Amiodarone Induced Torsades De Pointes with Intravenous Therapy for Atrial Fibrillation

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July 1, 2020

Abstract

The case of a 71 year-old woman with rapid atrial fibrillation who underwent electrical cardioversion and amiodarone therapy. QT interval prolongation was observed with the development of torsades de pointes that required recurrent electrical cardioversion and temporal pacemaker implantation.

Key Clinical Message : Amiodarone induced torsade de pointes is very rare tachyarrhythmia developed during intravenous therapy for atrial fibrillation. Careful monitoring of the QT interval can prevent development of this life-threatening arrhythmia.

Introduction: Amiodarone is a class III antiarrhythmic agent with a low frequency of pro-arrhythmic effect and an incidence of torsade de pointes (TdP) of <1.0% (1-3). Intravenous (IV) amiodarone is useful for the treatment of atrial fibrillation and ventricular tachyarrhythmias (4). We presented the rare case of a woman with rapid atrial fibrillation (AF) who developed TdP on day 3 of intravenous amiodarone therapy.

Case Report: A 71 year-old woman with a history of diabetes mellitus and hypertension treated with metformin and bisoprolol 2.5 mg, admitted with palpitations and dyspnea for two days. An examination revealed wheezing on the base of the lungs and rapid AF on ECG with QT interval of 326 msec, QTc 405 msec (Fig.1). Transoesophageal echocardiogram excluded left atrial thrombus and showed preserved left ventricular function. Electrical cardioversion to NSR was done and infusion of amiodarone 1 mg/min for 6 hours, then 0.5 mg/min for 18 hours started after 150 mg bolus. Soon atrial fibrillation developed again. For treatment after the first 24h, the maintenance infusion of amiodarone 0.5 mg/min was continued. Electrolytes were in the normal ranges. On the third day of hospitalization, an electrical cardioversion to NSR was done again after an additional 150 mg bolus of amiodarone. In the evening recurrent episodes of TdP were developed and need to electrical cardioversion (Fig.2). ECG revealed QT prolongation (511 msec, QTc 531 msec). The amiodarone was discontinued. An insertion of temporal pacemaker for 2 days was required to suppress this life-threatening arrhythmia (Fig.3). The QT interval gradually decreased to its baseline value after cessation of the intravenous amiodarone.

Discussion: Although therapy with amiodarone is generally considered safe, torsades de pointes can be developed after intravenous use of the drug (5-6). Amiodarone act by blocking different ion channels involving in the action potential with a dominant effect on potassium channels and therefore can prolong QT interval. The drug also causes bradycardia by suppressing the sinus node and atrio-ventricular conduction. Intravenous amiodarone significant slow intraventricular conduction and does not prevent the inducibility of ventricular tachycardia (7-8). In addition to route, dose and rate administration, other predisposing factors

to amiodarone induced TdP may be electrolyte disturbances and bradycardia due to concomitant drugs such as beta blockers and/or digoxin (9). The arrhythmia is more common in women (10).

Our patient was a woman treated with a low dose of beta-blocker on the day of start amiodarone infusion. It may be preferable to avoid initiation of IV amiodarone with beta-blocker. The patient admitted with rapid atrial fibrillation and pulmonary congestion and we need to do cardioversion and start antiarrhythmic drug. Soon after electrical cardioversion to normal sinus rhythm and intravenous amiodarone therapy, her heart rhythm again returned to rapid atrial fibrillation. We continued to treat our patient with IV amiodarone. During this time she was on ECG monitoring, but without regular measurement of QT interval. On the third day we gave additional bolus of amiodarone 150 mg and after that did cardioversion. In this evening amiodarone induced TdP was developed. Significant QT prolongation was revealed on ECG.

Usually amiodarone induced TdP occurs within 24h after initiation of the therapy. In our case it occurred on day 3 of the maintenance infusion of amiodarone. Perhaps we didn't have to give an additional bolus of amiodarone before the second cardioversion of atrial fibrillation.

Conclusions: C areful monitoring of the QT interval during intravenous amiodarone therapy can prevent development of amiodarone-induced TdP.

Author Contributions:

Irina Nordkin MD, MHA: treated the patient and wrote the manuscript.

Tatyana Levinas MD: treated the patient and revised the manuscript.

Inna Rosenfeld MD: treated the patient and revised the manuscript.

Majdi Halabi MD: treated the patient and wrote the manuscript.

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Figure legends:

Fig.1 ECG on admission revealing rapid atrial fibrillation.

Fig.2 ECG showing episodes of TdP and markedly prolonged QT interval.

Fig.3 ECG with temporal pacemaker at 75 bpm.