Opinion

Open Access
CrossMark

Electrocardiographic change: prolongation of the QT interval, imminent risk of sudden death

Volume 7 Issue 2 - 2024

Lígia Maria Oliveira de Souza
Polytechnic and Artistic University of Paraguay, Paraguay

Correspondence: Lígia Maria Oliveira de Souza, Polytechnic and Artistic University of Paraguay, Alto Paraná, Ciudad del Este, Paraguay, Email ligiamari2107@gmail.com

Received: April 14, 2024 | **Published:** April 19, 2024

Introduction

In this article, I will address important issues regarding pathological changes caused by QT interval prolongation and its main clinical repercussions. The QT interval on the electrocardiogram (ECG) as the time required for the heart to contract and recover electrically during each heartbeat. It begins at the beginning of the QRS complex, which represents ventricular depolarization (contraction), and ends at the end of the T wave, which represents ventricular repolarization (electrical recovery). Normally their values are less than 450ms in men, and values less than 470ms in women. In children, this value should be less than 440ms. It varies with heart rate, that is, it is shorter at faster heart rates and longer at slower heart rates. To compensate for this variation, it is common to adjust the QT interval to a standard resting heart rate (usually 60 beats per minute) using formulas such as Bazett's or others. This adjustment is made to aid in the clinical interpretation of the QT interval, as it allows comparison of values under different heart rate conditions. Therefore, the QT interval is very important in clinical practice, because its prolongation may be associated with an increased risk of serious arrhythmias, including torsades de pointes, which can lead to sudden cardiac death.

Changes that can cause a prolonged qt interval on the electrocardiogram include

Congenital Long QT Syndrome: A genetic condition that affects the repolarization of the heart, leading to an increase in the QT interval. According to Krahn AD et al.[1] since its initial description in 1957, our understanding of LQTS has increased dramatically. The prevalence of LQTS is estimated at □1:2,000, with a slight female predominance. The diagnosis of LQTS is based on clinical, electrocardiographic and genetic factors. Risk stratification of patients with LQTS aims to identify those at increased risk of cardiac arrest or sudden cardiac death. Factors including age, sex, QTc interval, and genetic background contribute to current risk stratification paradigms. Management of LQTS involves conservative measures such as avoiding medications that prolong the QT interval, pharmacological measures with non-selective β-blockers, and interventional approaches such as device therapy or left cardiac sympathetic denervation.

Medications

Antiarrhythmics: Certain medications used to treat cardiac arrhythmias, such as amiodarone, sotalol, and dofetilide, can prolong the QT interval.

Antibiotics: Some antibiotics, such as erythromycin, clarithromycin, azithromycin (especially in high doses) and moxifloxacin, are associated with prolongation of the QT interval.

Antidepressants: Some antidepressants, in particular selective serotonin reuptake inhibitors (SSRIs) and serotonin norepinephrine reuptake inhibitors (SNRIs), such as citalopram, escitalopram, sertraline, venlafaxine and duloxetine, may prolong the interval QT.

Antipsychotics: Some antipsychotics, especially second-generation antipsychotics (atypical antipsychotics), such as ziprasidone, quetiapine, olanzapine, and clozapine, have been associated with QT prolongation.

Antihistamines: Some second-generation antihistamines, such as terfenadine and astemizole, have been withdrawn from the market due to the risk of QT prolongation and cardiac arrhythmias.

Antiemetics: Certain medications used to prevent nausea and vomiting, such as ondansetron, granisetron, and dolasetron, can prolong the QT interval.

Electrolyte disorders

Imbalances in electrolyte levels, such as potassium, calcium, and magnesium, can affect the length of the QT interval. El-Sherif N et al.[2] Potassium (K^+) is the most abundant intracellular cation and hypokalemia is the most common electrolyte abnormality found in clinical practice. The most significant manifestation of hypokalemia on the ECG is a prominent U wave. It is known that several cardiac and non-cardiac medications suppress the HERG K^+ channel and therefore I(K), and especially in the presence of hypokalemia, may result in prolonged action potential duration and QT interval, QTU alternation, early post-depolarizations and torsade ventricular tachyarrhythmia de pointes (VT TdP). Hyperkalemia affects up to 8% of hospitalized patients, mainly in the context of compromised renal function. The manifestation of hyperkalemia on the ECG depends on the serum K^+ level. At 5.5-7.0 mmol/LK^+, high-peaked, narrow-based T waves are observed. At > 10.0 mmol/LK^+, sinus arrest, marked delay in intraventricular conduction, ventricular tachycardia and ventricular fibrillation may occur. Isolated abnormalities of extracellular calcium (Ca^{++}) produce clinically significant PE effects only when they are extreme in either direction. Hypocalcemia, often seen in the setting of chronic renal failure, results in prolongation of the ST segment and QT interval, whereas hypercalcemia, often seen with hyperparathyroidism, results in shortening of both intervals. Although magnesium is the second most abundant intracellular cation, the significance of magnesium disorders is controversial, in part due to the frequent association of other electrolyte abnormalities. However,

Submit Manuscript | http://medcraveonline.com

©2024 de Souza. This is an open access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and build upon your work non-commercially.

Copyright: ©2024 de Souza

IV magnesium, by blocking L-type Ca(++) current, can successfully terminate VT TdP without affecting the prolonged QT interval. Finally, despite the frequency of sodium abnormalities, particularly hyponatremia, their PE effects are rarely clinically significant.

Hypothyroidism

Reduced thyroid function can lead to a prolonged QT interval.

Changes in metabolism: Hypothyroidism reduces the levels of thyroid hormones, such as triiodothyronine (T3) and thyroxine (T4), which play an important role in regulating cellular metabolism. This can affect cardiac function, including ventricular repolarization, which is reflected by the QT interval on the ECG.

Effects on ion channels: Thyroid hormones influence the expression and function of several ion channels in the heart, including potassium, calcium, and sodium channels. Changes in the function of these channels can lead to disturbances in cardiac repolarization, contributing to the prolongation of the QT interval.

Reduced sympathetic activity: Hypothyroidism is associated with reduced activity of the sympathetic nervous system, which plays an important role in regulating heart rate and the electrical conduction of the heart. This decrease in sympathetic activity may contribute to disturbances in ventricular repolarization and prolongation of the QT interval.

Accumulation of mucopolysaccharides: In some cases of congenital hypothyroidism, accumulation of mucopolysaccharides may occur in cardiac tissue, which may lead to abnormalities in electrical conduction and, consequently, prolongation of the QT interval.

Heart diseases

Certain heart conditions, such as cardiomyopathy, heart failure, and myocardial infarction, may be associated with QT prolongation. Welten SJGC et al.[3] prolongation of the heart rate-corrected QT interval (QTc) on electrocardiogram (ECG) has been extensively studied as a risk factor for CVD and CVD mortality. The increased risk of CVD caused by prolonged QTc interval may increase susceptibility to ventricular fibrillation and sudden death. However, results from observational studies regarding the association between QTc prolongation and different types of CVD are inconsistent across subgroups and difficult to compare due to differences in covariate adjustment, sample size, and population differences.

Several risk factors can cause prolongation of the QTc interval, such as age, high blood pressure, increased BMI, heart failure and specific pharmacological agents. Furthermore, genetic disorders and electrolyte disturbances (hypokalemia and hypomagnesemia) are associated. Furthermore, the prolonged QTc interval is longer in people with diabetes than in those without diabetes. People with diabetes have an imbalance in the autonomic nervous system and worse glycemic control, which can influence myocardial cells. We do not know whether glucose tolerance modifies the association between the QTc interval and CVD occurrence and mortality.

Torsades de pointes

Torsades de pointes is a specific form of polymorphic ventricular tachycardia characterized by a distinct pattern on the electrocardiogram (ECG). "Torsades de pointes" is a French term meaning "twisting of ends", describing the characteristic appearance of the waves on the ECG during this type of tachycardia. During a torsades de pointes, the ECG waves oscillate between a positive and negative position, creating a "twist" pattern in the ECG trace. This change in ECG morphology is associated with a prolongation of the QT interval, which is one of the main risk factors for the development of torsades de pointes.

Torsades de pointes is a potentially serious arrhythmia as it can degenerate into ventricular fibrillation, a lethal arrhythmia that can lead to sudden cardiac arrest if not treated quickly. Risk factors for developing torsades de pointes include, prolongation of the QT interval, which can be caused by various conditions such as certain medications, electrolyte disturbances (e.g., hypocalcemi hypomagnesemia), genetic syndromes such as Congenital Long QT Syndrome and other heart conditions. Use of medications that prolong the QT interval, including some antiarrhythmics, antibiotics, antipsychotics, antidepressants, and antihistamines. Environmental factors such as hypothermia, personal or family history of cardiac arrhythmias. Treatment for torsades de pointes aims to stop the arrhythmia quickly to prevent degeneration into ventricular fibrillation. This can be done through administration of antiarrhythmic medications, such as magnesium sulfate, and interventions to correct underlying triggers, such as stopping medications that prolong the QT interval or correcting electrolyte imbalances. In emergency situations, it may be necessary to perform electrical cardioversion to restore normal heart rhythm (Figure 1).

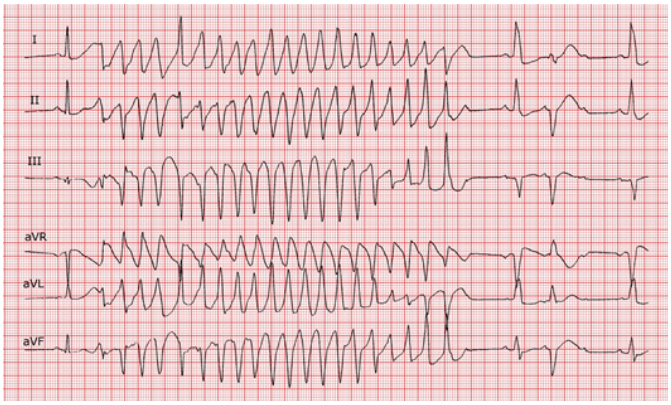

Figure 1 Cardio-fr.

Conclusion

Therefore, given the comprehensive analysis of changes, the study of QT prolongation is crucial for the prevention, diagnosis and treatment of potentially fatal cardiac arrhythmias, both in healthy individuals and in patients with underlying medical conditions.

Acknowledgments

None.

Conflicts of interest

The author declared that there are no conflicts of interest.

References

1. Krahn AD, Laksman Z, Sy RW, et al. Congenital long QT syndrome. *JACC Clin Electrophysiol*. 2022;8(5):687–706.
2. El Sherif N, Turitto G. Electrolyte disorders and arrhythmogenesis. *Cardiol J*. 2011;18(3):233–245.
3. Welten SJGC, Heijden AA, Remmelzwaal S, et al. Prolongation of the QTc interval is associated with an increased risk of cardiovascular diseases: the Hoorn study. *J Electrocardiol*. 2023;80:133–138.

Citation: de Souza LMO. Electrocardiographic change: prolongation of the QT interval, imminent risk of sudden death. *Hos Pal Med Int Jnl.* 2024;7(2):36–37. DOI: 10.15406/hpmij.2024.07.00236