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Torsade De Pointes: A Comprehensive Review of Pathophysiology and Therapeutic Approach

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Abstract

Torsade de pointes (TdP) is a rare form of polymorphic ventricular tachycardia, marked by fluctuating QRS complex amplitudes that twist around the isoelectric line on an electrocardiogram (ECG). It is commonly linked to prolonged QT intervals, which can be either acquired or congenital. While TdP often resolves spontaneously, it may lead to fatal ventricular fibrillation if untreated. Medications, electrolyte imbalances, and genetic conditions like Jervell and Lange-Nielsen syndrome contribute to QT prolongation, increasing TdP risk. Women are more susceptible, as they tend to have longer QT intervals. Clinical evaluation includes ECG and risk assessment for QT prolongation, while treatment focuses on correcting underlying electrolyte imbalances and, in severe cases, electrical cardioversion or pharmacological intervention. Prevention strategies emphasize careful medication management and the use of decision-support tools to minimize QT-prolonging drug use.

Keywords: Drug-induced QT prolongation, Overdrive pacing, QT prolongation, Romano-Ward syndrome, Tisdale QT risk score, Torsade de pointes (TdP)

INTRODUCTION

Torsades de Pointes is a kind of polymorphic ventricular tachycardia that is distinguished by the QRS complexes' slow amplitude change and twisting around an isoelectric line on the ECG. QTc prolongation, which refers to the heart rate-adjusted extension of the QT interval, is associated with Torsades de Pointes [1]. When a male's QTc exceeds 450 ms and a female exceeds 460 ms, it is deemed lengthy. A two- to three-fold increase in risk for Torsades de Pointes has been linked to a QTc larger than 500 ms. The beat could end on its own or turn into ventricular fibrillation [2–4].

ETIOLOGY

QTc prolongation, which can be acquired or congenital, causes Torsades de pointes. The most common cause of acquired QTc prolongation is medication. A long list of drugs can make someone more susceptible to Torsades. Antiarrhythmics, antipsychotics, antiemetics, antifungals and antimicrobials are among the medications on the list, although they are not the only ones. QTc prolongation can be exacerbated by substances that delay the hepatic metabolism of these drugs, increasing the risk of Torsades de pointes. Jervell and Lange-Nielsen and Romano-Ward syndrome are the two hereditary variations of congenital extended QT. Congenital deafness is linked to the former. Several risk variables, such as being older (over 65), female, having hypokalaemia, hypocalcaemia, hypomagnesaemia, bradycardia, heart disease and using diuretics are also linked to prolonged QTc and Torsades [5, 6].

Jervell and Lange Nielsen syndrome and Romano-Ward syndrome are two uncommon congenital long QT disorders.

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Keys

QT Interval

- The QT interval is the time between the start of the Q wave and the end of the T wave.
- Electrocardiogram (ECG) An ECG (electrocardiogram).
- QRS stands for the sequence of Q, R and S waves in an electrocardiogram (ECG) that represents ventricular depolarization.

Epidemiology of Torsade

The exact prevalence of Torsade de pointes remains unclear. This potentially life-threatening arrhythmia can cause sudden cardiac death, even in people with structurally healthy hearts [7]. In the U.S., there are approximately 300,000 sudden cardiac deaths annually, with torsade likely responsible for less than 5% of these cases.

The corrected QT interval tends to be longer in White individuals compared to Black individuals, which may contribute to a lower risk of acquired torsade in the Black population.

Additionally, Brugada syndrome is more commonly seen in Southeast Asians [8].

Torsade de pointes is two to three times more prevalent in women than in men, as women generally exhibit longer QT intervals [9] and are more susceptible to QT prolongation due to medications. Congenital long QT syndrome is inherited in an autosomal manner but is expressed more frequently and results in a more significant QT interval increase in women than in men.

Torsade can affect individuals across all ages, from newborns to the elderly, with the highest incidence observed in those aged 35 to 50. When it occurs in younger patients, it is often due to congenital long QT syndrome, while in older patients, it typically stems from acquired long QT syndrome[10]. A systematic review found that just over half (50.8%) of elderly patients (aged 80 and older) who experienced drug-induced Torsade did so because of “reckless administration” of a medication that prolongs the QT interval. Common instances of such reckless administration involved using another QT-prolonging agent (51.6%) or doing so despite awareness of existing QT prolongation (25.8%) [10].

Pathophysiology

The delayed rectifier potassium current is inhibited as part of the Torsades de Pointes mechanism. A protracted repolarisation phase results from an overabundance of positive ions in the cellular membrane. This extended repolarisation phase can cause Torsades de Pointes if an ectopic beat, or R on T phenomenon, is produced. Both congenital and drug-induced QT prolongation interfere with the potassium channel, leading to similar effects on the cellular membrane. The ability of Torsades de Pointes to settle on its own makes it slightly distinct from ventricular fibrillation. If treatment for Torsades de Pointes is not received, it may eventually develop into venticular fibrillation [11].

PRESENTATION

Patient History

Patients experiencing Torsade de pointes typically report recurrent episodes of palpitations, dizziness and syncope that align with arrhythmia occurrences; however, it is important to note that sudden cardiac death may transpire during the initial episode. Additional symptoms, such as nausea, cold sweats, shortness of breath, and chest pain may also manifest, though these are nonspecific and can arise from various forms of tachyarrhythmia.

In young people diagnosed with torsade, it’s important to check for congenital long QT syndrome, especially if there is a family history of sudden cardiac death or sudden infant death syndrome.

In these cases, episodes of Torsade may be precipitated by adrenergic stimuli, including stress, fear, or physical activity, while other contributing factors should also be considered. Refer to Long QT Syndrome for further details [12].

A family history of congenital deafness may provide additional insight, although a prolonged QT interval is observed in only 0.25–0.3% of deaf-mute children. Patients with Jervell and Lange-Nielsen syndrome often exhibit congenital sensorineural deafness, which follows an autosomal dominant inheritance pattern for cardiac anomalies, while deafness in other cases typically follows an autosomal recessive pattern.

Romano-Ward syndrome is another form of familial or congenital long QT syndrome, distinguished by normal hearing and inherited in an autosomal dominant manner.

Acquired long QT syndrome patients generally experience Torsade during episodes of bradycardia. Medications and electrolyte imbalances, like hypokalemia and hypomagnesemia are the main causes of acquired long QT syndrome. Although drug-induced Torsade de pointes are relatively uncommon, its prevalence is increasing, with incidence rates reaching 2–3% for certain medications. Therefore, it is crucial to inquire about all current medications the patient is taking.

Physical Examination

The physical signs of Torsade de pointes depend on the tachycardia's rate and duration, as well as the degree of reduced blood flow to the brain. Common symptoms include a rapid pulse, low or normal blood pressure and brief or prolonged loss of consciousness. These may be preceded by bradycardia or premature ventricular contractions, which can lead to palpitations. An increase in premature ventricular contractions in patients with a prolonged QTc interval may help predict an imminent episode of Torsade [13].

Additional signs like pallor and sweating may be observed, particularly during a sustained episode. Other physical symptoms can vary depending on the underlying cause of Torsade.

Evaluation

To diagnose Torsades de Pointes, an ECG is crucial. The key feature is the twisting of the QRS complexes around the isoelectric line. Torsades de Pointes is triggered by a PVC occurring on a previous T wave. Typically, a series of short-long-short R-R intervals precedes the onset. The risk of Torsades de Pointes in cases of drug-induced QTc prolongation can be accurately assessed using a QT nomogram, with the patient being at risk if any value falls above the nomogram's threshold [14, 15].

Treatment/Management

The initial approach to managing Torsades de Pointes involves the prevention of its occurrence by addressing modifiable risk factors. This entails the cessation of any medications that prolong the QT interval and the optimization of the patient's electrolyte balance. Correcting deficiencies in potassium, magnesium and calcium is crucial in averting the onset of Torsades. A limited number of studies suggest a potential prophylactic advantage of administering oral or intravenous magnesium for patients experiencing drug-induced QT prolongation. However, the overall efficacy remains uncertain and there is scant evidence indicating that magnesium influences the QT interval itself.

The management of Torsades de Pointes begins with assessing the patient's hemodynamic stability. Most instances of Torsades are self-resolving; however, the risk escalates for those who may progress to ventricular fibrillation. For patients exhibiting hypotension or those in cardiac arrest due to Torsades de Pointes, electrical cardioversion is indicated. Synchronized cardioversion should be employed for hemodynamically unstable patients with a pulse, utilizing 100J for monophasic and 50J for biphasic shocks. In cases of pulseless Torsades, defibrillation is required.

Intravenous magnesium is the primary pharmacological treatment for Torsades de Pointes. It has been demonstrated to stabilize the cardiac membrane, although the precise mechanism remains unclear. The recommended initial administration is a slow intravenous push of 2g of magnesium. Following this, an infusion of 1 g to 4 g per hour should be initiated to maintain magnesium levels above 2 mmol/L.

Once levels exceed 3 mmol/L, the infusion may be discontinued. Severe magnesium toxicity can develop when levels exceed 3.5 mmol/L, leading to symptoms like confusion, respiratory depression, coma and cardiac arrest.

Additionally, it is essential to address any hypokalemia, ensuring that serum potassium levels are maintained between 4.5 mmol/L and 5 mmol/L during the treatment of Torsades de Pointes.

For a patient experiencing recurrent episodes of Torsades de Pointes, despite receiving magnesium treatment, increasing the heart rate may provide additional benefit. This can be done pharmacologically with medications like isoproterenol. Studies show that isoproterenol can effectively prevent Torsades de Pointes in patients with prolonged QT intervals who do not respond to magnesium treatment. As a non-selective beta agonist, isoproterenol elevates the heart rate and reduces the QT interval, thereby decreasing the risk of the R-on-T phenomenon, which can precipitate TdP. The medication can be administered as an intravenous push of 10 mcg to 20 mcg or via an infusion adjusted to maintain a heart rate of 100 bpm. However, isoproterenol is contraindicated in individuals with congenital prolonged QT syndrome, as it may inadvertently prolong the QT interval. An alternative approach for terminating Torsades de Pointes is overdrive pacing.

Although there are limited studies on the efficacy of pacing for this condition, numerous case reports suggest it is a feasible option. Overdrive pacing can be utilized in cases of frequent Torsades de Pointes and those resistant to magnesium treatment. The pacing rate should be set to surpass the patient's intrinsic ectopic rate, with ventricular rates typically ranging from 90 bpm to 110 bpm being adequate, though some patients may require rates as high as 140 bpm. Overdrive pacing is advised for both drug-induced and chemically induced Torsades de Pointes [16–18].

Prevention of Torsade

Raising awareness of congenital QT syndrome, along with implementing systematic approaches to assess, monitor and manage patients with drug-induced QTc prolongation, can help prevent this uncommon yet serious condition. An example is a 2019 literature-based algorithm that provides a stepwise approach for assessing and managing drug-induced QTc prolongation in psychiatric patients, which can be a useful resource for pharmacists assisting mental health professionals [19].

Given the rapidly changing information on medications that can prolong QTc, incorporating a clinical decision support tool into electronic health records at the point of care is a useful strategy for preventing torsade de pointes [20–23].

The Tisdale QT risk score assigns different weightings to factors, such as age, gender, hypokalemia, baseline QTc interval, use of loop diuretics, number of QT-prolonging drugs, sepsis, heart failure and acute myocardial infarction, to estimate the risk of QT prolongation.

By integrating this risk score into clinician alerts, the prescription of QT-prolonging medications has been reduced, which has helped lower the incidence of significant QT prolongation in cardiac care units [21].

Additionally, decision support tools in outpatient settings that emphasize the risk of QT prolongation may further decrease the likelihood of acquired long QT syndrome and consequently, the risk of Torsade.

CONCLUSIONS

Torsades de Pointes is a form of polymorphic ventricular tachycardia, identified by varying QRS complex amplitudes on an electrocardiogram that appear to twist around the isoelectric line. This condition is linked to QTc prolongation, which refers to a heart rate-adjusted extension of the QT interval. The arrhythmia may resolve spontaneously or progress to ventricular fibrillation. This activity covers the assessment and treatment of Torsades de Pointes and highlights the importance of teamwork among healthcare professionals to provide coordinated care and improve patient outcomes.

REFERENCES

1. Davies RA, Ladouceur VB, Green MS. The 2023 Canadian Cardiovascular Society clinical practice update on management of the patient with a prolonged QT interval. Can J Cardiol. 2023 Oct;39(10):1285–301.
2. Van Laecke S. Hypomagnesemia and hypermagnesemia. Acta Clin Belg. 2019 Feb;74(1):41–47.
3. Wilders R, Verkerk AO. Long QT syndrome and sinus bradycardia—a mini review. Front Cardiovasc Med. 2018;5:106.
4. Khan Q, Ismail M, Haider I. High prevalence of the risk factors for QT interval prolongation and associated drug-drug interactions in coronary care units. Postgrad Med. 2018 Nov;130(8):660–665.
5. De Vecchis R, Ariano C, Di Biase G, Noutsias M. Acquired drug-induced long QTc: New insights coming from a retrospective study. Eur J Clin Pharmacol. 2018 Dec;74(12):1645–1651.
6. Salem JE, Dureau P, Bachelot A, Germain M, Voiriot P, Lebourgeois B et al. Association of oral contraceptives with drug-induced QT interval prolongation in healthy nonmenopausal women. JAMA Cardiol. 2018 Sep 01;3(9):877–882.
7. Nikolic G, Bishop RL, Singh JB. Sudden death recorded during Holter monitoring. Circulation. 1982 Jul;66(1):218–225.
8. Antzelevitch C. Brugada syndrome. Pacing Clin Electrophysiol. 2006 Oct;29(10):1130–1159.
9. Lehmann MH, Timothy KW, Frankovich D. Age-gender influence on the rate-corrected QT interval and the QT-heart rate relation in families with genotypically characterized long QT syndrome. J Am Coll Cardiol. 1997 Jan;29(1):93–99.
10. Jackobson G, Carmel NN, Lotan D, Kremer A, Justo D. Reckless administration of QT interval-prolonging agents in elderly patients with drug-induced torsade de pointes. Z Gerontol Geriatr. 2018 Jan;51(1):41–47.
11. Baldzizhar A, Manuylova E, Marchenko R, Kryvalap Y, Carey MG. Ventricular tachycardias: Characteristics and management. Crit Care Nurs Clin North Am. 2016 Sep;28(3):317–329.
12. Cho Y. Management of patients with long QT syndrome. Korean Circ J. 2016 Nov;46(6):747–752.
13. Marill KA, Lopez S, Hark D, Spahr J, Kapadia N, Liu SW. Increased ventricular ectopy precedes Torsades de pointes in patients with prolonged QT. J Electrocardiol. 2023 Sep–Oct; 80:17–23.
14. Groffen AJ, Bikker H, Christiaans I. Long QT syndrome overview. In: Adam MP, Feldman J, Mirzaa GM, Pagon RA, Wallace SE, Amemiya A, editors. GeneReviews® [Internet]. University of Washington, Seattle; Seattle (WA): Feb 20, 2003;
15. Turner JR, Rodriguez I, Mantovani E, Gintant G, Kowey PR, Klotzbaugh RJ et al. Cardiac safety research consortium. Drug-induced proarrhythmia and Torsade de pointes: A primer for students and practitioners of medicine and pharmacy. J Clin Pharmacol. 2018 Aug;58(8):997–1012.
16. De Lemos ML, Kung C, Kletas V, Badry N, Kang I. Approach to initiating QT-prolonging oncology drugs in the ambulatory setting. J Oncol Pharm Pract. 2019 Jan;25(1):198–204.
17. Porta-Sánchez A, Gilbert C, Spears D, Amir E, Chan J, Nanthakumar K et al. Incidence, diagnosis and management of QT prolongation induced by cancer therapies: A systematic review. J Am Heart Assoc. 2017 Dec 07;6(12).
18. Al-Khatib SM, Stevenson WG, Ackerman MJ, Bryant WJ, Callans DJ, Curtis Ab et al. 2017 AHA/ACC/HRS guideline for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: Executive summary: A report of the Am College of Cardi/Am Heart Association Task Force on ClinPractice Guidelines and the Heart Rhythm Society. Heart Rhythm. 2018 Oct;15(10):

19. Zolezzi M, Cheung L. A literature-based algorithm for the assessment, management and monitoring of drug-induced QTc prolongation in the psychiatric population. Neuropsychiatr Dis Treat. 2019; 15:105–114.
20. Gallo T, Heise CW, Woosley RL. Clinician responses to a clinical decision support advisory for high risk of Torsades de pointes. J Am Heart Assoc. 2022 Jun 7;11(11):
21. Tisdale JE, Jaynes HA, Kingery JR. Effectiveness of a clinical decision support system for reducing the risk of QT interval prolongation in hospitalized patients. Circ Cardiovasc Qual Outcomes. 2014 May;7(3):381–390.
22. Sorita A, Bos JM, Morlan BW, Tarrell RF, Ackerman MJ, Caraballo PJ. Impact of clinical decision support preventing the use of QT-prolonging medications for patients at risk for Torsade de pointes. J Am Med Inform Assoc. 2015 Apr;22(e1):
23. Trinkley KE, Pell JM, Martinez DD, Maude NR, Hale G, Rosenberg MA. Assessing prescriber behavior with a clinical decision support tool to prevent drug-induced long QT syndrome. Appl Clin Inform. 2021 Jan;12(1):190–197.

