Proarrhythmic Potential of Amiodarone: An Underestimated Risk?

Potential proarrítmico de la amiodarona: ¿un riesgo subestimado?

To the Editor,

Because of its known safety and efficacy, amiodarone is a first-line drug for the treatment of atrial and ventricular arrhythmias in patients with structural heart disease. Its proarrhythmnic side effects are usually underestimated because they are less common than those experienced with other antiarrhythmics. We describe a case of corrected QT interval prolongation (QTc) and torsade de pointes (TdP) secondary to amiodarone.

A 59-year-old woman with hypertension and chronic obstructive pulmonary disease with a significant bronchospastic component was attended at home for palpitations and dizziness. The electrocardiogram (ECG) showed self-limiting, regular, wide-QRS tachycardia at a rate of 210 bpm before admission and sinus rhythm at a rate of 90 bpm with a QRS duration of 90 ms and QTc of 415 ms on hospital admission (Fig. 1A). The cardiologic study revealed dilated cardiomyopathy with moderate ventricular dysfunction, but no major coronary lesions.

On the third day of hospitalization, the patient experienced well-tolerated sustained monomorphic ventricular tachycardia (SMVT) at a rate of 270 bpm (Fig. 2A) that reverted once intravenous perfusion of amiodarone was initiated.

These recommendations do not, however, apply for fibroelastomas at atypical sites. The reasonable approach when faced with such masses is to rule out as many diagnoses as possible by noninvasive means. Gradual enhancement of magnetic resonance data may help in tumor diagnosis. Once diagnosed, surgical resection (provided the risk is not too high) can provide an indication of etiology, prognostic evaluation, and specific treatment.

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Several small series found no correlation between amiodarone-induced QTc prolongation and the onset of TdP, which indicates that it is safe even in patients with this arrhythmic complication.1

The association was also detected in experimental animal studies, from which it can be inferred that the absence of TdP with amiodarone may be related to homogeneous lengthening of the action potential duration and the absence of early afterdepolarizations.2

Although the incidence of TdP with amiodarone is low compared to other class III antiarrhythmic agents, it is known to be twice as high in women.3,4 Some published case reports have

**Figure 1.** A, baseline electrocardiogram during sinus rhythm (90 bpm, QRS 90 ms, corrected QT interval 415 ms). B, electrocardiogram during sinus rhythm 24 h post-amiodarone dosing (75 bpm, QRS 146 ms, corrected QT interval 714 ms). C, pacemaker with pacing at 100 bpm. D, electrocardiogram during sinus rhythm 1 week after amiodarone discontinuation (75 bpm, QRS 110 ms, corrected QT interval 449 ms).

**Figure 2.** A, sustained monomorphic ventricular tachycardia. B, torsade de pointes. Note the sinus beat with long corrected QT interval preceding it (arrow). C, electrical cardioversion of ventricular fibrillation.
detected other associated factors that could prolong QTc (electrolyte abnormalities, bradycardia, etc.), which could enhance the proarrhythmic effect of the drug.5 In our patient, QTc prolongation showed a clear temporal relationship with amiodarone dosing both in terms of onset (24 h afterwards) and disappearance (1 week after the drug was discontinued). Although slight hypokalemia was observed, TdP did not cease when the levels were corrected, which suggests that although the condition could have enhanced the proarrhythmic effect of the drug it was not the main cause. In conclusion, although amiodarone is considered safe for the treatment of ventricular arrhythmia, its arrhythmogenic potential should not be underestimated, particularly in women and in the presence of concomitant factors that could prolong the QTc. Careful monitoring of the QTc interval and these factors can lower the risk of proarrhythmia.

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Extreme QT Interval Prolongation and Helicoid Ventricular Tachycardia (Torsade de Pointes) in Non-ST-Elevation Acute Coronary Syndrome

Prolongación extrema del intervalo QT y taquicardia helicoidal (torsade de pointes) en el síndrome coronario agudo sin elevación del ST

To the Editor,

A prolonged corrected QT interval (cQT) during coronary ischemia is a well known sign that has even been incorporated into the parameters tested for the assessment of ischemic risk in acute coronary syndrome (ACS).1 There is also a correlation between a long QT and helicoidal ventricular tachycardia, or torsade de pointes (TdP), that has been described in this context.2 Here, we present a case of ACS with a very prolonged QT interval with giant negative T-waves and a later development of TdP.

A 79-year-old woman sought emergency treatment for diffuse pain in the anterior thorax and dyspnea with 2 days evolution. She had a background of type-2 diabetes mellitus, systemic arterial hypertension, and rheumatoid arthritis. She was under treatment with metformin, vidalgliptin, losartan, and indometacin. Upon admission to the hospital, the patient was dyspneic with a blood pressure of 219/96 mmHg, an O2 saturation of 86%, and a regular pulse at 98 bpm, with notable bilateral basal crepitation. The abdomen was without abnormalities, with intact peripheral pulse. The initial electrocardiogram (ECG) (Fig. 1A) demonstrates a sinus

Figure 1. A, test results upon admission: RR, 640 ms; corrected QT interval, 510 ms. B, test results after 24 h: RR, 780 ms; corrected QT interval, 745 ms. C, test results after 13 days: RR, 760 ms; corrected QT interval: 504 ms. (Corrected QT intervals derived using Bazett’s formula).