Amiodarone induced electrical storm: a nightmare in emergency room

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Abstract

We present a case of an iatrogenic electrical storm with atrial fibrillation in whom amiodarone was administered to attempt chemical cardioversion, resulting in an unnoticed prolongation of the QT interval, with subsequent repeated polymorphic ventricular tachycardia, managed with isoproterenol. Concomitant drugs and slight electrolyte disturbances potentiated this phenomenon.

Given the widespread use of this drug in the emergency department, our case highlights a pertinent matter for all medical emergency practitioners. In addition, it stresses the significance of potential precipitating factors, such as electrolyte imbalances, which are clinical conditions very frequent in the emergency context, along with the importance of recognizing drug interactions. Finally, this case also emphasizes the vital importance of closely monitoring the patient’s receiving amiodarone.

Categories: Internal Medicine, Cardiology, Emergency Medicine
Keywords: drug-induced long qt syndrome, iatrogenic event, torsade des pointes, electrical storm, side effects of amiodarone

Introduction

Amiodarone is one of the most widely used antiarrhythmic drugs in the emergency room due to its perceived safety and effectiveness in managing a broad range of arrhythmias, namely for atrial fibrillation [1]. Nevertheless, it is crucial to acknowledge its potential proarrhythmic effects, which are often underestimated as they are less common compared with other antiarrhythmic drugs [2-4]. Although rare, amiodarone-induced QT prolongation can occur and degenerate in polymorphic ventricular tachycardia, also known as torsade de pointes (Tds), a potentially lethal complication [1-3,5,6].

Atrial fibrillation is the most prevalent arrhythmia in adults [1], and its high incidence in emergency departments requires not only cardiologists but also internal medicine and intensive care clinicians to master its treatment in the acute context.

Case Presentation
A 49-year-old male with alcoholic cirrhosis Child-Pug B under tiapride (100mg/day) and oxazepam (15mg/day) for alcohol abuse was admitted to the emergency department in haemorrhagic shock due to bleeding from esophageal varices. Endoscopic treatment was performed, and an infusion of proton pump inhibitors (PPIs) was initiated. Blood transfusion and fluid therapy were given for volume replacement. Due to periods of rapid new-onset atrial fibrillation (AF), intravenous amiodarone was started. Then, he presented with frequent non-sustained polymorphic ventricular tachycardia. An infusion of magnesium was administered, and the amiodarone was maintained (total dose of 2100mg in 36h).

Given the presence of AF and repolarization changes (T-wave flattening), a significant corrected QT interval (QTc) prolongation was not noticed (Figure 1). Later, he developed recurrent sustained polymorphic ventricular tachycardia with cardiac arrest requiring defibrillation several times. At this point, his EKG showed sinus rhythm and revealed a massive QTc prolongation with > 700ms with T-wave alternans (Figure 2). Blood tests (see Table 1) disclosed normal renal function with mild hypokalemia (K+ 3.2mmol/L) and hypomagnesemia (Mg2+ 0.7mmol/L). After multiple defibrillations, the transthoracic echocardiography exhibited moderate global systolic dysfunction of the left ventricle.

Iatrogenic QTc prolongation due to the use of amiodarone and tiapride with concomitant electrolytic disturbances was assumed. Amiodarone was stopped, electrolytic disturbances were promptly corrected, and isoproterenol with a target heart rate of 100 bpm was initiated. Ventricular dysrhythmia terminated, and QTc progressively normalized (Figure 3).

The patient was admitted to the intensive care unit for monitoring and further stabilization. However, a few days later, he developed severe aspiration pneumonia complicated by septic shock and died after ineffective resuscitation measures with broad-spectrum antibiotics and fluid therapy.
Discussion

The long QT syndrome is characterized by prolongation of the QT interval and by the occurrence of life-threatening tachyarrhythmias. It can be congenital or acquired [5-8]. Amiodarone is a class III antiarrhythmic agent effective in treating atrial and ventricular arrhythmias. It benefits patients with known or suspected structural heart disease [5,6]. Although rare, amiodarone-induced polymorphic ventricular tachycardia is a potentially fatal complication [1-4,8-10]. It acts mainly by blocking the potassium channels, which results in the prolongation of myocardial repolarization, represented by prolongation of the QT interval, particularly in the setting of predisposing conditions such as left ventricular hypertrophy, bradycardia, hypokalemia, and hypomagnesemia [2,3]. Therefore, we hypothesized that the use of intravenous amiodarone and the presence of multiple precipitating risk factors (such as hypokalaemia and hypomagnesemia) put our patient at increased risk of developing prolonged QT and, subsequently, potentially life-threatening arrhythmic storm.
Despite the need to monitor the QTc interval, a careful analysis in the emergency department is often tricky. In addition, the QT interval measured on the EKG varies with the RR interval, which makes the calculation of the QTc in AF challenging [11]. In our case, AF plus repolarization changes hindered the recognition of a significant prolongation of the QTc interval.

The coadministration of amiodarone and tiapride may also have contributed to prolonged QT and the further development of an electrical storm [12].

Traditionally, the treatment for an acquired QT long syndrome is to correct the cause, and in some cases, transvenous pacing can be used [5,13]. Notably, current European guidelines included isoproterenol for managing acquired LQT syndrome and recurrent dysrhythmias despite the correction of precipitating conditions [5]. It is known that amiodarone exerts its electrophysiologic effects by prolonging the action potential duration in cardiac cells [causing rectifier potassium channel (Ikr) block] and, consequently, lengthening the effective refractory period and repolarization, manifesting as a prolongation of the QT interval on the surface EKG. Isoproterenol induces adrenergic stimulation, increasing the expression of IKs, which shortens the QTc interval [13-15]. It is a less invasive alternative to temporary pacing overdrive in the prevention of ventricular dysrhythmias [5,13-15].

**Conclusions**

Finally, we present a case of an uncommon acquired arrhythmic storm form due to amiodarone - a drug widely used in the emergency department. This case highlights the importance of being alert to the arrhythmogenic potential of this drug, which should not be underestimated. Additionally, it stresses the potential risk factors that should be promptly identified and treated. Careful electrocardiographic monitoring in patients receiving amiodarone is crucial to avoid such complications. Thus, our clinical case stands for a reminder to a broad spectrum of physicians.

**Additional Information**

**Author Contributions**

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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**Disclosures**

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