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QT Prolongation: What is the actual risk?

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Objectives

1. Interpret the ECG to accurately measure the QT interval.
2. Explain the diagnostic pathway for evaluating long QT interval.
3. Differentiate the risk of QT prolongation and Torsades de Pointes among high-risk medications.
4. Formulate a strategy to manage drug-induced QT prolongation.

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Long QT

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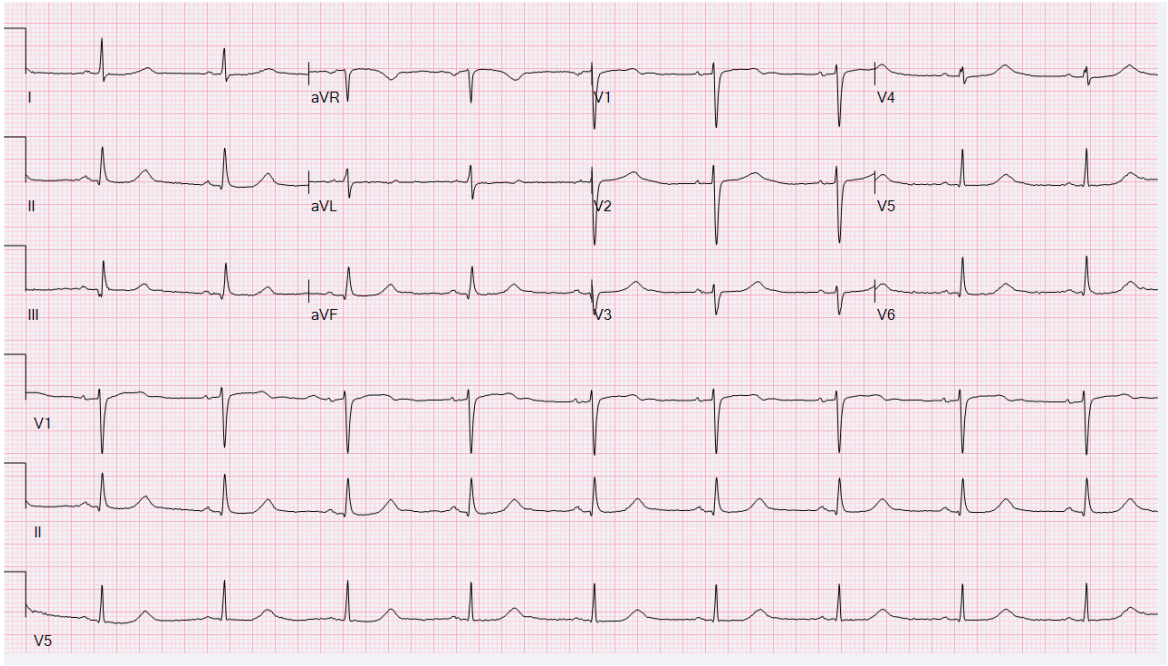
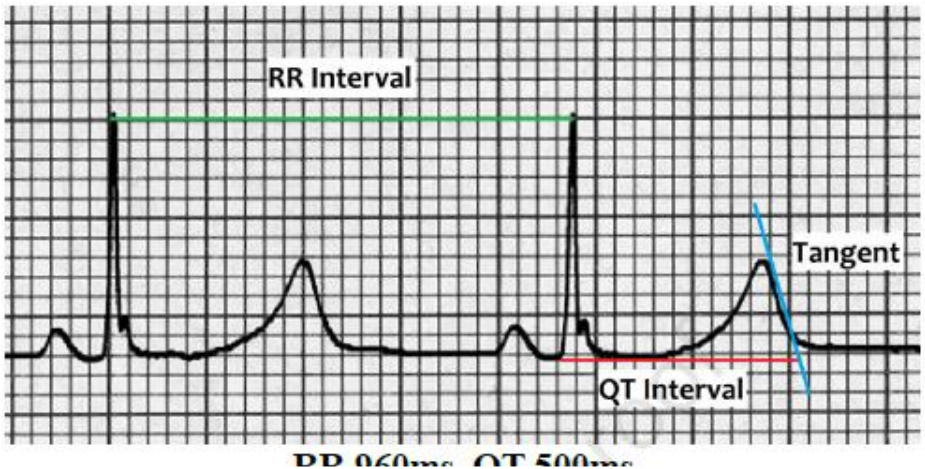
Clinical case

- 65-year-old female with a past medical history of hypertension, hyperlipidemia, and depression
- Symptoms of palpitations and dizziness
- Comes to the office for pre-op EKG prior to hip replacement



How do we accurately measure QTc

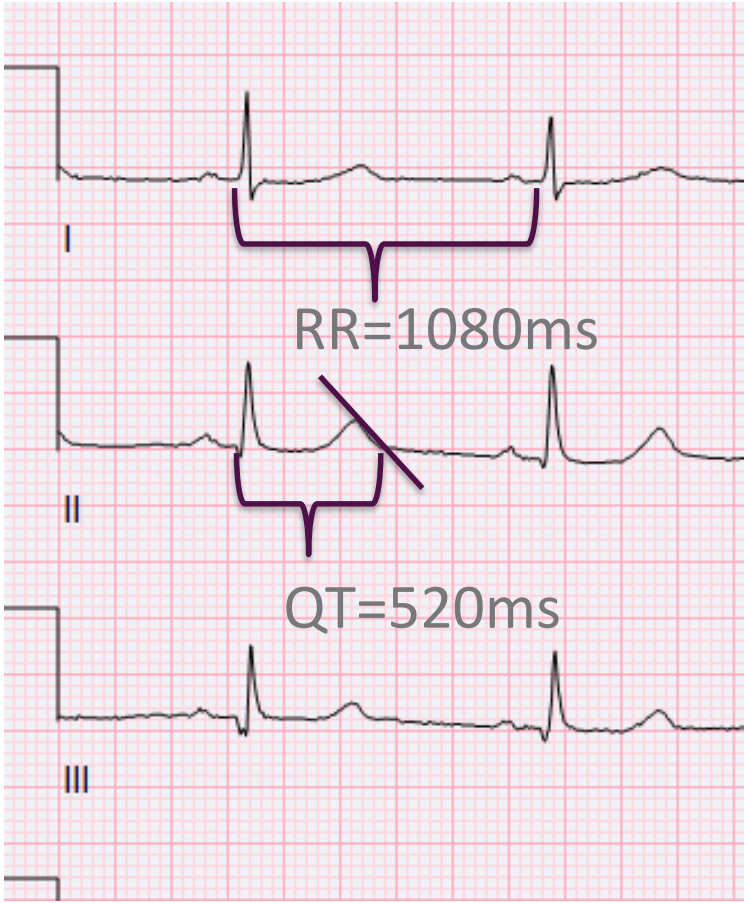
Figure 1. Tangent method of measuring QT interval



Normal values: Before puberty < 460msec, Males <470, Females <480

Pitfalls

- Paced rhythms
  - Calculated QTC -(Patients QRS-120)
- Bundle branch blocks
  - Calculated QTC -(Patients QRS-120)
- Atrial fibrillation
  - I take 3 QT intervals and average
- Sinus arrhythmia
  - Average of QT of longest and shortest RR interval



QTc interval= QT/ $\sqrt{RR}$

QTc interval= 0.52/ $\sqrt{1.08}$

QTc interval= 500

Differential diagnosis of long QT

- Hypokalemia
- Hypomagnesaemia
- Hypocalcemia
- Hypothermia
- Myocardial ischemia
- ROSC Post-cardiac arrest
- Raised intracranial pressure
- Congenital long QT syndrome
- Medications/Drugs

What to do next?

- Gather additional history
  - Syncope history
  - Palpitations
  - Family history
- Check the patient's medication list for QT prolonging drugs
- Lab work
- Holter monitor
- Stress testing

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Drug-Induced QT Prolongation

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A rare problem…

FDA-approved medications known to prolong QT interval: **210**[1]

- Known risk of Torsades de Pointes (TdP): **41**
- Conditional risk of TdP: **53**

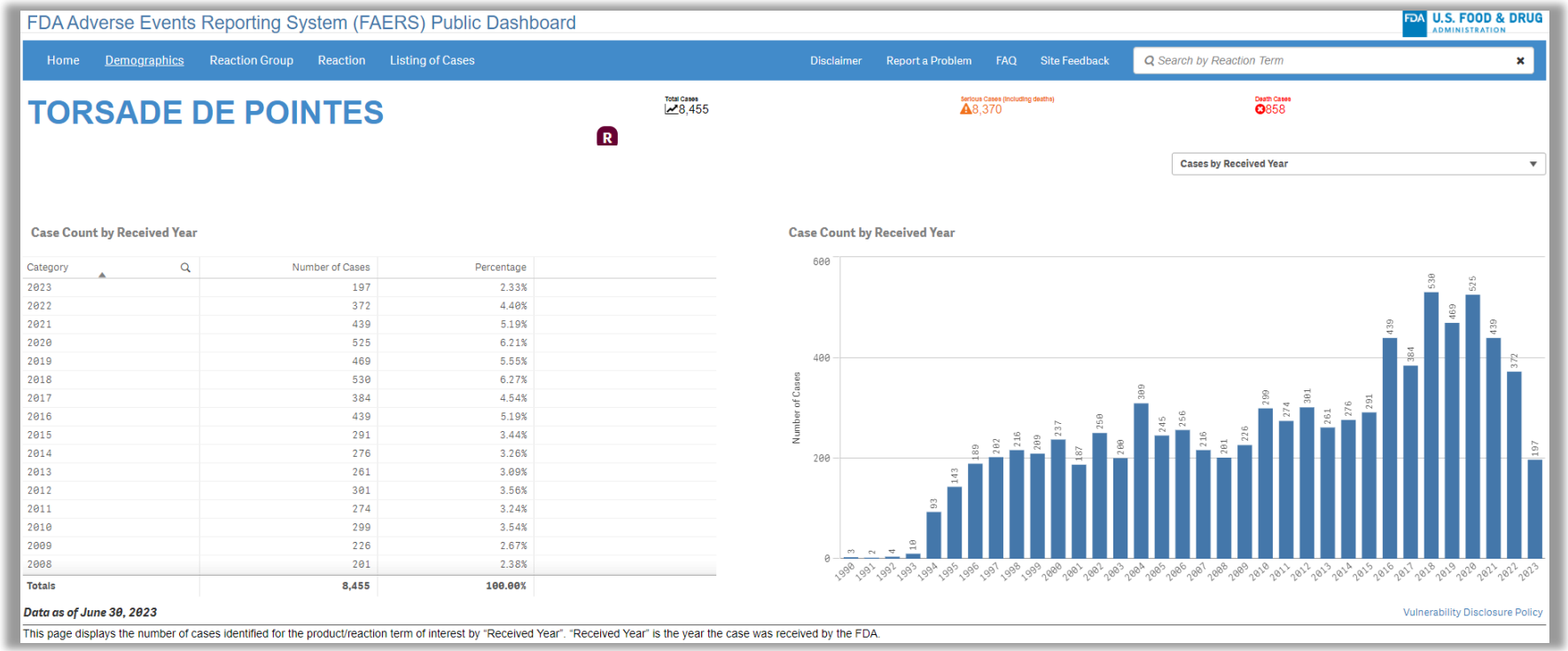
Inpatient evaluation of 167,546 patients identified 288 cases (0.17%) of drug-induced QT prolongation[2]

- 49/288 cases (17%) resulted in arrhythmia

Drug-induced QT prolongation is rare, but carries relatively high incidence of arrhythmia

1 Source: www.crediblemeds.org/druglist (Accessed 8/25/2023)
2 Eur J Clin Pharmacol 2023; 79: 759-765

…which still frequently causes arrhythmia



FDA Adverse Events Reporting System (FAERS) Public Dashboard

U.S. FOOD & DRUG ADMINISTRATION

Home | Demographics | Reaction Group | Reaction | Listing of Cases | Disclaimer | Report a Problem | FAQ | Site Feedback | Search by Reaction Term

TORSADE DE POINTES

Total Cases 8,455 | Serious Cases (including deaths) 8,370 | Death Cases 858

Cases by Received Year

Case Count by Received Year

Category	Number of Cases	Percentage
2023	197	2.33%
2022	372	4.40%
2021	439	5.19%
2020	525	6.21%
2019	469	5.55%
2018	530	6.27%
2017	384	4.54%
2016	439	5.19%
2015	291	3.44%
2014	276	3.26%
2013	261	3.09%
2012	301	3.56%
2011	274	3.24%
2010	299	3.54%
2009	226	2.67%
2008	201	2.38%
Totals	**8,455**	**100.00%**

Case Count by Received Year

Data as of June 30, 2023

Vulnerability Disclosure Policy

This page displays the number of cases identified for the product/reaction term of interest by "Received Year". "Received Year" is the year the case was received by the FDA.

Source: FDA Adverse Events Reporting System (FAERS) Public Dashboard. Accessed 8/29/2023

QT prolongation and TdP: Commonly Implicated Medications*

Known Risk of TdP *medications that prolong QT interval and are clearly associated with TdP, even when taken as recommended*		Possible Risk of TdP *medications that cause QT prolongation, but currently lack evidence of causing TdP when taken as recommended*		Conditional Risk of TdP *medications that are associated with TdP, but only under certain conditions (e.g., excessive dose, presence of drug interactions, patients with predisposing conditions)*	
Amiodarone	Levofloxacin	Aripiprazole	Paliperidone	Amantadine	Paroxetine
Azithromycin	**Methadone**	Bicalutamide	Promethazine	Amitriptyline	Posaconazole
Ciprofloxacin	Moxifloxacin	Buprenorphine	Remdesivir	Diltiazem	Propafenone
Citalopram	**Ondansetron**	Capecitabine	Tacrolimus	Doxepin	**Quetiapine**
Clarithromycin	Oxaliplatin	Clozapine	Tamoxifen	Esomeprazole	Ranolazine
Disopyramide	Procainamide	Dasatinib	Tizanidine	Fluoxetine	Risperidone
Dofetilide	Quinidine	Efavirenz	Tramadol	Hydroxyzine	Sertraline
Donepezil	Sotalol	Granisetron	Venlafaxine	Ivabradine	Trazodone
Dronedarone		Imipramine		Ketoconazole	
Erythromycin		Levetiracetam		Lansoprazole	
Escitalopram		Mirtazapine		Metoclopramide	
Fluconazole		Nicardipine		Metronidazole	
Haloperidol		Nortriptyline		Olanzapine	

*List not comprehensive

Source: https://www.crediblemeds.org/druglist

QT Prolongation:
When & How to Intervene

Interrupt QT prolonging therapy when:

- QT prolongation ≥ 500 msec
- Increase ≥ 60 msec from baseline

Be advised!
baseline *depolarization* abnormality may alter this threshold

Additional medication interventions:

- Assess potential for alternate medication
- Assess for presence of drug-drug interactions
- Correct electrolyte abnormalities

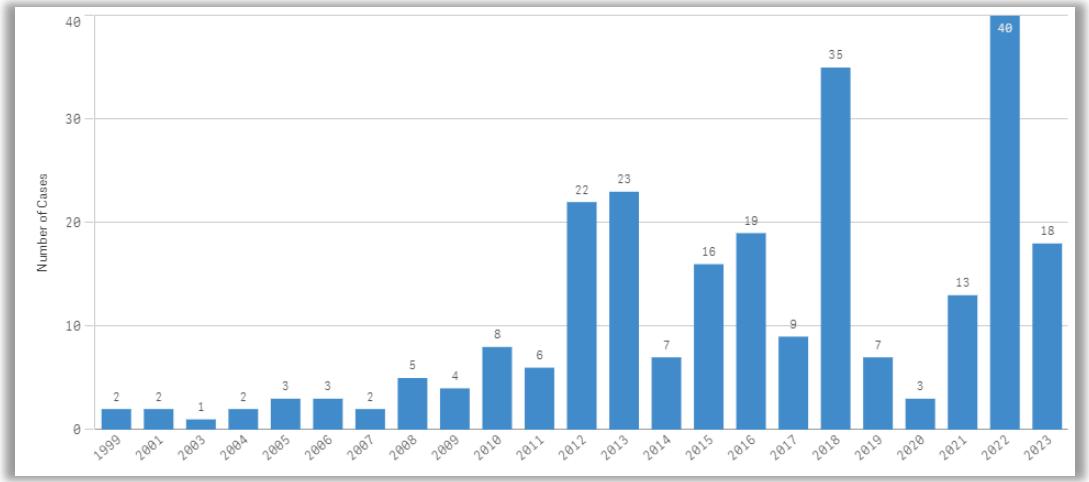
J Am Coll Cardiol. 2010 Mar 2;55(9):934-47

QT Prolongation Snapshot: Quetiapine

Credible Meds Risk Category: Conditional Risk of TdP

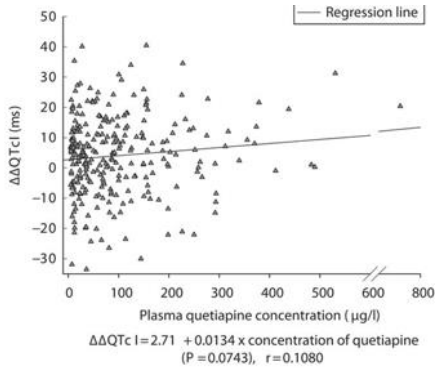
Risk Factors: bradycardia, hypokalemia, hypomagnesemia, excess dose, drug interactions

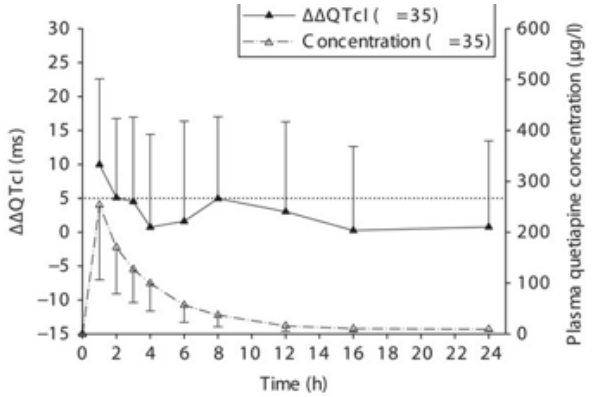
FAERS Database Reports:



QT Prolongation Snapshot: Quetiapine

Healthy Volunteers[1]:





QT Prolongation in CICU population[2]:

- Computer generated: **18**%
- Manually confirmed: **9**%

1 International Clinical Psychopharmacology 2016; 31(4):210-217
2 Am J Cardiol 2022; 163: 117-123

QT Prolongation Snapshot: Quetiapine

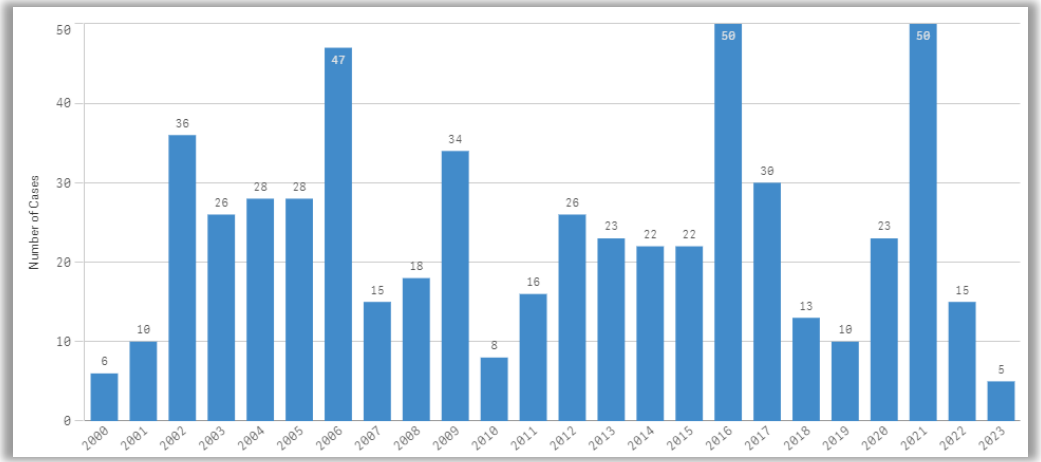
QT takeaway:

- Must account for population risk
- Know alternative therapy options

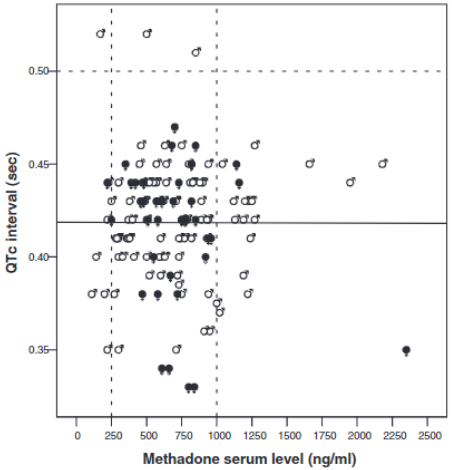
QT Prolongation Snapshot: Methadone

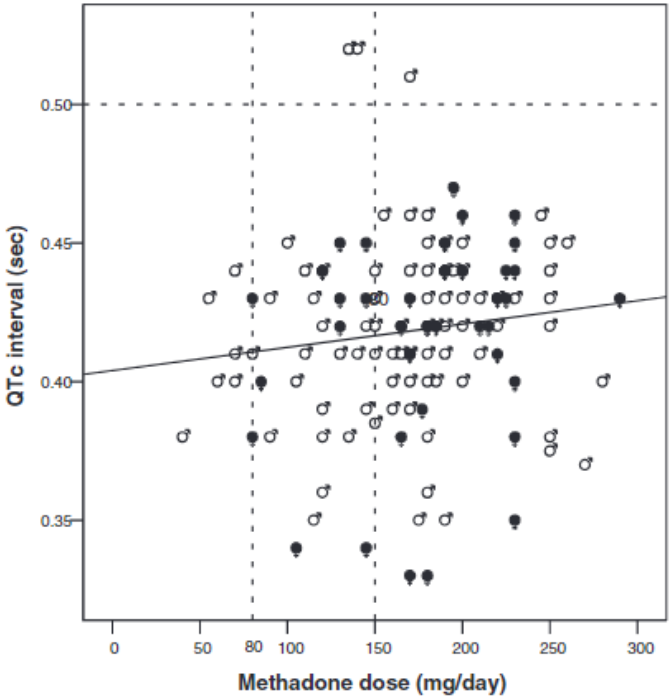
Credible Meds Risk Category: Known Risk of TdP

FAERS Database Reports :



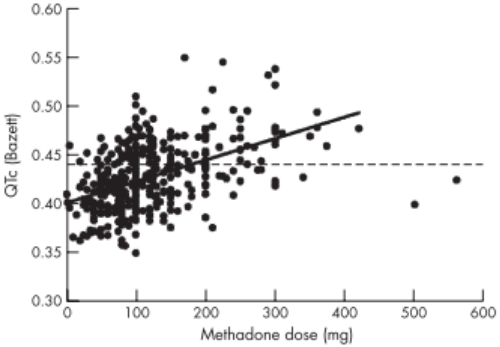
QT Prolongation Snapshot: Methadone





r=0.13

p=ns



Stronger dose-response

Addiction 2007; 102: 289–300
Heart 2007; 93: 1051-1055

QT Prolongation Snapshot: Methadone

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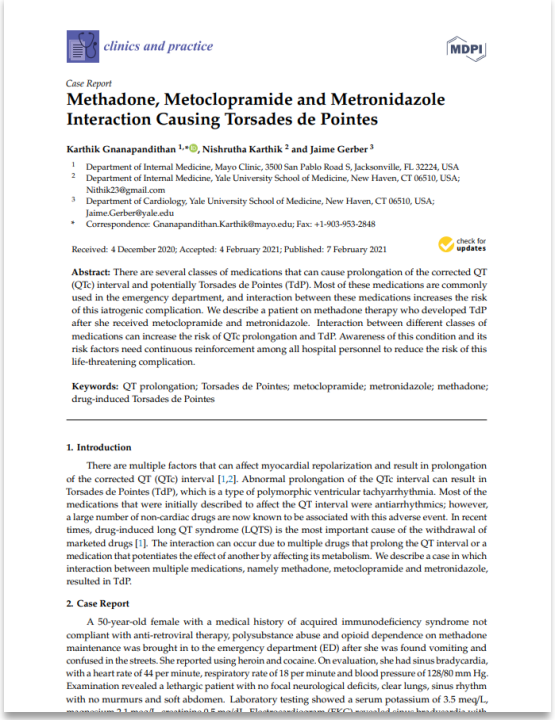

clinics and practice

MDPI

Case Report

Methadone, Metoclopramide and Metronidazole Interaction Causing Torsades de Pointes

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check for updates

Abstract: There are several classes of medications that can cause prolongation of the corrected QT (QTc) interval and potentially Torsades de Pointes (TdP). Most of these medications are commonly used in the emergency department, and interaction between these medications increases the risk of this iatrogenic complication. We describe a patient on methadone therapy who developed TdP after she received metoclopramide and metronidazole. Interaction between different classes of medications can increase the risk of QTc prolongation and TdP. Awareness of this condition and its risk factors need continuous reinforcement among all hospital personnel to reduce the risk of this life-threatening complication.

Keywords: QT prolongation; Torsades de Pointes; metoclopramide; metronidazole; methadone; drug-induced Torsades de Pointes

1. Introduction

There are multiple factors that can affect myocardial repolarization and result in prolongation of the corrected QT (QTc) interval [1,2]. Abnormal prolongation of the QTc interval can result in Torsades de Pointes (TdP), which is a type of polymorphic ventricular tachyarrhythmia. Most of the medications that were initially described to affect the QT interval were antiarrhythmics; however, a large number of non-cardiac drugs are now known to be associated with this adverse event. In recent times, drug-induced long QT syndrome (LQTS) is the most important cause of the withdrawal of marketed drugs [1]. The interaction can occur due to multiple drugs that prolong the QT interval or a medication that potentiates the effect of another by affecting its metabolism. We describe a case in which interaction between multiple medications, namely methadone, metoclopramide and metronidazole, resulted in TdP.

2. Case Report

A 50-year-old female with a medical history of acquired immunodeficiency syndrome not compliant with anti-retroviral therapy, polysubstance abuse and opioid dependence on methadone maintenance was brought in to the emergency department (ED) after she was found vomiting and confused in the streets. She reported using heroin and cocaine. On evaluation, she had sinus bradycardia, with a heart rate of 44 per minute, respiratory rate of 18 per minute and blood pressure of 128/80 mm Hg. Examination revealed a lethargic patient with no focal neurological deficits, clear lungs, sinus rhythm with no murmurs and soft abdomen. Laboratory testing showed a serum potassium of 3.5 meq/L, magnesium 2.1 meq/L, creatinine 0.5 mg/dL. Electrocardiogram (ECG) revealed sinus bradycardia with

Addiction

CASE REPORT

doi:10.1111/j.1360-0443.2008.02390.x

Ciprofloxacin-induced torsades de pointes in a methadone-dependent patient

Murali K. Nair, Krunal Patel & Perry J. Starer

Department of Internal Medicine, Mount Sinai School of Medicine, Elmhurst Hospital Center, New York, NY, USA

ABSTRACT

Background Methadone has been associated with QT prolongation and Torsades de pointes. Ciprofloxacin may prolong QT interval and induce Torsades de pointes when other risk factors are present. **Case description** A case is described in which a patient receiving methadone treatment developed Torsades de pointes following the addition of ciprofloxacin. **Conclusion** Ciprofloxacin should be used with caution in patients receiving methadone.

Keywords Arrhythmia, ciprofloxacin, drug-drug interactions, methadone, QT prolongation, torsades de pointes.

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Submitted 21 July 2008; initial review completed 20 August 2008; final version accepted 19 September 2008

INTRODUCTION

Torsades de pointes (TdP) is a potentially fatal ventricular tachyarrhythmia which often occurs in association with a prolonged QT interval [1,2]. The QT interval, which is measured on an electrocardiogram from the beginning of the QRS complex to the end of the T wave, varies with the heart rate and can be corrected for the QTc [2]. Numerous medications can prolong QT prolongation and induce Torsades de pointes [3,4].

Methadone, a medication used as a replacement therapy in the management of opioid dependence [5,6], has been associated with QT prolongation and Torsades de pointes [4,6,7].Risk factors of TdP during methadone treatment include high-dose methadone, human immunodeficiency virus (HIV) infection, hypokalemia, female sex, liver cirrhosis, renal failure and heart disease [5]. In addition, medications that increase the serum levels of methadone are risk factors for TdP among patients with methadone-associated TdP [5]. Some medications might themselves trigger TdP [5].

Fluoroquinolones, widely used and well-tolerated antibacterial agents, may cause prolonged QT intervals [1,8]. Although ciprofloxacin is less likely to prolong QT and induce TdP among other quinolones [1,8,9], it can have these effects, especially when other risk factors are present [10].

Although an interaction between methadone and ciprofloxacin causing respiratory depression has been reported previously [11], an interaction between methadone and ciprofloxacin causing Torsades de pointes has not been described previously.

We report a case of a patient receiving methadone treatment who developed Torsades de pointes following addition of ciprofloxacin.

CASE DESCRIPTION

A 56-year-old man was brought to the emergency department following an episode of witnessed syncope. His past medical history included hypertension, hepatitis C and intravenous drug use previously. In addition to receiving methadone 120 mg orally, he had been self-medicating for upper respiratory infection symptoms with his wife's ciprofloxacin (400 mg twice daily) prior to this hospitalization. Examination demonstrated a lethargic man who was responsive to painful stimuli. There were no cardiovascular or other abnormalities. Serum potassium was 3.3 mEq/l. Other routine blood tests, including magnesium, aspartate aminotransferase (AST), alanine aminotransferase (ALT), gamma glutamyltransferase (GGT), albumin, prothrombin time and international normalized ratio (INR), were normal. Electrocardiogram showed a sinus rhythm with a QTc interval of 456 msec (milliseconds). (An electrocardiogram obtained 3 years prior to admission, when the patient was receiving methadone 80 mg by mouth daily,

© 2008 The Authors. Journal compilation © 2008 Society for the Study of Addiction

CASE REPORT

Sudden Cardiac Arrest in a Patient on Chronic Methadone After the Addition of Azithromycin

John C. Winton, MD and Jennifer D. Twilla, PharmD

Abstract: Corrected QT-interval (QTc) prolongation with increased risk of fatal arrhythmia is a well-established toxicity of methadone. In this study, a case of sudden cardiac arrest in a patient on chronic methadone therapy is presented. A 47-year-old man presented unresponsive to the emergency department after pulseless arrest at his home. The patient's wife revealed he was taking methadone as part of an ongoing opioid dependency treatment and that he was prescribed azithromycin for an upper respiratory tract infection 3 days before his presentation. A 12-lead electrocardiogram at the time of presentation showed sinus tachycardia and a QTc of 490 milliseconds. It was concluded that the patient experienced a fatal arrhythmia because of QTc prolongation, precipitated by azithromycin in the setting of ongoing methadone use.

Key Indexing Terms: Arrhythmia; Methadone; Azithromycin. **[Am J Med Sci 2013;345(2):160–162.]**

Methadone is a long-acting opioid used primarily for the treatment of opioid dependence and, for this indication, is dispensed by Food and Drug Administration-approved facilities only. It can also be prescribed by independent practitioners for patients with chronic pain. Methadone carries a "black box" warning of corrected QT-interval (QTc) prolongation with potential for fatal arrhythmia including torsades de pointes. It is the only opioid that carries such a warning. The risk of fatal arrhythmia is significant enough that specific guidelines have been proposed regarding electrocardiogram (ECG) monitoring and careful attention to drug interactions.[1] Our review explores one particular case of methadone-induced arrest, the mechanism of QTc prolongation with regard to methadone and other factors potentiating fatal arrhythmia.

CASE REPORT

A 47-year-old man was brought to our emergency department from an outlying hospital for unresponsiveness. Family was *en route* at the time of presentation to our hospital, so initial history was limited to third-hand information obtained from the transfer report. According to the report, the patient was found cyanotic and obtunded by his wife. Emergency medical services quickly arrived and transported the patient to the outlying facility, whereupon he was transferred to our hospital for higher level of care. Medical history was significant for hypertension and prescription opioid addiction, for which he was enrolled in an accredited treatment program. Home medications were initially reported as methadone 10 mg 3 times daily, lisinopril/hydrochlorothiazide 20/12.5 mg once daily, benzonatate 100 mg every 8 hours as needed, and an over-the-counter cold medicine consisting of acetaminophen, dextromethorphan and doxylamine. The patient had no known allergies. Social history was reportedly negative for tobacco or alcohol use. Review of systems was otherwise unobtainable, and the rest of the known history was unremarkable.

Initial examination revealed a moderately obese man, obtunded with irregular respirations. He had no discernible response to voice or tactile stimulation, and he was aphasic except for sporadic moaning. His body temperature was 36.8°C, heart rate around 130 beats per minute and the rhythm regular. The respiratory rate was 25 breaths per minute and blood pressure 120/80 mm Hg by manual cuff measurement. Oxygen saturation was 100% via non-rebreather mask. His pupils reacted normally to light and his extraocular movements appeared intact. Oral mucosa was moist. Heartbeat was tachycardic but regular; a soft systolic murmur was detected at the left sternal border. He had no jugular venous distention, normal peripheral pulses and no lower extremity edema. His lungs were clear bilaterally. He was mildly tachypneic without retractions. Abdomen was protuberant because of central obesity but otherwise benign. Musculoskeletal examination revealed mildly increased tone throughout with full range of passive motion and no deformities. Neurological examination was limited but revealed no focal deficits other than the aforementioned increased tone; deep tendon reflexes were normal, Babinski reflexes were downgoing and gag was intact. The Glasgow coma scale score was 10. Laboratory evaluation revealed lactic acidosis (lactate 15 ng/dL, pH 7.11) and hyperammonemia (74 mg/dL). Other than a low serum bicarbonate level, electrolytes and renal function were normal. Liver function tests were normal as well, as were cardiac isoenzymes. ECG revealed sinus tachycardia and a QTc of 490 milliseconds. No previous ECGs were available. Chest radiography was normal.

Because of the lack of an immediately discernible cause for his symptoms, and because of his history of drug abuse, the patient's initial diagnosis was presumed to be toxic metabolic encephalopathy. Extended toxicology screening, however, was negative for all substances other than opiates, and his presenting symptoms were not consistent with opiate overdose. Magnetic resonance imaging of his brain was normal, and all cultures (blood, urine and cerebrospinal fluid) were negative. Echocardiogram revealed normal ventricular function and no structural abnormalities. His mental status gradually improved without any therapy other than a few days of empiric antibiotics and supportive care. He had no significant arrhythmias during the rest of his hospitalization.

Details surrounding his precipitating event were elucidated on hospital day 12, after the patient had improved enough to leave the intensive care unit and his wife was re-interviewed. She was present at the time of his arrest. Just before bedtime the

From the Methodist University Hospital, Inpatient Physicians (icw) and Clinical Pharmacy (dzt), Memphis, Tennessee; and Departments of Medicine (icw) and Clinical Pharmacy (dzt), University of Tennessee Health Science Center, Memphis, Tennessee.

Submitted May 25, 2012; accepted in revised form June 27, 2012.

Disclosure: The authors state that they have no financial or conflicts of

QT Prolongation Snapshot: Methadone

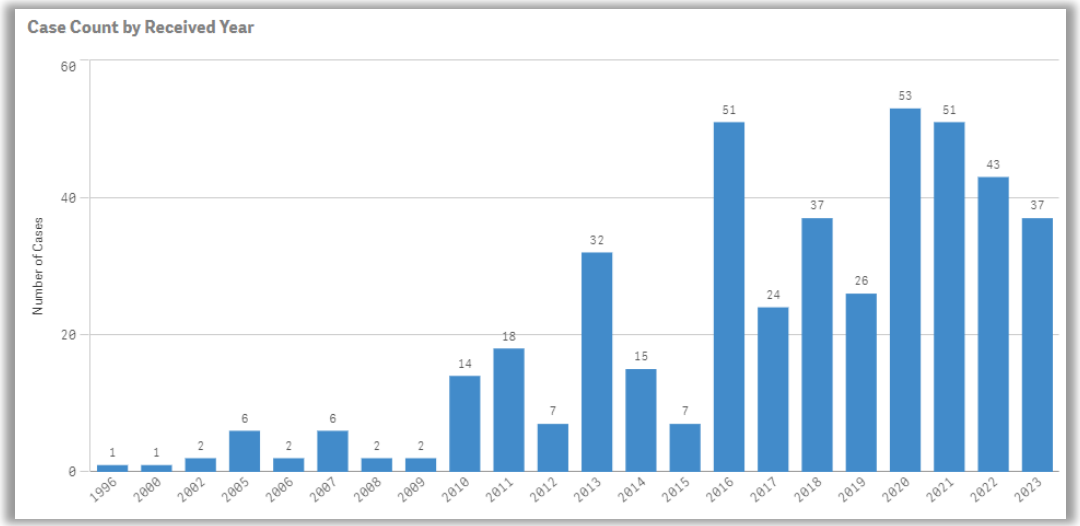
QT takeaway:

- Drug interactions can make chronic stable QT prolongation into unstable QT prolongation

QT Prolongation Snapshot: Ondansetron

Credible Meds Risk Category: Known Risk of TdP

FAERS Database Reports :



QT Prolongation Snapshot: Ondansetron

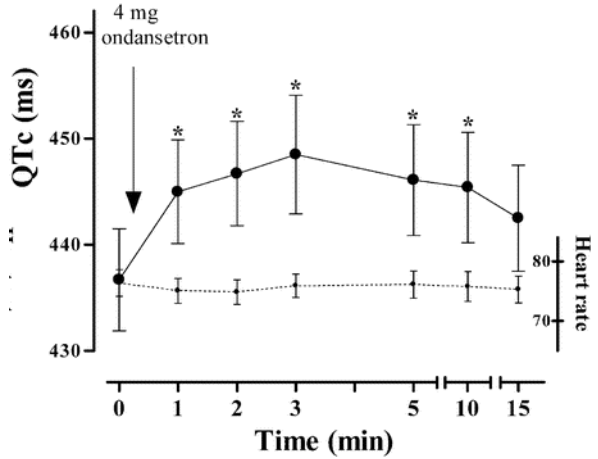
2011: FDA issues warning about safety concerns over Zofran® and the associated risk of QT prolongation and TdP; review is initiated

2012: FDA announces labeling updates for Zofran®, removing 32 mg single IV dose from approved recommendations

- No single dose should exceed 16 mg
- Announcement specifically notes that recommendations for low-dose post-op nausea and all oral regimens are not changed

QT Prolongation Snapshot: Ondansetron

Intravenous 4 mg dose linked with 12-20 msec increase in QT interval[1-4]



Limited reporting on oral dose and TdP

- 1 single case report in published literature[5]
- Patient with concomitant bradycardia and hypomagnesemia

1 Acad Emerg Med 2016;23(1):102-5
2 Drug Healthc Patient Saf. 2011; 3: 53–58
3 Anesthesiology 2005;102(6):1094-100
4 Am J Health-Syst Pharm. 2018; 75:276-82
5 Clin Case Rep. 2019;7:1557–1558

QT Prolongation Snapshot: Ondansetron

QT takeaway:

- QT prolonging medications can still be used safely
  - Important to know dose, route of administration on QT response

- Knowing the history of QT prolongation warnings can provide reassurance for safe use

QT Prolongation Management

Know when to interrupt (*or enhance monitoring of*) therapy

- Absolute QT threshold
- Change in QT interval

Correct the correctable

- Electrolytes
- Heart rate
- Drug interactions

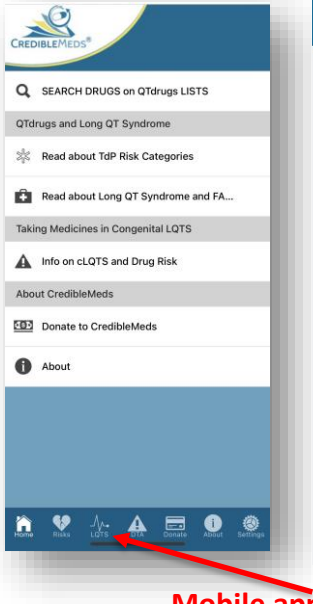
No need to re-invent the wheel

- Use available tools to assess
  - www.CredibleMeds.org

Credible Meds Database

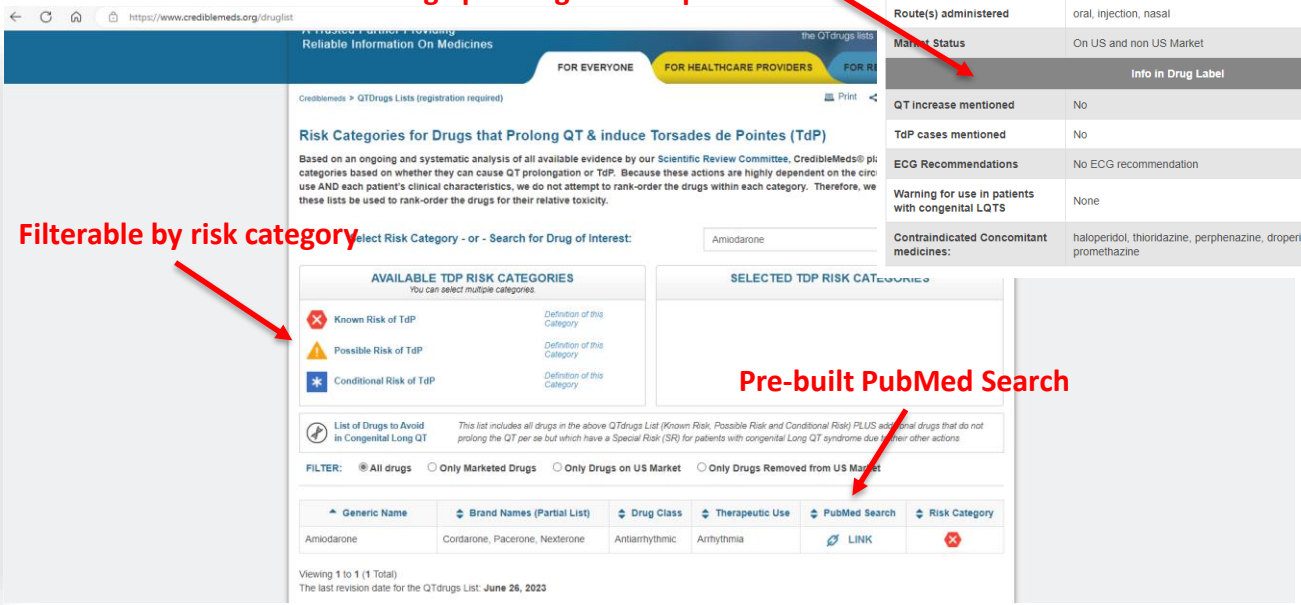
Electronic resource to promote safe medication use

- Best known for QTdrugs List



Mobile app available

Drug-specific guidance provided



Filterable by risk category

Pre-built PubMed Search

Generic Name(s)	Metoclopramide
Brand Names (Partial List)	Reglan, Gimoti, Afipran, Maxolon, Cerucal, Clopamon, Clopra, Maxeran, Maxolon, Metozolv, Plasil, Pramin, Primperan, Perinorm
Current TdP risk category	Drugs with conditional TdP risk
Conditions for TdP if Conditional Risk Drug	Bradycardia, Low serum K or Mg, Used with concomitant QT/TdP drug, Use in cLQTS patients
Main Therapeutic Use(s)	Nausea, vomiting
Route(s) administered	oral, injection, nasal
Market Status	On US and non US Market
Info in Drug Label	
QT increase mentioned	No
TdP cases mentioned	No
ECG Recommendations	No ECG recommendation
Warning for use in patients with congenital LQTS	None
Contraindicated Concomitant medicines:	haloperidol, thioridazine, perphenazine, droperidol, prochlorperazine, promethazine

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LIFE CHANGING MEDICINE

Long QT

Ann Canterbury, MD

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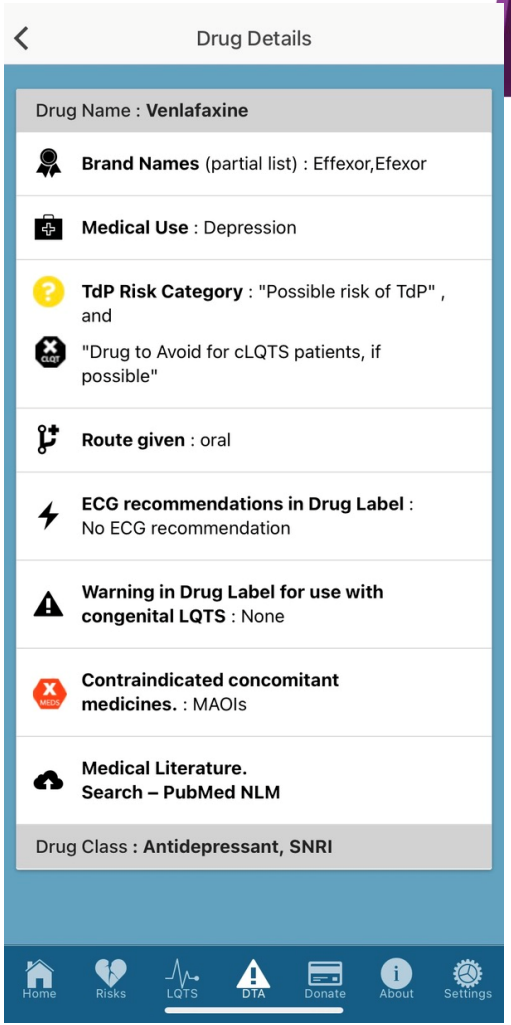
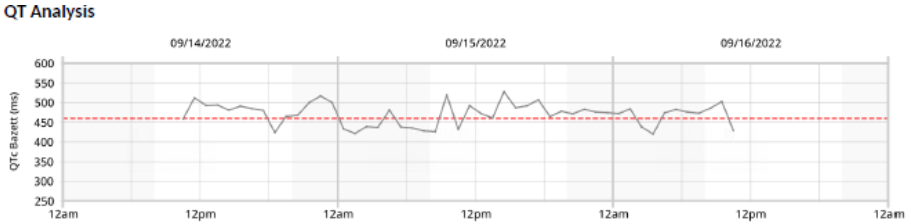
Case Continued

- Admitted to a history of syncope
  - While exercising during pregnancy
- No history of seizures
- Family history of sudden cardiac death in her father at age 50
- The patient had basic lab work with normal electrolytes
- Meds include: lisinopril and venlafaxine
- Venlafaxine discontinued and QTc went from 500 to 487

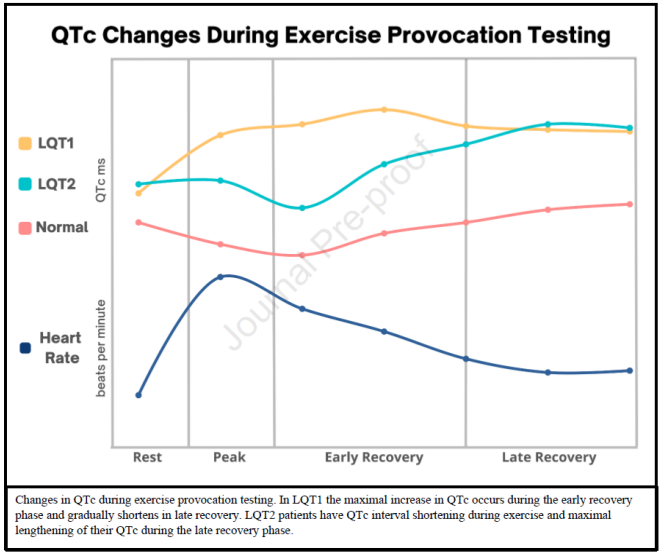


Additional work up

- Holter monitors can give QT analysis
  - Length of time with long QT during monitoring
  - Can see changes in T wave morphology
  - Assess for brady and tachyarrhythmias
- Stress testing
  - Typically QT shortens with exercise
  - If it gets longer, suggestive of pathology



QTc changes in LQT1 and LQT2 during exercise provocation testing



Changes in QTc during exercise provocation testing. In LQT1 the maximal increase in QTc occurs during the early recovery phase and gradually shortens in late recovery. LQT2 patients have QTc interval shortening during exercise and maximal lengthening of their QTc during the late recovery phase.

[27]

Additional work up continued

- Genetic testing
  - High clinical suspicion (Class I)
  - Schwartz score of 1.5 or higher (Class II)
  - Asymptomatic patients without a family history of congenital LQTS but who have serial ECGs with QTc ≥480 milliseconds before puberty or ≥500 milliseconds post-puberty (Class I)
  - Asymptomatic patients without a family history of congenital LQTS but who have serial ECGs with QTc ≥460 milliseconds before puberty or ≥480 milliseconds post-puberty (Class II)

Table 2. 1993-2011 LQTS diagnostic criteria[a]

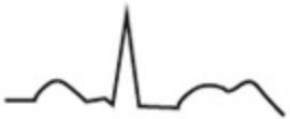
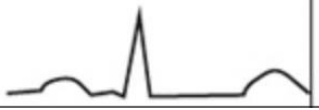
	Points
Electrocardiographic findings	
QTc[b] ≥480 ms 460-479 ms 450-459 ms (in males)	 3 2 1
QTc[b] 4th minute of recovery from exercise stress test ≥480 ms	1
Torsades de pointes[c]	2
T-wave alternans	1
Notched T-wave in 3 leads	1
Low heart rate for age[d]	0.5
Clinical history	
Syncope[c] With stress Without stress	 2 1
Congenital deafness	0.5
Family history	
Family members with definite LQTS[e]	1
Unexplained sudden cardiac death below age 30 years among immediate family members[e]	0.5

Case Continued

- Ultimately underwent genetic testing
- Positive for mutation in KCNQ1 (LQTS 1)

Congenital Long QT

- Prevalence of congenital LQTS is at least 1 in 2000 live births
- Asymptomatic patients who exceed 470ms (males) or 480 ms (females) on serial ECGs, and do not have acquired QT-prolonging factors, consider further evaluation for congenital LQTS
  - 10% chance of having LQTS
- Majority of individuals with LQTS are asymptomatic at diagnosis and remain so their entire life
- Present most commonly with LQTS-triggered syncope or syncope followed by generalized seizures

Type	Current	Functional Effect	Frequency Among LQTS	ECG	Triggers Lethal Cardiac Event	Penetrance*
LQTS1	K	↓	30%-35%		Exercise (68%) Emotional stress (14%) Sleep, response (9%) Others (19%)	62%
LQTS2	K	↓	25%-30%		Exercise (29%) Emotional stress (49%) Sleep, response (22%)	75%
LQTS3	Na	↑	5%-10%		Exercise (4%) Emotional stress (12%) Sleep, response (64%) Others (20%)	90%

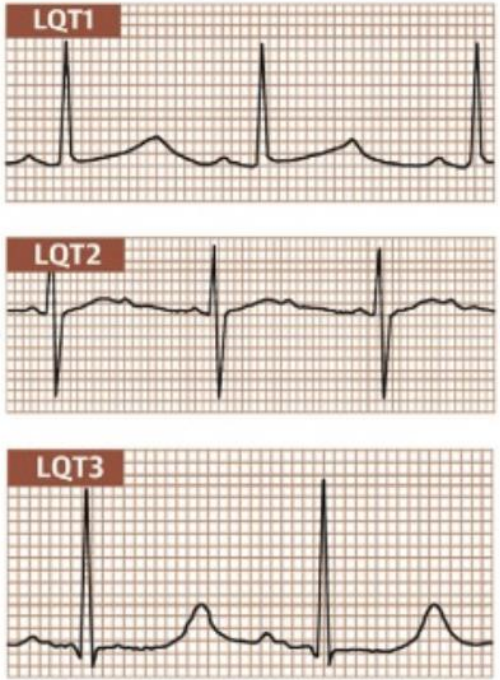
Assessment

Evaluation

- History and pedigree
- ECG
- Exercise ECG
- Genetic testing

Diagnosis

- Schwartz score ≥3.5
- Genetic testing
- QTc ≥480ms in females or ≥470ms in males



Risk Stratification

Established Markers

- Cardiac arrest
- QTc
- Cardiogenic syncope

Other Markers

- Age
- Sex (affected by age)
- Genotype

Treatment

Conservative

- Avoid QT prolonging drugs
- Safe exercise

Pharmacological

- β-blockers
- Mexiletine

Interventional

- Atrial pacing
- Left cardiac sympathetic denervation
- ICD

Krahn AD, et al. J Am Coll Cardiol EP. 2022;8(5):687-706.

HVI Inherited Arrhythmia Clinic

Where: UPMC Presbyterian Heart and Vascular Office (5B)/UPMC Passavant (Tuesday)

Who: Patients and families with suspected or confirmed inherited arrhythmic syndromes (LQTs, BrS, CPVT, SQTs, ERS) or ARVC, young SCA survivors or family members of SCD victim/SCA survivors.

What: Comprehensive family based phenotypic and genotype evaluation with pre and post test genetic counselling and arrhythmic risk stratification along with familial cascade genetic/screening (with collaboration of Children's CHP Electrophysiology).

Phone: 1 877 426-8762 (answered daily M-F 8AM-4:30PM) or 412 647-6000

Fax: 412 647-0595

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Direct contact number/ email of scheduling person (if needed): Toni Jones 412-647-9767; jonestm2@upmc.edu

RN: Katherine Davison RN Ph.412 647-9820, Fax 412 647-0595. davisonkl@upmc.edu

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UPMC
LIFE CHANGING MEDICINE

QT Prolongation: What is the actual risk?

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Clinical Pharmacist
UPMC Mercy