Class III Antiarrhythmics and Periprocedural **Torsades de Pointes: A Case Series**

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BACKGROUND

- Torsades de pointes (TdP) is a type of polymorphic ventricular tachycardia characterized by oscillatory changes in amplitude of the QRS complexes around the isoelectric line on electrocardiogram (ECG)
- TdP is associated with QT interval prolongation
- TdP may terminate spontaneously or degenerate into ventricular fibrillation
- Class III antiarrhythmics are commonly used for rhythm control in patients with atrial fibrillation but are associated with the development of torsades de pointes polymorphic ventricular tachycardia due to prolongation of cardiac repolarization duration, manifested as a prolonged QTc interval on the electrocardiogram
- Risk is increased with bradycardia, fluid shifts, and electrolyte imbalances
- This case series describes two women on sotalol or dofetilide with initially normal QTc who developed TdP during or early after catheter ablation for atrial fibrillation

CASE 1

- A 68-year-old female with atrial fibrillation on dofetilide presented with episodes of lightheadedness, chest pressure, and nausea three days after catheter ablation for atrial fibrillation
- Initial ECG showed a rate of 86 bpm and QTc of 426 ms
- The next day, ECG showed sinus bradycardia with a rate of 55 bpm and prolonged QTc of 577 ms (Figure 1)
- Her telemetry showed brief episodes of TdP
- Dofetilide was discontinued, and magnesium and potassium were administered
- She had no subsequent ventricular arrhythmias, and QTc normalized

CASE 2

- A 70-year-old female with atrial fibrillation on sotalol presented for catheter ablation for atrial fibrillation
- ECG showed sinus bradycardia with a rate of 54 bpm and QTc of 467 ms
- After induction of anesthesia, her rate was 34 bpm with QTc of 640 ms, and she developed TdP (Figure 2)
- Defibrillation at 200 J was successful
- Pacing from the coronary sinus was performed to prevent bradycardia, and she underwent successful ablation for atrial fibrillation
- Sotalol was discontinued, and she was monitored for 2 days without recurrence of ventricular arrhythmias.

Torsades de pointes is a type of polymorphic ventricular tachycardia that is associated with QT prolongation.

In patients taking Class III antiarrhythmics, risk of TdP is increased by periprocedural bradycardia, fluid shifts, and electrolyte imbalances.

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Figure 1: Case 1 EKG. EKG demonstrates bradycardia with a rate of 55 beats per minute and a prolonged QT interval of 577 milliseconds, corrected with Bazett's formula.

Figure 2: Case 2 Telemetry Strip. Telemetry strip demonstrates initiation of polymorphic ventricular tachycardia consistent with torsades de pointes.

These two cases illustrate the importance of carefully monitoring patients with risk factors for QTc prolongation, particularly those taking class III antiarrhythmics, during and after surgical procedures, as they are at risk for developing TdP precipitated by electrolyte shifts and bradycardia.



DISCUSSION

 Both patients developed TdP periprocedurally despite previously normal QTc and no prior ventricular arrhythmia

 Acute fluid shifts and bradycardia likely contributed to precipitating acutely prolonged QTc and development of TdP, despite normal electrolyte levels and no administration of other QTc prolonging medications

The QT prolonging effect of class III antiarrhythmics is caused by inhibition of the rapid component of the delayed rectifier potassium current (IKr)

mediated by the potassium channel encoded by the KCHN2 gene. This results in prolongation of the action potential duration and increased susceptibility to afterdepolarizations that can trigger TdP.

• Women are more prone to drug-induced TdP for unclear reasons, but it is postulated that hormonal differences may play a role.

Bradycardia is a major risk factor for drug-induced TdP due to the reverseuse dependency of inhibition of lkr.

• In both cases, TdP was quickly identified and treated, electrolytes were replaced, and the antiarrhythmic drug was appropriately withdrawn



CONCLUSION