Sevoflurane as a Cause of Torsade de Pointes in Patient with the Long QT Syndrome

Case Report
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Background: Long QT syndrome (LQTS) is a rare condition that in certain circumstances can lead to severe and potentially lethal cardiac arrhythmia known as Torsade de Pointes (TdP). Inhalational anesthetics are among many medications and conditions known to prolong QT and thus potentially predispose the patient to TdP. Although studies have shown that sevoflurane should be safe for the healthy patients, the situation is unclear in patients with LQTS. We present a case of 14-year-old Caucasian female with the diagnosis of LQTS who developed TdP during sevoflurane inhalational induction. At the end, an anesthetic plan for patients with LQTS will be suggested.

Introduction

The QT interval represents a period of ventricular depolarization and repolarization in the cardiac cycle, beginning with QRS complex and ending with T wave (Figure 1)

Fig. 1
QT interval
Long QT syndrome (LQTS) is a rare medical condition affecting the heart that can lead to a potentially fatal heart dysrhythmia-Torsade de Pointes (TdP). Although 10-15% of population has a genetic variation in LQTS genes\(^1\) with 13 mutations identified\(^2\), the majority of the patients affected remain asymptomatic. Fewer have non-specific symptoms such as palpitations, syncope, seizures or sudden cardiac arrest. QT prolongation can also be acquired by medication or electrolyte abnormalities.

QT is not a fixed interval but shortens or lengthens with the HR. Therefore, QT should be corrected for the HR. The Bazett’s formula is the most widely used for this purpose.

\[
QT_c = \frac{QT}{\sqrt{RR}}
\]

In men, QTc>460 ms is prolonged and 440-460 ms borderline prolonged. In women those values are QTc>470 ms and QTc of 450-470 ms respectively. Cardiac rhythm disturbances usually do not occur until QTc exceeds 500 ms\(^3,4,5\).

We present a case of TdP that occurred on inhalational induction in a patient with LQTS. We also review existing data on safety of anesthetic agents in long QT patients.

This study was founded from departmental resources. The authors have no conflicts of interest to disclose.

**Case report**

A 14 year-old ASA II Caucasian female (43.5 kg, 159 cm) with LQTS and intermittent 2 degree AV block presented for pacemaker replacement and heart catheterization. Patient had no allergies and took her atenolol in the morning. Physical exam was unremarkable. ECG from 2 months before showed paced rhythm of 60 bpm, QTc of 540 ms.

Patient was extremely afraid of IV start. Therefore, inhalational induction was offered. Maintenance was planned with propofol TIVA. Premedication was omitted and patient was taken to the cardiac catheterization lab.

Standard ASA monitors were applied. Single breath inhalational induction with sevoflurane was performed. After successful induction, while IV was started, VT in form of TdP occurred (Figure 2).

Help was called, sevoflurane was stopped. Shortly after, spontaneous resolution of VT was observed. Since vital signs were stable and an IV had been started, propofol, fentanyl and vecuronium were given and the trachea successfully intubated. Case continued uneventfully under propofol TIVA. At the end of the case patient had TOF 4/4 -91%; no reversal was given. Recovery was uneventful and patient was discharged next day.

**Discussion**

TdP happens when early depolarization occurs during prolonged repolarization of the ventricle. There are numerous risk factors that predispose to QT prolongation: medications, female sex, elderly age, electrolyte deficiencies (low potassium, magnesium, calcium), slow heart rhythms, complete AV block, structural heart diseases.

Many drugs cause a QT interval prolongation, however there is no linear relationship between the length of QT and risk of arrhythmia. Recently, transmural dispersion of repolarization (TDR) has been
found to be a better predictor of which medication can cause TdP. (evaluation of difference between T-wave peak and end of action potential in endocardial, epicardial and mid-myocardial cells). Shortened TDR, despite prolonged QTc is protective against TdP.

Medications prolonging the phase 3 of cardiac cycle are proarrhythmic. Those affecting phase 2 will possess antiarrhythmic properties.

The list of medications known to be a risk factor for TdP is extensive (http://www.azcert.org/). Out of anesthetic gases and volatile anesthetics, only sevoflurane is listed. Medications such as thiopental, succinylcholine, some NDMR (atracurium, pancuronium) prolong QT, without predisposing patient to TdP. Some studies show no effect of volatile anesthetics on the length of QT at all. Majority of studies, however, show that halothane, isoflurane, enflurane, sevoflurane and desflurane do prolong the QT interval. Whether that makes them torsadogenic, remains questionable. In some studies, sevoflurane was found to decrease the TDR. In some case reports sevoflurane induced TdP, however always in presence of other predisposing factors. Propofol has been shown to prolong QT severely and cause TdP. Other papers showed minimal change in QT interval or reversal of QT prolongation in healthy patients. Not much can be stated about the benzodiazepines.

Conclusion

The review of data collected so far is inconclusive. Current opinion on taking patients with LQTS through the anesthetic safely includes:

- Obtaining baseline ECG (assess QTc)
- Continuing beta-blockers
- Providing calm environment, premedication, measures to decrease sympathetic stimulation
- Availability of defibrillator
- Using general anesthesia, anesthetic of choice (propofol, fentanyl, vecuronium). The safest volatile anesthetic is isoflurane. Avoidance of muscle relaxation reversal.
- Intra-operative temperature monitoring and hypothermia prevention.
- Regional anesthesia with epinephrine free local anesthetics.
- Providing comfortable environment, adequate pain control, QTc monitoring and avoidance of medications known to prolong the QT interval in post-op period.

Should TdP occur, stop offending drugs and address risk factors. The cornerstone of TdP therapy includes IV magnesium. If that is not successful, appropriate steps from ACLS should be followed.

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References


BRIDION— for optimal neuromuscular blockade management and improved recovery

Predictable and complete reversal

- 98% of BRIDION patients recovered to a TOF* ratio of 0.9 from reappearance of T2 at within 5 minutes2
- 97% of BRIDION patients recovered to a TOF* ratio of 0.9 from 1 to 2 PTCs at within 5 minutes2

Rapid reversal

- BRIDION rapidly reversed patients from reappearance of T2 at in 1.4 minutes1
- BRIDION rapidly reversed patients from 1 to 2 PTCs at in 2.7 minutes3

BRIDION is indicated for the reversal of neuromuscular blockade induced by rocuronium or vecuronium. In children and adolescents (aged 2-17 years), BRIDION is only recommended for routine reversal of moderate rocuronium-induced neuromuscular blockade.

Important safety information

BRIDION is not recommended in patients with severe renal impairment. Studies in patients with hepatic impairment have not been conducted and, therefore, patients with severe hepatic impairment should be treated with caution. Caution should be exercised when administering BRIDION to pregnant women as no clinical data on exposed pregnancies are available.

BRIDION has not been investigated in patients receiving succinylcholine in the intensive care unit (ICU) setting.

If neuromuscular blockade is required within 24 hours of BRIDION administration, a neuromuscular blocking agent should be used instead of rocuronium or vecuronium. The most commonly reported adverse reactions were dyspnea (mild or bitter taste) and anesthetic complications (movement, coughing, cramping, or twitching on the endotracheal tube). In patients treated with BRIDION, few cases of awareness were reported. The relation to BRIDION was uncertain. In few individuals, allergic reactions (e.g., flushing, urticarial rash) following BRIDION were reported. Children should be prepared for the possibility of allergic reactions and have the necessary treatments. In a trial of patients with a history of pulmonary complications, bronchospasm was reported in 2 patients and a causal relationship could not be fully excluded.

 Volunteer studies have demonstrated a slight (17%-22%) and transient (<3 minutes) prolongation of the postoperative intermediate part of the electromyographic reflex (PMC) with BRIDION; however, clinical studies have demonstrated no clinically relevant effect on postoperative bleeding complications with BRIDION alone in combination with anticoagulants. BRIDION has demonstrated no in vitro pharmacodynamic interaction with anticoagulants, caution should be exercised in patients on anticoagulants for a pre-existing or co-mordial condition. This pharmacodynamic interaction is not clinically relevant for patients receiving routine postoperative prophylactic anticoagulation. Although formal interaction studies have not been conducted, drug interactions were observed in clinical trials. Preliminary data suggest that clinically significant drug interactions are unlikely with the possible exceptions of bortezomib, fadilacid, and the oral contraceptives.

1. Trial of four
2. Post-hoc analysis
3. Second twitch


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