MEDDOCS Open Access Publisher

ISSN: 2639-4383

Annals of Cardiology and Vascular Medicine

Open Access | Case Report

A Rhythm-Control Dilemma: Torsades Following Progressive QT Prolongation in Severe Cardiomyopathy with Long QT Family History

Tolga Kiyici; Sumeeta Bhogal; Berke Cenktug Korucu*

Rutgers Health/ Jersey City Medical Center, Jersey City, NJ, USA.

***Corresponding Author(s):** **Berke Cenktug Korucu**
Jersey City Medical Center Department of Medicine 355 Grand St. Jersey City, NJ 07302, USA.
Email: cenkorucu@gmail.com

Received: May 23, 2026

Accepted: June 08, 2026

Published Online: June 15, 2026

Journal: Annals of Cardiology and Vascular Medicine

Publisher: MedDocs Publishers LLC

Online edition: http://meddocsonline.org/

Introduction

Torsades de pointes is a polymorphic ventricular tachycardia associated with delayed myocardial repolarization and QT interval prolongation. Its development is often multifactorial, with recognized contributors including structural heart disease, bradycardia, electrolyte abnormalities, inherited repolarization disorders, and QT-prolonging medications [1-3]. In adults with cardiomyopathy and coexisting atrial and ventricular arrhythmias, distinguishing inherited susceptibility from acquired QT prolongation can be clinically challenging.

Amiodarone is frequently used for rhythm control in patients with structural heart disease, but its repolarization effects require careful monitoring when QT prolongation evolves during therapy [3,4]. We present a case of progressive QT prolongation culminating in torsades de pointes during rhythm management in a patient with severe cardiomyopathy, mixed atrial and ventricular arrhythmias, and strong family history of confirmed long QT syndrome requiring ICD placement in multiple relatives.

Case presentation

A 71-year-old male with a history of heart failure with moderately reduced ejection fraction, hypertension, type 2 diabetes mellitus, and prior atrial and ventricular arrhythmias presented with progressively worsening shortness of breath for two weeks. Four months prior to presentation, outpatient cardiac event monitoring performed after exercise stress testing demonstrated premature atrial and ventricular beats with episodes of nonsustained ventricular tachycardia and supraventricular tachycardia with aberrancy. Family history was notable for long QT syndrome with implantable cardioverter-defibrillator placement in multiple family members.

On presentation, vital signs were notable for temperature 97.7°F, heart rate 141 beats/minute, respiratory rate 22 breaths/minute, blood pressure 118/78 mmHg, and oxygen saturation above 94% on room air. Physical examination was notable for bilateral crackles and jugular venous distension. Initial electrocardiogram demonstrated atrial fibrillation with repolar-

Cite this article: Kiyici T, Bhogal S, Cenktug Korucu B. A Rhythm-Control Dilemma: Torsades Following Progressive QT Prolongation in Severe Cardiomyopathy with Long QT Family History. Ann Cardiol Vasc Med. 2026; 9(1): 1092.

ization abnormalities and QTc ranging from 381–504 ms. Laboratory studies revealed AST 60 U/L, ALT 91 U/L, creatinine 1.2 mg/dL, glucose 134 mg/dL, troponin 20 ng/L, and proBNP 2033 pg/mL. Potassium, magnesium, and calcium levels remained within normal range at 4.0 mmol/L, 2.0 mg/dL, and 9.0 mg/dL, respectively. Bedside echocardiography demonstrated reduced ejection fraction compared to prior imaging with a collapsible inferior vena cava. The patient was treated with intravenous furosemide and metoprolol and admitted to the cardiovascular critical care unit for further management.

Transthoracic echocardiography demonstrated an ejection fraction of 20% with global hypokinesis. Digoxin therapy failed to restore sinus rhythm. Subsequent transesophageal echocardiography-guided cardioversion was unsuccessful. QTc prior to cardioversion was 406 ms and measured 382 ms immediately afterward. Amiodarone infusion was initiated and later transitioned to oral therapy. Cardiac catheterization demonstrated nonobstructive coronary artery disease. Guideline-directed medical therapy including sacubitril/valsartan, spironolactone, dapagliflozin, and nadolol was initiated due to concern for possible inherited long QT syndrome.

During hospitalization, serial electrocardiograms demonstrated progressive QT prolongation with QTc increasing to 540 ms on follow-up electrocardiography and subsequently to 645 ms prior to development of torsades de pointes. The patient developed polymorphic ventricular tachycardia consistent with torsades de pointes requiring intravenous magnesium sulfate 4 g and 200-J electrical cardioversion. Post-event electrocardiography continued to demonstrate marked repolarization abnormalities with QTc measuring 592 ms. Given malignant ventricular arrhythmia and concern for inherited arrhythmogenic substrate, dual-chamber implantable cardioverter-defibrillator placement was performed for secondary prevention.

Following ICD placement, the patient experienced recurrent supraventricular tachycardia managed with intravenous metoprolol and adenosine. Subsequent atrial lead dislodgement was identified, and the patient underwent atrial lead revision with restoration of sinus rhythm. Amiodarone was transitioned to mexiletine, and nadolol was changed to metoprolol succinate 50 mg daily following ICD placement to assist with management of paroxysmal supraventricular tachycardia. The patient remained stable afterward and was discharged with outpatient electrophysiology follow-up arranged.

Discussion

This case highlights progressive repolarization instability culminating in torsades de pointes during rhythm management in a patient with severe cardiomyopathy, mixed atrial and ventricular arrhythmias, and a strong family history of confirmed long QT syndrome requiring ICD placement in multiple relatives. Although the family history supported an inherited repolarization disorder, the marked QTc progression during hospitalization suggests that the malignant ventricular arrhythmia was likely multifactorial rather than attributable to a single trigger.

Torsades de pointes is associated with delayed ventricular repolarization and QT prolongation, but its development often reflects the interaction of multiple risk factors, including inherited repolarization abnormalities, structural heart disease, bradycardia, electrolyte disturbances, and QT-prolonging medications [1-3]. In this patient, severe electrolyte derangement was unlikely to be the primary driver, as electrolyte levels were within normal range around the torsades event. Instead, serial electrocardiograms demonstrated progressive QTc prolongation from 382 ms after cardioversion to 540 ms on follow-up and 645 ms before torsades de pointes, supporting dynamic repolarization instability as the central feature of the case.

This case also illustrates the difficulty of distinguishing inherited susceptibility from acquired QT prolongation in adults with cardiomyopathy and complex arrhythmias. The patient had a strong family history of confirmed long QT syndrome, but also had severe left ventricular systolic dysfunction and exposure to rhythm-control therapy. In such patients, QT prolongation may reflect reduced repolarization reserve, where an inherited substrate becomes clinically apparent after additional stressors are introduced [3,4].

Amiodarone is commonly used for rhythm control in patients with atrial arrhythmias and structural heart disease and is generally considered less torsadogenic than many other QT-prolonging antiarrhythmic agents [5-7]. However, amiodarone can prolong repolarization, and torsades de pointes has been reported, particularly in susceptible patients with structural heart disease, left ventricular dysfunction, bradycardia, electrolyte abnormalities, or concomitant rate-controlling/QT-prolonging therapy [5-7]. In this patient, amiodarone exposure may have contributed to progressive QT prolongation in the setting of severe cardiomyopathy and inherited repolarization susceptibility. However, the overall presentation is best interpreted as multifactorial repolarization instability rather than isolated drug-induced torsades.

The coexistence of atrial and ventricular arrhythmias was another important feature of this case. The patient presented with atrial fibrillation with rapid ventricular response, had prior outpatient monitoring demonstrating nonsustained ventricular tachycardia and supraventricular tachycardia with aberrancy, later developed torsades de pointes, and subsequently had recurrent supraventricular tachycardia. This pattern suggests a broader electrophysiologic vulnerability rather than a single isolated rhythm disorder. Similar published cases emphasize that inherited or acquired repolarization abnormalities may become clinically apparent when additional stressors such as myocardial disease, rhythm-control therapy, or cardioversion are present [8,9].

Management focused on acute termination of torsades and prevention of recurrent malignant arrhythmia. The patient received intravenous magnesium sulfate and 200-J electrical cardioversion. Magnesium is recommended in torsades de pointes even when serum magnesium is not frankly low, as its therapeutic role is not limited to correction of hypomagnesemia [2,3]. Given the malignant ventricular arrhythmia and inherited arrhythmogenic background, dual-chamber ICD implantation was performed for secondary prevention. Amiodarone was subsequently transitioned to mexiletine, and after ICD placement, nadolol was changed to metoprolol succinate to assist with management of paroxysmal supraventricular tachycardia.

The main teaching point is that QT prolongation may evolve progressively during rhythm management before malignant ventricular arrhythmia occurs. In patients with structural heart disease, inherited repolarization susceptibility, and mixed atrial and ventricular arrhythmias, serial ECG monitoring should guide antiarrhythmic decisions. Rather than attributing torsades to a single factor, clinicians should consider the combined effects of inherited substrate, cardiomyopathy, rhythm-control therapy, and dynamic repolarization changes.

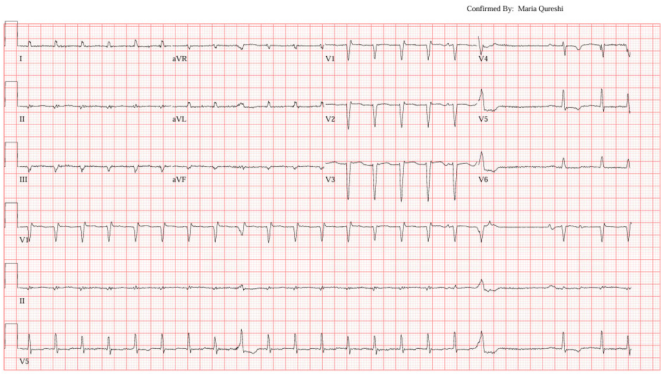

Figure 1: Initial ECG demonstrating atrial fibrillation with rapid ventricular response and frequent wide-complex beats, favored to represent premature ventricular complexes versus aberrant conduction, along with repolarization abnormalities.

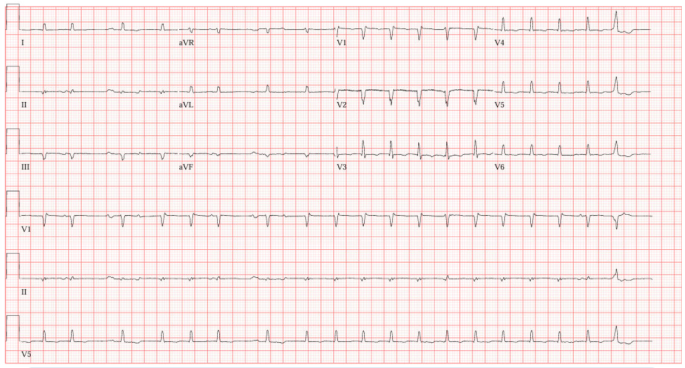

Figure 2: Latest ECG prior to torsades de pointes showing sinus rhythm with premature supraventricular complexes in a pattern of bigeminy; QTc reported 645 ms.

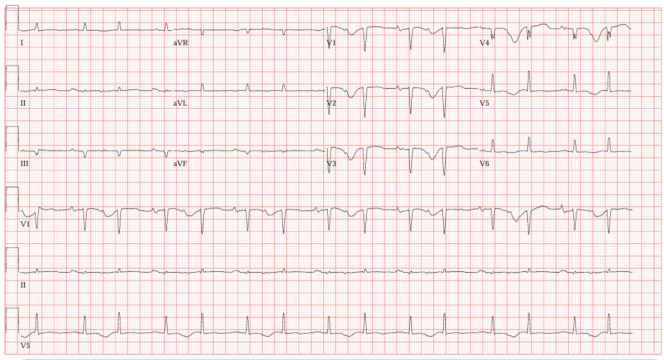

Figure 3: Latest ECG prior to torsades de pointes showing sinus rhythm with premature supraventricular complexes in a pattern of bigeminy; QTc reported 645 ms.

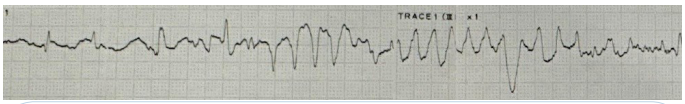

Figure 4: Latest ECG prior to torsades de pointes showing sinus rhythm with premature supraventricular complexes in a pattern of bigeminy; QTc reported 645 ms.

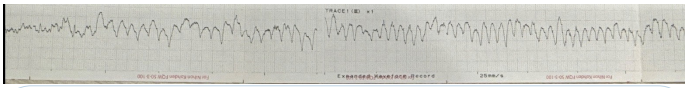

Figure 5: Telemetry strip showing torsades de pointes.

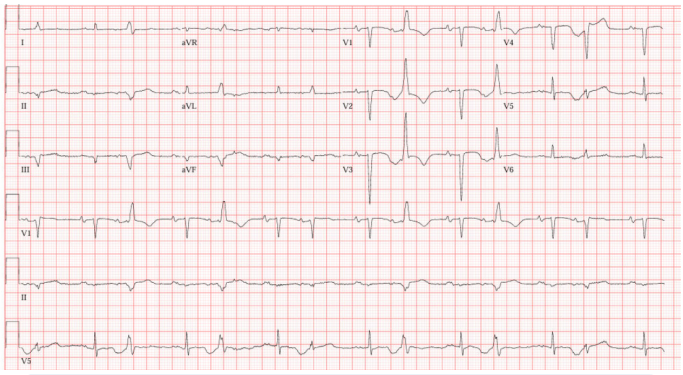

Figure 6: Post-event ECG showing persistent repolarization abnormalities; QTc reported 592 ms.

Conclusion

Progressive QT prolongation should be recognized as a dynamic marker of repolarization instability rather than an isolated ECG measurement. In patients receiving rhythm-control therapy or other QT-prolonging medications, serial ECG monitoring and reassessment of modifiable risk factors are essential to prevent malignant ventricular arrhythmias. Early recognition of worsening QT prolongation can guide timely intervention, including electrolyte optimization, medication adjustment, acute torsades management, and consideration of secondary prevention strategies when clinically indicated.

References

1. Cohagan B, Brandis D. Torsade de Pointes. StatPearls.
2. Li M, Ramos LG. Drug-Induced QT Prolongation and Torsades de Pointes. P T. 2017; 42: 473-477.
3. Drew BJ, Ackerman MJ, Funk M, et al. Prevention of Torsade de Pointes in Hospital Settings. Circulation. 2010; 121: 1047-1060.
4. Wilde AAM, Amin AS. Diagnosis, management and therapeutic strategies for congenital long QT syndrome. Heart. 2022; 108: 332-338.
5. Schrickel JW, Schwab JO, Yang A, et al. "Torsade de pointes" in patients with structural heart disease and atrial fibrillation treated with amiodarone. Pacing Clin Electrophysiol. 2006; 29: 363-366.
6. Shenthar J, Rachaiah JM, Pillai V, et al. Incidence of drug-induced torsades de pointes with intravenous amiodarone. Indian Heart J. 2017; 69: 707-713.
7. Siddoway LA. Amiodarone: Guidelines for Use and Monitoring. Am Fam Physician. 2003; 68: 2189-2197.
8. Lawin D, et al. Case report of a patient with congenital long QT syndrome Type 2 presenting with electrical storm: do not judge a book by its cover! Eur Heart J Case Rep. 2022.
9. Lypourlis D, Mundisugih J, Chia YV. Early afterdepolarizations and electrical storm after cardioversion for atrial fibrillation. HeartRhythm Case Rep. 2022; 8: 254-258.