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Prognostic significance of prolonged corrected QT interval in cerebral contusion

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***Background & objectives*: Cerebral contusion (CC) results in a release of catecholamines, autonomic dysfunction and neural stimulation that can lead to a number of cardiac adverse events, so it is critical to determine these. So the objective of this study was to investigate the prognostic significance of electrocardiographic changes, particularly the effects of a prolonged corrected QT (QTc) interval in CC.**

***Methods*: In this retrospective cohort study, 110 patients with CC were evaluated. Age, sex, concomitant diseases, Glasgow Coma Scale on admission, radiological assessment of the contusion (location, size, course and presence of cerebral oedema), need for surgical intervention, length of hospital stay and the extended Glasgow Outcome Scale (GOS-E) were statistically analysed within the QTc interval by routine electrocardiography (ECG) on admission.**

***Results*: The prolonged QTc interval was found to be associated with a higher incidence of cerebral oedema and a significantly higher risk of needing surgery. Patients with a prolonged QTc interval had a significantly larger contusion volume, greater midline shift and longer hospital stay, so their GOS-E score was significantly lower. A prolonged QTc interval on admission resulted in a hospital stay of more than eight days (sensitivity: 0.97 and specificity: 0.86), a higher risk of midline shift of more than 0.45 cm (*P*=0.006, sensitivity: 0.80 and specificity: 0.99) and a GOS-E score of <7 (sensitivity: 0.97 and specificity: 0.85).**

***Interpretation & conclusions*: ECG changes on admission showing a prolonged QTc interval have prognostic significance in CC. This simple and easily applicable information should be taken into consideration at the time of clinical decision making which may prevent an adverse events survivor.**

Key words Cardiac side effects - cerebral contusion - ECG - prognostic significance - corrected QT interval - traumatic brain injury

Traumatic brain injury (TBI) is a serious condition that can lead to lifelong disability and persistent impairment of quality of life and includes various types of injuries to the brain parenchyma[1]. One of the most severe mechanisms of injury is haemorrhagic cerebral contusion (CC), which occurs in approximately 35 per

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cent of cases of TBI[2,3]. CC is defined as permanent vascular and parenchymal damage to the grey matter and subcortical areas of the brain[4]. It can lead to severe neurological and neuropsychological impairment due to damage to nervous tissue from excitotoxicity, acidotoxicity, ionic imbalance, oxidative stress, activation of the inflammatory cascade and apoptosis[5,6]. These can also trigger secondary damage outside the central nervous system, to the cardiovascular, respiratory, haematologic, endocrine and immune systems[7-13].

The cardiac side effects of various intracranial lesions, especially TBI, have been studied[14,15]. Neurogenic cardiac injury is also reportedly associated with catecholamine and neuroinflammatory responses triggered by a brain injury[16,17]. After traumatic haemorrhage CC, catecholamine release, autonomic dysfunction and neural stimulation cascades cause a range of adverse cardiac events, including cardiogenic shock, arrhythmias and dysfunction[18,19]. Therefore, the identification and elimination of these cardiac lesions are crucial at CC.

To achieve favourable outcomes in patients with CC and overcome neural and extra-neural injuries, it is important to apply effective treatment standards and know the prognostic factors that may affect patient outcomes[3]. In this single-centre cohort study, we examined a total of 110 patients with CC and retrospectively evaluated their electrocardiography (ECG) on admission, particularly the association between corrected QT (QTc) interval, lesion severity and outcome.

Material & Methods

This single centre retrospective study was conducted by the department of Neurosurgery, Prof. Dr. Cemil Tascioglu State Hospital, Istanbul, Turkey, from January 1, 2017 to October 31, 2020. The study was approved by the Institutes Ethics Committee of the University of Health Sciences, Prof. Dr. Cemil Tascioglu State Hospital. A written informed consent was obtained from participants included in this study.

Study design: A total of 110 patients with only CC, treated according to the recommendations of the Brain Trauma Foundation TBI Guidelines[20], were evaluated in the study.

Selection of participants: Individuals over 20 yr of age who were admitted to the Prof. Dr. Cemil Tascioglu State Hospital within one hour of head trauma with a radiologically diagnosed CC on brain computed tomography (CT) were evaluated. Patients younger than 20 yr of age, admitted later than one hour after head trauma or transferred from another institution were excluded. A total of 283 patients with additional extracranial or intracranial injuries besides CC at CT (traumatic subarachnoid haemorrhage, epidural and subdural haematomas, skull fractures and pneumocephalus) were excluded. In addition, patients with hypokalaemia, hypomagnesaemia and hypocalcaemia on routine blood tests at admission and with known cardiovascular disease (previous myocardial ischaemia, congestive heart failure and cardiomyopathy) or arrhythmias were excluded. The use of other drugs causing prolongation of QT (anti-arrhythmics, antipsychotics, antidepressants, antihistamines, quinolones and antifungals) was also established as an exclusion criterion.

Study parameters: All demographic characteristics and clinical parameters at admission were used as primary parameters: age, sex, comorbidities, Glasgow Coma Scale (GCS) scores, radiological characteristics from CC *via* CT scans (location, size and presence of cerebral perilesional oedema) and routine ECG data with QTc interval *via* the Bazett's formula. Cardiology consultations were controlled through the hospital patient record quality system.

It has been shown that CC progresses in the first six to 24 h on an average after head trauma[21]. In the absence of neurological deterioration, routine control scans CT were performed six and 24 h after admission, according to our department protocol. The presence of progression of contusion volume based on follow up CT scans and the need for surgical intervention were the secondary parameters. Length of hospital stay and extended Glasgow Outcome Scale (GOS-E) scores were evaluated as the outcome parameters.

Statistical analysis: Data were tested for a normal distribution by the Shapiro-Wilk test. Those with a normal distribution and two independent groups were tested with the Student's t test. Results without a normal distribution and independent two groups were examined with the Mann-Whitney U test. Exact and Pearson Chi-square tests were used for the analysis between categorical variables. QTc intervals >440 ms in men and 460 ms in women were accepted as prolonged. Age, GCS, hospitalization and other clinical characteristics, as well as laboratory and treatment methods (variables

whose cut-off values were determined by the receiver operating characteristic [ROC] analysis were modelled at the categorical level) were first analyzed using the univariate logistic regression (LR) method, and variables found to be significant were then reassessed using the stepwise multivariate LR method. SPSS v25.0 (Statistical Packages for the Social Sciences 25, SPSS inc., IBM Corp., Somers, NY, USA) was used for statistical analysis, and $P<0.05$ was considered as significant.

Results

Of the 110 study participants, 74 were male and 36 were female. The age of the participants ranged from 20 to 84 yr, with a mean of 45.29 (±18.15). A total of 42 (38%) of the participants had a previous comorbidity (Table I). Of these, 13 had a history of hypertension, seven had diabetes mellitus and six had both. Eight participants had chronic obstructive pulmonary disease, six had pulmonary disease and hypertension (HT) and four had chronic renal failure. The median GCS score of all the participants on admission was 11 (Table II).

The localization of the contusions was also evaluated: frontal lobe in 45 (41%), occipital lobe in 17 (15%), parietal lobe in 12 (11%), temporal lobe in eight (7%) and with involvement of multiple lobes (extensive) in 28 (25%) participants. The largest contusion volumes ranged from two to 36 cm^3, with a mean of 15.49 (±9.4). Cerebral perilesional oedema was noted in 44 (40%) participants.

ECG abnormalities were found in 53 (52%) participants on admission: S-T abnormality in 23 (20.9%), T negativity in 18 (16.4%), both in 11 (10%) patients. After cardiological consultation, no additional treatment was performed in the patients with ECG abnormalities. A total of 38 participants had a prolonged QTc interval (Table I).

In all, 11 study participants required surgical intervention: decompressive craniectomy was performed in seven and external ventricular drainage was replaced in four according to the recommendations of the Brain Trauma Foundation TBI Guidelines[21]. An increase in the volume of contusion was observed in 34 (31%) with an average of 10.09 (±4.94) cm^3. The length of hospital stay ranged between two to 35 days (median of five days). The median of GOS-E value at discharge was seven (Table II).

A significant difference was found between prolonged QTc interval with cerebral oedema ($P<0.001$) and the need for surgery ($P<0.001$). More cerebral oedema was noted in participants with prolonged QTc intervals. A prolonged QTc interval was found to be associated with a higher risk of needing surgery (Table I).

Individuals with a prolonged QTc interval were found to have a larger contusion volume compared with normal QTc (24.39±7.15 *vs*. 10.79±6.65; $P<0.001$). In addition, a prolonged QTc interval resulted in a higher midline shift ($P<0.001$) and a longer hospital stay ($P<0.001$). Furthermore, we found a significant difference between prolonged QTc interval and GOS-E score ($P<0.001$). The GOS-E score was significantly higher in individuals with normal QT interval as compared to those with prolonged QTc interval (Table II). The rate of good recovery observed in subjects with normal QTc was 84.7% and was significantly higher than in subjects with prolonged QTc (2.6%; Table I).

The relationship between the progression of contusion volume, length of hospital stay, GOS-E, midline shift within the QTc interval was investigated using the ROC test, and a cut-off value for the QTc interval was determined. A prolonged QTc interval had a higher risk of midline shift above 0.45 cm ($P=0.006$, sensitivity: 0.80 and specificity: 0.99). Prolonged QTc interval resulted in hospitalization for >8 days ($P<0.001$, sensitivity: 0.97 and specificity: 0.86). Finally, it was found that participants with prolonged QTc interval were discharged at a GOS-E score of <7 ($P=0.029$, sensitivity: 0.97 and specificity:0.85; Table III).

Progression of contusion volume of <11 cm^3, presence of cerebral oedema, length of hospital stay of >8 days and non-recovered outcomes (GOS-E<7) independently had a significant effect on prolonged QTc ($P<0.05$). Only the presence of cerebral oedema had a significant effect on prolonged QTc values. It was observed that the prolonged QTc value was 7.42 times higher in participants with cerebral oedema than in those without oedema [odds ratio (95% CI: 7.42 (1.22 - 44.93); Table IV].

Discussion

Traumatic brain injuries are a serious socioeconomic public health problem often requiring long hospital stays and affect participants' quality of life[22]. TBI includes various types of damage to

Table I. Clinical and outcome parameters I

Study parameters	Normal QTc interval, n (%)	Prolonged QTc interval, n (%)	Total, n (%)
Sex			
Male	47 (65.3)	27 (71.1)	74 (67.3)
Female	25 (34.7)	11 (28.9)	36 (32.7)
Comorbidities			
Yes	28 (38.9)	14 (36.8)	42 (38.2)
No	44 (61.1)	24 (63.2)	68 (61.8)
ECG changes			
S-T abnormality	16 (22.2)	7 (18.4)	23 (20.9)
T negativity	13 (18.1)	5 (13.2)	18 (16.4)
T negativity, S-T abnormalities	5 (6.9)	6 (15.8)	11 (10)
None	38 (52.8)	20 (52.6)	58 (52.7)
Cerebral oedema			
Yes	10 (13.9)	34 (89.5)***	44 (40)
No	62 (86.1)	4 (10.5)	66 (60)
Requirement of surgery			
Yes	0	11 (28.9)***	11 (10)
No	72 (100)	27 (71.1)	99 (90)
Outcomes			
Dead	1 (1.4)	9 (23.7)	10 (9.1)
Vegetative state	0	4 (10.5)	4 (3.6)
Severe disability	1 (1.4)	10 (26.3)	11 (10)
Moderate disability	9 (12.5)	14 (36.8)	23 (20.9)
Good recovery	61 (84.7)***	1 (2.6)	62 (56.4)

***P<0.001. P value was obtained from Mann-Whitney U or Chi-square test. ECG, electrocardiography; QTc, corrected QT

Table II. Clinical and outcome parameters II

Study parameters	Normal QTc interval		Prolonged QT interval		Total	
	Mean±SD	Minimum-maximum	Mean±SD	Minimum-maximum	Mean±SD	Minimum-maximum
Age	45.63±18.35	20-84	44.66±18.01	20-81	45.29±18.15	20-84
GCS	10.28±3.65	3-15	11.16±3.06	5-15	10.58±3.47	3-15
Highest contusion volume (cm^3)	10.79±6.65	2-30	24.39±7.15***	10-36	15.49±9.40	2-36
Progression of contusion volume (cm^3)	12.33±3.93	7-18	9.61±5.06	2-24	10.09±4.94	2-24
Midline shift (cm)	0.29±0.08	0.2-0.4	0.81±0.38***	0.20-1.50	0.66±0.40	0.20-1.50
Days of hospitalization	4.76±3.80	2-22	16.29±6.15***	6-35	8.75±7.25	2-35
GOS-E	7.25±1.15***	1-8	3.58±1.88	1-7	5.98±2.27	1-8

***P<0.001. P value was obtained from Mann-Whitney U test. GCS, Glasgow Coma Scale; GOS-E, Extended Glasgow Outcome Scale; SD, standard deviation; QTc, corrected QT

the brain parenchyma, with one of the most severe forms being haemorrhagic CC, which is closely associated with high morbidity and mortality rates and disability after severe injury[2,3]. Primary injuries of neural and cerebral microvascular tissue and secondary injuries of the extracranial organs may occur due to contusions[4,23]. Excitatory amino acids in the extracellular membrane lead to inflammatory and

Table III. Receiver operating characteristic analysis for corrected QT intervals

Study parameters	Cut-off	AUC (95% CI)	SE	Sensitivity	Specificity
Contusion volume progression	<11	0.687 (0.475-0.900)	0.155	0.68	0.67
Duration of hospitalization***	>8	0.952 (0.915-0.988)	<0.001	0.97	0.86
Midline shift**	>0.45	0.909 (0.802-0.999)	0.001	0.80	0.99
GOS-E*	<7	0.960 (0.925-0.995)	<0.001	0.97	0.85

P *<0.05, **<0.01, ***<0.001. AUC, area under curve; GOS-E, Extended Glasgow Outcome Scale; SE, standard error; CI, confidence interval

Table IV. Univariate and stepwise multivariate logistic regression analysis of prediction of corrected QT level

Variables	Univariate LR, OR (95% CI)	Stepwise multivariate LR, OR (95% CI)
Age	0.99 (0.95-1.02)	
Male	1.31 (0.56-3.06)	
Comorbidities	0.92 (0.41-2.06)	
GCS	1.08 (0.96-1.22)	
Contusion volume progression <11 cm^3	5.28 (1.50-18.51)**	2.13 (0.19-24.27)
Cerebral oedema	52.70 (15.36-180.78)***	7.42 (1.22-44.93)$^{\delta}$
Contusion volume (follow up maximum)	1.28 (1.17-1.39)***	1.09 (0.97-1.24)
Duration of hospitalization >8 days	229.40 (28.22-1865.03)***	24.13 (0.50-1172.59)
GOS-E <7 (not recovery)	205.18 (28.44-1654.66)***	1.65 (0.03-95.54)
ECG changes		
ST abnormality	0.83 (0.29-2.35)	
T-negativity	0.73 (0.23-2.34)	
T-negativity and ST abnormality	2.28 (0.62-8.40)	

P **<0.01, ***<0.001 for univariate LR; $^{\delta}P$<0.05 for stepwise multivariate LR values. Multivariable analysis was used stepwise forward selection. All selected variables were presented with OR, 95% CI and OR, odds ratio; ECG, electrocardiography; LR, logistic regression

apoptotic cascades through neurotransmitters, heat shock proteins, imbalance of sodium, potassium and calcium entering the blood–brain barrier leading to severe multisystem damage[5,6,23]. In addition to primary damage to the nervous system, these pathways can also have devastating effects on the cardiovascular, pulmonary, endocrine, haematologic and immune systems[7,13]. These multisystem secondary injuries may compromise cerebral perfusion or increase intracranial pressure and lead to a vicious cycle that negatively affects the patient mortality[23,24].

Although the effects of CC on the cardiovascular system have long been a topic of interest for researchers, there is no consensus on how to avoid these. The hypothesis that most researchers have agreed upon is the catecholamine release after CC, which leads to autonomic discharge and neurally induced cardiac injury[14,18,25,26]. These adverse cardiac events include mitochondrial dysfunction, petechial subendocardial haemorrhage, focal myofibrillar degeneration, myocyte death, repolarization changes of myocardial infarction, arrhythmias and cardiac shock[15,19].

Each repolarization abnormality (prolonged QTc interval, ischaemia-like ECG changes and morphological end repolarization abnormalities) had characteristic predisposing effects on survival and morbidity in participants with TBI[27]. In addition, increased intracranial pressure can lead to ECG changes in the form of T-depression, prolonged QTc interval and sinus bradycardia[28]. Hjalmarsson *et al*[29] studied the effects of ECG abnormalities and elevated cardiac markers on survival in intracerebral haemorrhage and found that only a prolonged QTc interval was an important factor for poor prognosis. This is in concordance to the present study where we conclude that the presence of a prolonged QTc interval on admission has poor prognostic significance in individuals with CC. Given the correlation between

CC and adverse cardiac events *via* neural pathways, we emphasize that early treatment of cerebral injury in CC reduces the incidence of cardiac complications and offers a better prognosis.

This study had several limitations. Because of the retrospective design, causal risk factors could not be inferred. The patient group without CC was not included as a control group. This affected the results in the causality part of the risk factor analysis. Furthermore, ECG was routinely evaluated only at admission. Therefore, serial ECG assessments that would have allowed us to see whether the prolonged QTc interval was normalizing could not be reviewed. For the same reason, we could not find an answer to the question of whether troponin levels are elevated in patients with prolonged QTc interval. The sample size of the study was limited primarily due to the presence of known cardiovascular disease or the use of other drugs causing prolongation of QT which were accepted as exclusion criteria. We believe that a higher predictive value can be obtained if the frequency of follow up is increased and a prospective study is designed with a healthy control group in a larger sample size. Despite these limitations, our study provides a correlation between CC and the QTc interval.

Overall this study showed that there is a significant relationship between QTc interval and volume of contusion on admission and cerebral oedema in patients with CC. A prolonged QTc interval on admission meant a larger volume of contusion and the presence of cerebral oedema. Moreover, the same prolonged QTc interval was found to be related to a higher risk of needing surgery, a longer duration of hospitalization and a remarkably lower GOS-E value.

We believe that it is important to look for ECG changes showing a prolonged QTc interval at admission which may provide better insight for the further prognosis of the CC patient. In addition, a prolonged QTc interval can be used as a marker of poor outcomes in CC. Further clinical studies are needed to verify our findings and clarify the prognostic role of QTc interval changes in CC.

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