

Worsening Conduction Delay in Hypertrophic Cardiomyopathy

What Drug Is Responsible?

ECG CHALLENGE

We present the case of a 64-year-old male who presented to the emergency room because of recent onset palpitations. He was diagnosed with a first episode of atrial fibrillation and was managed at the emergency room by direct current cardioversion. The patient's past medical record was unremarkable, and no relevant findings were discovered during the physical examination or laboratory tests. The baseline ECG is shown in Figure 1. On discharge the patient was referred for outpatient cardiology evaluation and was eventually seen 2 months later. The ECG at the outpatient clinic is shown in Figure 2.

Which drug was most likely prescribed to the patient to account for the differences between the first and second ECGs?

Please turn the page to read the diagnosis.

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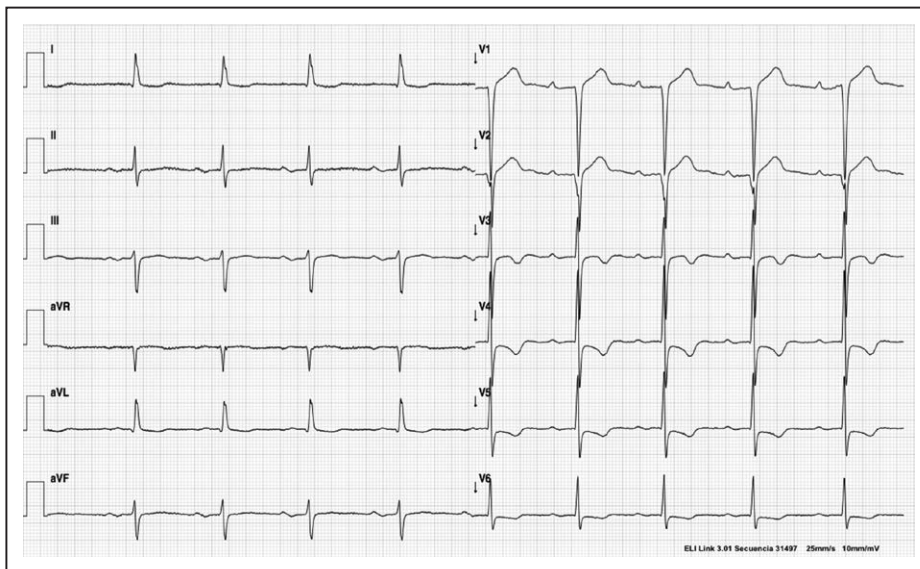


Figure 1. Baseline ECG.

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RESPONSE TO ECG CHALLENGE

The ECG shows an intraventricular conduction delay in the context of hypertrophic cardiomyopathy, which is worsened by propafenone administration.

Baseline ECG (Figure 3A) shows sinus rhythm with first atrioventricular block; the PR interval is 0.280 seconds long. The QRS complex is wide (0.110 seconds), showing a nonspecific conduction delay. An R wave is absent on leads V1 and V2 (QS morphology) with a notch in the descending part of the Q wave in lead V2. Small Q waves also appear in leads I, and aVL and frontal QRS axis is at -20° . Alterations of the repolarization were also present, such as asymmetrical and negative T waves in lateral leads (V3–V6, I, and aVL). Compared with baseline ECG, the one on propafenone (Figure 3B) shows conduction intervals increase by $\approx 30\%$ (PR 360 msec and QRS 140 msec). The patient had been on a daily dose of 450 mg (150 mg/8 hours) of propafenone; when it was discontinued, the ECG returned to baseline. Propafenone was initiated at the emergency department and was continued in the presence of good clinical tolerance and maintenance of sinus rhythm. Later on, ECG and cardiac magnetic resonance (Figure 4) confirmed the structural diagnosis, showing asymmetrical hypertrophy of the ventricular walls, a maximum thickness of 23 mm at the mid-septum, and disorganized fibrosis along the septum.

Proarrhythmic properties of antiarrhythmic drugs have been known for years. Some antiarrhythmics have

the ability to slow electric conduction through the myocardium, which is the mechanism proposed to cause sudden cardiac arrhythmic death reported in selected patients taking antiarrhythmics (IC) agents. This is usually observed in the presence of structural abnormalities predisposing to reentry phenomena.¹ In addition, IC antiarrhythmic drugs are the most powerful sodium channel blockers. As a consequence, they produce the strongest myocardial conduction delay and therefore prolongation in conduction intervals.²

The prolongation of conduction intervals is dose-dependent, and it can reach up to 20% of baseline duration at maximum doses of propafenone.³ Higher prolongations should worry clinicians because it can reflect a risk of drug toxicity and proarrhythmic effects. Dose reduction may be sufficient to deal with mild prolongations, often observed in the face of poor metabolizers or drug interactions at the cytochrome P450 pathway. However, in our case, absolute discontinuation was mandatory as soon as hypertrophic cardiomyopathy was diagnosed. Other side effects of propafenone, such as dizziness or metallic taste, may be tolerable, but some patients may experience hypotension or ventricular dysfunction, which can be especially harmful in hypertrophic cardiomyopathy.

In summary, we present a case of a worsening conduction delay because of propafenone in the context of hypertrophic cardiomyopathy. Intraventricular conduction delay is frequently observed with IC agent therapy. However, excessive QRS prolonga-

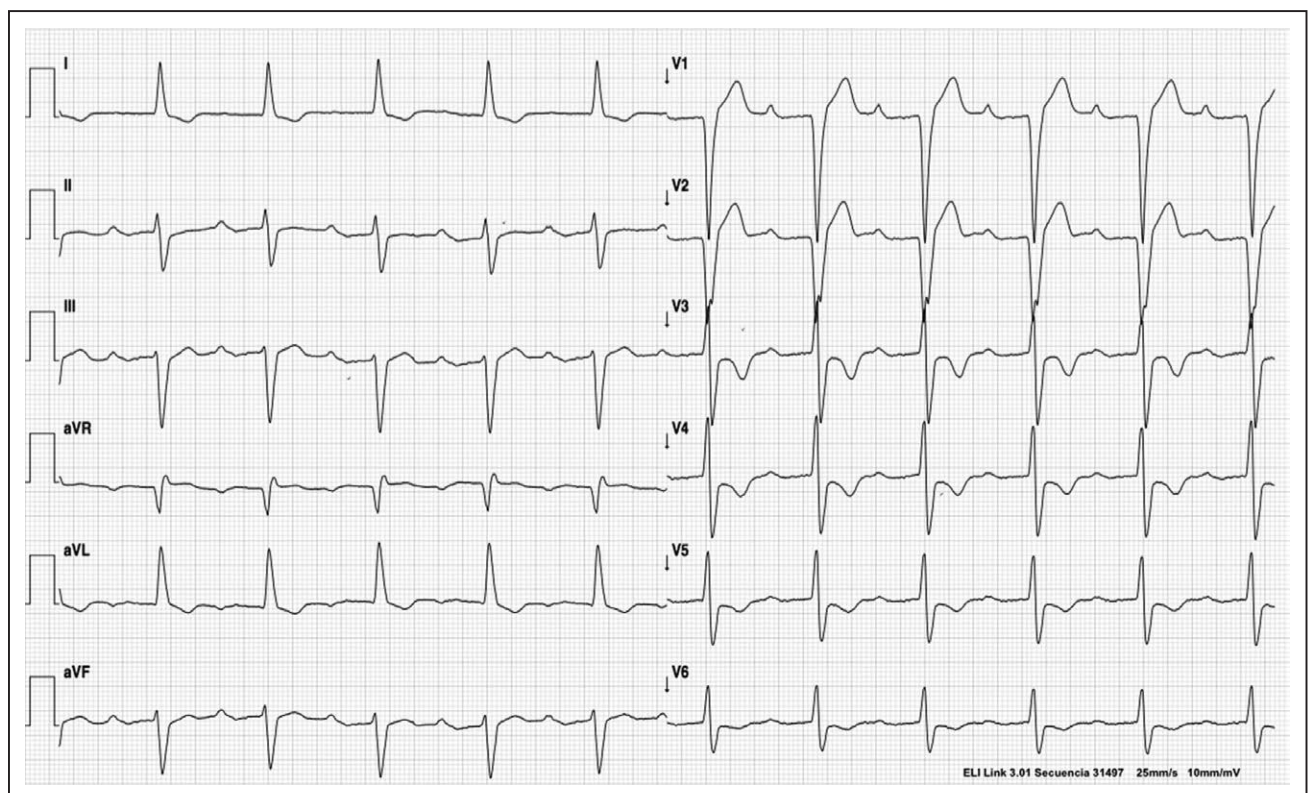


Figure 2. ECG at the cardiology outpatient clinic.

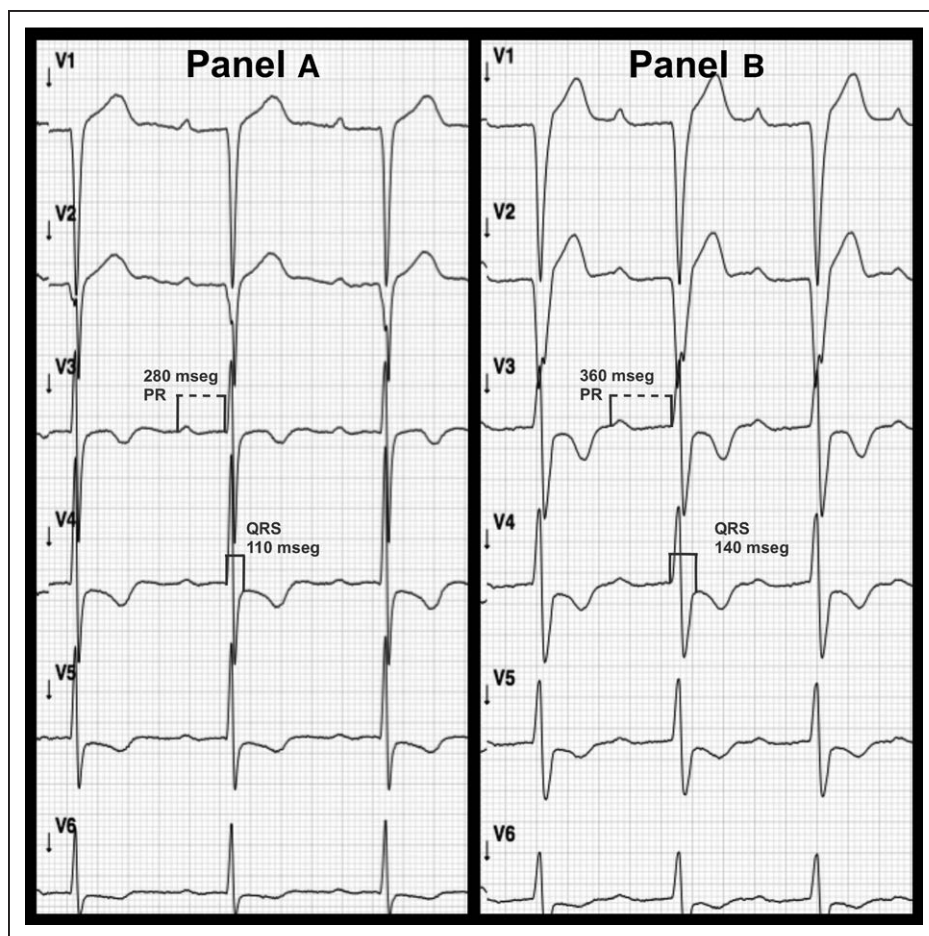


Figure 3. ECG comparison. **A**, Baseline ECG shows sinus rhythm with first atrioventricular block, PR 0.280 seconds. QRS is wide (0.110 seconds) with a nonspecific conduction delay and asymmetrical and negative T waves. **B**, ECG under propafenone treatment. PR and QRS intervals are significantly prolonged compared with baseline (360 msec and 140 msec, respectively).

tions should warn about potential drug toxicity, and dosage should be adjusted. Structural heart disease is a general contraindication for IC antiarrhythmics

because of its intrinsic predisposition to ventricular arrhythmias.



Figure 4. Cardiac magnetic resonance in a 4-chamber view. There is an asymmetrical hypertrophy at the level of basal to mid-septum.

DISCLOSURES

None.

AFFILIATIONS

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FOOTNOTES

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