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Original Article

DRUG-INDUCED LONG QT: CASE REPORT

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ABSTRACT

D*rug-induced long-QT syndrome is a potentially catastrophic complication in the hospital setting. Ventricular arrhythmias such as torsade de Pointes (TdP) are more likely to develop in patients with underlying heart disease, comorbidities, electrolyte abnormalities and conduction system disease. We report a clinical case of drug-induced long QT in which polymorphic ventricular tachycardia and atrial fibrillation are triggered, causing the death to acute neurological deterioration secondary to ischemic cerebrovascular event.*

Keywords: *Ventricular Tachycardia, Drug-Induced Long QT, Atrial Fibrillation*

1. INTRODUCTION:

Drug-induced long QT refers to the prolongation of the QT interval coupled with T wave alterations, secondary to the delay in ventricular repolarization, which is associated with an increased risk of fatal arrhythmias and sudden death. Torsade de Pointes type polymorphic ventricular tachycardia (TdP) can cause sudden death in patients receiving drugs that lengthen the QT. The incidence of non-cardiac drug-induced TdP is typically low (less than 1:10,000 or 1: 100,000). Atrial fibrillation (AF) has been associated with a higher incidence in the context of long QT, an increased risk of complications such as heart failure, embolic events, and mortality. With the aforementioned, we present the case of a patient with acquired long QT interval with TdP, who after pharmacological resolution, presents an episode of AF, with repercussions at the central nervous system level, secondary to cardio embolicS phenomenon.

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2. PRESENTATION OF THE CASE

We present the case of a 76-year-old woman, with no significant medical history, who is referred from a private clinic with a diagnosis of community-acquired pneumonia, where levofloxacin 1.0 gram was administered intravenously every 24 hours. 48 hours after the start of treatment, she presented a syncopal episode, with no apparent cause. As a study protocol, a 12-lead electrocardiogram was requested, which showed a prolonged QT interval. He was admitted to the emergency medical service, where he was requested laboratory studies that reported the following, hemoglobin 9.01 g / dL, hematocrit 25.4%, glucose 84 mg / dL, urea 34 mg / dL, BUN 15.8 mg / dL, creatinine 0.7 mg / dL, sodium 147 mEq / dL, potassium 4.0 mEq / dL and chlorine 109 mEq / dL. A new electrocardiogram was requested, which confirmed prolongation of the QT interval, with QT corrected with Bazzet's formula of 610 ms (Figure 1). Later, polymorphous ventricular tachycardia type TdP is evidenced by telemetry; 2 grams of magnesium sulfate were administered intravenously, with remission of the same. During his stay in the Intensive Care Unit, he presented paroxysmal atrial fibrillation, with a CHA2DS2-VASc score of 3 points and HAS-BLED 1 point, treatment with amiodarone infusion and enoxaparin in

prophylactic doses was started, however, he presented acute neurological deterioration, secondary to ischemic-type cerebral vascular event, which conditions death.

3. DISCUSIÓN

Drug-induced long QT refers to the prolongation of the QT interval coupled with T wave alterations, secondary to the delay in ventricular repolarization, which is associated with an increased risk of fatal arrhythmias and sudden death, this variant being (acquired form), the most common in hospitalized patients.1 The most frequent clinical manifestations are syncope and lipothymia, less frequently palpitations and seizures. In the electrocardiogram, the QT measurement in DII is recommended, from the beginning of the QRS, to the end of the T wave, subsequently it must be corrected by the heart rate, the most used formula being Bazzet's, being the reference measurement in women a QTc greater than 470 ms and in men a QTC greater than 460 ms, according to the recommendations of the European Society of Cardiology.2 Regarding the probability of risk of presenting drug-induced long QT, James Tisdale et al. published and they validated the Tisdale scale, which consists of 10 variables, with different scores, which gives the probability that hospitalized patients have of manifesting this electrocardiographic alteration (Table 1) .[3]

Another aspect to consider in the genesis of this arrhythmia is the so-called "repolarization reserve", which is defined as the compensatory mechanism generated by other repolarizing currents typical of the cardiac cell, when secondary to the action of a drug, congenital alteration or acquired from a type of transmembrane ion channel can result in excessive changes in the cellular repolarization mechanism, that is, when an individual presents, for example, involvement of the IKs channel or in the calcium current, they may not show changes in QT, until block the IKr channel. In the mechanism generated by pharmacological action, it can appear in the patient with a weak repolarization reserve, which cannot compensate for the inhibition of potassium channels; in other cases, this weak reserve in the face of pharmacological inhibition of the mentioned channels is secondary to additional factors specific to the patient, such as genetic mutation or pathological electrophysiological remodeling.[4]

On the other hand, AF continues to be an important cause of ischemic cerebrovascular accidents (20 to 30%) due to cardioembolic phenomena; the electrocardiographic diagnosis is characterized by presenting an "f" wave, an irregular RR interval, with a minimum duration of 30 seconds.5 A meta-analysis carried out in 2018 that included 8 cohort studies, of which three related the presence of long QT with atrial fibrillation persistent and five with first-time diagnostic atrial fibrillation, including a total of 309,676 participants, demonstrated a significant association between the prolonged QTc interval and the risk of atrial fibrillation.6 The BEAT-AF study concluded in a patient with prolonged QT and AF , increased major adverse cardiovascular events and mortality, the QTc interval was significantly associated with an increase of 30% for heart failure, 21% for major adverse cardiovascular events and 31% for mortality from all causes.7 Finally, a study published in the 2013, performed a statistical analysis of the relationship between the long QT interval and the incidence of AF, inc with a total of 21,679 participants, concluding a higher risk of AF presentation in patients with prolonged QTc, suggesting that a longer QT interval may directly reflect a greater propensity to AF as a manifestation of parallel refractory aberrations in the atrium and the ventricle. [8]

4. CONCLUSIÓN

Drug-induced long QT is a rare entity, however, with a high probability of arrhythmogenic complications and potential risk of death. It is important to identify and correct potential risk factors associated with a prolonged QT interval in hospitalized patients to avoid catastrophic outcomes.

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