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ORIGINAL ARTICLE

Effects of sevoflurane or ketamine on the QTc interval during electroconvulsive therapy

Feray Erdil · Zekine Begeç · Gülay Erdoğan Kayhan · Saim Yoloğlu · Mehmet Özcan Ersoy · Mahmut Durmuş

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Abstract
Purpose To evaluate the effect of sevoflurane or ketamine on the corrected QT (QTc) interval and the interval from the peak to the end of the T wave (Tp-e) during electroconvulsive therapy (ECT) in patients with major depression.
Methods This prospective, randomized, double-blinded study included 24 patients that were randomly allocated to receive sevoflurane (group S) or ketamine (group K) for ECT session. Group S patients received 8 % sevoflurane for anesthesia induction, which was maintained at 2−4 % until delivery of the electrical stimulus. Group K patients received a bolus of ketamine (1 mg/kg). The mean arterial pressure (MAP) and heart rate (HR) and the electrocardiogram (ECG) were recorded before (T1) and after induction of anesthesia (T2) and 0, 1, 3, and 10 min after the electrical stimuli ended (T3, T4, T5, and T6, respectively).
Results In both groups, the QTc interval was significantly longer at T2, T4, T5, and T6 than at baseline. The QTc interval was longer at T4, T5, and T6 in group S compared to that in group K, the Tp-e interval was significantly longer at T4 in group K both baseline and group S. The HR in group S was increased at T4 compared with group K. MAP was significantly higher after induction of anesthesia in group K compared to those in group S at all time points.
Conclusions Although group S showed a prolonged QTc interval after ECT compared to group K, the Tp-e interval in both groups was not significantly affected clinically. Sevoflurane blunted MAP and peak HR.

Keywords Electroconvulsive therapy · Ketamine · Major depression · Sevoflurane · QTc interval

Introduction

Electroconvulsive therapy (ECT) is a well-established treatment for severe depression in patients who do not respond to pharmacotherapy. Patients with major depression have altered autonomic nervous system activity as indicated by a higher resting heart rate (HR) and reduced HR variability compared to nondepressed controls [1]. In addition, ECT may cause an acute rise in QT dispersion [2], which may increase the risk of Torsades de Pointes (TdP) arrhythmias and sudden cardiac death in these patients.

Recent clinical studies have suggested that a more reliable predictor of TdP is the interval between the peak and the end of the T wave (Tp-e), which is a surface electrocardiogram (ECG) marker of transmural dispersion of repolarization (TDR) [3, 4]. It has been reported that some drugs (droperidol, ondansetron) that increase the QTc interval do not exaggerate TDR more than saline, as measured by the Tp-e interval. Thus, they appear unlikely to increase the risk of TdP in healthy pediatric patients [5].

The effect of an anesthetic agent on the QT interval may play a role in the patients' susceptibility to arrhythmias during ECT. There has been growing interest in recent

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F. Erdil (✉) · Z. Begeç · G. E. Kayhan · M. Ö. Ersoy · M. Durmuş
Department of Anaesthesiology and Reanimation, Medical Faculty, Inonu University, Malatya, Turkey
e-mail: feray.erdil@inonu.edu.tr

S. Yoloğlu
Department of Biostatistics, Medical Faculty, Inonu University, Malatya, Turkey

years in the use of ketamine in psychiatric patients as an anesthesia for ECT [6–8]. However, due to the sympathomimetic feature of ketamine, increased plasma catecholamine concentrations may increase the QTc and Tp-e intervals, which may add to the hemodynamic effect of ECT.

Sevoflurane provides a fast induction and emergence of anesthesia due to its low blood-gas solubility. It can be helpful for intravenous (iv) catheter application in patients who are frightened, sad, or psychiatrically agitated in a way that affects their ability to cooperate [9]. However, some studies have shown that sevoflurane increases the QTc interval during anesthesia induction by inhalation [10–12].

We evaluated the effects of sevoflurane and ketamine on the QTc and Tp-e intervals during ECT in patients with major depression.

Materials and methods

The study protocol was approved by the Ethics Committee of Our Medical Faculty (number 2011/196). The ClinicalTrials.gov Identifier was NCT01870219. All patients provided written informed consent prior to the study. This prospective, randomized, double-blinded study included 24 patients categorized as American Society of Anesthesiologists I−II patients, who were each scheduled for six ECT sessions for major depression. Patients were excluded from this study if they were younger than 18 years; pregnant; had a permanent pacemaker, diabetes mellitus, atrial fibrillation, flutter, or electrolyte imbalance; were taking anti-arrhythmias or β-blockers; or were unable to provide informed consent. All long-term psychiatric medications were continued.

Noninvasive arterial blood pressure, ECG, and pulse oximetry-measured oxygen saturation were monitored before the induction and throughout the duration of the anesthesia. None of the patients were premedicated. The seizure threshold is determined at the first ECT session and this session was not included in any group. For the second to seventh treatments, patients were randomly allocated to receive sevoflurane (group S) or ketamine (group K) using computer-generated random numbers. In group S, sevoflurane was initiated at 8 % for anesthesia induction and maintained at 2−4 % until the electrical stimulus was delivered, at which time it was turned off. In group K, ketamine was given as a 1 mg/kg iv bolus. An inflated tourniquet was applied to the upper arm to isolate circulation to the arm, which allowed an accurate assessment of motor seizures. Then succinylcholine (1 mg/kg) was administered iv, and ventilation was assisted with 100 % oxygen by a face mask in both groups. Investigators collecting the data were blinded to the identity of the anesthesia used. This blinding was achieved by applying peppermint oil to the facemask of the investigator and shielding the vaporizer part of the anesthetic machine with a green towel. A fruity scent was added to the patients' facemasks.

Electrical stimuli were delivered via bifrontotemporal electrodes using a Thymatron System IV ECT Instrument (Somatics Inc., Lake Bluff, IL, USA). The seizure threshold was titrated during the first session, beginning at 5 % charge (frequency 10 Hz; pulse width 0.5 ms) and restimulated at 5 % increments until evidence of generalized seizure activity was observed (or up to 25 % charge, at which point stimulus was given at 100 % charge). These data were not included in the study. Subsequent treatments were administered at a level of 150 % of the determined seizure threshold. The duration of the motor seizure was recorded as the time from ECT stimulus to the cessation of the tonic–clonic motor activity in the isolated arm. The EEG tracing was recorded continuously from two frontal electrodes. The duration of the EEG seizure was recorded from the EEG trace, and the peak HR during the convulsion was recorded from the ECG. The times were recorded from the end of succinylcholine administration until patients breathed spontaneously, opened their eyes, and obeyed commands. The interval between successive ECT sessions was 2–3 days.

The mean arterial pressure (MAP), HR, and ECG were recorded before anesthetic induction (T1), after anesthetic induction (T2), and 0, 1, 3, and 10 min after the electrical stimuli ended (T3, T4, T5, and T6, respectively). The patients were discharged from the recovery room when they met the discharge criteria.

The QT and Tp-e intervals were measured by one author (FE) who was blinded to the identity of the group allocation. The QT intervals were measured manually from the onset of the QRS complex to the end of the T wave (defined as the intersection of the isoelectric line and the tangent of the maximal downward limb of the T wave). The Tp-e interval was measured from the peak of the T wave to the end of the T wave. When U waves were present, the end of the T wave was defined as the nadir of the curve between the T and U waves. QT and Tp-e intervals were calculated for three consecutive complete P-QRS-T cycles in lead II and V5 that were averaged to give a mean QT interval for the lead II and Tp-e interval for V5. Fridericia's formula: $QT_C = QT/RR^{1/3}$ was used for corrections of the QT intervals [13].

A total sample size of 24 achieved to 97 % power to detect a mean difference of 20 ms in QTc interval, with an estimated SD of 24, and with a significance level of 0.05, using paired sample *t* test. Statistical analyses were performed using SPSS for Windows, version 10.0 (SPSS Inc. Chicago, IL, USA). Categorical variables are reported as

number (n) and percent (%). Continuous variables are reported as means (±SD). The normality for continuous variables in the groups was confirmed by the Shapiro–Wilk test. Baseline and post-baseline measurements were compared with paired t test or Wilcoxon test within each group and unpaired t test or Mann–Whitney U test were used for between-group comparisons at each time point. The Chi-square was used for categorical variables. p values of <0.05 were considered statistically significant.

Results

Four patients were excluded during the study; one patient refused further study treatments after the first study treatment in group S and three patients due to problems associated with the ECG machine in group K. In total, 13 male and 11 female subjects, with a mean (SD) age of 35.9 (11.6) years (range, 19–56 years) and weight of 69.7 (13) kg (range, 50–90 kg), received a total of 144 ECTs in six successive sessions. The distribution of the antidepressant drug used was sertraline in eight (33.3 %), fluoxetine in ten (41.7 %), escitalopram in six (25 %) patients. The recovery times after ECT were not different between groups. The durations of motor and EEG seizures were significantly longer in group K ($p < 0.05$) (Table 1).

In both groups, the QTc interval was significantly longer at T2, T4, T5, and T6 than at the baseline (for group S, $p = 0.0001$, for all comparisons, for group K, $p = 0.0001$, $p = 0.0001$, $p = 0.026$, $p = 0.0001$) (Table 2). The Tp-e interval in group S was not different at any time point compared to baseline values, whereas the Tp-e interval was significantly longer at T4 in group K ($p = 0.045$) (Table 2). In between-group comparisons, the QTc interval in group S was significantly longer at T4, T5 than in group K. The Tp-e interval was significantly longer at T4 in group K than in group S (Table 2).

Cardiac arrhythmias occurred during ECT-induced seizures in four (33.3 %) patients in group S and seven (58.3 %) patients in group K ($p > 0.05$). The cardiac arrhythmias resolved immediately before the spontaneous end of the seizure.

HR increased significantly at T3, T4, T5, and T6 in group S ($p = 0.031$, $p = 0.0001$, $p = 0.0001$, $p = 0.0001$, respectively) and at T2, T3, T5, and T6 in group K ($p = 0.0001$, for all comparisons). The HR in group S was increased at T4 compared with group K (Table 3). In group S, MAP decreased at T2 and increased at T3, T4, T5, and T6 compared to baseline ($p = 0.0001$ for T2−T5, and $p = 0.002$ for T6). The MAP increased at all measurement times in group K compared to baseline ($p = 0.0001$, for all comparisons) (Table 3). The MAP values in group K were higher at T2, T3, T4, T5, and T6 compared with group S (Table 3). Peak HR increased from the baseline in both groups ($p = 0.0001$), and was significantly higher in group K (144.4 ± 7.8) than in group S (130.7 ± 14.6) ($p = 0.0001$).

Table 1 Seizure durations and recovery times in patients

	Group S	Group K	p value
Seizure activity (s)			
Motor seizure	19.8 (4.4)	34.9 (10.6)	0.0001
EEG seizure	27.7 (4.4)	43.7 (12.6)	0.0001
Recovery time (min)			
Spontaneous breathing	2.6 (0.9)	2.7 (1.2)	0.710
Open eyes	5.8 (1.5)	6.5 (1.7)	0.119
Obeying commands	6.7 (1.4)	6.8 (1.8)	0.877

Values are mean (SD)

Group S sevoflurane group, *group K* ketamine group, p values are for between-group comparisons (paired t test or Wilcoxon test)

Table 2 QTc and Tp-e intervals in both groups

	QTc interval (ms)			Tp-e interval (ms)		
	Group S	Group K	p value	Group S	Group K	p value
T1	412.5 (13.6)	410.9 (12.8)	0.412	95.0 (10.3)	96.0 (10.9)	0.324
T2	432.5 (19.2)[a]	431.6 (16.3)[a]	0.716	95.1 (10.2)	95.9 (10.8)	0.361
T3	411.5 (26.2)[a]	409.1 (17.3)	0.580	97.1 (11.5)	97.6 (11.6)	0.792
T4	434.1 (20.8)[a]	422.5 (15.4)[a]	0.004	95.2 (9.6)	97.3 (11.5)[b]	0.035
T5	436.0 (19.8)[a]	416.8 (16.0)[b]	<0.001	95.1 (9.6)	96.9 (11.7)	0.098
T6	433.8 (18.4)[a]	424.9 (14.1)[a]	0.016	94.6 (9.7)	95.9 (11.3)	0.130

Values are mean (SD)

Group S sevoflurane group, *group K* ketamine group, p values are for between-group comparisons (paired t test or Wilcoxon test)

[a] $p < 0.001$ compared with T1 (paired t test or Wilcoxon test)

[b] $p < 0.05$ compared with T1 (paired t test or Wilcoxon test)

Table 3 Hemodynamic values in both groups

	Heart rate (bpm)			Mean arterial pressure (mmHg)		
	Group S	Group K	*p* value	Group S	Group K	*p* value
T1	79.4 (11.1)	78.6 (10.8)	0.426	93.0 (10.2)	95.1 (9.6)	0.096
T2	83.2 (10.5)	85.7 (12.2)[a]	0.280	83.0 (10.6)[a]	104.9 (10.9)[a]	<0.001
T3	86.5 (12.4)[b]	87.4 (13.5)[a]	0.677	104.6 (15.8)[a]	117.6 (9.2)[a]	0.001
T4	90.3 (12.2)[a]	83.2 (9.5)	0.012	105.8 (13.8)[a]	116.1 (8.3)[a]	<0.001
T5	92.9 (12.7)[a]	89.1 (9.2)[a]	0.125	104.2 (10.8)[a]	113.3 (7.7)[a]	0.001
T6	91.8 (11.6)[a]	89.7 (8.5)[a]	0.308	99.0 (11.0)[b]	105.6 (9.5)[a]	0.004

Values are mean (SD)

Group S sevoflurane group, *group K* ketamine group, *p* values are for group comparisons (paired *t* test or Wilcoxon test)

[a] $p < 0.001$ compared with T1 (paired *t* test or Wilcoxon test)

[b] $p < 0.05$ compared with T1 (paired *t* test or Wilcoxon test)

Discussion

In the present study, the QTc interval increased following the induction of anesthesia and ECT from the first minute in both groups; this increase was significant for 10 min after electrical stimulation ended in group S. The Tp-e interval was not different at any time point compared with the baseline values in the sevoflurane group; however, the Tp-e interval was significantly longer at T4 in the ketamine group. The peak HR and MAP were suppressed in group S compared to those in group K.

The Tp-e interval consists of the interval between the peak and the end of the ECG T wave and is an indicator of TDR. TDR is a physiological phenomenon and occurs due to inhomogeneity of repolarization rates across the myocardial wall. Physiological TDR is produced when repolarization occurs asynchronously across the myocardial wall. The morphology of the ECG T wave [4, 14, 15] results from the differential time course of repolarization across the myocardial wall. The first repolarization is that of the epicardial cells, and the completion of epicardial repolarization corresponds with the peak of the T wave. The total duration of the action potential is determined by the midmyocardial (M) cells, which are the last to repolarize whereas the end of the T wave corresponds with the full recovery of these cells [4, 14]. A Tp-e increase by 25 ms has been suggested to be clinically significant [5].

The QT interval represents the period of depolarization of the cardiac ventricles. This interval is usually reported as the corrected QT (QTc), which is the QT interval corrected for heart rate. Several anesthetic drugs prolong the QTc interval. A prolonged QT interval in ECG is associated with TdP, a malignant polymorphic ventricular tachyarrhythmia. In healthy subjects, the normal QTc interval is 380–450 ms [16], and the acceptable upper limit is 440 ms [17]. Recently, it has been proposed that prolongation of the QTc interval is a poor predictive factor for TdP. The Tp-e interval has been suggested to be a more reliable indicator of the occurrence of TdP. The normal range for Tp-e interval has not been defined.

Because medications such as antidepressants, benzodiazepines, and major tranquilizers exert an influence on the increases of QTc interval, patients with major depression may already have higher risks of ventricular arrhythmias and cardiac events before anesthesia [18]. Tezuka et al. found that the QTc interval, QTD, and QTcD increased significantly before anesthetic induction in patients with major depression, and were associated with an increased risk of ventricular arrhythmias [19]. In our study in patients with depression, the baseline QTc interval was below 440 ms, and the baseline Tp-e interval was 95 ms. The impact of the patients' medications on ECG was minimized by maintaining a fixed drug regimen throughout the entire course of ECT.

Although the effect of ketamine on the QTc interval has not been described, its use for patients with long QT syndrome is not recommended due to its sympathomimetic properties [20]. In two recent studies, ketamine facilitated the induction of isoproterenol-refractory idiopathic ventricular tachyarrhythmias in one patient, and injection with ketamine probably induced a wide-complex dysrhythmia in another patient [21, 22]. In our study, ketamine increased the QTc interval at T2, T4, T5, and T6, whereas the Tp-e interval increased significantly at T4. However, this increase in the Tp-e interval may not be significant clinically. Thus, subanesthetic dose ketamine can be used safely without concern for arrhythmias.

All inhalation agents trigger myocardial repolarization of the heart. Clinical studies have indicated that sevoflurane increases the QTc interval in all age groups during anesthesia induction by inhalation [11, 23]. In our study, the QTc interval increased in the sevoflurane group in a manner similar to that of the ketamine group; however, this increase was more significant for the sevoflurane group at

T4, T5, and T6. This result could be due to the inhibition of the K channel by sevoflurane [24]. However, no significant change in the Tp-e interval was detected in our study or in other studies [3, 4]. This discrepancy may be explained by the fact that sevoflurane has an equal effect on repolarization in epicardial, endocardial, and M cells. This situation prolongs the duration of repolarization (QTc prolongation) but does not affect TDR [3]. Additionally, the fact that there is no change in the Tp-e interval despite the statistically significant prolongation of the QTc interval in group S may indicate that the risk of TdP development with anesthetic induction via sevoflurane may be lower.

Cardiac arrhythmias such as sinusal and ventricular tachycardias and premature atrial and ventricular contractions are common during seizures or in the immediate postictal period [25]. In our study, we observed sinus tachycardia, or premature ventricular contractions, as cardiac arrhythmias during ECT-induced seizures. Because ECGs were not recorded during seizures, the QTc and Tp-e intervals were not measured in patients with cardiac arrhythmias. The peak HR was greater during seizures in the ketamine group, which may be due to the sympathetic effects of ketamine and ECT. In addition, a significant relationship between myocardial ischemia and tachycardia has been reported even without obvious hemodynamic changes such as hypotension or hypertension [26]. Hence, ketamine should be used with caution in patients with cardiovascular diseases.

All volatile anesthetic agents cause myocardial depression and some peripheral dilatations due to the dose used. In our study, MAP decreased compared to basal levels following induction in the sevoflurane group, similar to a previous report [27], and HR remained constant. Both MAP and HR increased in the ketamine group due to the sympathetic effect. The typical cardiovascular response to ECT consists of generalized autonomic nervous system stimulation, with an initial parasympathetic-induced bradycardia lasting 10–15 s, immediately followed by a more prominent sympathetic response that results in tachycardia and hypertension lasting 5 min or longer [28]. MAP and HR were high in both groups after ECT. However, the increase in MAP was greater in the ketamine group at all time points after ECT.

Although the exact mechanism of therapeutic action of ECT is unknown, the seizure duration is considered an indicator of the efficacy of ECT. It has been reported that only abortive or extremely short seizures (<15 s) can result in reduced therapeutic benefit [29]. The durations of EEG and EMG in our study were shorter than those reported by Calarge et al. [30]. This difference might be due to differences in study design, as their study included fewer patients and was not double-blind. Ketamine may cause less anticonvulsant activity when it is the only anesthetic agent used [6]. In the present study, the durations of both EEG and EMG in the ketamine group were similar to those in other studies, and longer than those in the sevoflurane group [31, 32]. Because ECT is frequently administered in an outpatient setting, the anesthetic agents used should have rapid recovery profiles. Recovery times were similar after both sevoflurane and ketamine treatment.

The use of ketamine did not result in obvious adverse effects, but it is possible that side effects of smaller magnitude may have been detected if more detailed questionnaires were used to screen for psychotomimetic effects, and this may be the main limitation of our study.

Although sevoflurane prolonged the QTc interval after ECT more than ketamine in patients with major depression, the Tp-e intervals in both groups were not significantly affected clinically. Assuming that increased TDR is a reliable indicator of the risk of TdP, these results suggest that both sevoflurane and ketamine induction may not induce TdP for ECT in patients with major depression. The peak HR and MAP were blunted with sevoflurane.

Conflict of interest The authors have no conflicts of interest to declare.

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