Negative Inotropy from Flecainide Toxicity

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Abstract

Flecainide is a potent sodium channel inhibitor that slows phase 0 of the cardiac action potential. Rapid heart rates cause greater drug binding to the sodium channel, a phenomenon termed use dependence. High concentrations of bound drug result in adverse effects due to the physiological effects of sodium channel blockade, including conduction velocity slowing and negative inotropy. The negative inotropic effect of flecainide toxicity, which can cause or exacerbate heart failure, is an underappreciated phenomenon. We report a case of flecainide toxicity where the negative inotropic effects of flecainide were demonstrated echocardiographically. Given the frequency with which flecainide is utilized, it is important for all healthcare providers to recognize this potentially life-threatening consequence of flecainide toxicity.

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Key Words

Flecainide toxicity • Negative inotropy • Heart failure

Introduction

A 78-year-old man with paroxysmal atrial fibrillation that was well-controlled for 2 years on flecainide was admitted with severe dyspnea and fatigue. He had a baseline normal left ventricular ejection fraction (LVEF), no known coronary artery disease, and no prior adverse events related to flecainide. The electrocardiogram on admission revealed atrial fibrillation with rapid ventricular rates and left bundle branch block

Video 1. Comparison of left ventricular function by echocardiography. Two transthoracic echocardiogram clips are shown side by side for comparison. On the left is a parasternal long axis view recorded in atrial fibrillation with left bundle branch block (LBBB) aberration in the setting of flecainide toxicity at which time the left ventricular ejection fraction (LVEF) was estimated to be 20%. On the right is a parasternal long axis view recorded after the patient had been cardioverted to sinus rhythm with resolution of at which time the LVEF was estimated to be 50%. CV=cardioversion, RV = right ventricle, LV=left ventricle, LA=left atrium, Ao=aorta, LVEF=left ventricular ejection fraction, LBBB=left bundle branch block.

(LBBB) with a QRS duration of 150 ms (Figure 1). The transthoracic echocardiogram while in this rhythm revealed an LVEF of 20% with global hypokinesis (Video 1, left panel). The patient was electrically cardioverted to sinus rhythm, and flecainide was discontinued. A repeat electrocardiogram revealed sinus rhythm at 62 beats per minute with resolution of LBBB (Figure 2). T wave inversions in leads V1–V4 after cardioversion were seen due to T wave memory after resolution of

Figure 1. Presenting electrocardiogram. The 12 lead electrocardiogram at the time of presentation demonstrated atrial fibrillation with a ventricular rate of 120 beats per minute and left bundle branch block with a QRS duration of 150 ms.

Figure 2. Electrocardiogram post cardioversion. The 12 lead electrocardiogram post cardioversion demonstrated sinus rhythm at 62 beats per minute with resolution of left bundle branch block (LBBB) and T wave inversions in leads V1 through V4 consistent with T wave memory seen after resolution of LBBB.

LBBB. The transthoracic echocardiogram was repeated within 24 hours and showed an improved LVEF of 50% ([Video 1, right panel](#)).

Flecainide is a potent sodium channel inhibitor that slows phase 0 of the cardiac action potential. Rapid heart rates cause greater drug binding to the sodium channel, a phenomenon termed use dependence. High concentrations of bound drug result in adverse effects due to the physiological effects of sodium channel blockade, including conduction velocity slowing and negative inotropy. Slowed conduction velocity manifest by QRS widening on the 12 lead ECG is a well-recognized consequence of flecainide toxicity. However, the negative inotropic effect of flecainide toxicity, which can cause or exacerbate heart failure, is less well-recognized. In our patient, the degree of left ventricular dysfunction seen on echocardiogram was felt to be out of proportion to his tachycardia and LBBB-induced dyssynchrony, and

was attributed to the use-dependent negative inotropy of flecainide.

Several previous studies have reported hemodynamic compromise in the setting of flecainide toxicity [1-4]. To our knowledge, this is the first case to document the negative inotropic effects of flecainide toxicity echocardiographically. It is important for all healthcare providers to recognize this potentially life-threatening consequence of flecainide toxicity given the frequency with which this drug is utilized.

Conflict of Interest

The authors have no conflict of interest relevant to this publication.

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