Amiodarone: Adverse Effects and the Clinical Picture of Torsades de Pointes. A Narrative Review

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ABSTRACT

Introduction: Amiodarone is a Class III antiarrhythmic drug widely used in the treatment of supraventricular and ventricular arrhythmias. However, its pharmacokinetic profile is associated with a wide range of adverse effects, including QT interval prolongation, a condition that predisposes patients to the development of *Torsades de Pointes* (TdP), a potentially fatal ventricular arrhythmia.
Objective: To analyze the adverse effects associated with the use of amiodarone and their relationship to the development of TdP, highlighting risk factors and drug interactions.
Methods: A narrative review was conducted by searching databases such as PubMed, Cochrane, LILACS, and SciELO, including studies published between 2010 and 2026 in English, Spanish, and Portuguese. Observational studies, systematic reviews, and case reports were included, while studies not available via open access and those involving pediatric and animal populations were excluded.
Results: Amiodarone acts primarily by blocking HERG-type potassium channels, prolonging cardiac repolarization. Its use is associated with multiple systemic adverse effects and significant drug interactions. QT interval prolongation was identified as the main mechanism associated with the development of TdP, especially in the presence of risk factors such as electrolyte imbalances, comorbidities, and concomitant use of QT-prolonging drugs.
Conclusions: Although the incidence of amiodarone-associated TdP is low, its potential severity makes early identification of risk factors, clinical and electrocardiographic monitoring, and appropriate management of drug interactions essential in order to prevent major adverse events.

Keywords: amiodarone, torsades de pointes, QT prolongation, drug interactions.

INTRODUCTION

Amiodarone is used to treat supraventricular and ventricular arrhythmias [1,2]. Its adverse effects (AEs) include liver, thyroid, pulmonary, dermatological, ocular, neurological, and even cardiac complications [3,4,5]. Although rare, it is associated with QT interval prolongation on the electrocardiogram (EKG), causing *Torsades de Pointes* (TdP), a polymorphic ventricular arrhythmia that carries a risk of sudden cardiac death [6,7]. To prevent the potential proarrhythmic effects of amiodarone, it is necessary to monitor underlying diseases, cardiac and extracardiac adverse events, the route of administration, and the concomitant use of drugs that also prolong the QT interval [2,5,8,9].

METHODS

A search was conducted in the PubMed, Cochrane, LILACS, and SciELO databases, and studies published in English, Spanish, and Portuguese between 2010 and 2026 were included using the terms “amiodarone”, “adverse effects”, “QT prolongation”, “Torsades de Pointes”, among others, with the Boolean operators AND and OR.

Cohort studies, systematic reviews, case reports, case-control studies, narrative reviews, translational studies, and observational studies were considered; studies that were not open access, as well as those involving pediatric and animal populations, were excluded. Additionally, a manual search of scientific journals and a review of the references in relevant articles were conducted to identify additional literature.

RESULTS

1. Amiodarone

Amiodarone is a Class III antiarrhythmic drug, a derivative of iodinated benzofuran; its mechanism of action involves blocking HERG-type potassium channels (KCNH2), which are responsible for the IKr current (rapid delayed rectifying current) involved in the repolarization phase of the cardiac action potential, thereby prolonging its duration [10,11].

A clinically relevant aspect of amiodarone lies in its broad spectrum of action, as it also inhibits, to a lesser extent, cardiac Na+ (Class I) and Ca2+ (Class IV) channels and even acts as a beta-adrenergic receptor antagonist (Class II) [11,12].

Regarding its pharmacokinetics, it is highly lipophilic and is absorbed slowly, characterized by a bioavailability ranging from 30% to 80% [10], with

significant distribution in highly perfused tissues and muscle, and a tendency to accumulate in adipose tissue [12]. Additionally, it has a protein binding rate of over 95%. The onset of action varies depending on the route of administration; when administered orally, it ranges from 2 to 3 days and may be prolonged up to 3 weeks, whereas when administered intravenously (IV), it occurs within 1 to 2 hours, with a peak concentration reached after 12 to 24 hours [10,12]. It has a long half-life, ranging from 60 to 100 days [10], which can extend to 142 days during chronic treatment [13,14].

It acts as a cytochrome P450 inhibitor, which causes drug interactions with medications that rely on this system for their metabolism; such is the case with warfarin, an anticoagulant that, when administered with amiodarone, increases the risk of bleeding. It is metabolized by the CYP3A4 enzyme and produces an active metabolite, desethylamiodarone, which has an even longer half-life than the original drug, although the exact magnitude of its antiarrhythmic effect is unknown. This explains the occurrence of toxic effects following chronic use. It is excreted primarily via the bile and, to a lesser extent, via the kidneys. Therefore, no dose adjustment is required in patients with renal insufficiency [2,10,11,12,13].

The clinical use approved by the Food and Drug Administration (FDA) is for recurrent ventricular fibrillation and ventricular tachycardias in high-risk patients in whom other drugs are ineffective; although it is also used off-label for the treatment of supraventricular tachyarrhythmias, prevention of ventricular tachyarrhythmias, drug-induced arrhythmias, postoperative tachyarrhythmias, and atrial fibrillation [5,11,14,15].

2. Adverse effects of amiodarone

Although effective, amiodarone has been shown to be associated with adverse effects in 15% of patients after one year of treatment, and in up to 50% during chronic use. Doses exceeding 400 mg carry a higher risk of complications [13,16]. Continuous patient follow-up and monitoring of any adverse effects that develop are essential, as are careful management and laboratory testing. Fortunately, most adverse effects tend to improve upon discontinuation of the drug, and in 20% of cases, they are associated with a clinically relevant risk [17]. The main adverse effects are listed below.

2.1. Cardiac

Cardiac adverse effects primarily include sinus bradycardia (12%) and atrioventricular block. In cases of atrioventricular block with a risk of worsening conduction, symptoms typically resolve when the dose is reduced; if this is not the case, a pacemaker is implanted. In addition, an increase in the pacing threshold of monophasic and biphasic defibrillators of up to 62% has been reported. Hypotension is the main adverse event following IV administration of amiodarone after the first few hours; as a preventive measure, it is recommended that initial infusion rates not exceed 30 mg/min [5,12]. QT interval prolongation, defined as a value greater than 460 ms, and TdP, a potentially fatal arrhythmia with a prevalence of less than 1%, are the main focus of this manuscript and will be discussed later [18].

2.2. Thyroid

Amiodarone consists of 37.5% iodine, a dose that exceeds the maximum daily intake; it also bears a resemblance to the thyroid hormones triiodothyronine (T3) and thyroxine (T4). As a result, the prevalence of thyroid disorders is 15-20% [19]. Amiodarone-induced thyrotoxicosis (AIT) is classified into two categories. Type 1 AIT is due to increased synthesis of thyroid hormones, generally in patients with pre-existing thyroid disease, whereas Type 2 AIT occurs more frequently in patients without prior thyroid dysfunction and results from drug-induced cytotoxicity [20,21]. On the other hand, hypothyroidism and hyperthyroidism are caused by a decrease in T3 and an increase in inactive T3, effects preceded by the inhibition of the conversion of T4 to T3. Hypothyroidism (1-32%) is associated with iodine-sufficient areas, in women, and in patients with positive antiperoxidase antibodies. Hyperthyroidism (1-23%) is found in iodine-deficient areas. To prevent the patient's condition from worsening, monitoring of T3 and T4 is recommended before and 6 months after treatment with amiodarone [5,21,22].

2.3. Pulmonary

Clinically, pulmonary toxicity and pulmonary fibrosis are the most concerning adverse events due to their potentially life-threatening severity. Pulmonary toxicity, with an incidence of 4-17%, can manifest as organized pneumonia that responds favorably to corticosteroids, chronic interstitial pneumonia, pulmonary nodules, and eosinophilic pneumonia; the latter is considered the primary radiographic finding of this AEs. The progression of pulmonary toxicity can even lead to acute respiratory distress syndrome [5,10,23,24,25]. Pulmonary fibrosis is reported in 1 out of every 1,000 patients following IV administration of amiodarone and is a serious condition, as it can occur within the first 3 months after the start of amiodarone treatment. The progression of pulmonary fibrosis is alarming, as there is a risk of developing respiratory failure and, in fatal cases, death. In such cases, discontinuation of the drug should be considered [5,10,26].

2.4. Liver-related

Amiodarone can cause elevated levels of aspartate aminotransferase (AST) and alanine aminotransferase (ALT) in 25% of cases, although these levels tend to decrease when the drug is discontinued. In addition, it can cause liver damage and steatosis, as its continuous administration places stress on the endoplasmic reticulum in the liver and adipocytes, leading to lipid accumulation, reduced lipogenesis, and induced lipolysis. Hepatotoxicity manifests in 15-20% of cases in two forms: severe acute hepatitis, occurring 24 hours after IV administration of amiodarone; and chronic toxicity resulting from prolonged oral therapy, with a marked increase in AST and ALT. In severe cases with multiple comorbidities, it may even progress to liver failure. As previously mentioned, reducing or discontinuing amiodarone is the recommended course of action in these cases [5,10,27,28].

2.5. Ocular

Among the adverse effects, ocular adverse effects are recognized as a key manifestation. More than 90% of patients treated with amiodarone develop corneal

microdeposits, which constitute the primary sign of ophthalmic toxicity. In 10% of cases, this may occur alongside visual halos. Optic neuritis is considered one of the most severe complications, as it causes unilateral or bilateral vision loss, with a recurrence rate of less than 1-2%; it usually resolves upon reduction of the drug, although in a minority of these cases it may be permanent. Discontinuation of the drug should be considered [5,29,30,31].

2.6. Dermatological

At the skin level, "blue man syndrome", which, as its name suggests, is characterized by a bluish-gray discoloration of the skin, has been documented in less than 3% of cases, while the yellowish-brown variant has a prevalence of 10%. These manifestations result from the accumulation of amiodarone, and although they may cause cosmetic concerns, both are benign. The use of protective creams is recommended, and in some cases, treatment may even be suspended [5,32,33].

3. QT interval prolongation

QT prolongation is defined as a corrected QT (QTc) interval greater than 460 ms in women and 450 ms in men. The Bazett formula is the most commonly used in clinical practice to determine the QTc based on heart rate,

$$(QTc = QT/\sqrt{RR})$$

where RR is the interval between two consecutive R waves and QT is the interval between a Q wave and a T wave.

There are various causes; however, in the case of amiodarone administration, this prolonged QTc is due to the inhibition of potassium ion channels, which are responsible for cardiac repolarization. These values vary depending on the patient's age and sex. High doses of amiodarone, associated comorbidities, electrolyte imbalances, and the coadministration of other drugs that also prolong the QT interval, among other factors, tend to increase the likelihood of developing TdP. QT interval prolongation creates an electrophysiological environment that promotes the onset of polymorphic ventricular arrhythmia due to an abnormal ventricular complex that triggers it (Figure 1) [34,35,36,37].

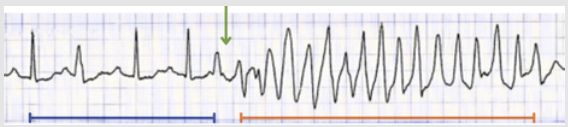


Figure 1. Transition from QT interval prolongation to the onset of Torsades de Pointes. The blue line represents the preliminary phase with QT interval prolongation. The green arrow highlights the onset of Torsades de Pointes, corresponding to the first abnormal ventricular complex. The orange line delineates the polymorphic ventricular tachycardia. *Adapted from: Lee B et al. Rare case of Torsades de Pointes in severe hypothyroidism: literature review and challenges in management. Int J Emerg Med. 2022;15(1):11.*

4. *Torsades de Pointes*

TdP is recognized as a polymorphic ventricular arrhythmia that manifests as oscillations in the amplitude of the QRS complexes around the isoelectric line, associated with QT interval prolongation (Figure 2); a QTc interval greater than 500 ms increases the risk of developing TdP. This arrhythmia may resolve or progress to ventricular fibrillation.

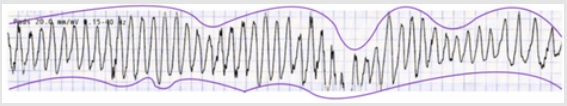


Figure 2. Characteristic twisting pattern of *Torsades de Pointes*. The upper and lower purple envelope lines highlight the tachycardia and the cyclic variation in the amplitude of the QRS complexes, creating the twisting appearance around the isoelectric line characteristic of *Torsades de Pointes* ("twisting of the tips"). *Adapted from: Lee B et al. Rare case of Torsades de Pointes in severe hypothyroidism: literature review and challenges in management. Int J Emerg Med. 2022;15(1):11.*

Fifty percent of patients who develop TdP are asymptomatic, although reported symptoms include syncope, palpitations, and dizziness. Fortunately, in the context of amiodarone therapy, the incidence is less than 1% of cases. Approximately 10% of TdP cases result in sudden cardiac death. Prevention of this arrhythmia involves correcting modifiable risk factors, such as discontinuing the coadministration of drugs that prolong the QTc interval and maintaining the patient's electrolyte balance. The primary treatment for TdP is IV magnesium, as it stabilizes the cardiomyocyte membrane and corrects hypokalemia. Because TdP is bradycardia-dependent, another option is to increase the heart rate, typically with drugs such as isoproterenol or by implanting a pacemaker [39,40,41].

5. Risk factors associated with amiodarone treatment

In light of the above, the risk factors include the following (Table 1).

Table 1. Risk factors for TdP during amiodarone therapy.

Factors	Examples
Electrocardiographic	• QTc prolongation greater than 460 in women and 450 in men • Bradycardia
Electrolyte-related	• Hypokalemia • Hypomagnesemia • Hypocalcemia
Patient related	• Patient's age (>65 years) • Female gender • Liver dysfunction • Renal dysfunction • Duration of treatment • Underlying heart conditions (congenital long QT syndrome, heart failure, left ventricular hypertrophy, acute myocardial infarction) • Obesity • Impaired hepatic metabolism
Pharmacological	• Treatment with diuretics • Concomitant use with drugs that prolong the QT interval • IV administration (15%)

Distribution of factors that contribute to the development of this polymorphic ventricular arrhythmia during amiodarone administration [5,36]. TdP = torsade de pointes; QTc = corrected QT interval; IV = intravenous.

6. Drug interactions

With regard to drug interactions, we analyzed the drugs that interact with amiodarone and verified that they are currently available in Mexico (Table 2).

Table 2. Drugs available in Mexico that are associated with an increased risk of TdP when coadministered with amiodarone.

Group	Drugs	
Antibiotics	• Erythromycin • Azithromycin • Clarithromycin • Ciprofloxacin • Trimethoprim/sulfamethoxazole	• Levofloxacin • Moxifloxacin • Gatifloxacin • Metronidazole
Antifungals	• Fluconazole • Itraconazole	• Ketoconazole
Cardiovascular	• Dronedarone • Ivabradine	• Ranolazine
Antipsychotics	• Haloperidol • Chlorpromazine • Olanzapine	• Risperidone • Aripiprazole • Clozapine
Antidepressants	• Fluoxetine • Paroxetine • Citalopram • Escitalopram	• Imipramine • Amitriptyline • Mirtazapine • Trazodone
Dementia	• Donepezil	• Galantamine
Antineoplastic	• Lapatinib • Lenvatinib • Sorafenib • Sunitinib • Osimertinib • Arsenic trioxide	• Vandetanib • Crizotinib • Dabrafenib • Dasatinib • Tamoxifen
Antimalarials	• Chloroquine • Hydroxychloroquine	
Antihistamines	• Loratadine	• Hydroxyzine
Urological	• Solifenacin • Tolterodine	• Alfuzosin
Muscle relaxants	• Cyclobenzaprine	
Inhaled anesthetics	• Isoflurane	• Sevoflurane
Migraine	• Zolmitriptan	
Antiparkinsonian	• Apomorphine	
Laxatives	• Sodium phosphate (mono/dibasic)	
Bronchodilators	• Formoterol	• Vilanterol
Antiemetics	• Ondansetron	• Granisetron
Antiretrovirals	• Efavirenz	• Atazanavir
Anti-ulcer	• Famotidine	

This table lists the main drugs that have a valid marketing authorization from COFEPRIS and increase the risk of TdP following administration of amiodarone [5,42]. TdP = torsade de pointes.

DISCUSSION

Amiodarone is a Class III antiarrhythmic drug used in the treatment of ventricular and supraventricular arrhythmias; however, its pharmacokinetic and pharmacodynamic profile is associated with a wide range of adverse effects and drug interactions. Its primary mechanism of action involves the inhibition of HERG-type potassium channels, which are responsible for the repolarization of the cardiac potential, leading to QT interval prolongation. This phenomenon, in and of itself, represents a risk factor for the development of early post-depolarizations and, in the presence of comorbidities and other predisposing factors, may contribute to the onset of TdP, a potentially fatal polymorphic ventricular arrhythmia, with an incidence of less than 1%.

Despite its low frequency, amiodarone-associated TdP has significant clinical implications, as adverse effects and drug interactions may necessitate adjusting, reducing, or discontinuing treatment. In this context, early detection via electrocardiogram, as well as the identification and correction of risk factors, are essential for preventing adverse outcomes. Furthermore, it is essential to avoid the coadministration of drugs that prolong the QT interval and to perform an individualized risk assessment for each patient.

Among the main limitations of this study are the scarcity of evidence in the Mexican population, as well as the lack of specific records on amiodarone-induced TdP in this context. Consequently, future studies should focus on developing therapeutic alternatives with an effective antiarrhythmic profile and a lower incidence of adverse effects, with the goal of reducing the risk of QT prolongation and the development of TdP [10,11,36].

CONCLUSION

Amiodarone therapy and the development of TdP pose a relatively low risk compared to other antiarrhythmic drugs in the same class; however, this risk is not negligible. Furthermore, the presence of comorbidities in the patient increases this risk. It is essential to identify risk factors early; to identify drugs that interact with amiodarone; to perform the laboratory tests deemed necessary by the cardiologist before and during administration of the drug; to identify and manage adverse events; and, if necessary, to reduce or discontinue the amiodarone dose.

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Conflict of interest

The author declares no conflicts of interest.

Ethical considerations

This study did not involve the participation of humans or animals, nor the collection of primary data; therefore, it did not require approval from a research ethics committee. All data used were obtained from previously published and publicly accessible secondary sources.

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