Review and Management of the Dental Patient With Long QT Syndrome (LQTS)

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Long QT syndrome (LQTS) is a unique cardiovascular condition, with both congenital and acquired forms that afflict patients. These patients show a lengthening of the repolarization phase of the cardiac cycle, which can be best visualized on an electrocardiogram (ECG). The ECG changes can include QT interval (the time between the start of the Q wave and the end of the T wave, as seen on an ECG) and T wave abnormalities, as well as progression to torsades de pointes and ventricular fibrillation. The ECG changes are most commonly elicited by physical activity, emotional stress, and certain medications. This condition represents a challenge for the oral and maxillofacial surgeon. Patients with LQTS must receive proper medical management and a controlled and anxiety-free surgical environment. The purpose of this article was to present a review of LQTS and provide recommendations for effective surgical management. Additionally, a case report of a patient with LQTS, treated by one of the authors, has been included.

Key Words: Long QT syndrome; Torsades de pointes; Ventricular fibrillation.

Long QT syndrome (LQTS) is a cardiovascular disorder resulting from improper functioning of cardiac ion channels. The malfunction can be attributed to either genetic mutation, as seen with congenital LQTS, or from exposure to drugs or metabolic abnormalities, as seen with acquired LQTS in patients who carry the LQTS gene. The resultant effect is a lengthening of the ventricular repolarization phase of the cardiac cycle. Patients with LQTS frequently show significant changes on the electrocardiogram (ECG) that can include prolongation of the QT interval (the time between the start of the Q wave and the end of the T wave, as seen on an ECG) and T wave abnormalities, with subsequent transformation to torsades de pointes and ventricular fibrillation (Figure). Physical activity, emotional stress, and drugs can elicit these ECG changes. The resultant impact on these patients is considerable, with clinical outcomes that include syncope, seizures, torsades de pointes, and sudden cardiac

death. The age of patients that can be affected by LQTS ranges from in utero to adulthood.

ECG

The QT interval on ECG represents both the depolarization and repolarization phases of ventricular action potential. Unlike the QRS segment, the normal QT interval shows circadian variation, and it is affected by changes in heart rate and autonomic tone.¹ In order to correct for the influence of heart rate, the following formula developed by Bazett is commonly utilized (all values measured in seconds):²

$$QT_c = \frac{QT}{\sqrt{RR'}}$$

where:

QT = measured QT interval from the beginning of the Q wave to the end of the T wave

RR' = measured R to next R' interval

QTc = heart rate – corrected QT interval

When analyzing QTc, one finds that the normal values for men and women are slightly different. This has

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Table 1. QT Intervals in Males and Females

	Males	Females
Normal	Below 430 ms	Below 450 ms
Borderline	430–450 ms	450–470 ms
Prolonged	450 ms	470 ms

been attributed to females' showing a slightly longer baseline QTc than men in clinical and animal trials. In general, it is accepted that a value of QTc greater than 0.440 seconds is considered prolonged; however, more specific values as they apply to LQTS have been identified (Table 1).³ These values are based on corrections derived from Bazette's formula because this formula has been more widely used and studied.

The most dreaded outcome of LQTS is progression to polymorphic ventricular tachycardia (torsades de pointes). An individual episode of torsades is usually short-lived; it may go unnoticed on the ECG. However, the tendency to recur in rapid succession is what can lead to syncope and death in these patients. ECG findings emerge after a prolonged QT interval, followed by progressive twisting of the QRS complex around the baseline with an associated heart rate ranging from 150 to 300 bpm.⁴ Clinical and experimental studies suggest that the progression to torsades is rarely associated with a QTc of less than 500 milliseconds.¹

Additional ECG findings include T-wave and U-wave abnormalities. The T waves may be larger or prolonged, or they may have a notched, bifid, or biphasic appearance.^{3,5} An additional feature of LQTS encountered on ECG is the so-called T-wave alternans, where there is beat-to-beat variation in T-wave amplitude.³ Although pathognomonic for LQTS, this ECG alteration has high specificity, but it lacks sensitivity.^{3,6} Additionally, U-wave abnormalities may be seen that include prominent and bizarre U waves and U-wave alternans.

CONGENITAL LONG QT SYNDROME

Currently there are 8 major recognized genotypes of congenital LQTS. The most common types are LQT1 (42%) and LQT2 (45%), which together account for almost 90% of all patients affected by congenital LQTS. Both these types affect the function of cardiac potassium channels, causing ventricular depolarization to be delayed and the QT interval to be lengthened. A complete discussion of all 8 types of this condition is beyond the scope of this article; for more information, the March 2006 review of LQTS in *Genetics in Medicine* by Modell and Lehman⁷ can be reviewed. Of particular interest in this review is the study cited by Schwartz et al of 670 patients with LQTS, which showed that 62% of cardiac events in LQT1 patients oc-

Normal Sinus Rhythm

Long QT Syndrome

Torsades de Pointes

Top: ECG showing normal sinus rhythm with a normal QT interval. Middle: ECG showing a lengthening of the QT interval, as seen in long QT syndrome. Bottom: ECG showing polymorphic ventricular tachycardia. Note the characteristic twisting of QRS complexes of torsades de pointes. Images are for illustration purposes only and are not to scale.

curred during exercise, 43% in LQT2 were connected with episodes of emotional stress, and 39% in LQT3 occurred during rest or sleep.⁷

ACQUIRED LONG QT SYNDROME

The etiology of acquired LQTS is linked to various medications, as well as drug use, electrolyte deficiencies related to diet, medical conditions, and recent cardioversion of atrial fibrillation. The medications that prolong the QT interval directly block potassium channels in a way that closely mimics the LQT2 genotype of congenital LQTS.⁷ The same loss in function of potassium ion channels is seen, with a resultant prolonged QT interval.

GENERAL TREATMENT CONSIDERATIONS

The treatment of patients with LQTS presents a challenge for dentists and dental specialists. Two common triggers that elicit prolongation of the QT interval in these patients are emotional stress and auditory stimuli. So, for effective and safe management of these patients, it is crucial to provide a treatment environment that is as stress free as possible. Among the considerations in planning surgical procedures, attention must be given to preoperative manage-

ment, intraoperative monitoring, and postsurgical evaluation. Perhaps an even greater treatment challenge is for those individuals having undiagnosed LQTS. Currently in the United States it is speculated that 10 to 15% of patients with LQTS have normal QTc duration, which helps explain why the condition is believed to be underdiagnosed. The true prevalence of this disorder is difficult to estimate, but it is expected to occur as often as 1 in every 10,000 individuals.⁸ Although this article addresses the needs of known LQTS patients, the same information is applicable for undiagnosed patients. Knowledge of management strategies may prove useful for practitioners, should the condition initially present in the office as a sudden cardiac event in response to stress or auditory stimuli.

Preoperative

Beta-blockers. The standard treatment for LQTS is beta-blockers, with propranolol being the most widely used.³ Preoperatively, all patients with LQTS should be on beta-blocker therapy, which is continued throughout the perioperative period with their normal dose given on the day of surgery. Beta-blockers lower the maximal heart rate and provide permanent pacing, which eliminates pauses that can predispose the patient to the onset of torsades de pointes.

Electrolyte Management. Deficiencies in potassium, magnesium, or calcium can predispose a patient to a delay in repolarization of the heart. Therefore, attention must be given to the assessment of serum electrolytes prior to surgery, should intervention for replacement be required. It is considered preferable for potassium levels to be within the higher range of normal (4.5 to 5.0). If indicated, 10–40 mEq/L potassium can be given either orally or via infusion over 2 to 3 hours. Even if normal electrolyte values are identified, pretreatment with magnesium infusion of 30 mg/kg over 2 to 5 minutes is considered beneficial because of its effects on potassium and sodium currents implicated in generating early after-depolarization.⁹

Pacemakers and ICDs. Discussion with patients regarding pacemakers and implantable cardioverter defibrillators (ICDs) is absolutely necessary. These treatment modalities are generally reserved for those patients who have not responded to pharmacologic therapy, and as a result they should be considered to be at higher risk for the development of torsades. The use of certain instruments such as electrocautery can interfere with the function of pacemakers and ICDs. Between the 2 forms of electrocautery available, bipolar is preferred over monopolar for patients with pacemakers and ICDs. In the bipolar form, the active and returning electrodes are together at

the site of cauterization, whereas the monopolar form utilizes the patient's body as a ground. The grounding pad usually is positioned on the thigh, which carries the returning current back to the machine. The main concern with the use of electrocautery is that the current can interfere with the functioning of the pacemaker or ICD. Potential interactions include inducing ventricular arrhythmias or fibrillation, asynchronous pacing, inhibition of pacing, inappropriate shock therapy, and electrical reset.¹⁰ The likelihood of these interactions is greatly reduced with the use of bipolar electrocautery, largely because there is no current going through the patient; however, the preoperative recommendations for these devices are universal. It is necessary in the preoperative phase to set these devices to an asynchronous pacing mode and for the defibrillating function of the ICDs to be deactivated. For ICDs, tachycardia therapy must be deactivated or, if available, settings such as Electrocautery Protection Mode or Off-Electrocautery can be used.^{10,11} These precautions prevent the ICD from detecting the cautery electrical signal and delivering an inappropriate shock.¹² If a specific mode exists for electrocautery use, tachyarrhythmia detection and therapy features are deactivated, and the pacing mode switches to an asynchronous pacing mode—Ventricular Asynchronous, Atrial Asynchronous, or Dual Chamber Asynchronous.^{10,12} If tachycardia therapy is simply programmed to Off or a magnet is placed over the device to temporarily inhibit or deactivate tachycardia therapy; the bradycardia pacing mode remains as programmed.¹⁰ For proper programming of pacemakers before surgery, a magnet can be placed over the device to pace asynchronously at the magnet rate, or the device can be programmed to an asynchronous pacing mode.¹⁰ To avoid compromising patient safety, pacemaker or defibrillator patches can be applied before induction of anesthesia. During the procedure, the external pacemaker or defibrillator should be immediately available and ready for use; postoperatively, the ICD should be turned back on while the patient is being monitored in recovery. As described previously, permanent pacing eliminates pauses, which can predispose a patient to an onset of torsades de pointes. Without the benefit of the ICD during the procedure, it is mandatory to have an ECG connected for proper patient monitoring.⁹ Additionally, we feel that the patient's cardiologist should be contacted concerning recommendations for programming of the ICD or pacemaker both during the procedure and postoperatively.

Medications That Prolong the QT Interval. In the event the patient is taking medications known to prolong the QT interval or elicit torsades de pointes, they must be identified preoperatively (Tables 2 and 3).⁹ Consideration can be given to discontinue the medication during the procedure if it will not compromise the patient's health.

Table 2. Drugs That Prolong the QT Interval or Induce Torsades⁹

<i>Well Documented Association</i>	<i>Moderate Association or Case Reports</i>	<i>Increased Risk With LQTS</i>	<i>Unclear Association</i>
Cardiac			
Amiodarone	Flecainide	Dobutamine	Mexiletine
Bepridil	Moexipril/HCTZ	Dopamine	
Disopyramide	Nicardipine	Ephedrine	
Dofetilide	Isradipine	Epinephrine	
Ibutilide		Isoproterenol	
Procainamide		Midodrine	
Quinidine		Norepinephrine	
Sotalol		Phenylephrine	
Antimicrobials			
Clarithromycin	Azithromycin		Ampicillin
Erythromycin	Foscarnet		Ciprofloxacin
Grepafloxacin	Gatifloxacin		Fluconazole
Pentamidine	Levofloxacin		Itraconazole
Sparfloxacin	Moxifloxacin		Ketoconazole
	Telithromycin		TPM/SMX
Psychotropics			
Chlorpromazine	Lithium		Amitriptyline
Haloperidol	Quetiapine		Amoxapine
Mesoridazine	Risperidone		Clomipramine
Pimozide	Venlafaxine		Desipramine
Thioridazine	Ziprasidone		Doxepin
			Fluoxetine
			Galanthamine
			Imipramine
			Nortriptyline
			Paroxetine
			Protriptyline
			Sertraline
Miscellaneous			
Arsenic Trioxide	Amantadine	Albuterol	
Chlorpromazine	Astemizole	Cocaine	
Cisapride	Chloral Hydrate	Fenfluramine	
Domperidone	Dolasetron	Levalbuterol	
Droperidol	Felbamate	Metoproterenol	
Halofantrine	Fosphenytoin	Phentermine	
Haloperidol	Granisetron	Phenylpropranolamine	
Levomethadyl	Indapamide	Probuco	
Mesoridazine	Octreotide	Pseudoephedrine	
Methadone	Ondansetron	Ritodrine	
Organophosphates	Salmeterol	Terbutaline	
Primozide	Tacrolimus		
Thioridazine	Terfenadine		
	Tamoxifen		
	Tizanidine		
	Voriconazole		

Should this be contraindicated, patient safety can be maintained through close monitoring throughout the procedure as well as during the postoperative period.⁹

Preoperative Medication. It has been well documented that stress and loud noises can trigger LQTS. Emotional stress prior to surgery can therefore be greatly reduced by oral or IV benzodiazepine. Midazolam decreases the sympathetic activity in unstimulated patients and has been shown not to affect QTc or cause dysrhythmias on ECG when used in combination with fen-

tanyl. It has been recommended that ketamine be avoided in patients with LQTS because of stimulating the sympathetic nervous system.⁹

INTRAOPERATIVE

Intravenous Agents

Among the benzodiazepines, midazolam has been studied the most and does not seem to adversely affect

Table 3. Additional Medications to Avoid for Patients with LQTS⁹

Ketamine
Succinylcholine
Atropine
Glycopyrrolate

the QT interval when used as an anxiolytic or an induction agent.⁹ However, all studies that confirm these findings were done on patients without LQTS, so the true effect of benzodiazepines on patients with LQTS is unknown.

Propofol has been shown to have little or no effect on the QTc interval for those patients undergoing minor surgical procedures.^{3,9,13} Additionally, it has proven to be an effective and rapid means of reversing sevoflurane-induced QTc prolongation in healthy patients.¹⁴

Local Anesthetics

The use of lidocaine on patients with LQTS during vaginal delivery and cesarean section without complications has been documented. Unfortunately, little information exists pertaining to its use in oral surgical procedures. The use of epinephrine has been avoided in patients with LQTS because it can prolong the QT interval in some of these patients. Further, it might be prudent to use topical anesthesia prior to the administration of local anesthesia, to further decrease stress and anxiety.⁹

Inhalation Agents

Of the inhalation anesthetics, isoflurane has been reported as the agent of choice because of its apparent safety.⁹ Despite some prolongation of the QTc interval, these agents (halothane, enflurane, isoflurane, sevoflurane) have been used safely in patients with LQTS who were taking beta-blockers.^{9,15–18} Nitrous oxide has been used in patients with LQTS without any adverse effects reported and therefore may be a useful agent; furthermore, it may serve to reduce anxiety.⁹

Paralytics

Succinylcholine use as a muscle relaxant should be avoided because of its autonomic effects and potassium release. It will prolong the QT interval in patients with LQTS unless the patient is treated with a priming dose of tubocurarine.^{9,18,19}

Reversal Agents

Anticholinergic medications such as atropine and glycopyrrolate should be avoided because of negative effects on LQTS patients. Atropine can elicit torsades de pointes, and glycopyrrolate can lengthen the QT interval in healthy patients. The use of anticholinergic medications in conjunction with anticholinesterases (neostigmine or edrophonium) has been shown to prolong the QT interval in healthy patients. Their use together in patients with LQTS should be done with extreme caution to avoid abrupt and profound changes in heart rate.⁹

Narcotics

Fentanyl and morphine have proven effective in treating patients with LQTS. A balanced dual-drug approach best avoids sympathetic stimulation and adverse effects for patients, as documented by Kies.⁹

Also, those patients being treated with methadone for drug addiction are at risk for torsades de pointes. The dosage level of methadone is a determining factor in terms of their risk, with the higher dosages causing greater risk of complications.

POSTOPERATIVE

Antiemetics

Depending on the nature of the procedure and the tolerance of the patient, postoperative nausea and vomiting may be encountered. Droperidol has been documented as a drug that can prolong the QT interval and also carries a “black box” warning for putting patients at risk for fatal arrhythmias.^{9,20,21} For these reasons this drug should be avoided. Instead, ondansetron can be used if an antiemetic is necessary. However, it must be noted that ondansetron is included on the list of medications having a risk for torsades de pointes; it must be utilized carefully and the patient monitored closely.⁹

Monitoring

During the postoperative period, close monitoring of the patient is imperative. It is crucial to maintain a calm and quiet environment as the patient begins to recover from the effects of anesthesia. Attention must be given to the QT interval as the patient returns to a state of wakefulness, ensuring that the QT interval returns to its baseline, preoperative level. It should also be stressed that adequate pain management during

the postsurgical period is critical, thus preventing the patient from becoming agitated and emotionally stressed. Since the local anesthetics utilized should not contain epinephrine, the desired level of pain control may need to be achieved by using intravenous analgesics. Furthermore, it is absolutely necessary to have an external defibrillator (manual or automated) bedside in the event the patient shows arrhythmic ECG changes requiring intervention.

Management of Torsades de Pointes

The most dreaded complication in the treatment of patients with LQTS is the development of torsades de pointes because of its ability to progress to ventricular fibrillation and sudden death. The treatment of choice, should this complication be encountered, is an immediate IV bolus of magnesium sulfate at 30 mg/kg over a 2–3 minute period, which is then followed by an infusion of 2–4 mg of magnesium sulfate per minute. After a period of 15 minutes, if torsades persists, it is then recommended to repeat the bolus. Magnesium does not shorten the QT interval, but the exact mechanism by which it suppresses torsades de pointes is unknown. During the supplementation with magnesium, it is crucial to monitor total dosages; in general it is recommended not to exceed 30–40 g in a 24-hour period. It is also recommended to maintain higher levels of potassium and, if needed, slow IV infusion of 10–40 mEq of potassium over 2–3 hours can be used to maintain levels at 4.5–5 mmol/L. If the initial effort to restore normal rhythm with magnesium sulfate proves ineffective, unsynchronized defibrillation is indicated. Should the administration of both IV magnesium sulfate and defibrillation prove ineffective, temporary transvenous pacing can be used to manage torsades de pointes.³ The QT interval shortens with a faster heart rate, so temporary transvenous pacing can be effective in terminating torsades. It is effective in both forms of LQTS because it facilitates the repolarizing potassium currents and prevents long pauses, suppressing early after-depolarizations and decreasing the QT interval.^{22,23} Atrial pacing is the preferred mode because it preserves the atrial contribution to ventricular filling; it should be instituted at a rate of 90–110 bpm until the QT interval is normalized.²³ In general, cardioversion is kept as a last resort for acute management of torsades because of the tendency for frequent recurrences following cardioversion.²³

CASE REPORT

G.S., an 8-year-old female weighing 22 kg, was admitted to the hospital for removal of her “extra tooth”

located between and palatal to her maxillary central incisors. The dentist referred the patient to the oral and maxillofacial surgeon for removal of the supernumerary tooth because of the patient's known medical history of LQTS. The treatment plan was for the patient to be admitted to the hospital and be sedated by a pediatric anesthesiologist, with a pediatric cardiologist present in the operating room. The patient was admitted to the hospital and taken to the same-day surgery wing. Her preadmission tests indicated normal levels of Mg. She was taking nadolol, a beta-blocker. She was not being treated with a pacemaker or an ICD. At the time of the procedure, every effort was made to create an environment free of noise and interruption by any staff not immediately involved in the procedure. The patient was fully monitored including blood pressure, ECG, temperature, and pulse oximetry. The parents were permitted to stay with the child while midazolam was administered IV. Once the child was sedated, the parents were asked to leave the room, and propofol and fentanyl were administered IV and 100% oxygen was administered by way of a nasal cannula. The patient was then prepared and draped in the usual manner for an intraoral procedure. Local anesthesia, mepivacaine 3% without a vasoconstrictor, was administered and the supernumerary tooth was removed uneventfully. There were no abnormal ECG findings noted throughout the procedure. The patient tolerated the procedure well and awoke comfortably, with her parents at her side in the operating room. The patient was next taken to the postanesthesia care unit for continued monitoring of vitals and ECG. After 2 hours, there were no abnormal ECG findings, so she was considered to be in a stable condition for discharge. The patient left the hospital later that day with her parents. She was seen for a follow-up visit 1 week later, when adequate healing of the surgical site was noted.

Summary

Prior to surgery, all medications that affect the QT interval should be discontinued if possible, following proper consultation with the patient's physician. Additionally, the use of a preoperative beta-blocker is strongly recommended, and benzodiazepine is a viable option to reduce anxiety. Electrolyte values must be analyzed and corrected if necessary. It is recommended that magnesium be given preoperatively to optimize the effects of sodium and potassium currents on the heart.

The armamentaria used during IV sedation cases by oral and maxillofacial surgeons for accurate monitor-

ing of patients suffice for patients with LQTS but must include pulse oximetry, capnograph, and ECG monitor. Anesthetic and narcotic agents of choice for the procedure include nitrous oxide, propofol, fentanyl, and a local anesthetic without epinephrine.

Following the procedure, the patient should be transported to a comfortable and low-stress area during the initial recovery period. Careful monitoring of the patient during this time period is crucial, and the surgeon must be prepared to recognize and manage episodes of torsades de pointes should they arise.

CONCLUSIONS

The management of patients afflicted with LQTS by a dentist or dental specialist (in this case the oral and maxillofacial surgeon) requires a sound understanding of this unique cardiac ion channelopathy because of its dangerous sequelae and risk of sudden death. Successful treatment begins with preoperative patient assessment and planning of the procedure. Elimination—or at least reduction—of precipitating factors that affect the QT interval or elicit torsades de pointes is imperative. Consideration must be given to the operator to be utilized during the procedure to ensure a quiet and anxiety-free treatment setting for the patient. Although the procedure can be performed in a private office, it may be prudent to manage such patients in a hospital setting with the obvious benefit of increased medical resources and assistance from other medical specialists. The decision of where best to treat dental patients with LQTS depends on the overall physical and mental status of the patient; the complexity of the procedure, and the ability of the dentist to provide the patient with a suitable environment free of disruption.

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