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CASE REPORT

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Torsades de pointes after prolonged intravenous amiodarone therapy for atrial fibrillation

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Abstract

Amiodarone can induce TdP; therefore, it should be avoided as a first choice for therapy in patients without heart disease. Careful QT interval monitoring, especially during intravenous use, can prevent development of this life-threatening arrhythmia.

KEYWORDS

amiodarone, atrial fibrillation, QT prolongation, torsades de Pointes

1 | INTRODUCTION

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

Amiodarone is a class III antiarrhythmic agent with a low frequency of pro-arrhythmic effects and an incidence of TdP of <1.0%.[1-3] Intravenous amiodarone is useful for the treatment of AFib and ventricular tachyarrhythmias.[4] We presented the rare case of a woman with rapid AFib who developed TdP on day 3 of intravenous amiodarone therapy.

2 | CASE REPORT

A 71-year-old woman with a history of diabetes mellitus and hypertension treated with metformin and bisoprolol 2.5 mg was admitted with palpitations and dyspnea which she has been suffering for the two previous days. An examination revealed wheezing on the base of the lungs and rapid AFib on ECG with QT interval of 326 msec, QTc 405 msec (Figure 1). Electrolytes were in the normal ranges. Transesophageal echocardiogram (TEE) excluded left atrial thrombus and showed preserved left ventricular function. Electrical cardioversion to normal sinus rhythm (NSR) was done and an infusion of amiodarone 1 mg/min for 6 hours and then 0.5 mg/min for 18 hours was started after administrating 150 mg bolus. Usually, we administer intravenous amiodarone for 24 hours after sinus rhythm restoration and perform at least one ECG every 24 hours. In this patient soon after cardioversion, the AFib developed again. Therefore, treatment with the maintenance infusion of amiodarone 0.5 mg/min was continued after the first 24-hour period. On the third day of the therapy, the rhythm on the ECG was AFib 87/min with slightly prolonged QT/QTc intervals (444 msec, QTc 488 msec) (Figure 2). An electrical cardioversion to normal sinus rhythm (NSR) of 60-70/min was done again after additional 150 mg bolus of amiodarone had been given. In the evening, recurrent episodes of TdP developed and a number of electrical cardioversions were required (Figure 3). The ECG revealed a markedly prolonged QT interval of 511 msec (QTc 531 msec). The amiodarone was discontinued. An insertion of a temporary pacemaker for 2 days was required to

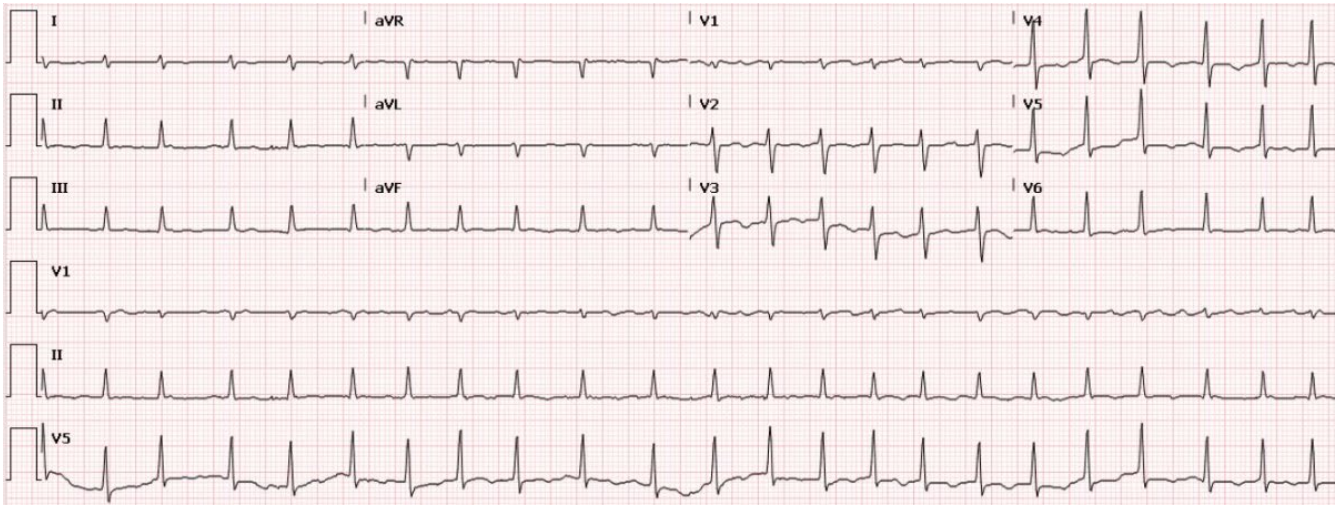

FIGURE 1 ECG on admission revealing rapid atrial fibrillation

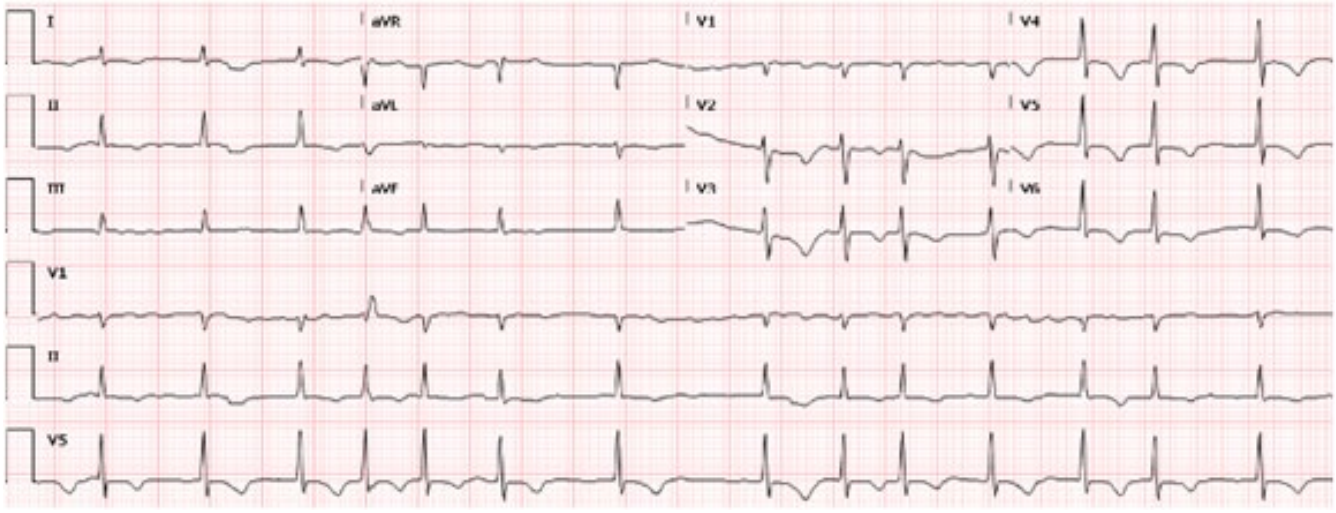

FIGURE 2 ECG with AFib on the third day of the hospitalization

suppress this life-threatening arrhythmia (Figure 4). The QT interval gradually decreased to its baseline value after cessation of the intravenous amiodarone.

3 | DISCUSSION

Amiodarone a class III antiarrhythmic agents is useful for the treatment of atrial and ventricular tachyarrhythmias. This drug has pro-arrhythmic effects and TdP can develop with a higher incidence after intravenous use.[5,6] Amiodarone acts by blocking different ion channels involved 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 atrioventricular conduction. Intravenous amiodarone significantly slows 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 who was being treated regularly with a low dose of beta-blocker and had already taken it on the day when the amiodarone infusion was started. It may be preferable to avoid initiation of IV amiodarone with concomitant beta-blocker. The patient was admitted with rapid AFib and signs of left heart failure. Her hemodynamic state was stable. She did not have a history of ischemic heart disease and TEE showed a normal left ventricular function.

The medical management of AFib includes rate or rhythm control. In patients with signs of left heart failure, treatment with beta-blockers or calcium channel blockers can worsen

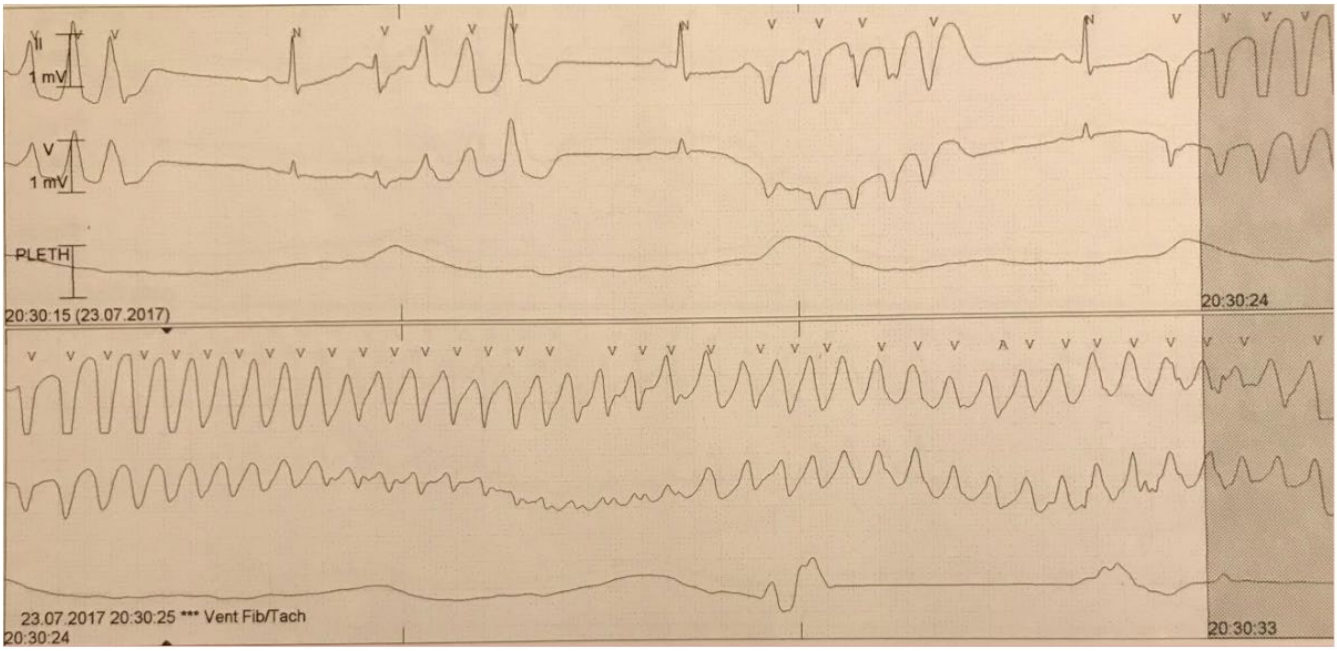

FIGURE 3 ECG showing episodes of TdP and markedly prolonged QT interval

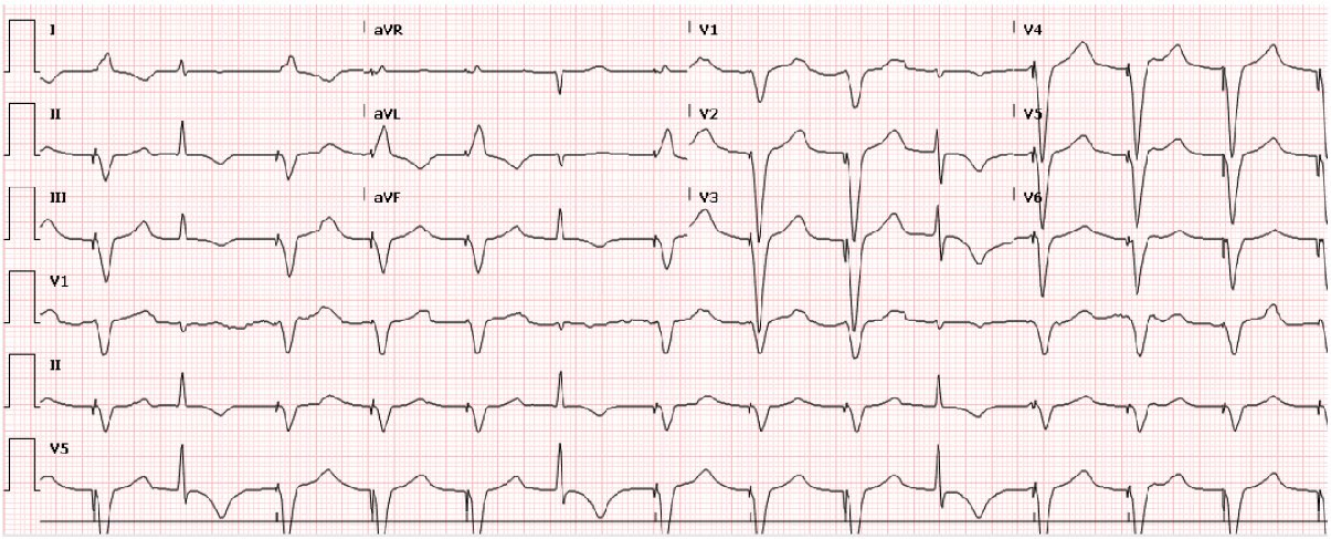

FIGURE 4 ECG with temporal pacemaker at 75 bpm

this condition. In this patient, we preferred rhythm control with the goal of reducing AFib-related symptoms and improving her quality of life over time.

In stable patients, either pharmacological or electrical cardioversion can be attempted, but pharmacological cardioversion is less effective.

According to current guidelines for the management of atrial fibrillation, pretreatment with amiodarone, flecainide, ibutilide, or propafenone should be considered to facilitate the success of electrical cardioversion. The choice of a specific drug is based on the type and severity of associated heart disease. Owing to extracardiac toxicity of amiodarone, other antiarrhythmic drugs should be considered first whenever possible. Amiodarone is mainly indicated in patients with heart failure.[11]

As already mentioned, for our patient we decided to do electrical cardioversion and start an antiarrhythmic drug. Soon after electrical cardioversion to NSR 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. That evening amiodarone-induced TdP developed. Significant QT prolongation was revealed on the ECG.

According to literature, amiodarone-induced TdP more commonly occurs within 24 hours after initiation of the therapy.[5] In our case, it occurred on day 3 of the maintenance infusion of amiodarone. On that morning the ECG revealed prolongation of QTc 488 msec as in comparison with ECG on admission (QTc 405 msec). Perhaps at that time we should have stopped the infusion of amiodarone, as well as not giving an additional bolus of amiodarone before the second cardioversion. After restoration of NSR, the rate was in the low normal range. It would have been useful to assess QT/QTc intervals during that day. An excessive QTc prolongation above 500 msec caused the development of TdP in the patient.

4 | CONCLUSIONS

Intravenous amiodarone therapy requires careful monitoring of the QT interval and timely discontinuation of the treatment in case of excessive prolongation. It can prevent the development of amiodarone-induced TdP.

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CONFLICT OF INTEREST

None declared.

AUTHOR CONTRIBUTIONS

Irina Nordkin MD, MHA, treated the patient and wrote the manuscript. Tatyana Levinas, MD, and Inna Rosenfeld, MD, treated the patient and revised the manuscript. Majdi Halabi, MD, treated the patient and wrote the manuscript.

ETHICAL APPROVAL

Written consent for publication was obtained from the patient and is available upon request.

DATA AVAILABILITY STATEMENT

All data used during the case report are available from the corresponding author on reasonable request.

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