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Review Article

REVIEW ARTICLE

Perioperative management of patients with congenital or acquired disorders of the QT interval

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Abstract

QT prolongation can be attributable to various causes that can be categorised as acquired or congenital. Arrhythmias related to QT prolongation can result in clinical presentations, such as syncope and sudden cardiac death. The perioperative period presents a number of issues that may affect a patient's risk of developing polymorphic ventricular tachycardia or torsades de pointes. Although most patients may have an unremarkable perioperative course, some may have complications; this review article aims to help clinicians avoid potential complications, and to help them address treatment for perioperative issues that may occur.

Keywords: anaesthesia; long QT syndrome; perioperative care; torsades de pointes

Editor's key points

- The authors review the phenomenon of QT prolongation and provide advice on the management of these patients presenting for anaesthesia.

QT prolongation can be acquired or originate from genetically mediated long QT syndrome (LQTS). QT prolongation and LQTS can increase susceptibility to syncope, cardiogenic seizures, aborted cardiac arrest (ACA), and sudden cardiac death (SCD) because of its characteristic polymorphic ventricular tachyarrhythmia, termed *torsades de pointes* (TdP) or 'twisting of points'. QT prolongation and LQTS have significant implications for patient care and introduce an additional level of complexity to the perioperative care of affected patients. Here, we review the definition, diagnostic criteria, pathophysiology, prognosis, and perioperative management of patients with either acquired QT prolongation or congenital LQTS.

Definition of prolonged QT interval

The normal range of heart-rate-corrected QT intervals (QTc) varies by age and sex, with females having a slightly longer QT interval than males.[1] Prolonged QTc is defined as >450 ms in

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males and >460 ms in females.[1] However, the 99th percentile values are ≥460 ms (prepuberty), ≥470 ms (males), and ≥480 ms (females); these values are probably the most useful and actionable. With increasingly longer QTc values, the pre-test probability of a patient having an LQTS-causing mutation also increases. In fact, genetic testing for LQTS is a Class I indication for asymptomatic, post-pubertal individuals if an otherwise idiopathic QTc of ≥500 ms (≥480 ms prepuberty) is detected and persists on serial ECGs.[2] In addition, increasingly prolonged QTc values portend a higher risk of potentially lethal arrhythmic events.[3,4] Of note, the QT interval on an ECG can vary in an individual, depending on autonomic state, circadian rhythm, medication effect, or electrolyte imbalance.[5]

The American Heart Association and American College of Cardiology have recommended standardised methods[1] of measuring the QT interval. Any computer-generated measurement should be confirmed manually. To confirm the QTc, Leads II and V5 optimally are used.[6] The preferred QT measurement method draws a tangent line along the maximum slope of the descending limb of the T wave; the end of the QT interval is the point at which the tangent line intersects the isoelectric line defined by the TP segment (Fig. 1). Care should be taken to exclude the U wave, when present, unless it exceeds half of the T-wave amplitude.[7] Care should also be taken to avoid inadvertent inclusion of the P wave at higher heart rates. When either atrial fibrillation or sinus arrhythmia is present, all of the QT intervals and the preceding RR intervals in the QRS complexes should be averaged to minimise overestimation of the QTc. Specifically, simply taking the longest QT interval and dividing by the square root of the shortest RR interval will result in a gross overestimation of the true QTc.[8]

The formula classically used to correct for heart rate is the Bazett formula.[9] However, this formula may overestimate the true QTc at faster heart rates, and conversely, it may underestimate the true QTc at slower heart rates.[1,10]

$$\text{Corrected QT} = \text{QT}/\sqrt{\text{RR}}$$

Congenital LQTS *vs* acquired QT prolongation

Congenital LQTS is a potentially inherited cardiac channelopathy that manifests with a prolonged QT interval and polymorphic ventricular tachycardia, characterised by TdP, with an estimated prevalence rate of one in 2500.[2,11] To date, 17 LQTS-susceptibility genes have been identified (Table 1), and among patients with a high index of suspicion for LQTS, the LQTS genetic test will be positive for about 75–80%. Almost all the genotype positivity is confined to three canonical LQTS-susceptibility genes: *KCNQ1* [for long QT Type 1 (LQT1), 35%], *KCNH2* [for long QT Type 2 (LQT2), 30%], and *SCN5A* [for long QT Type 3 (LQT3), 10%].

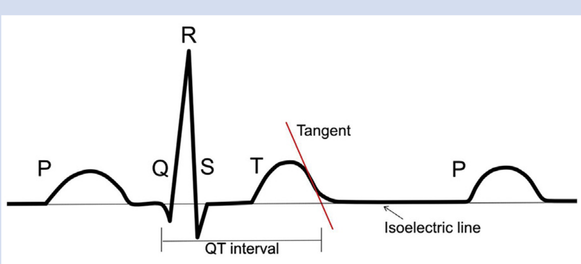

Fig 1. Measurement of the QT interval.

QT prolongation and subsequent TdP (Fig. 2) caused by medications, electrolyte disturbances, or disease was previously termed *acquired LQTS*. However, *acquired QT prolongation* is the preferred expression. Although most cases of acquired QT prolongation are not genetically based, the first manifestation of congenital LQTS may occur in the setting of acquired QT prolongation. For example, approximately 5–10% of patients with drug-induced QT prolongation may have an underlying LQTS-causative mutation.[29]

Before pursuing a diagnosis of congenital LQTS, reversible causes of QT prolongation must be excluded. Possible causes may include medication, myocardial ischaemia, hypothermia, and electrolyte abnormalities (e.g. hypokalaemia, hypomagnesaemia, or hypocalcaemia).

Genetics of congenital LQTS

In congenital LQTS, at least 17 types with associated gene mutations have been identified (Table 1). Among the described phenotypes of congenital LQTS is Jervell and Lange-Nielsen syndrome, an autosomal recessive trait often resulting in severe LQTS and concomitant sensorineural hearing loss.[30] Another is Romano–Ward syndrome, a more common autosomal-dominant condition not associated with hearing impairment.[31,32]

The aforementioned canonical LQTS-susceptibility genes encode the critical pore-forming α subunits of cardiac ion channels, and most are inherited in an autosomal-dominant pattern.[2,12] Other mutations involve proteins interacting with the ion channels; many mutations identified in these components have unknown effects and are thus termed *variants of unknown significance*.[33]

Clinical features of congenital LQTS

The phenotypic presentation of LQTS can be variable. Patients may be asymptomatic, with evidence of LQTS only on a 12-lead ECG at rest or with repolarisation stress (e.g. sympathetic stimulation during exercise testing) or other forms of testing (e.g. epinephrine stress testing).[34] The presentation of congenital LQTS can be symptomatic, ranging from palpitations to an event, such as SCD. The index event for patients with a known disease-causing mutation varies by age and sex. Male patients most commonly present with an LQTS event before age 18 yrs, whereas female patients are more likely to present with an event after puberty. The history must be obtained carefully from patients who present with syncope to exclude neurocardiogenic syncope and orthostatic intolerance—these are not considered relevant LQTS-attributable events. The history should also focus on identifying possible triggers of LQTS events.

A number of triggers have been identified that are typically associated with LQT1, LQT2, and LQT3, the most common genotypes.[14] Exercise is a trigger most commonly associated with LQT1. A heightened adrenergic state results in prolongation of the QTc; thus, exercise testing is used to provoke ECG manifestation in individuals with suspected LQT1.[14] In patients with LQT2, situations of acute emotion or startling (e.g. from an alarm clock) can provoke an event. Additionally, pause-dependent events with TdP, similar to those seen in patients with drug-induced QT prolongation, are typical of

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