Case Report

Amiodarone-Induced *Torsade de Pointes* in a Patient with Wolff-Parkinson-White Syndrome

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Amiodarone is generally regarded to have a high safety profile with a low incidence of arrhythmias. However, there have been reports of *torsades de pointes* under certain conditions, such as electrolyte imbalance or concomitant antiarrhythmic therapy. We describe a case of amiodarone-induced *torsade de pointes* early after initiation of intravenous amiodarone in the setting of T-wave *alternans*.

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Case presentation

An approximately 60-year-old African American male was evaluated for complaints of palpitations and dyspnea of 2 days' duration. The electrocardiogram revealed wide complex tachycardia (Figure 1) with intermittent loss of atrioventricular conduction, best seen in lead V_1 (arrows). The duration of the QRS complex was prolonged, measuring approximately 155 ms, with positive concordance in the precordial leads. The patient was transferred to the coronary care unit on intravenous amiodarone at 1 mg/min following a loading dose of 150 mg. After 24 hours of intravenous amiodarone exposure (approximate total dose of 1 g), sinus rhythm was restored. The analysis of the ECG upon cardioversion revealed a short PR interval with QT interval prolongation (QTm 582 ms, QTc 582 ms) with evidence of pre-excitation (delta waves) in the precordial leads (Figure 2) and macroscopic T-wave *alternans*. In light of his electrocardiographic findings, it was suspected that the patient had experienced atrial fibrillation due to rapid anterograde conduction over the accessory pathway. Intravenous amiodarone was continued and an electrophysiological study with possible catheter ablation was planned. However, that night the patient's telemeter monitoring recorded frequent asymptomatic multifocal premature ventricular complexes (PVCs). Eventually, the patient complained of "not feeling well" and was found to be pale, light headed and shivering. Telemetry recordings at this time showed a short run of sustained polymorphic ventricular tachycardia with undulation of the QRS complex compatible with *torsade de pointes* (Figure 3). Serum electrolytes and magnesium levels were all within normal limits. Intravenous amiodarone was discontinued and

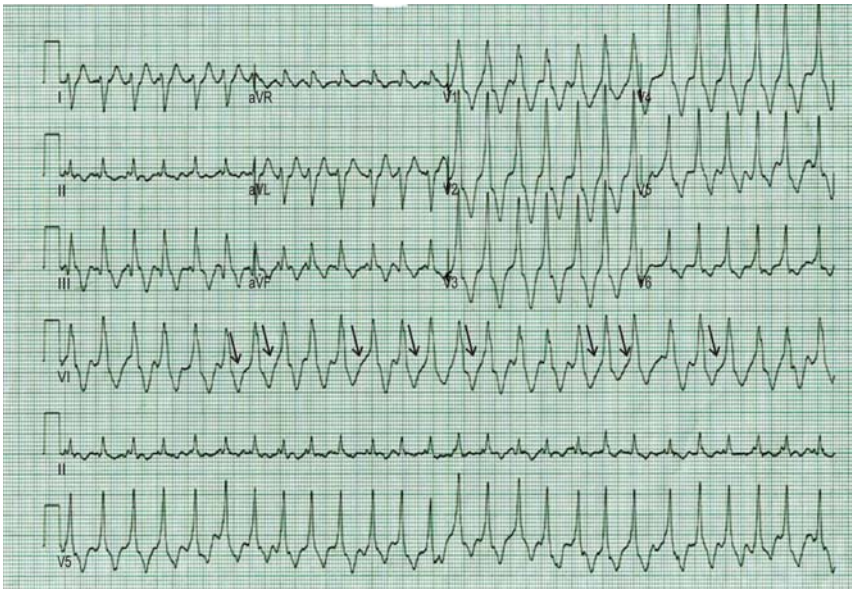

Figure 1. Presenting 12-lead ECG with wide-QRS complex tachycardia. There is intermittent loss of atrioventricular conduction best seen in lead V_1 (arrows). The QRS width was measured to be approximately 155 ms with positive concordance in the pre-cordial leads.

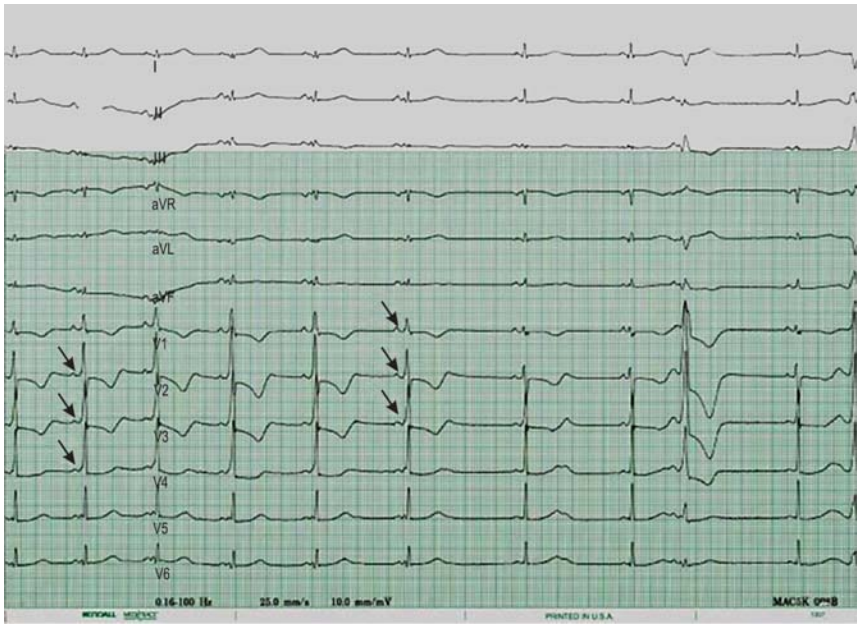

Figure 2. A 12-lead ECG after intravenous amiodarone cardioversion demonstrates sinus rhythm with a short PR interval and QRS prolongation. Also present is shortening of the PR interval with slurred slow rising onset to the QRS or the delta waves in leads V_1-V_4 (arrows). Significant macroscopic T-wave *alternans* is also evident on the chest leads (V_2-V_4).

the patient was promptly taken to the electrophysiology laboratory where mapping confirmed a left lateral accessory pathway, which was ablated successfully. At 6 months of follow up, he was clinically stable with the QT interval prolongation no longer present (QT ~ 418 ms, QTc ~ 438 ms). Nor was any delta wave visible at this time.

Discussion

This case illustrates the proarrhythmic potential of amiodarone, a commonly prescribed and potent antiarrhythmic agent. Amiodarone has certain unique pharmacological properties that differentiate it from other anti-arrhythmics,[1] such as the ability to block the fast sodium channel and the slow I_{Ca} current mediated through the L-calcium channel.[2] This calcium-channel blocking action is an important factor explaining the relatively low incidence of *torsade de pointes* associated with amiodarone. However, electrophysiological changes and ventricular tachyarrhythmia have sometimes been observed during high-dose amiodarone loading within the first day of therapy under certain clinical conditions.[3,4] This appears to be the case with our pa-

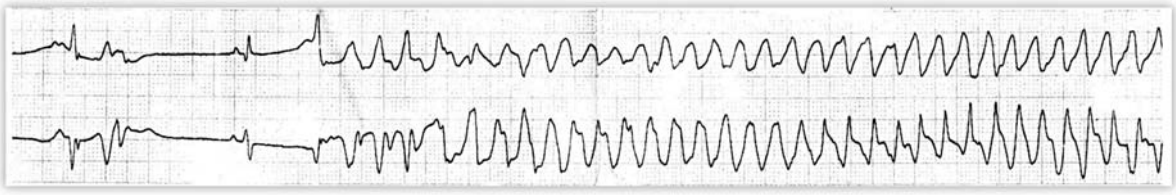

Figure 3. Telemetry strip revealing bradycardia with a long QT interval preceding a run of polymorphic ventricular tachycardia with undulation on the QRS axis *(torsade de pointes)*. The rhythm self-resolved before any medical therapy could be initiated.

tient, who was started on a maintenance dose of amiodarone (1 mg/ min) following a bolus (150 mg). Upon conversion, his ECG revealed certain highly arrhythmogenic milieus that significantly increased his risk for the development of ventricular arrhythmias. These included QT interval prolongation and rare macroscopic T-wave *alternans*. Continuation of intravenous amiodarone despite these changes eventually led to *torsade* in our patient. He had no history of ventricular tachycardia before amiodarone administration and developed *torsade* within 24 hours of initiation of intravenous therapy. Our case illustrates the importance of recognizing the proarrhythmic properties of amiodarone in certain high-risk situations. In our patient it was the highly arrhythmogenic combination of prolonged QT interval and T-wave *alternans*. Clinicians must therefore be aware of the importance of careful patient monitoring during amiodarone therapy, even with short-term use, in high risk patients.

References

1. Podrid PJ. Amiodarone: reevaluation of an old drug. Ann Intern Med. 1995; 122: 689-700.
2. Nattel S, Talajic M, Quantz M, DeRoode M. Frequency-dependent effects of amiodarone on atrioventricular nodal function and slow-channel action potentials: evidence for calcium channel blocking activity. Circulation. 1987; 76: 442-449.
3. Lim HE, Pak HN, Ahn JC, Song WH, Kim YH. Torsade de pointes induced by short-term oral amiodarone therapy. Europace. 2006; 8: 1051-1053.
4. Tomcsányi J, Merkely B, Tenczer J, Papp L, Karlócai K. Early proarrhythmia during intravenous amiodarone treatment. Pacing Clin Electrophysiol. 1999; 22: 968-970.