (heart-rate corrected QT interval) QTc, and QT dispersion) QTd intervals in individuals with (temporal lobe epilepsy) TLE and those without TLE using a standard 12-lead electrocardiogram.

Patients & methods: This cross-sectional research was undertaken on 100 cases aged 10 to 20 diagnosed with epilepsy in accordance with 2017 ILAE criteria. The patients’ informed written permission was acquired. In our study, we included 100 cases: 50 with TLE and 50 with non-TLE verified by seizure semiology. All patients were subjected to a comprehensive history, clinical examination (heart rate, pulse, and blood pressure), and clinical evaluation, which included a comprehensive epilepsy history. On the basis of neurology service documents or the initial publication of the international classification of diseases, 9th revision (ICD9) diagnostic or 10th revision (ICD10) codes for epilepsy, diagnostic age for epilepsy was calculated.

Results: The mean QT interval in group I was 418.30±25.48 ms while that of group II was 406.20±27.63 ms, the mean QTc of group I was 513.60±51.94 ms and was 488.70±50.65 ms in group II. The calculated QTd was with a mean of 57.60±25.05 ms while that of group II was 43.60±31.89 ms. It means that the QT interval, QTc, and QTd values were considerably greater in the group I (temporal epilepsy) contrasted with group II (non-temporal epilepsy); (p=0.025, 0.030, and 0.016, respectively). The mean QT, QTc, and QTd values for FE were 409.20±20.80, 500.70±55.60, and 52.60±29.70 ms, respectively. QT, QTc, and QTd mean values for patients with widespread epilepsy were 412.00±25.60, 505.00±68.60, and 46.20±28.70 ms. QT, QTc, and QTd interval were insignificantly different between focal and generalized epilepsy. The longer an illness progresses, the longer the QT and QTc intervals, as there was a substantial positive correlation among illness’s course and QT interval (r=0.391, p=0.001) and QTc interval (r=0.289, p=0.011), but there was no noticeable impact on QTd due to the illness’s duration, as we found an insignificant correlation among duration of illness and QTc and QTd.

Conclusions: Our findings indicate that; QTc interval and QTd are longer in epilepsy cases more among TLE cases contrasted with non-TLE. Since there was no distinction among different epilepsy types (focal and generalized).

Keywords: corrected QT interval, QT dispersion, temporal lobe epilepsy

INTRODUCTION

Sudden unexpected death in epilepsy (SUDEP) is a potentially fatal complication of seizures that occurs in people with epilepsy who have no history of trauma or drowning and whose epilepsy is extensively documented yet who die suddenly and unexpectedly [1]. Autonomic and respiratory degradation are regarded as the primary pathophysiologic processes driving SUDEP, with cardiac abnormalities constituting a substantial proportion of the autonomic aspect [2].

Several studies indicated that persons with epilepsy had a two- to three-fold greater incidence of sudden cardiac arrest (SCA), regardless of conventional cardiac hazard factors for SCA. 10.0%-50.0% of deaths in people with persistent...
refractory epilepsy are caused by SUEP [3, 4]. Those between the ages of 20 and 40 are most frequently affected. Despite strong efforts to understand the pathophysiology of SUEP today, it is still not completely comprehended [5].

Comorbidities of FE include cardiac autonomic impairment and irregular heart rhythms, particularly TLE, due to the amygdala’s crucial role in regulating cardiovascular autonomic activity and the transmission of seizure activity through the insular cortex [6]. Many investigations focusing on rhythm anomalies and their predictions [7, 8] have been conducted on this patient population. Unfortunately, there are very few studies that focus on people with generalized epilepsy (GE). This is feasible for clinical investigations since the relevant cohorts consist mostly of in-hospital candidates for TLE surgery who have undergone at least a preliminary cardiac evaluation (ECG) [9, 10]. Abnormal cardiac repolarization is reflected on the ECG as a shorter or prolonged QT interval and high QT dispersion (QTd) [11]. The QT interval is the period that passes between when the ventricles depolarize and repolarize [12]. Ventricular arrhythmias [13] are potentially fatal and are associated with prolonged ventricular repolarization [8]. Intercal ECG lengthening of the corrected QT interval (QTc) was more common in adults with epilepsy than in those without epilepsy, and it was also detected in certain pediatric patients two hours after generalized seizures [14]. During prolonged, unobserved night-time seizures with associated hypoxia, in [15], it was theorized that some persons are more prone to develop a cardiac arrhythmia due to the effects of QT hysteresis and more persistent or exacerbated prolongation of the QTc. Autonomic dysfunction is commonly disrupted by both partial and generalized seizures [16]. While convulsive status epilepticus is a frequent neurological disorder in children, it remains unstudied whether protracted seizures in healthy children might result in chronic ventricular depolarization–repolarization abnormalities. Investigations of children with epilepsy, nevertheless, have shown that short-term seizures are associated with lengthening QTc intervals and higher spatial variability, as well as considerable heart rate (HR) oscillations and premature ventricular beats [17, 18]. In children, like in adults, epileptic episodes can be followed by ventricular instability due to abnormal ventricular depolarization-repolarization [19]. Convulsive status epilepticus is a frequent juvenile neurological disorder; however, it has not yet been determined whether protracted seizures in otherwise healthy children might result in chronic ventricular depolarization-repolarization abnormalities [20]. In order to evaluate if there is a distinction in QTc or QTd among TLE and non-TLE, we designed this research to assess QTc and QTd in epileptic cases using a standard 12-lead ECG and to determine whether there is a variation in QTc or QTd among TLE and non-TLE.

PATIENTS & METHODS

This cross-sectional investigation was done on 100 cases in Al-Hussein Hospital, and Sayed Galal Hospital, Al-Azhar University. Participants’ written informed consent was acquired. The research was done after approval from the Ethical Committee of Al-Azhar University, Al-Hussein, and Sayed Galal Hospitals, from January 2022 to February 2023.

Inclusion Criteria

Our included patients aged 10-20 years old and above, diagnosed with epilepsy in respect to 2017 ILAE criteria [21].

Exclusion Criteria

Exclusion criteria were epileptic cases on medication that is associated with extended QT interval (chlorpromazine; haloperidol; droperidol; quetiapine; olanzapine; amisulpride; thiourazine; types of IA, IC, and III antiarrhythmic; tricyclic antidepressants; antihistamines; chloroquine; hydroxychloroquine; quinine; and macrolides) in addition to epileptic cases who suffer hypokalemia, cardiac, hepatic, or renal illnesses, pregnancy and lactation.

In our research, we enrolled 100 cases: 50 with TLE and 50 with non-TLE confirmed by seizure semiology. All patients were treated to a comprehensive history, clinical examination (HR, pulse, and blood pressure), and clinical evaluation, which included a comprehensive epilepsy history.

The age at diagnosis of epilepsy was established founded on neurology service documents or the 1st manifestation of ICD, 9th revision (ICD-9) diagnostic or 10th revision (ICD-10) codes for epilepsy. Based on the neurology service’s data, as an operational definition, refractory epilepsy was defined as the inability to control seizures while trying two different, effective ASMs [22].

TLE was identified based on seizure semiology, magnetic resonance imaging (MRI), and electroencephalogram (EEG) findings. ECG recordings were performed on every patient in the research (CM 300 A, Comen, China). In leads with stable isoelectric lines, QT interval was recorded from the start of the QRS until the end of the T wave. In the company of U waves, QT interval was determined by going to the lowest point on the graph among the T and U waves and computed manually [23]. Bazett’s formula, QT/[(RR)]1/2, was used to calculate QTc. The variance among the highest and minimum QT intervals was calculated to estimate the QTd [24]. A cardiologist evaluated every ECG. QT interval value was determined in accordance with the criteria of European Society of Cardiology [25], which define a prolonged QT interval as more than 470 ms in females and more than 450 ms in men. It was defined prolonged QTd as a value greater than 50 ms [23].

Statistical Analysis

The statistical analysis was done using SPSS v27 (IBM, Armonk, NY, USA). To assess the normality of the data distribution, the Shapiro–Wilks test and histograms were employed. Parametric quantitative data were given as the mean and standard deviation (SD) and analyzed using the unpaired student t-test. Non-parametric quantitative data were provided as the median and interquartile range and analyzed using Mann Whitney-test. Where applicable, Chi-square test or Fisher’s exact test was used to analyze qualitative variables provided as frequency and percentage (%). Pearson correlation was used to measure the correlation between two quantitative variables. A two-tailed p-value of 0.050 was regarded as statistically substantial.

RESULTS

The mean age was 15.60±3.250 years in group I and 15.90±3.04 in group II. There were 58.0% males and 42.0%
Table 1. Basic descriptive data of researched groups

<table>
<thead>
<tr>
<th></th>
<th>Group I (n=50)</th>
<th>Group II (n=50)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>15.6±3.25</td>
<td>15.9±3.04</td>
<td>0.527</td>
</tr>
<tr>
<td>Sex</td>
<td>Male 29 (58.0%)</td>
<td>27 (54.0%)</td>
<td>0.687</td>
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<tr>
<td></td>
<td>Female 21 (42.0%)</td>
<td>23 (46.0%)</td>
<td></td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>46.90±11.47</td>
<td>48.50±10.36</td>
<td>0.471</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.51±0.06</td>
<td>1.51±0.07</td>
<td>0.695</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>20.70±5.10</td>
<td>21.50±5.20</td>
<td>0.437</td>
</tr>
<tr>
<td>Duration of illness (years)</td>
<td>6.10±1.48</td>
<td>5.90±1.34</td>
<td>0.724</td>
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<tr>
<td>Types of epilepsy</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Focal</td>
<td>33 (66.0%)</td>
<td>36 (72.0%)</td>
<td>0.665</td>
</tr>
<tr>
<td>Generalized</td>
<td>17 (34.0%)</td>
<td>14 (28.0%)</td>
<td></td>
</tr>
<tr>
<td>Focal</td>
<td>25 (75.8%)</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Temporal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Extra temporal</td>
<td>8 (24.2%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Seizure attack duration (s)</td>
<td>63 (36.7-93.7)</td>
<td>76.5 (50.2-98.7)</td>
<td>0.373</td>
</tr>
</tbody>
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Table 2. Vital signs & ECG data between researched groups

<table>
<thead>
<tr>
<th></th>
<th>Group I (n=50)</th>
<th>Group II (n=50)</th>
<th>p-value</th>
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</thead>
<tbody>
<tr>
<td>HR (beats/min)</td>
<td>85.20±8.70</td>
<td>84.40±9.23</td>
<td>0.665</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>133.20±12.36</td>
<td>129.00±13.13</td>
<td>0.103</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>85.00±15.68</td>
<td>87.40±14.11</td>
<td>0.423</td>
</tr>
<tr>
<td>ECG</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EF (%)</td>
<td>57.0±4.91</td>
<td>56.20±5.46</td>
<td>0.425</td>
</tr>
<tr>
<td>LVEDD (cm)</td>
<td>4.14±0.15</td>
<td>4.08±0.14</td>
<td>0.029*</td>
</tr>
<tr>
<td>LVESD (cm)</td>
<td>2.40±0.22</td>
<td>2.50±0.19</td>
<td>0.596</td>
</tr>
<tr>
<td>p-max (ms)</td>
<td>151.70±14.11</td>
<td>147.30±12.54</td>
<td>0.106</td>
</tr>
<tr>
<td>p-min (ms)</td>
<td>121.40±10.93</td>
<td>119.70±12.61</td>
<td>0.478</td>
</tr>
<tr>
<td>p-wave dispersion (ms)</td>
<td>33.40±11.03</td>
<td>29.60±8.82</td>
<td>0.063</td>
</tr>
</tbody>
</table>

females in group I and 54.0% males and 46.0% females in group II. The mean weight was 46.90±11.47 kg in group I and 48.50±10.36 kg in group II. The mean height was 1.51±0.06 m in group I and 1.51±0.07 m in group II. The mean BMI was 20.70±5.10 kg/m² in group I and 21.50±5.20 kg/m² in group II. The cases had been afflicted with the illness for a mean of 6.10±1.48 years in group I and for a mean of 5.90±1.34 years in group II. 66.0% of group I had focal epilepsy and 34.0% had generalized type, whereas 72.0% of group II had focal epilepsy and 28.0% had generalized type. In patients with focal epilepsy in group I, 75.8% cases were temporal and 24.2% were extratemporal. The median duration of seizure attack was 63.0 (36.7-93.7) in group I and 76.5 (50.2-98.7) in group II.

Basic descriptive data (age, sex, weight, height, BMI, period of illness, types of epilepsy, and duration of seizure attack) were insignificantly different between the studied groups (Table 1).

Regarding the vital signs, the mean HR was 85.2±8.7 beats/min in group I and 84.40±9.230 beats/min in group II. The mean SBP was 133.20±12.36 mmHg in group I and 129.00±13.13 mmHg in group II. The mean DBP was 85.00±15.68 mmHg in group I and 87.40±14.11 mmHg in group II. On ECG data, EF was with a mean of 57.0±4.91% in group I and 56.20±5.46% in group II. LVEDD was with a mean of 4.14±0.15 cm in group I and 4.08±0.14 cm in group II.

LVESD was with a mean of 2.40±0.22 cm in group I and 2.50±0.19 cm in group II. p-max was with a mean of 151.70±14.11 ms in group I and 147.30±12.54 ms in group II. p-min was with a mean of 121.40±10.93 ms in group I and 119.70±12.61 ms in group II. p-wave dispersion was with a mean of 33.40±11.03 ms in group I and 29.60±8.82 ms in group II. Vital signs (HR, SBP, and DBP) were not distinguishable amongst two groups. ECG data had no discernible effect on either group. Except LVEDD was considerably greater in group I contrasted with group II (p=0.029; Table 2).

Patients were screened using a 12-lead electrocardiogram for comparability purposes of the QT interval, QTc, and QTd among the studied groups. The mean QT interval in group I was 418.30±25.48 ms while that of group II was 406.20±27.63 ms, the mean QTc of group I was 513.60±61.94 ms and was 488.70±50.65 ms. The calculated QTd was with a mean of 57.60±25.05 ms while that of group II was 43.60±31.89 ms. It means that the QT interval, QTc and QTd values were considerably greater in the group I (temporal epilepsy) contrasted with group II (non-temporal epilepsy); (p=0.025, 0.030, and 0.016, respectively; Table 3; Figure 1).

In our research, 69.0% of our cases had focal seizures, and 31.0% had generalized seizures. The mean QT, QTc, and QTd values for FE were 409.2±20.8, 500.7±55.6, and 52.6±29.7 ms, respectively, QT, QTc, and QTd mean values for patients with widespread epilepsy were 412.0±25.60, 505.0±68.60, and 46.2±28.70 ms. QT, QTc, and QTd interval were insignificantly different amongst focal and GE.

Among the studied patients, 12 cases exhibited MRI pathology, including 10 cases with signs of temporal sclerosis, two cases with cortical dysplasia in the right TLE and one case with a low-grade glioma in the right upper parietal area.

There was an insignificant relation between sex and QT as there was no discernible gender difference in QT (Table 4). The period of sickness had a substantial effect on the QT interval and QTc. The longer an illness progresses, the longer the QT and QTc intervals, as there was a substantial positive correlation among illness’s course and QT interval (r=0.391, Table 3).

Table 3. Contrast between researched groups as regard QT, QTc, & QTd intervals

<table>
<thead>
<tr>
<th></th>
<th>Group I (n=50)</th>
<th>Group II (n=50)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>QT interval (ms)</td>
<td>418.30±25.48</td>
<td>406.20±27.63</td>
<td>0.025*</td>
</tr>
<tr>
<td>QTc interval (ms)</td>
<td>513.60±61.94</td>
<td>488.70±50.65</td>
<td>0.030*</td>
</tr>
<tr>
<td>QTd interval (ms)</td>
<td>57.60±25.05</td>
<td>43.60±31.89</td>
<td>0.016*</td>
</tr>
</tbody>
</table>

Figure 1. Contrast between researched groups as regard QT, QTc, & QTd intervals (Source: Authors’ own elaboration)

Table 4. Comparison of QT according to focal & generalized epilepsy & relation between sex & QT data of studied patients

<table>
<thead>
<tr>
<th></th>
<th>Focal epilepsy (n=50)</th>
<th>Generalized epilepsy (n=50)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>QT interval (ms)</td>
<td>409.20±20.80</td>
<td>412.00±25.60</td>
<td>0.586</td>
</tr>
<tr>
<td>QTc interval (ms)</td>
<td>500.70±55.60</td>
<td>505.00±68.60</td>
<td>0.738</td>
</tr>
<tr>
<td>QTd interval (ms)</td>
<td>52.60±29.70</td>
<td>46.20±28.70</td>
<td>0.314</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Male (n=56)</th>
<th>Female (n=44)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>QT interval (ms)</td>
<td>411.20±22.70</td>
<td>408.60±21.90</td>
<td>0.569</td>
</tr>
</tbody>
</table>
Table 5. Correlation between duration of illness & different QT data of studied patients

<table>
<thead>
<tr>
<th>Duration of illness</th>
<th>Correlation coefficient</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>QT interval (ms)</td>
<td>0.391</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>QTc interval (ms)</td>
<td>0.289</td>
<td>0.011*</td>
</tr>
<tr>
<td>QTd interval (ms)</td>
<td>0.059</td>
<td>0.560</td>
</tr>
</tbody>
</table>

Figure 2. Significant positive correlation between QT interval & duration of illness (Source: Authors’ own elaboration)

Figure 3. Significant positive correlation between QTc interval & duration of illness (Source: Authors’ own elaboration)

p<0.001) and QTc interval (r=0.289, p=0.011), but there was no noticeable impact on QTd due to the illness’s duration, as we found an insignificant correlation among duration of illness and QTc and QTd (Table 5; Figure 2, and Figure 3).

DISCUSSION

Epilepsy is a chronic condition that causes central autonomic dysfunction, which can lead to cardiac electrophysiological remodeling [26]. Autonomic function is frequently affected during partial and generalized seizures, in addition to during the interictal and postictal phases [27]. During most seizures, whether temporal or extra-temporal in origin, sympathetic reactions predominate, resulting in tachycardia, tachypnea, elevated BP, pupillary dilatation, diaphoresis, and facial flushing [28].

Much research indicate lateralization of cardiovascular autonomic regulation on the hemispheres. The right hemisphere regulates sympathetic tone more than parasympathetic tone [29, 30]. After temporal seizures, tachycardia can be more prevalent [31]. Although epileptic seizures raise the hazard of cardiac arrhythmias, we intended to better understand the pathophysiology of SUDEP by evaluating cardiac repolarization indices in interictal epileptic cases, particularly those with TLE, versus non-TLE cases using a regular 12-lead surface ECG. In fact, it is simpler to examine indicators of cardiac repolarization using a 12-lead surface ECG [32] even if Holter monitoring could properly monitor QTc and QTd with additional data on temporal distinction in QTc and QTd as opposed to merely spatial variation detected by ECG.

Heart electrical alterations and observed ECG abnormalities in children with epilepsy have yet to be fully characterized for their clinical importance. To completely define the cardiac electrical changes represented by the minor ECG variations and to assess their likely long-term effect on the overall pediatric epilepsy population, more prospective studies are needed [12, 33].

The results of our research showed that epileptic patients had a significantly longer QT interval, which increased their hazard of ventricular arrhythmia, particularly in the company of cardiac structural changes such as ischemic heart disease, heart failure, ventricular hypertrophy, or autonomic nervous system dysfunction, and could even lead to sudden death.

Previous research has shown that epileptic patients have prolonged repolarization indices compared to healthy controls. For example, in [19], it was researched cases with generalized tonic-clonic seizures during the interictal period and discovered that epileptic patients had a prolonged QTd in comparison to the healthy group. Interictal QT prolongation and QTc prolongation were also observed in epileptic patients [34].

Moreover, in [35], it was discovered that QTc considerably increased in epileptic patients during convulsions across all types of epilepsy. Animal studies demonstrating altered heart representation of various Na, K, Ca, and cationic channels may provide an explanation for the increased arrhythmic hazard and potential for sudden death seen in epileptic individuals due to abnormalities in ventricular repolarization [36].

The correlation among epilepsy severity and ECG alterations has been established, and this correlation might be due to a number of causes. The presence of inotropes, irregular electrolytes, and chronic seizure types were all shown in univariant analysis to be linked with ECG changes [37]. In their research of 127 children, it was observed that the older children had epilepsy, the higher the risk of an irregular ECG [38]. QT interval and QTc were shown to be significantly longer in the current research with increasing illness duration, but QTd was not significantly affected by illness length.

This agreed with the results of [39], which studied 100 people with epilepsy (50 with temporal and 50 with non-temporal forms) and 50 healthy age- and gender-matched controls and discovered a substantially extended QTc with greater illness duration. Similarly, children with Dravet syndrome who have had epilepsy for five years or more had longer QTc intervals and more severe spatial temporal ventricular repolarization heterogeneity [40].

Epilepsy severity and ECG abnormalities may be related in several ways. For instance, changes in HR variability (a metric for sympathy-vagal balance) have been widely reported as a hallmark of epilepsy [41]. Long-term epileptic adults who have sympathetic dominance may experience worse cardiac
function, including increased heart stiffness and reduced exercise tolerance [42].

A similar molecular remodeling of the heart, this time in mice with both newly developed and long-standing epilepsy, has been documented. These results indicate that epilepsy may have negative effects on the heart by causing myocardial remodeling due to abnormal autonomic control. Further research into the mechanism among epilepsy duration and cardiac abnormalities [43, 44] is warranted.

Cases with TLE were discovered to have significantly longer QTc and QTd contrasted with non-TLE cases, which may be accounted for by [45] description of a mechanism termed the lockstep phenomenon, because of a research in which an anesthetized cat received pentylenetetrazol (a substance that, at large amounts, induces convulsions) and the cardiac postganglionic sympathetic and vagal nerve discharges were associated with the onset of convulsions it has been demonstrated that sympathetic activity is altered during an epileptiform discharge, leading to a shift in peripheral efferent output to the heart. Increased autonomic dysregulation seen in epileptic individuals is a possible explanation for this [46].

Autonomic dysregulation is a hallmark of TLE with elevated hazard of SUDEP due to disrupted neuro-respiratory and neuro-cardiac connections caused by sympathetic and parasympathetic overactivation [47]. Increased sympathetic activity has been linked to a prolonged QT interval and this association has been linked to early following depolarization [48]. Uncontrolled sickness is associated with an increase in sympathetic activity due to parasympathetic dysfunction, which decreases once the disease is under control [6]. This may explain why QTc prolongation is more common when the disease is not under control. Further to the arrhythmic hazard, which may be the most well-known cause of sudden cardiac mortality in the elderly, elevated sympathetic activity with TLE is associated with increased myocardial stiffness, atrial volumes and ventricular pressures [42].

Consistent with our findings, it was contrasted TLE and non-TLE groups for QTc and QTd and found that the former were considerably greater in the former (p=0.025 and p=0.038) [39]. In addition, it was studied 80 epileptic cases (40 controlled epileptic cases and 40 refractory epileptic cases) and discovered that controlled and refractory epileptic cases had elevated average QTc and QTd values contrasted with the placebo group [49].

Being a single-center research with a very limited sample size, our research had several limitations. Our research did not have a placebo group. TLE was diagnosed solely on the basis of semiology and interictal EEG; nevertheless, autonomic seizures may result from the activation of extratemporal regions.

CONCLUSIONS

Our findings indicate that the QTc interval and QTd are longer in epilepsy cases with TLE contrasted with those without TLE. As there was no distinction among the distinct forms of epilepsy (focal and GE), QTc prolongation, and QTc dispersion may be the result of epilepsy itself, independent of its cause. More prospective, bigger investigations are required to elucidate the underlying processes and prognostic significance of elevated QTc and QTd in TLE relative to non-TLE. In epileptic cases, a follow-up ECG is advised, as is the use of a QT interval diary, especially for TLE.

REFERENCES


