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The other Face of Amiodarone

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Abstract

The risk of Amiodarone-associated TdP is not common but possible and often underestimated. We present a case of a patient who had a history of AF that was converted to sinus rhythm by Amiodarone. However, the patient developed TdP few days after loading and on maintenance Amiodarone treatment. We documented a sudden significant increase in QTc interval by 400 ms within 24 hours which led to the presentation of patient to Emergency Department (ED) with pulseless polymorphic ventricular tachycardia. QTc prolongation showed a clear temporal relationship with Amiodarone dosing and disappearance (6 days after the drug was discontinued).

Introduction

Amiodarone is commonly used for the conversion and maintenance of atrial fibrillation to sinus rhythm in patients with structural heart disease. In this report, we describe a case of Amiodarone-induced polymorphic ventricular tachycardia subsequently developed in the same patient. Although that is a well-documented complication but yet still unrecognised and we hope to raise awareness to acute physicians and ED colleagues of this less common but possibly fatal side effect of this drug.

Case presentation

A 75 year old female who is usually fit and well presented on the 2nd of January to the Emergency Department (ED) with history of intermittent palpitations and shortness of breath, she denied chest pain or syncope. Found to be in Atrial Fibrillation (AF) with rapid ventricular response, admission ECG figure 1 (AF, rate 168, QTc 423,T wave inversion V4-6) as she presented with few hours of the onset of symptoms she was given Amiodarone for chemical cardioversion with excellent results were she was cardioverted to normal sinus rhythm. Bloods were unremarkable with normal electrolytes, Thyroid function test, renal, liver profile and full blood count, the only abnormality was slight raised troponin of 113 (normal range < 40) which attributed to AF with rapid ventricular response in view of absence of chest pain and absence of regional wall motion abnormality on Transoesophageal Echocardiogram (TEE) which showed normal left ventricle, mild Aortic regurgitation and slightly dilated left atrium of 4.5cm. She was discharged home on Amiodarone and anticoagulated with DOAC with a follow up in cardiology clinic.

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She presented with similar episode of shortness of breath and palpitations which resolved spontaneously upon arrival to ED on the 14th of January, denied chest pain or syncope and did not show signs of infection, fluid over load or dehydration on examination. ECG-Figure 2 showed 1st Degree HB, rate 65, QTc 483 and static T wave inversion. Troponin, inflammatory markers and electrolytes were normal.

In view of resolution of symptoms, lack of red flag symptoms in history like presyncope or chest pain, haemodynamic stability with normal blood pressure, rate controlled AF and no oxygen requirement and recent discharge from cardiology after thorough investigations, the patient was discharged home after a stat dose of furosemide as impression was diastolic dysfunction precipitated by an attack of Paroxysmal AF with rapid ventricular response. A tape was arranged and discharged on furosemide and a copy of the discharge letter sent to cardiology department.

Unfortunately the patient presented 24 hours later after developing a pulseless Ventricular Tachycardia (VT), which responded to one shock by Ambulance crew on arrival with return of spontaneous circulation. ECG-Figure 3 showed sinus bradycardia rate 51 and QTc 632.

The patient recovered in Coronary care unit with no further events, Amiodarone was stopped and ECG –Figure 4 on the 20th of January showed sinus bradycardia, QTC 486.

Patient was discharged on Bisoprolol and impression was felt to be polymorphic VT secondary to Amiodarone induced prolonged VT.

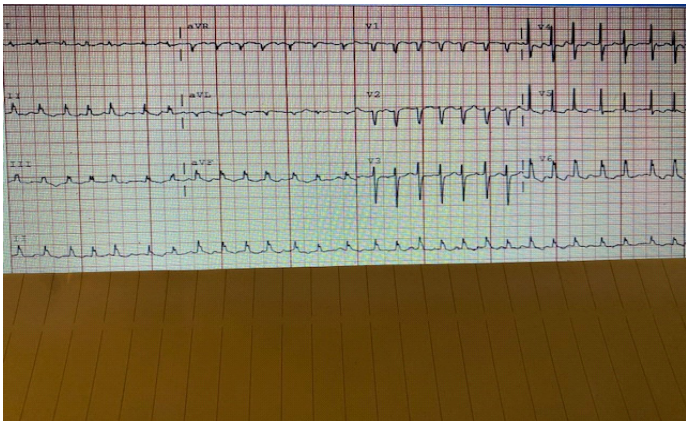

Figure 1: ECG

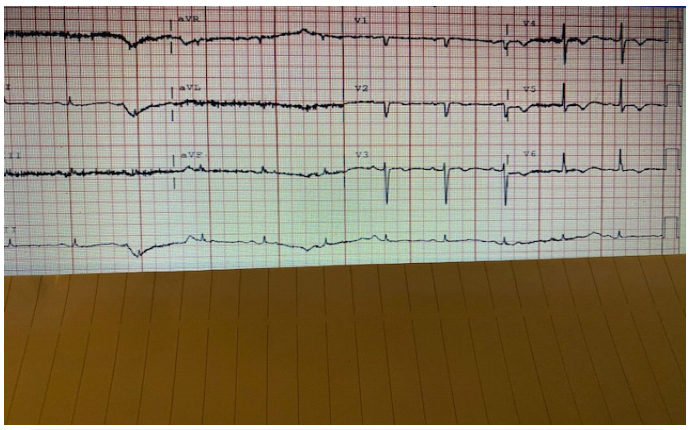

Figure 2: ECG

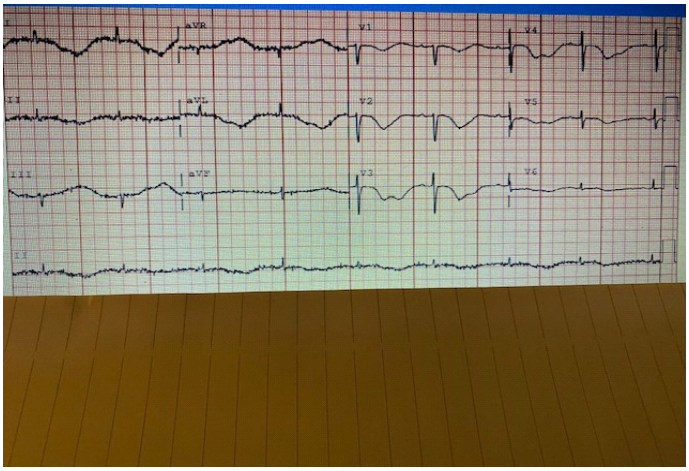

Figure 3: ECG

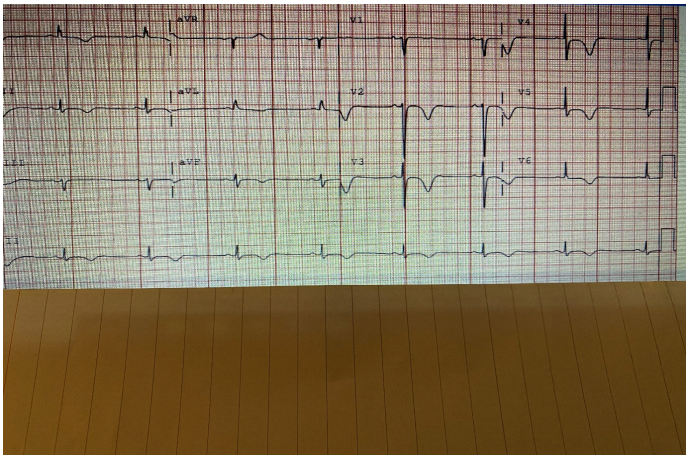

Figure 4: ECG

Case discussion

AF is common, incidence increase with age and can reach up to 10 % for patients over 80 years [1].

Amiodarone is commonly used in paroxysmal AF as it helps to restore and maintain sinus rhythm [2,3].

Amiodarone has effect on depressing sinus node and the AV node and prolongs the QT interval which can potentially cause symptomatic bradycardia and proarrhythmia [4,5].

Non cardiac side effects of Amiodarone which require observation are liver, thyroid function abnormality with more serious effects on lungs which can occur few months after initiation of medication [6]. The usual practice after starting patients on Amiodarone is to monitor Liver and thyroid function and consider chest X-ray +/- pulmonary function tests if respiratory symptoms occur.

Our patient was discharged on the usual oral maintenance dose of Amiodarone after having an intravenous loading dose in hospital which successfully reverted AF to sinus rhythm. However, in our case it has caused serious ventricular arrhythmias after ten days only of starting treatment.

No other anti-arrhythmic drugs or antibiotics were used concurrently in our patient. The most likely explanation of the TdP seen in our patient was the prolongation of the QT interval with Amiodarone which occurred 24 hours from a documented ECG with QTc of 483 to 623. QTc gradually reversed to more normal values after the drug was discontinued.

Learning point

Frequent monitoring with serial ECG for the potential serious cardiac side effects of Amiodarone

References

1. Roy D, Talajic M, Thibault B, Dubuc M, Nattel S, et al. Pilot study and protocol of the Canadian Trial of Atrial Fibrillation (CTAF). The American journal of cardiology. 1997; 80: 464-468.
2. Torres V, Tepper D, Flowers D, Wynn J, Lam S, et al. QT prolongation and the antiarrhythmic efficacy of amiodarone. Journal of the American College of Cardiology. 1986; 7: 142-147.
3. Patterson E, Eller BT, Abrams GD, Vasiliades J, Lucchesi BR et al. Ventricular fibrillation in a conscious canine preparation of sudden coronary death-prevention by short-and long-term amiodarone administration. Circulation. 1983; 68: 857-864.
4. Touboul PA, Atallah GE, Gressard AL, Kirkorian GI. Effects of amiodarone on sinus node in man. Heart. 1979; 42: 573-578.
5. Neuss HF, Nowak FG, Schlepper M, Wüsten B. The effects of anti-anginal drugs on AV-conduction in normal subjects. Arzneimittel-Forschung. 1974; 24: 213-216.
6. Hohnloser SH, Klingenheben T, Singh BN. Amiodarone-associated proarrhythmic effects: A review with special reference to torsade de pointes tachycardia. Annals of internal medicine. 1994; 121: 529-535.