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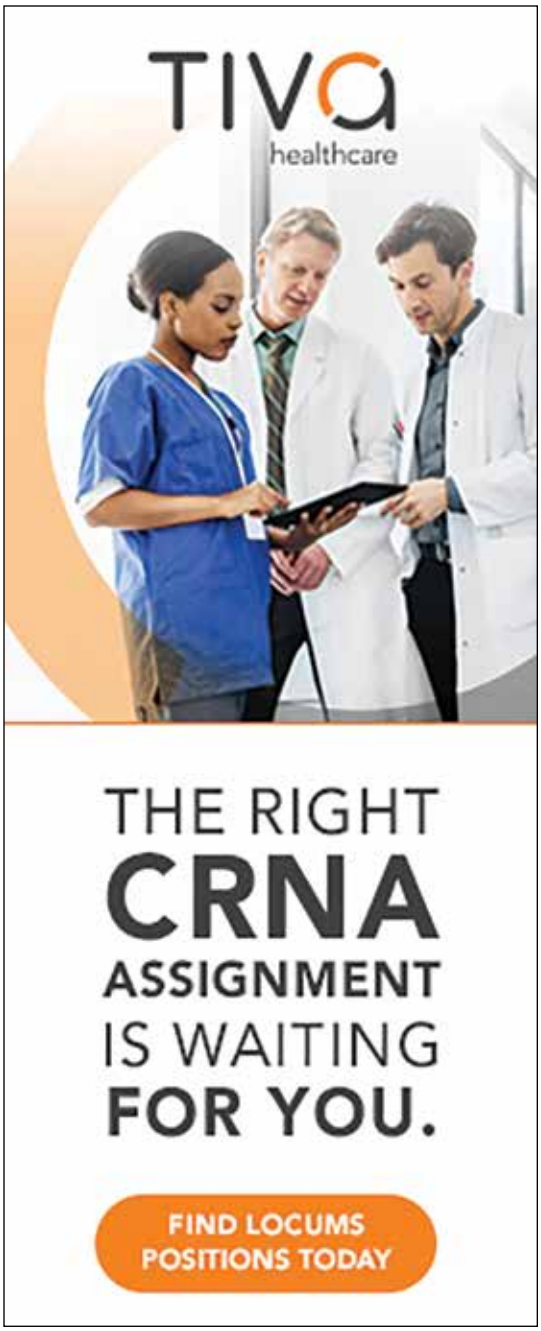
Anesthetic Management of Patients With Prolonged QT Syndrome: Case Study and Literature Review

Leah Coyle, BSN



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Anesthetic Management of Patients With Prolonged QT Syndrome: Case Study and Literature Review

Leah Coyle, BSN

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Learning Objectives

1. *Describe the pathophysiology of long QT syndrome and how it relates to the cardiac cycle.*
2. *Identify patients at risk for long QT syndrome.*
3. *Explain the pathophysiology, risk factors, and treatment for torsades de pointes (TdP).*
4. *Describe which anesthesia medications can be safely administered to patients with long QT syndrome*

INTRODUCTION

Congenital and acquired long QT syndrome (LQTS) occurs in 1 per 2500 to 10,000 individuals and is a leading cause of sudden cardiac death.[1] Sixty percent of all patients diagnosed are asymptomatic.[1] Patients undergoing surgery are exposed to a multitude of medications that can prolong the QT interval, including antibiotics, inhalation anesthetics, and antiemetics. The physiological stress and electrolyte imbalances common with surgery can contribute to QT changes.[2] Nurse anesthetists must be knowledgeable about the management of patients with LQTS. This article will provide current evidence-based recommendations for the anesthetic management of patients with LQTS in order to optimize patient safety throughout the perioperative period.

CASE SUMMARY

A 70-year-old man presented to a large academic medical facility with sepsis secondary to acute cholecystitis. He was transferred from an outside facility where a laparoscopic cholecystectomy was attempted and emergently aborted due to an episode of torsades de pointes (TdP). The patient's medical history included hypertension, aortic valve replacement (2008), chronic neuropathic pain, hyperlipidemia, and a transient ischemic attack. His daily medications included amlodipine, clopidogrel, duloxetine, metoprolol, rosuvastatin, trazodone, warfarin, and methadone. His allergies included allergies to nonsteroidal anti-inflammatory drugs (NSAIDs) and aspirin. Pre-procedure vital signs were as follows: blood pressure, 144/78; heart rate, 91; temperature, 37.3 °C; O_2 saturation, 96% on room air; and QTc (QT corrected for heart rate), 0.65 s. An echocardiogram obtained the day before surgery showed a normal mechanical aortic valve, mild left ventricular dysfunction, and a normal ejection fraction. Blood work on the day of surgery revealed the following findings: hemoglobin, 11 g/dL; hematocrit, 23%; platelets, 152,000/mcL; white blood cells, 16,000 cells/mcL; sodium, 132 mEq/L; potassium, 3.6 mEq/L; serum urea nitrogen, 12 mg/dL; creatinine, 0.6 mg/dL; and magnesium, 1.7 mEq/L. The patient received 2 g of magnesium sulfate before surgery and had stopped all anticoagulants in preparation for a laparoscopic cholecystectomy. His international normalized ratio on the day of surgery was 2.6; he received one unit of fresh-frozen plasma prior to incision.

The anesthetic plan for this patient involved avoiding QT-prolonging medications in an effort to prevent TdP and incorporating multimodal analgesia to control his chronic pain. Induction included 5 mg midazolam, 250 mcg fentanyl, and 100 mg succinylcholine. The patient was intubated and two 18-gauge peripheral intravenous lines and an arterial line were placed. A ketamine infusion at 200 mcg/kg/h was started prior to incision and 2% sevoflurane was used to anesthetize the patient. Rocuronium was given to keep 0-2/4 twitches using a standard train-of-four monitor. The patient received 1 g of intravenous acetaminophen. The emergency crash cart was readily available in the operating room and defibrillator pads were placed on the patient. The procedure was in progress with the intraoperative baseline QTc measuring between 0.43 and 0.48 s; vital signs were stable. The patient spontaneously converted into polymorphic ventricular tachycardia resembling TdP; chest compressions were started immediately. One biphasic defibrillation at 200 J was administered and the patient was converted into normal sinus rhythm. Two grams intravenous magnesium sulfate was administered over 5 minutes. The procedure was completed and reversal of neuromuscular blockade was achieved with sugammadex. The patient was appropriately following commands and was extubated and taken to the intensive care unit. The patient received an automated implantable cardioverter-defibrillator 2 days later and the rest of his hospitalization was uneventful.

The patient's prolonged QT interval was largely attributed to his chronic methadone use. Methadone is a mu agonist and *N*-methyl-D-aspartate (NMDA) antagonist.[1] It is an excellent analgesic for neuropathic pain. Methadone has a known side effect of QT prolongation.[3] When treatment is first initiated, serial electrocardiograms (ECGs) are obtained every 30 days to monitor the QT interval.[4] If methadone is used perioperatively for pain management, a baseline ECG should be obtained.[5] Normal dosing varies based upon patient response and tolerance; normal doses range from 60 to 100 mg/d.[5] This patient had been on methadone for several years and had developed a tolerance to the medication; at the time of surgery he was taking 200 mg 4 times a day, for a total of 800 mg daily. The patient's baseline prolonged QT interval, in combination with the sevoflurane and ketamine used during the surgery, are speculated to have caused TdP.

PATHOPHYSIOLOGY

A basic understanding of the electrophysiology of the heart

is needed to comprehend the implications of LQTS. The QT interval involves several integral components of the cardiac cycle: the QRS complex, which denotes ventricular depolarization, and the ST segment and T wave, or ventricular repolarization.[6] The QRS complex normally measures 0.05 to 0.10 s; it is considered prolonged when it measures greater than 0.12 s.[7] The QT interval is measured from the beginning of the QRS complex to the end of the T wave and typically is averaged over 3 to 5 beats.[8] See Figure 1 for a visual representation of a cardiac electrical complex.[9] The Bazett formula is most commonly used to calculate the QTc; it is calculated by dividing the QT interval by the square root of the R-R interval.[10] The QT interval varies inversely with heart rate; an increase in the heart rate shortens the amount of time for the ventricles to depolarize and repolarize. In contrast, bradycardia gives the ventricles more time for the electrical signal to pass through cardiac tissue.[11] Because the QT interval is dependent on the heart rate, normal values are described by using the QT interval corrected for heart rate, or the QTc.[8] A prolonged QT interval translates to a prolonged repolarization of the ventricles. During repolarization, the cardiac myocytes are susceptible to reentry ventricular rhythms such as premature ventricular contractions, or PVCs. A PVC during repolarization of the ventricles can put the electrical signals of the heart into chaos and cause a deadly, nonperfusing cardiac rhythm to ensue.[11] A review of normal QTc values for men, women, and children is shown in Table 1.[11] Research has been done on why QTc intervals differ between males and females. The QT interval varies during menstrual cycles, which supports the idea of hormonal influences on cardiac conductivity.[8] Males are at higher risk for LQTS during childhood and females are higher risk from the teen years into adulthood.[12] Testosterone has been shown to decrease the QTc; this explains why the incidence in teenage males is much lower than in females.[8]

Table 1: QTc Values in Men, Women, and Children[10]

QTc (s)	Men	Women	Children
Normal	<0.43	<0.45	<0.45
Prolonged	>0.45	>0.47	>0.46

In 1996 François Dessertenne, a French physician, coined the term *torsades de pointes* after seeing an ECG exhibiting a specific form of polymorphic ventricular tachycardia.[4] *Torsades de pointes* translates to "twisting peaks," which describes the QRS complexes twisting around a single isoelectric line in a rhythmic fashion.[8] TdP is often self-limiting and causes momentary syncope. It can quickly progress into ventricular fibrillation and cause death. Clinicians do not know what causes TdP to cease spontaneously.[13] The immediate pharmacological treatment for TdP is intravenous magnesium sulfate.[8,14] A 2-g bolus of intravenous magnesium sulfate should be given over 2 to 5 minutes.[14] Although the exact mechanism of action of TdP is unclear, it is speculated that magnesium sulfate works through blocking sodium and calcium channels.[8] If the patient is hypokalemic, administering intravenous potassium to normalize serum levels is recommended.[8] A second method to treat TdP is to increase the heart rate either pharmacologically via dobutamine, atropine, or isoproterenol or mechanically via a temporary external pacemaker.[14] Overdrive pacing is effective because it shortens the QTc by increasing the heart rate.[8]

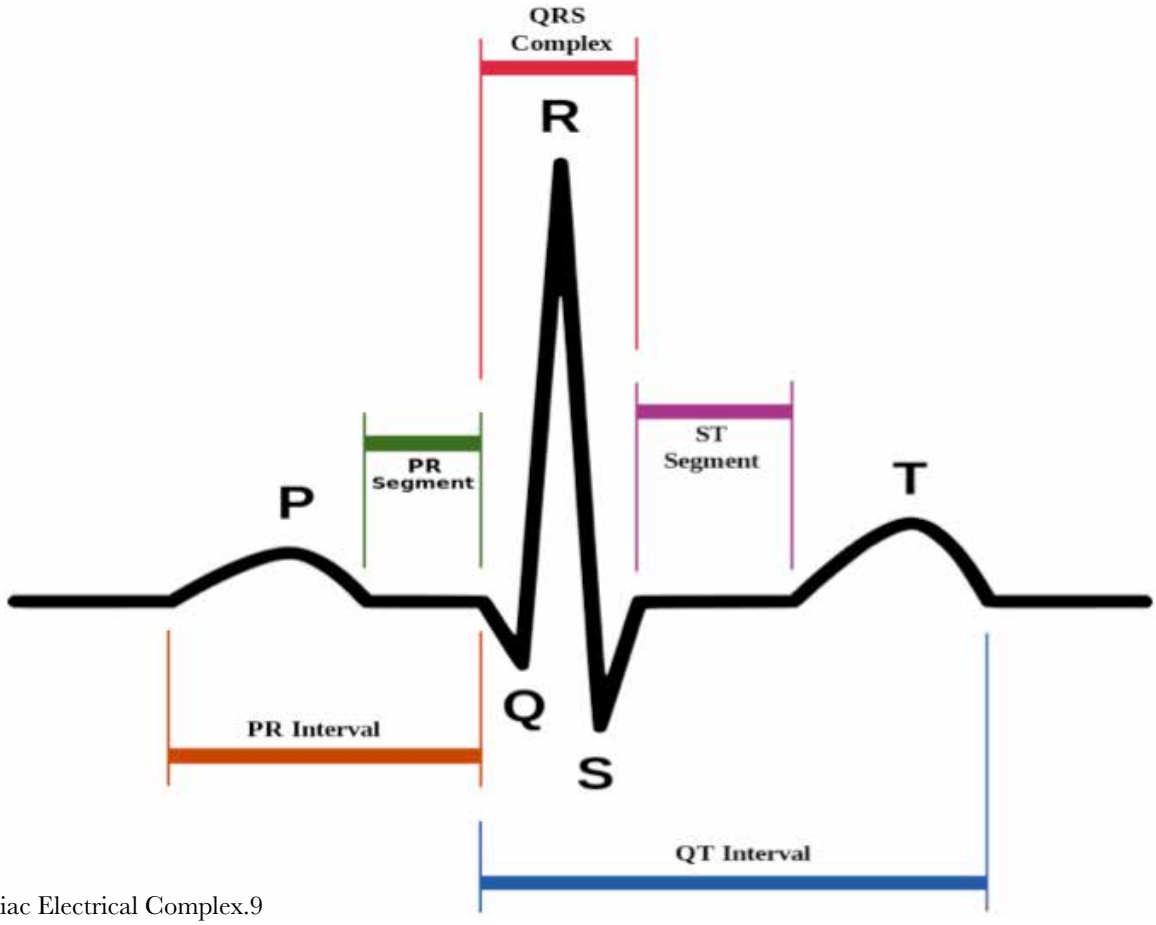

Figure 1: Cardiac Electrical Complex.9

Lidocaine blocks sodium channels and prolongs the cardiac action potential and is another option for treatment of TdP. First-line treatment is always magnesium sulfate.[7,14] See Figure 2 for an example of TdP captured by a 12-lead ECG.[15]

CONGENITAL LQTS

Historically, LQTS was separated into 2 categories: congenital and acquired. Congenital LQTS is the result of a genetic mutation that alters cardiac myocyte function.[12] Seventy percent of patients with a diagnosis of LQTS will have a gene mutation in 1 of 12 genes linked to this condition.[12] More specifically, 90% of all genotype-linked cases can be caused by 1 of 3 genes: *KCNQ1*, which causes LQT type 1 (LQT1); *KCNH2*, which causes LQT type 2 (LQT2); and *SCN5A*, which causes LQT type 3 (LQT3).[13] Determining the specific gene mutation allows clinicians to predict physiological manifestations; see Table 2. Patients with LQT1 often have negative cardiac events during physiological stress or exercise, with a large number of events occurring after swimming.[13] Patients with LQT2 experience events after auditory stimuli such sudden loud noises, or even a telephone ringing.[13] Events for patients with LQT3 occur most often when the patient is at rest or asleep.[13]

Table 2: Types of LQTS[12,13]

Type	Gene	Cardiovascular events are seen:
LQT1	*KCNQ1*	With physical exercise, especially swimming
LQT2	*KCNH2*	With auditory stimulation, such as a telephone ringing
LQT3	*SCN5A*	At rest, such as while sleeping

ACQUIRED LQTS

Acquired LQTS is attributed to electrolyte imbalances or specific medications. Once the electrolyte imbalances are corrected or the offending medications are stopped, the prolonged QT intervals will typically resolve. Patients with metabolic abnormalities that predispose them to electrolyte abnormalities should be assessed for LQTS. Examples include kidney failure and eating disorders.[11] Hypocalcemia leads to delayed ventricular repolarization and a prolonged QT interval.[6] In addition, hypokalemia and hypomagnesemia predispose the patient to cardiac abnormalities.[8] Other risk factors for acquired LQTS include female sex, bradycardia, congestive heart failure, and left ventricular hypertrophy.[8,16] The time period directly after conversion from atrial fibrillation is another time period during which the heart is susceptible to TdP.[8]

Over 50 medications can cause LQTS.[1] A study by Curtis et al[17] looked at pharmaceutical records and recorded how often a QT-prolonging drug was prescribed in the outpatient setting; nearly 23% of patients were prescribed one of these drugs, with one-half of the prescriptions being antidepressants.[17] Tricyclic antidepressants such as amitriptyline and nortriptyline are known to prolong the QT interval.[1] Another class of antidepressants, selective serotonin reuptake inhibitors (SSRIs), also prolongs the QT interval.[16] Macrolide and quinolone antibiotics such as erythromycin, clarithromycin, and azithromycin also prolong the QT interval.[16] Quinidine, procainamide, flecainide, sotalol, and amiodarone are examples of antiarrhythmics that are known to alter cardiac repolarization.[1] These medications, among others, should not be prescribed for individuals with LQTS.

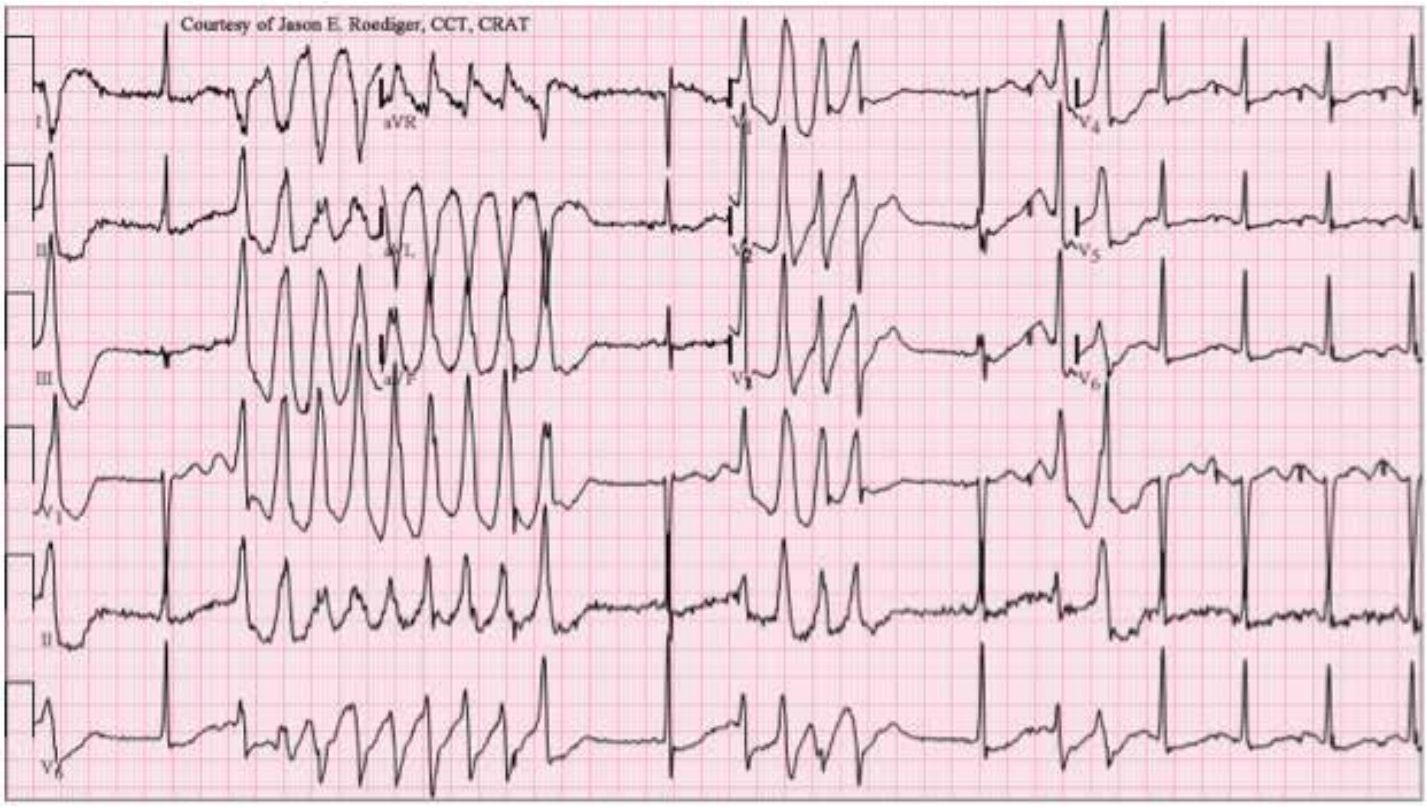

Figure 2: Twelve-Lead ECG Showing TdP.15

ANESTHETIC MANAGEMENT

Managing the patient with LQTS perioperatively can be challenging. Many patients with LQTS will have normal baseline ECGs; they only exhibit symptoms when exposed to a drug that prolongs the QT interval.[8] Also, patients may have intermittent QT prolongation; a symptomatic patient can have a normal ECG.[10] A comprehensive preoperative evaluation should include a physical examination, medication review, and medical history. If a patient has a family history of sudden cardiac death, a genetic LQTS should be suspected. If the patient has been prescribed beta-blockers, ensure that they are taken on the day of surgery.[14] Obtaining a 12-lead ECG preoperatively can provide the anesthetist with the baseline QTc.[18] Electrolytes should be checked and optimized before the start of surgery. If possible, avoid any medication that prolongs the QT interval. Intraoperative monitoring of the QTc is encouraged; many modern cardiac monitors have this ability.[14] Heart rate should be maintained at less than 120 beats/min by using beta-blockers.[1] Blood pressure should be maintained within 20% of the patient's pre-procedure measurements.[6] Electrolytes should be checked frequently during surgery if large blood loss or fluid shifts occur. The patient's body temperature should be closely monitored and hypothermia should be avoided; shivering can cause sympathetic activation.[14] A defibrillator and emergency medications should be readily available. After the procedure the patient should have continuous ECG monitoring.

Anesthetic management of patients with LQTS should focus on reducing sympathetic stimulation; any intervention that blunts the sympathetic response prevents prolongation of the QT interval. For example, the insertion of a Laryngeal Mask Airway can attenuate the sympathetic stimulation and subsequent QTc prolongation that occurs with direct laryngoscopy and intubation.[19] In healthy patients, spinal anesthesia will transiently cause an increase in the QTc that will eventually return to baseline after 15 minutes.[20] This can be attributed to sympathetic stimulation that occurs when a nervous patient receives spinal anesthesia. As that stimulation gradually resolves, the QTc returns to normal.[20] Proper premedication before neuraxial anesthesia can help to mitigate this.[20] Spinal blocks and epidurals have both been safely used in patients with LQTS.[18] Local anesthetics, both esters and amides, have no impact on the cardiac cycle when injected locally; utilizing a peripheral nerve block is an excellent option for these patients.

Anesthetists must use clinical judgment when developing the anesthetic plan for patients with LQTS. All volatile anesthetics including isoflurane, desflurane, and sevoflurane cause a dose-dependent, reversible prolongation of the QT interval.[3,18,21] Thiopental causes signification QT prolongation because it causes increased plasma norepinephrine concentrations.[3] Propofol is recommended for the induction of anesthesia; it has been proven to have minimal impact on the QTc interval.[14] Propofol has been shown to decrease QT prolongation caused by inhalation agents.[14] Although it is known for being cardiovascularly stable, etomidate can cause QTc prolongation.[3] Dexmedetomidine, an alpha-2 agonist, has been shown to significantly shorten QTc intervals in adults by decreasing sympathetic activity.[20] However, the bradycardia that often occupancies dexmedetomidine use can have negative cardiac effects.[3] In children, dexmedetomidine has been shown to have the opposite effect and can prolong the QT interval.[22] Benzodiazepines have no effects on the repolarization time of the heart and are excellent anxiolytics in this patient population.[14,18] Most opioid analgesics, including fentanyl, morphine, alfentanil, and remifentanil, have no impact on the QT interval.[3] The only opioid that has been shown to increase repolarization time is sufentanil.[18] Using narcotics to help blunt sympathetic stimulation prior to direct laryngoscopy is beneficial to all patients, especially those with LQTS.[3] Meperidine has been shown to prolong the QT interval.[16] Due to its propensity to cause sympathetic stimulation, ketamine can also cause prolongation of the QT interval.[3,14]

Careful consideration should be given when deciding if a patient requires neuromuscular blockade for surgery. The depolarizing neuromuscular blocker succinylcholine does cause significant QT prolongation.[3,14] Studies of the impact of nondepolarizing neuromuscular blockers on the QTc vary. Several studies of vecuronium, atracurium, and rocuronium show that they can be safely used in patients with LQTS.[3] Other studies reveal that rocuronium, when dosed at 0.6 mg/kg and 1.2 mg/kg, does cause prolongation of the QTc interval.[21] The confounding research is attributed to the fact that a depolarizing paralytic is never given systemically without other pharmacologic agents. When considering a reversal agent, sugammadex produces no change in the QTc.[23] The anticholinergics atropine and glycopyrrolate cause QT prolongation and have been implicated in cases involving TdP.[14]

Controlling postoperative nausea and vomiting (PONV) is a challenge for patients with LQTS because many medications used to prevent PONV affect the QTc. All serotonin-blocking drugs can cause QT prolongation.[1] Examples include ondansetron, dolasetron, granisetron, and palonosetron.[24] One study by Kim et al showed that a 4-mg dose of ondansetron did not cause the QTc to prolong; an 8-mg dose did cause a significant change.[25] This was confirmed by further research.[2] The Food and Drug Administration issued a black box warning for droperidol, a dopamine receptor blocker, concerning QT prolongation. Doses greater than 25 mg can cause instantaneous QT prolongation and can lead to TdP or ventricular fibrillation. The typical dose for PONV, 0.625 mg to 2.5 mg intravenous, has not been shown to prolong the QT interval.[24] Metoclopramide, a dopamine antagonist and gastrointestinal tract prokinetic, does prolong the QT interval.[26] Dexamethasone has no impact on the QT interval, making it an excellent option to treat or prevent PONV.[8]

CONCLUSION

Many patients have a diagnosis of LQTS, and anesthetists must be knowledgeable of evidence-based recommendations concerning the anesthetic management of these patients. Optimizing patient safety throughout the perioperative period is key. Preoperative optimization, intraoperative medication selection, and postoperative monitoring are all important aspects of the anesthetic to consider when caring for a patient with LQTS.

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Questions:

POST TEST

1. The QT interval varies inversely with heart rate.
   - A. True
   - B. False

2. Patients with LQT2 will often have adverse cardiac events after which event?
   - A. Auditory stimuli
   - B. Sleep
   - C. Psychological stress
   - D. Exercise

3. Which narcotic can prolong the QT interval?
   - A. Remifentanil
   - B. Morphine
   - C. Fentanyl
   - D. Sufentanil

4. Which of the following is NOT a risk factor for acquired LQTS?
   - A. Congestive heart failure
   - B. Tachycardia
   - C. Left ventricular hypertrophy
   - D. Electrolyte imbalances

5. Which medication given intravenously is a first-line treatment for TdP?
   - A. Lidocaine
   - B. Atropine
   - C. Magnesium sulfate
   - D. Dobutamine

6. The incidence of LQTS is higher in teenage males than in teenage females.
   - A. True
   - B. False

7. In the pediatric population, the QT interval is considered prolonged when it exceeds what value?
   - A. >0.43 s
   - B. >0.44 s
   - C. >0.45 s
   - D. >0.46 s

8. All doses of droperidol prolong the QT interval.
   - A. True
   - B. False

9. The QRS complex is considered prolonged when it reaches what value?
   - A. >0.08 s
   - B. >0.10 s
   - C. >0.12 s
   - D. >0.14 s

10. Which of the following antiarrhythmics can be safely given intravenously to a patient with LQTS?
    - A. Amiodarone
    - B. Procainamide
    - C. Quinidine
    - D. Lidocaine

11. Benzodiazepines such as midazolam can prolong the QT interval.
    - A. True
    - B. False

12. The QRS complex corresponds to what portion of the cardiac cycle?
    - A. Atrial depolarization
    - B. Ventricular repolarization
    - C. Ventricular depolarization
    - D. None of the above

13. Which of the following volatile anesthetics prolongs the QT interval?

A. Isoflurane

B. Desflurane

C. Sevoflurane

D. All of the above

14. Patients with LQTS should take all beta-blockers as prescribed on the day of surgery.

A. True

B. False

15. Which drug is recommended for the induction of general anesthesia in patients with LQTS?

A. Propofol

B. Thiopental

C. Etomidate

D. Sufentanil

16. Which drug does not prolong the QT interval?

A. Amitriptyline

B. Meperidine

C. Succinylcholine

D. Dexamethasone

17. Which percentage of patients with LQTS has a specific gene mutation that is linked to this syndrome?

A. 40%

B. 50%

C. 60%

D. 70%

18. Which antibiotic does not prolong the QT interval?

A. Clarithromycin

B. Azithromycin

C. Vancomycin

D. Erythromycin

19. Which reversal agent does not alter the QT interval?

A. Neostigmine + Glycopyrrolate

B. Sugammadex

20. What is the name of the formula used to calculate the QTc?

A. LaPlace

B. Starling

C. Hagen-Poiseuille

D. Bazett

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