# Recurrent Torsades de Pointes in an Elderly Woman with a Long QT Syndrome

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## Introduction

- The QT interval represents the duration of ventricular depolarization and repolarization.
- Prolongation of the QT interval may be congenital or acquired.
- This increases the risk of polymorphic ventricular tachycardia (i.e., torsades de pointes) and cardiac arrest.
- Recognition, understanding and appropriate and timely intervention can improve survivability in these patients.
- We describe an illustrative case in which acquired prolongation of the QT interval secondary to electrolyte derangement and administration of ciprofloxacin resulted in cardiac arrest due to torsade de pointes.

## Patient Description

- A 78-year-old female with a past medical history of atrial fibrillation, ischemic heart disease, congestive heart failure, nonischemic cardiomyopathy, and hypertension.
- She was recently admitted for atrial fibrillation, during which she was treated with heparin and amiodarone.
- While admitted she had a syncopal episode which was diagnosed as Torsades de Pointes (TdP) thought secondary to QT prolongation by amiodarone and electrolyte abnormalities.
- Amiodarone and furosemide were discontinued with stabilization of QT interval.
- She had no recurrence of TdP and fitted for a LifeVest prior to discharge (Figure 1).
- The day following hospital discharge, she presented to the emergency department (ED) after being shocked three times by her LifeVest.
- Review of electrocardiogram demonstrated prolonged QTc of 559 milliseconds (Figure 2).
- She had a witnessed syncopal episode while in the ED with identification of TdP and was defibrillated (Figure 3).

**Figure 1. Zoll LifeVest consisting of garment, electrode belt, and a monitor**


**Figure 2.** ECG in the emergency department showing prolonged QTc of 559 milliseconds

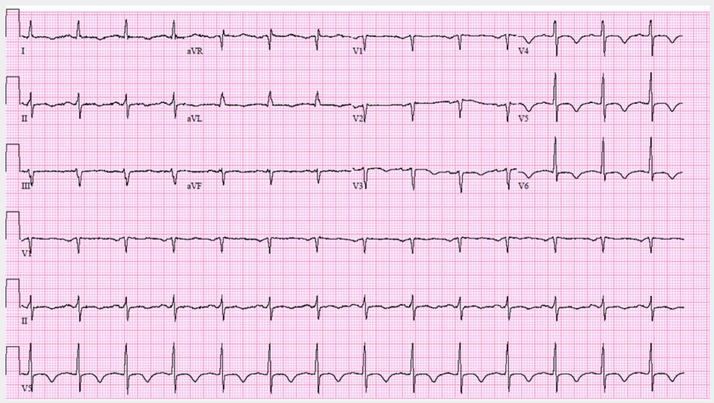
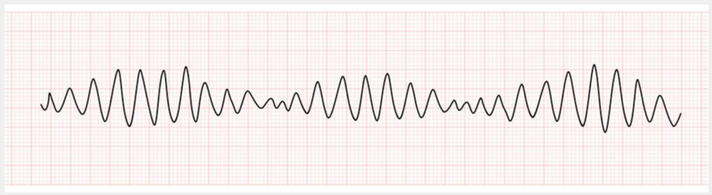



**Figure 3.** Rhythm strip showing Torsades de Pointes, a type of polymorphic ventricular tachycardia characterized by oscillatory changes in amplitude of the QRS complexes around the isoelectric line.



## Intervention

- On admission she was started on lidocaine and underwent left heart catheterization that identified an anomalous right coronary artery without evidence of obstructive coronary pathology.
- It was felt that her cause for recurrent TdP was due to R on T phenomenon associated with frequent premature ventricular contractions.
- She ultimately underwent successful placement of a dual chamber internal cardiac defibrillator (DCICD) with atrial pacing.
- She has had no recurrences of symptoms since placement of the defibrillator and discontinuing the lidocaine.

## Discussion

- Acquired causes of long QT syndrome include structural heart disease (heart failure, ischemia, left ventricular hypertrophy), drug-induced, hyponatremia, hypokalemia, hypocalcemia, starvation, hypothermia and elevated ICP .
- The first step in managing TdP is preventing its onset by targeting modifiable risk factors.
- This includes discontinuing any QT prolonging drugs and optimizing a patient's electrolyte profile. Correcting hypokalemia, hypomagnesemia, and hypocalcemia can all help to prevent the onset of torsades.
- Magnesium 1-2 grams should be administered in conjunction with electrical defibrillation over one to two minutes.
- For a patient that continues to have intermittent runs of Torsades de Pointes, despite treatment with magnesium, temporary transvenous overdrive pacing can be employed.

## Conclusions

- Causes of acquired prolongation of the QT interval are common in critically ill patients.
- The prognosis is good as long as the trigger medication or factor has been identified and discontinued.
- Patients should be taught how to monitor their pulses and note for the presence of any adverse effects from the medications. In addition, the family should be taught basic life support.