Left bundle branch block and severe ventricular dysfunction due to flecainide toxicity: a case report

Bloqueo de rama izquierda y disfunción ventricular grave secundarios a toxicidad por flecainida: a propósito de un caso

To the editor:

Flecainide is a class I antiarrhythmic drug belonging to subgroup IC of the Vaughan-Williams classification, which used incorrectly can lead to potentially serious adverse effects.

We present the case of a 61-year-old woman with multiple cardiovascular risk factors. In 1991 she underwent an electrophysiological study for palpitations at another center, with diagnoses of double accessory pathway with inducible isthmic flutter. None of the substrates were ablated, and treatment was started with flecainide 100 mg daily, with no subsequent follow-up.

In February 2021, she attended the emergency department of another center for the debut of atrial fibrillation (AF) and heart failure (HF), and effective pharmacological cardioversion with 300 mg of oral flecainide was started. After clinical improvement, discharge home was decided. The patient consulted our center 3 weeks later due to persistent clinical symptoms of dyspnea on minimal effort and edema in the lower limbs.

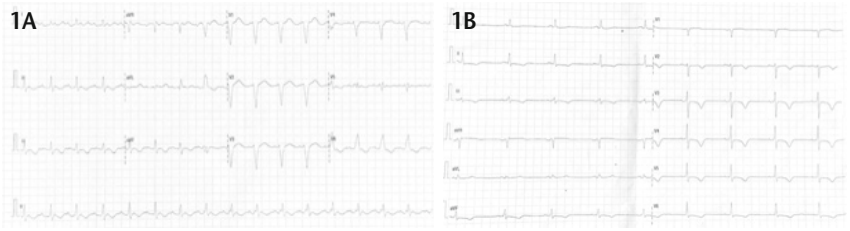

Figure 1. 1A. Ambulatory ECG of the patient on flecainide treatment. 1B. ECG at hospital discharge, after discontinuation of flecainide.

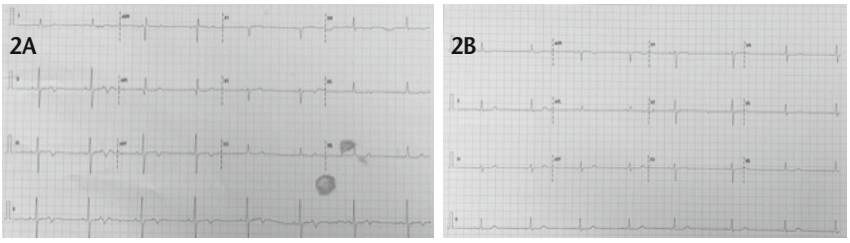

Figura 2. ECG in HF consultation 20 days (2A) and 3 months (2B) after hospital discharge and discontinuation of flecainide.

The electrocardiogram (ECG) showed sinus tachycardia with left bundle branch block (LBBB) with QRS of 150 milliseconds (ms) and normal QTc interval (Figure 1A) and a bedside echocardiogram showed severe ventricular dysfunction, indicating hospital admission to our center. Flecainide was withdrawn and intravenous diuretic treatment, HF modifying treatment, and oral anticoagulation were started. Laboratory tests showed renal and hepatic function in the normal range. A new echocardiogram showed a slightly dilated left ventricle, left ventricular ejection fraction (LVEF) of 30% and great ventricular asynchrony. The study was completed with coronary angiography which showed no significant lesions. In view of the clinical improvement, it was decided to discharge the patient home, maintaining sacubitril/valsartan 49 mg/51 mg every 12 h, furosemide 40 mg every 24 h, spironolactone 25 mg every 24 h, edoxaban 60 mg every 24 h and bisoprolol 10 mg every 24 h and referral to the HF clinic. The ECG at discharge showed sinus bradycardia at 52 bpm with narrow QRS, asymmetric and deep generalized negative T-neg with prolonged QTc (505 msges), and it was decided not to initiate class III antiarrhythmic drugs for AF rhythm control strategy (Figure 1B). After 20 days, she came for consultation, being asymptomatic and presenting partial improvement of LVEF and electrocardiographic disorders, with persistence of slightly elongated QTc (473 msecs) and asymmetrical negative T in inferior face (Figure 2A). At 3 months, LVEF normalized (57%) and all electrocardiographic disturbances (Figure 2B), with no AF paroxysms.

Flecainide decreases ionic inward currents in voltage-dependent sodium and potassium channels[1] and generates both negative chronotropic and inotropic effects. This results in conduction slowing of cardiac fibers (atrial, ventricular and conduction system) and is evidenced on the ECG as prolongation of the PR, QRS and QT intervals.[2]

Intraventricular conduction disturbances are a frequent adverse effect and their morphology is related to QRS duration, with LBBB being the most frequent with QRS > 200 msecs[3] and the one with the worst prognosis, since it is capable of inducing ventricular dysfunction.[4]

It is a drug with a narrow therapeutic margin5 and with a potential risk of causing symptoms attributable to its toxicity such as the one presented in our case. Proper patient selection, as well as close follow-up including periodic monitoring of drug levels, are essential to avoid the appearance of these problems.[5]

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References

1 Smith A, Gerasimon G. An electrocardiographic series of flecainide toxicity. Indian Pacing Electrophysiol J. 2019;19:75-8.
2 Puga M, Gallardo-Medina Y, Castro-Hevia J, Quirós-Luis J. Toxicidad por flecainida: aparición de signo de Brugada y bloqueo de rama izquierda. Revista Cubana de Cardiología y Cirugía Cardiovascular [Internet]. 2018 [citado 17 Jul 2022]; 24 (1): [aprox. 5 p.]. Disponible en: http://www.revcardiologia.sld.cu/index.php/revcardiologia/article/view/725
3 Ghataoura R, Patil S. Flecainide toxicity: a presentation to the emergency department with literature review. BMJ Case Rep. 2020;13:e232691.
4 Valentino MA, Panakos A, Ragupathi L, Williams J, Behzad BP. Flecainide toxicity: a case report and systematic review of its electrocardiographic patterns and management. Cardiovasc Toxicol. 2017;17:260-6.
5 Tamargo J, Le Heuzey JY, Mabo P. Narrow therapeutic index drugs: a clinical pharmacological consideration to flecainide. Eur J Clin Pharmacol. 2015;71:549-67.