ORIGINAL RESEARCH



QTc changes in elderly patients: a comparison of spinal and general anesthesia

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Abstract

Background: Prolongation of the corrected QT (QTc) interval is associated with an elevated risk of ventricular arrhythmias and sudden cardiac death. Both spinal and general anesthesia are known to influence QTc duration; however, their differential effects in elderly patients remain inadequately defined. Methods: This prospective randomized study evaluated QTc interval changes in patients aged over 65 years undergoing elective surgery under either spinal or general anesthesia. QTc intervals were measured at multiple perioperative time points and compared within and between groups. General anesthesia was maintained with sevoflurane, while spinal anesthesia was administered using hyperbaric bupivacaine. Results: No intraoperative QTc prolongation was observed in either group. In the early postoperative period, the general anesthesia group showed a significant increase in QTc compared to baseline (432 \pm 24 ms vs. 443 \pm 29 ms, p = 0.023), whereas the spinal group exhibited no such change $(427 \pm 24 \text{ ms } vs. 425 \pm 28 \text{ ms}, p = 0.974)$. Despite this, postoperative QTc values were significantly higher in the spinal anesthesia group compared to the general anesthesia group (p = 0.019). Conclusions: General anesthesia with sevoflurane may contribute to postoperative QTc prolongation in elderly patients, whereas spinal anesthesia appears not to exert such an effect. Spinal anesthesia may thus be preferable for patients with heightened arrhythmia risk. Clinical Trial Registration: This study was registered at ClinicalTrials.gov (NCT06375863).

Keywords

Spinal anesthesia; General anesthesia; Sevoflurane; Electrocardiography; QTc interval

1. Introduction

The QT interval (QT), defined as the duration between the onset of the QRS complex (QRS) and the conclusion of the T wave (T) on an electrocardiogram (ECG), represents the time required for both ventricular depolarization and repolarization [1]. An extended heart rate-corrected QT (QTc) interval reflects electrical instability within the ventricles and is closely linked to a heightened risk of severe arrhythmias such as torsades de pointes, ventricular fibrillation and sudden cardiac death [2, 3].

Spinal anesthesia has been associated with QTc prolongation, likely due to its interference with sympathetic outflow in the thoracolumbar region caused by subarachnoid blockade [4–6]. Similarly, various anesthetic agents—including volatile agents like sevoflurane, isoflurane and desflurane, as well as intravenous drugs such as propofol, thiopental, etomidate and ketamine—have been reported to increase QTc duration [7–9]. Procedural maneuvers, notably laryngoscopy and endotracheal intubation, may further aggravate QTc prolongation by stimulating the sympathetic nervous system [10]. interventions continues to rise, the potential for ventricular arrhythmias becomes an increasing clinical concern, even among those without prior cardiac disease. Age-related decline in physiological resilience and diminished anesthetic tolerance further emphasize the importance of careful anesthetic technique selection in this demographic group [11].

Although numerous investigations have explored the influence of individual anesthetic agents on QTc dynamics, direct comparative analyses between spinal anesthesia and general anesthesia with sevoflurane in geriatric patients remain lacking. Therefore, the current study was designed to assess the impact of spinal anesthesia on QTc interval and to compare it with the effects of sevoflurane-based general anesthesia in elderly individuals.

2. Materials and methods

2.1 Study design and setting

This prospective randomized trial was designed to compare QTc interval changes in elderly patients undergoing spinal or general anesthesia. The study was approved by the Ethics Committee of Health Science University Haseki Training and

As the proportion of elderly patients undergoing surgical

Research Hospital (Istanbul, Turkey; approval date: 29 March 2023; approval number: 44-2023) and was registered at ClinicalTrials.gov (NCT06375863). The research adhered to the principles of the Declaration of Helsinki, and written informed consent was obtained from all participants. The study was conducted at a 700-bed tertiary care hospital in Istanbul, Turkey, between 29 March 2023 and 29 May 2023.

2.2 Patient selection and grouping

Among the 82 consecutive patients aged over 65 years scheduled for lower abdominal, extremity or urological surgery, 56 met the inclusion criteria and were enrolled. The study exclusion criteria were: having preoperative ECG abnormalities, a baseline QTc interval of \geq 450 ms, a family history of long QT syndrome, medications affecting QTc intervals, electrolyte imbalances, contraindications to spinal anesthesia (*e.g.*, coagulation disorders), unstable angina pectoris, chronic obstructive pulmonary disease, hepatic or renal failure, an American Society of Anesthesiologists (ASA) physical classification >III or obesity (Body Mass Index (BMI) >30).

The patients were randomly assigned to either the spinal anesthesia group (Group SA) or the general anesthesia group (Group GA) using a sealed envelope method in a 1:1 ratio. Randomization and group allocation were conducted by an independent researcher who was not involved in data collection.

2.3 Anesthesia procedure

In the preoperative care unit, all patients received 10 mL/kg of Ringer's lactate solution via a peripheral vein over 30 minutes. Premedication consisted of intravenous administration of 0.015 mg/kg midazolam and 1 μ g/kg fentanyl. In Group GA, general anesthesia was induced with 2.0 mg/kg propofol, and endotracheal intubation was facilitated by administering 0.6 mg/kg rocuronium to achieve neuromuscular blockade. Next, the patients were ventilated in Volume Control Ventilation (VCV) mode using an anesthesia machine (Dräger Primus, Dräger Medical Systems, Inc., Danvers, MA, USA) with a tidal volume of 6-8 mL/kg. The respiratory rate was adjusted to maintain an end-tidal CO₂ (PETCO₂) level between 32 and 36 mmHg. Anesthesia was maintained with an end-tidal concentration of 1.5-2% sevoflurane in an oxygen-air mixture (Fraction of inspired oxygen $(FiO_2) = 0.4$), with additional doses of rocuronium (0.15 mg/kg) administered as needed. At the end of the surgery, residual neuromuscular blockade was reversed with 4 mg/kg sugammadex.

In Group SA, spinal anesthesia was administered at the L3-4 or L4-5 intervertebral space using a 25-gauge Whitacre spinal needle (pencil-point) under strict aseptic conditions. Before the procedure, local anesthesia was applied with intradermal injection of 1% lidocaine hydrochloride. After confirming correct needle placement through cerebrospinal fluid outflow, 3–4 mL of 0.5% hyperbaric bupivacaine (Buvasin Spinal 0.5% Heavy; 3123001, VEM İlaç San. ve Tic. A.Ş., Istanbul, Turkey) was injected into the subarachnoid space. Immediately after drug administration, the patient was positioned supine. Sensory block levels were assessed using a pinprick test, and motor block was evaluated with the modified Bromage scale. Surgery started once a sensory block at the T10

level was achieved.

2.4 Monitoring techniques and data collection

Non-invasive blood pressure, heart rate (HR), peripheral oxygen saturation (SpO₂), and continuous ECG monitoring were performed for all patients throughout the procedure using the Mindray BeneView T8 system (patient monitor, Shenzhen Mindray Bio-Medical Electronics Co., LTD, Shenzhen, China). The QT interval was automatically measured in lead II, and the QTc interval was calculated using Bazett's formula (QTc = QT/ $\sqrt{R-R}$ interval (RR) (sec)) based on readings from the ECG monitoring system.

QTc intervals were measured and recorded at the following time points: before anesthesia induction in the general anesthesia group (Group GA; QTc-pre) or before subarachnoid injection in the spinal anesthesia group (Group SA; QTc-pre); at 1, 5, 10 and 15 minutes after endotracheal intubation or subarachnoid injection; and immediately following surgery (QTcpost). The occurrence of arrhythmias was also documented.

Patient characteristics, including age, gender, height, weight, body mass index (BMI), comorbidities and ASA physical status classification, were recorded. In Group SA, additional assessments included the maximum sensory block level and motor block recovery times. Cardiopulmonary adverse events such as hypotension (mean arterial pressure \leq 70 mmHg), hypertension (systolic arterial pressure \geq 160 mmHg or diastolic arterial pressure \geq 90 mmHg), bradycardia (HR \leq 50 beats per minute), tachycardia (HR \geq 100 beats per minute) and hypoxemia (SpO₂ <90%) were also monitored.

2.5 Statistical analysis

Data analysis was performed using the Statistical Package for Social Sciences software for Windows (SPSS, version 22.0; IBM, Chicago, IL, USA). Continuous variables are presented as mean \pm standard deviation (SD), and categorical variables are expressed as patient numbers and percentages. The normality of Continuous variables was assessed using the Kolmogorov-Smirnov and Shapiro-Wilk tests. For betweengroup comparisons of normally distributed variables, independent Student's t-tests were used. Categorical variables were analyzed using chi-squared or Fisher's exact tests as appropriate. QTc interval changes within each group were assessed using two-way analysis of variance (ANOVA), and post hoc multiple comparisons were performed using the twosided Dunnett test. Sample size calculation was based on previous research [4], which reported a QTc interval of 397.3 \pm 27.4 ms following spinal anesthesia in non-geriatric patients. A power analysis with $\alpha = 0.05$ and $\beta = 0.2$ determined that a minimum of 28 patients per group was required to detect a 20 ms increase in QTc interval with sufficient statistical power. A *p*-value of < 0.05 was considered statistically significant.

3. Results

A total of 82 consecutive patients scheduled for lower abdominal, extremity, or urological surgery were initially enrolled in this prospective randomized study. Of these, 17 patients were excluded for not meeting the inclusion criteria and five declined to participate. The final analysis included 30 patients in each group (Fig. 1). No significant differences were observed between the groups in terms of demographic characteristics, including age, gender, height, weight and BMI, as well as ASA physical status and the prevalence of comorbidities such as hypertension, diabetes mellitus, ischemic heart disease and chronic obstructive pulmonary disease (COPD) (Table 1). All surgical procedures were performed with patients in the supine position.

Throughout the study, no cardiopulmonary adverse events were recorded, and no arrhythmias or ST segment (ST) changes were detected on ECG. In Group SA, the maximum sensory block level was T4 in six patients, T7 in 14 patients, and T10 in 10 patients.

QTc intervals were similar between the two groups at most measurement points, except for QTc-post. The mean QTc-post was significantly higher in Group GA compared to Group SA (443 \pm 29 ms vs. 425 \pm 28 ms, p = 0.019). Additionally, in Group GA, QTc-post was significantly prolonged compared to QTc-pre, whereas no significant changes in QTc were observed in Group SA (Table 2).

4. Discussion

This prospective, randomized study compared the effects of spinal and general anesthesia on QTc interval changes in elderly patients. To the best of our knowledge, this is the first study to investigate this association.

The aim of this study was to compare the effects of spinal and general anesthesia on QTc interval changes as recorded on ECG. The main findings were that general anesthesia led to QTc prolongation in the early postoperative period, whereas spinal anesthesia did not. The results demonstrated that neither spinal nor general anesthesia caused QTc prolongation following induction or subarachnoid block in the intraoperative period. However, in the early postoperative period, general anesthesia resulted in a significant increase in QTc interval compared to baseline (from 432 ± 24 ms to 443 ± 29 ms, p = 0.023). Additionally, the mean QTc interval was significantly longer in the general anesthesia group than in the spinal anesthesia group (443 ± 29 ms *vs.* 425 ± 23 ms, p = 0.019).

Contrary to our findings, a study by Duma *et al.* [12] reported that anesthesia induction and airway management led to a significant increase in median QTc duration from 427 ms (412–442 ms) to 445 ms (429–468 ms). In contrast, their study found no QTc prolongation following spinal anesthesia, with QTc durations of 438 ms (425–453 ms) before spinal anesthesia and 439 ms (429–461 ms) after spinal anesthesia. However, Duma *et al.* [12] also noted that QTc was prolonged to 450 ms (433–473 ms) after the initiation of sedation following spinal anesthesia.

Ornek *et al.* [13] compared the effects of volatile induction and maintenance anesthesia (VIMA) with sevoflurane and spinal anesthesia on QT dispersion (QTd), QTc and QTd, and found no significant changes in any of these parameters at the measured time points. However, QTc values recorded at 3 minutes after induction, as well as at 1 and 3 minutes after intubation and incision, were reported to be significantly higher in the VIMA group than in the spinal anesthesia group.

Similarly, Silay *et al.* [14] observed that increasing sevoflurane concentrations from 0.5% to 5% during anesthesia induction did not result in QTc prolongation. However, QTc was significantly prolonged at 1 and 3 minutes after intubation,



FIGURE 1. Flowchart showing the study design.

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Variables	Group SA $(N = 30)$	Group GA $(N = 30)$	<i>p</i> value					
Age (yr)	68 ± 4	69 ± 4	0.291^{a}					
Sex M/F (n)	22/8	19/11	0.405^{b}					
Weight (kg)	77 ± 7	75 ± 10	0.443^{a}					
Height (cm)	170 ± 7	170 ± 8	0.837^{a}					
BMI (kg/m ²)	27 ± 2	26 ± 2	0.170^{a}					
ASA class I/II/III (n)	17/12/1	17/10/3	0.585^{b}					
Co-existing disease (n)	14 (46.7%)	12 (40.0%)	0.602^{b}					
Hypertension	8 (26.7%)	9 (30.0%)	0.774^{b}					
Diabetes mellitus	4 (13.3%)	6 (23.3%)	0.731 ^c					
IHD	5 (17.9%)	4 (14.3%)	1.000^{c}					
COPD	0 (0.0%)	3 (10.0%)	0.237^{c}					

TABLE 1 Patient characteristics

Values are expressed as mean \pm SD or percentage and number of patients. ^aStudent's t-test, ^bPearson chi-square test, ^cFisher's exact test. M: male; F: female; IHD: ischemic heart disease; COPD: chronic obstructive pulmonary disease; Group SA: spinal anesthesia group; Group GA: general anesthesia group; BMI: body mass index; ASA: American Society of Anesthesiologists.

TABLE 2. Comparison of Qie values between the groups.								
Measurement point	Group SA $(N = 30)$	Group GA $(N = 30)$	p_1	p_2	p_3			
QTc-pre	427 ± 24	432 ± 24	0.425	-	-			
QTc-1	425 ± 20	429 ± 26	0.448	0.943	0.945			
QTc-5	428 ± 18	430 ± 22	0.742	0.995	0.956			
QTc-10	425 ± 19	428 ± 22	0.598	0.951	0.713			
QTc-15	426 ± 23	434 ± 24	0.181	0.990	0.987			
QTc-post	425 ± 28	443 ± 29	0.019	0.974	0.023			

TABLE 2. Comparison of QTc values between the groups

Values were expressed as mean \pm SD. p_1 value: comparison between groups; p_2 value: comparison of QTc-post to QTc-pre value for group SA; p_3 value: comparison of QTc-post to QTc-pre value for group GA. Group SA: spinal anesthesia group; Group GA: general anesthesia group; QTc: corrected QT interval.

with increases of 31 ms and 21 ms, respectively.

Laryngoscopy, endotracheal intubation, and extubation have been well documented to trigger a sympathoadrenal response, leading to an increase in plasma catecholamine levels [15– 20], and elevated catecholamine levels have been associated with QTc interval prolongation [19, 21, 22], making QTc prolongation during periods of increased sympathetic activity, such as laryngoscopy and intubation, an expected finding [12, 13].

The influence of anesthesia agents on QTc duration remains difficult to determine, as most drugs used in anesthesia affect the QTc interval to some extent. Kleinsasser *et al.* [23] and Han *et al.* [24] investigated the specific effects of sevoflurane on QT and QTc intervals by designing studies in which anesthesia was induced and maintained with sevoflurane. After the initial measurements, the anesthesia was adjusted according to individual patient needs and surgical requirements using opioids, muscle relaxants and airway interventions such as tracheal intubation or laryngeal mask airway placement. Both studies concluded that sevoflurane significantly prolonged QT and QTc intervals [23, 24]. Despite these reports, conflicting results have been reported. Guler *et al.* [25] found no sig-

nificant changes in QTc duration with sevoflurane administration. Variations in inhaled anesthesia concentration, the time required to achieve the target minimum alveolar concentration (MAC) value, the use of premedication, and the physiological stress induced by laryngoscopy and tracheal intubation (LTI) have been proposed as potential factors contributing to discrepancies among studies [13, 14, 23, 25].

In the present study, QTc prolongation was not observed following anesthesia induction or at 1, 5, 10 and 15 minutes after endotracheal intubation, which could be attributed to the counteracting effect of propofol on sevoflurane-induced QTc prolongation and the attenuation of the sympathoadrenal response to LTI by fentanyl. Previous studies have reported that intravenous propofol can shorten the QTc interval during anesthesia induction [26, 27]. Consistent with our findings, the combined administration of propofol and fentanyl has been shown to counteract QTc prolongation associated with sevoflurane anesthesia and LTI-related sympathetic stimulation [7, 26, 28].

Spinal anesthesia has been reported to prolong the QTc interval in a dose-dependent manner [29]. However, Ornek *et al.* [13] found that selective spinal anesthesia with 5 mg

of bupivacaine did not alter the QTc interval. Similarly, Song *et al.* [4] reported that spinal anesthesia did not result in QTc prolongation in non-diabetic patients, with QTc changes of 8.5 \pm 19.9 ms (from 388.8 \pm 21.1 to 397.3 \pm 27.4 ms) following subarachnoid block. Additionally, their study classified patients into groups based on QTc interval changes, reporting that 29% exhibited no change, 57% showed moderate changes, 14% had marked changes and none experienced substantial changes in the QTc interval [4]. In contrast to these findings, studies that have used higher doses of hyperbaric bupivacaine have reported QTc prolongation following spinal anesthesia [5], which has been further supported by additional studies [5, 6], suggesting that QTc prolongation induced by spinal anesthesia primarily depends on the dose of local anesthesia and the level of the sensory block achieved [3, 5, 13, 29].

The QTc prolongation associated with spinal anesthesia can be explained by two potential mechanisms. The first mechanism involves compensatory sympathetic activation. When spinal anesthesia is administered below the T10 level, QTc prolongation may occur due to a compensatory increase in sympathetic tone in unblocked segments, including cardiac sympathetic fibers originating from T1–T4 [5, 6, 30, 31]. In previous studies where QTc prolongation was observed, the maximum sensory block level was at T10 [4, 6, 31]. In contrast, in the present study, the sensory block level ranged between T4 and T10, and no QTc prolongation was detected at any measurement point after spinal anesthesia. This finding may be attributed to the limited increase in compensatory sympathetic activity due to a smaller number of unblocked segments. The second mechanism of QTc prolongation may be related to spinal anesthesia-induced hypotension, which can enhance sympathetic outflow via baroreceptor activation [30, 31]. Differences in hemodynamic responses between studies may account for the discrepancies in findings. In contrast to our results, Akhlaghi et al. [32] reported that spinal anesthesia with hyperbaric bupivacaine resulted in QTc prolongation in elderly patients, and this difference may be attributed to a more significant decrease in blood pressure in their study population compared to ours.

Abnormal QTc prolongation is considered an independent risk factor for sudden cardiac death [33]. Several studies have demonstrated that QTc intervals increase with advancing age [34, 35]. Age-related QTc prolongation may be attributed to various factors, including cardiac hypertrophy and increased myocardial fibrosis, both of which can lead to abnormal cardiac action potential formation and conduction. Additionally, an imbalance between sympathetic and parasympathetic activity may alter myocardial repolarization, further contributing to QTc prolongation [35, 36].

The QTc interval in elderly patients has been reported to range from 418 ± 3 ms to 453.70 ± 43.77 ms, depending on factors such as comorbidities and medications [36, 37]. In the present study, the mean QTc interval in the total patient population was 429 ± 25 ms, which is consistent with previous findings.

Nakao *et al.* [37] reported that in patients over 70 years old, QTc intervals significantly increased from 434 ± 28 ms to 450 ± 37 ms within 60 minutes of sevoflurane exposure. In contrast, younger patients did not exhibit significant QTc

changes following sevoflurane administration, with values remaining stable (from 427 \pm 32 ms to 432 \pm 34 ms) [37]. However, our study did not observe QTc prolongation with sevoflurane at any intraoperative time point, and this may be explained by differences in study design, as some patients in Nakao's study [37] received epidural anesthesia in addition to general anesthesia. Additionally, variations in the timing of QTc measurements between studies may have contributed to the differing results.

This study had several limitations. First, an imbalance between sympathetic and parasympathetic activity might have influenced QTc interval changes; however, it should be noted that basal autonomic activity was not assessed. Second, plasma norepinephrine levels might have been increased due to sympathetic stimulation induced by anesthesia induction, endotracheal intubation and volatile or spinal anesthesia, yet these levels were not measured in the present study. Third, the sample size was relatively limited, which might have affected the statistical power of the findings.

5. Conclusions

In conclusion, the findings of this study indicate that neither spinal nor general anesthesia with sevoflurane caused QTc prolongation during the intraoperative period. However, general anesthesia was associated with prolongation of the QTc interval in the early postoperative period, whereas spinal anesthesia did not result in QTc prolongation in elderly patients. These results suggest that spinal anesthesia may be a preferable option for elderly patients with risk factors for arrhythmias.

AVAILABILITY OF DATA AND MATERIALS

The authors declare that all data supporting the findings of this study are available within the paper and any raw data can be obtained from the corresponding author upon request.

AUTHOR CONTRIBUTIONS

CY, SU—designed the research study. CY, SU, SK, MH, OC performed the research; wrote the manuscript. CY, MH, OC supervised the data collection. SU—analyzed and interpreted the data. SK, MH, OC—literature research. All authors have read and approved the final manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

The study protocol was approved by the Ethics Committee of Health Science University Haseki Training and Research Hospital, Istanbul, Turkey (date: 29 March 2023 and number: 44-2023) and registered at ClinicalTrials.gov (NCT06375863). This study was conducted in accordance with the Declaration of Helsinki, and written informed consent was obtained from all patients. ACKNOWLEDGMENT

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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