

Amiodarone-Induced Electrical Storm: A Nightmare in the Emergency Room

Andreia Campinas ¹, Mariana Pereira Santos ¹, Maria João Sousa ¹, Catarina Gomes ¹, Severo Torres ¹

¹. Cardiology, Centro Hospitalar Universitário de Santo António, Porto, PRT

Corresponding author: Andreia Campinas, andrea.campinas23@hotmail.com

Review began 11/18/2023

Review ended 11/21/2023

Published 11/27/2023

© Copyright 2023

Campinas et al. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Abstract

Drug-induced long QT syndrome (LQTS) is defined as prolonged corrected QT interval (QTc ≥ 460 ms) plus polymorphic ventricular arrhythmia fitting the description of torsades de pointes temporally associated with the administration of a drug or combination of drugs. Amiodarone therapy is a known uncommon cause of acquired QT interval prolongation that should not be underestimated. We present a case of an iatrogenic electrical storm with atrial fibrillation (AF) in which 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. Additionally, 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 because of its perceived safety and effectiveness in managing a broad range of arrhythmias, namely, for atrial fibrillation (AF) [1]. Nevertheless, it is crucial to acknowledge its potential proarrhythmic effects, which are often underestimated as it is less common compared to other antiarrhythmic drugs [2-4]. Although rare, amiodarone-induced QT prolongation can occur and degenerate into polymorphic ventricular tachycardia, also known as torsade de pointes, a potentially lethal complication [1-3,5,6].

Case Presentation

A 49-year-old male with alcoholic cirrhosis Child-Pugh B under tiapride (100 mg/day) and oxazepam (15 mg/day) for alcohol withdrawal syndrome was admitted to the emergency department in hemorrhagic shock because of bleeding from esophageal varices. Endoscopic treatment was performed, and an infusion of proton pump inhibitors (PPIs) was initiated. Moreover, blood transfusion and fluid therapy were provided for volume replacement (hemoglobin of 6 g/dL). Because of periods of rapid new-onset AF, intravenous amiodarone was started. He then presented with frequent non-sustained polymorphic ventricular tachycardia (Figure 1). An infusion of magnesium was administered, and the amiodarone was maintained (total dose of 2,100 mg in 36 h).

How to cite this article

Campinas A, Pereira Santos M, Sousa M, et al. (November 27, 2023) Amiodarone-Induced Electrical Storm: A Nightmare in the Emergency Room. Cureus 15(11): e49494. DOI 10.7759/cureus.49494

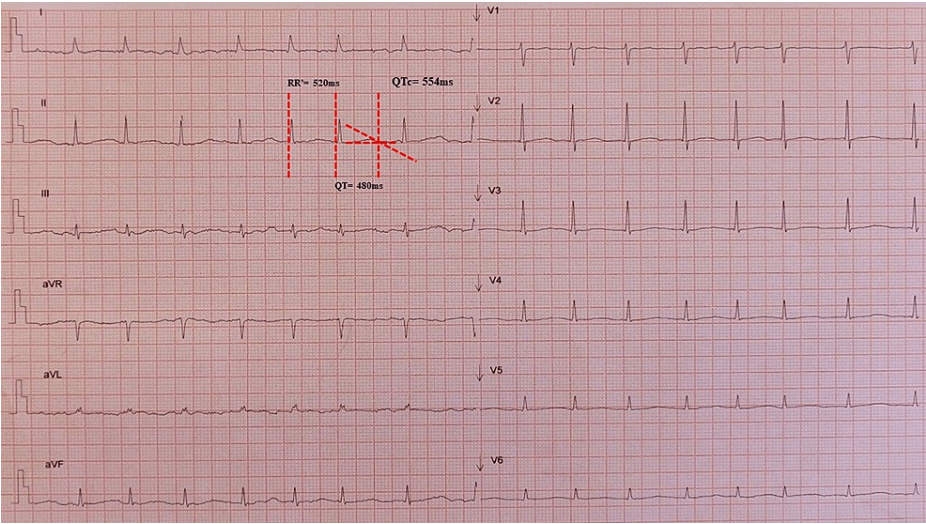


FIGURE 1: EKG performed between episodes of non-sustained polymorphic ventricular tachycardia.

Atrial fibrillation with controlled ventricular rate, flattened T waves, and prolonged corrected QT interval (QTc) of 554 ms.

Given the presence of AF and repolarization changes (T-wave flattening), a significant 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 >700 ms with T-wave alternans (Figure 2). Blood tests (see Table 1) disclosed normal renal function with mild hypokalemia (K+ 3.2 mmol/L) and hypomagnesemia (Mg2+ 0.9 mmol/L). After multiple defibrillations, the transthoracic echocardiography (TTE) exhibited moderate global systolic dysfunction of the left ventricle.

	Result	Reference range
Creatinine mg/dL	0.9	0.70-1.20
Urea mg/dL	50	10-50
Potassium mmol/L	3.2	3.50-5.30
Magnesium mmol/L	0.7	0.74-1.07

TABLE 1: Blood analysis results.

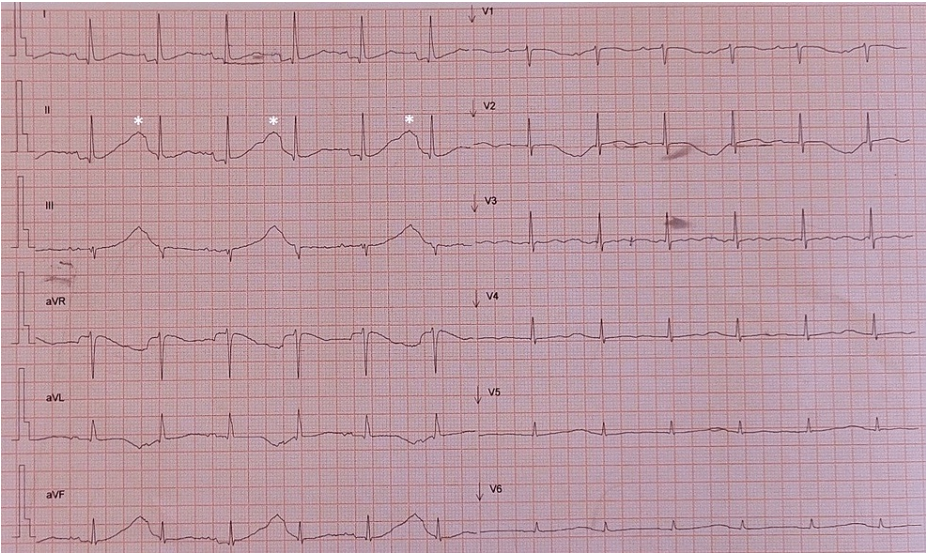


FIGURE 2: EKG performed between episodes of sustained polymorphic ventricular tachycardia.

Sinus rhythm, note the extreme QTc interval prolongation (more than 700 ms) and the presence of T-wave alternans (white stars).

Iatrogenic QTc prolongation because of 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).

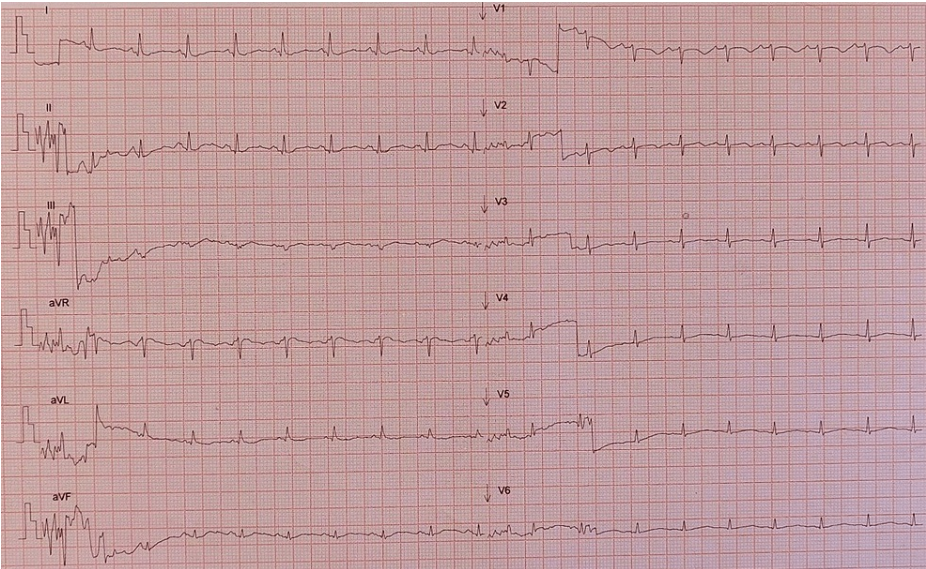


FIGURE 3: EKG showing improved QTc.

Corrected QTc.

The patient was admitted to the intensive care unit for monitoring and further stabilization. Three days later, the TTE was repeated, which showed preserved left ventricular systolic function. 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

Long QT syndrome (LQTS) is characterized by the prolongation of the QT interval and by the occurrence of

life-threatening tachyarrhythmias. It can be congenital or acquired [5-8]. According to the current guidelines, it is defined as a QTc ≥ 480 ms or a Schwartz risk score ≥ 3 for clinical diagnosis. In the presence of arrhythmic syncope or cardiac arrest, a QTc ≥ 460 ms is sufficient to consider a diagnosis of LQTS, as it is the presence of a pathogenic mutation in the genes associated with LQTS in asymptomatic patients with a normal QTc. The Schwartz score includes electrocardiographic findings (e.g., duration of the QTc interval, torsade de points, T-wave alternants, notched T-wave in three leads, and a low heart rate for age), clinical history (syncope or congenital deafness), and familial history [5,6]. Our patient met the criteria for LQTS owing to the QTc interval duration, the presence of T-wave alternants (white stars), and episodes of torsade de points.

Amiodarone is a class III antiarrhythmic agent effective in treating atrial and ventricular arrhythmias, which benefits patients with known or suspected structural heart disease [5,6]. It acts mainly by blocking the potassium channels, resulting 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]. Although rare, amiodarone-induced polymorphic ventricular tachycardia is a potentially fatal complication [1-4,7-9]. Therefore, we hypothesized that the use of intravenous amiodarone and the presence of multiple precipitating risk factors (e.g., 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. Additionally, the QT interval measured on the EKG varies with the RR interval, making the calculation of the QTc in AF challenging [10]. In our case, AF plus repolarization changes hindered the recognition of a significant prolongation of the QTc interval.

Tiapride, an atypical neuroleptic agent, is a selective dopamine D2-receptor antagonist. It shares the ability as the amiodarone to block the rectifier potassium channel (I_{Kr}), resulting in delayed repolarization of the cardiac action potential and prolongation of the QT interval. Thus, the coadministration of amiodarone and tiapride may also have contributed to prolonged QT and further development of an electrical storm [11].

Traditionally, the treatment for an acquired LQTS syndrome is to correct the cause, and in some cases, transvenous pacing can be used [5,12]. Notably, current European guidelines included isoproterenol for managing acquired LQTS 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 I_{Kr} 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 I_{Ks}, which shortens the QTc interval [12-14]. It is a less-invasive alternative to temporary pacing overdrive in the prevention of ventricular dysrhythmias [5,12-14].

Conclusions

Thus, our clinical case, underscored by its unique complexity and nuanced challenges, serves as a poignant reminder to a wide range of medical practitioners across diverse specialties, urging them to remain vigilant, adaptive, and collaborative in their approach to patient care.

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.

Concept and design: Andreia Campinas, Mariana Pereira Santos, Maria João Sousa, Catarina Gomes, Severo Torres

Acquisition, analysis, or interpretation of data: Andreia Campinas, Maria João Sousa, Catarina Gomes, Severo Torres

Drafting of the manuscript: Andreia Campinas, Mariana Pereira Santos, Maria João Sousa, Catarina Gomes, Severo Torres

Critical review of the manuscript for important intellectual content: Andreia Campinas, Mariana Pereira Santos, Maria João Sousa, Catarina Gomes, Severo Torres

Supervision: Severo Torres

Disclosures

Human subjects: All authors have confirmed that this study did not involve human participants or tissue.

Conflicts of interest: In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Hindricks G, Potpara T, Dagres N, et al.: 2020 ESC Guidelines for the diagnosis and management of atrial fibrillation developed in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS): The Task Force for the diagnosis and management of atrial fibrillation of the European Society of Cardiology (ESC) Developed with the special contribution of the European Heart Rhythm Association (EHRA) of the ESC. *Eur Heart J*. 2021, 42:373-498. [10.1093/eurheartj/ehaa612](https://doi.org/10.1093/eurheartj/ehaa612)
2. Sclarovsky S, Lewin RF, Kracoff O, Strasberg B, Arditti A, Agmon J: Amiodarone-induced polymorphous ventricular tachycardia. *Am Heart J*. 1983, 105:6-12. [10.1016/0002-8703\(83\)90270-3](https://doi.org/10.1016/0002-8703(83)90270-3)
3. Nkomo VT, Shen WK: Amiodarone-induced long QT and polymorphic ventricular tachycardia. *Am J Emerg Med*. 2001, 19:246-8. [10.1053/ajem.2001.22655](https://doi.org/10.1053/ajem.2001.22655)
4. Moro C, Romero J, Corres Peiretti MA: Amiodarone and hypokalemia. A dangerous combination. *Int J Cardiol*. 1986, 13:365-8. [10.1016/0167-5273\(86\)90121-X](https://doi.org/10.1016/0167-5273(86)90121-X)
5. Zeppenfeld K, Tfelt-Hansen J, de Riva M, et al.: 2022 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death. *Eur Heart J*. 2022, 43:3997-4126. [10.1093/eurheartj/ehac262](https://doi.org/10.1093/eurheartj/ehac262)
6. Al-Khatib SM, Stevenson WG, Ackerman MJ, et al.: 2017 AHA/ACC/HRS guideline for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. *Circulation*. 2018, 138:e272-391. [10.1161/CIR.0000000000000549](https://doi.org/10.1161/CIR.0000000000000549)
7. Jackman WM, Friday KJ, Anderson JL, Aliot EM, Clark M, Lazzara R: The long QT syndromes: a critical review, new clinical observations and a unifying hypothesis. *Prog Cardiovasc Dis*. 1988, 31:115-72. [10.1016/0033-0620\(88\)90014-X](https://doi.org/10.1016/0033-0620(88)90014-X)
8. Jurado Román A, Rubio Alonso B, Martín Asenjo R, Salguero Bodes R, López Gil M, Arribas Ynsaurriaga F: Proarrhythmic potential of amiodarone: an underestimated risk? *Rev Esp Cardiol (Engl Ed)*. 2012, 65:292-4. [10.1016/j.rec.2011.05.018](https://doi.org/10.1016/j.rec.2011.05.018)
9. Nordkin I, Levinas T, Rosenfeld I, Halabi M: Torsades de pointes after prolonged intravenous amiodarone therapy for atrial fibrillation. *Clin Case Rep*. 2021, 9:391-4. [10.1002/ccr3.3539](https://doi.org/10.1002/ccr3.3539)
10. Dash A, Torado C, Paw N, Fan D, Pezeshkian N, Srivatsa U: QT correction in atrial fibrillation - measurement revisited. *J Electrocardiol*. 2019, 56:70-6. [10.1016/j.jelectrocard.2019.06.009](https://doi.org/10.1016/j.jelectrocard.2019.06.009)
11. Iglesias E, Esteban E, Zabala S, Gascon A: Tiapride-induced torsade de pointes. *Am J Med*. 2000, 109:509. [10.1016/S0002-9343\(00\)00564-7](https://doi.org/10.1016/S0002-9343(00)00564-7)
12. Khan IA: Clinical and therapeutic aspects of congenital and acquired long QT syndrome. *Am J Med*. 2002, 112:58-66. [10.1016/S0002-9343\(01\)01011-7](https://doi.org/10.1016/S0002-9343(01)01011-7)
13. Suarez K, Mack R, Hardegree EL, Chiles C, Banchs JE, Gonzalez MD: Isoproterenol suppresses recurrent torsades de pointes in a patient with long QT syndrome type 2. *HeartRhythm Case Rep*. 2018, 4:576-9. [10.1016/j.hrcr.2018.08.013](https://doi.org/10.1016/j.hrcr.2018.08.013)
14. Shimizu W: Effects of sympathetic stimulation on various repolarization indices in the congenital long QT syndrome. *Ann Noninvasive Electrocardiol*. 2002, 7:332-42. [10.1111/j.1542-474x.2002.tb00182.x](https://doi.org/10.1111/j.1542-474x.2002.tb00182.x)