Texas Heart Institute Journal

Case Reports

Flecainide-Induced Atrial Flutter With 1:1 Conduction Complicated by Ventricular Fibrillation After Electrical Cardioversion

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Flecainide, a widely prescribed class IC agent used to treat atrial arrhythmias, can in rare cases cause 1:1 atrial flutter with rapid conduction. We describe the case of a 59-year-old man who was on a maintenance regimen of flecainide for refractory atrial fibrillation. When 1:1 atrial flutter with rapid conduction developed, emergency medical technicians attempted synchronized cardioversion, which caused ventricular fibrillation necessitating defibrillation. The patient ultimately underwent radiofrequency ablation and cryoablation to resolve his symptomatic atrial flutter. We discuss the atrial proarrhythmic effects of flecainide and how to mitigate complications in high-risk patients. ***(Tex Heart Inst J 2021;48(2):e197099)***

Citation:
Colangelo T, Johnson D, Ho R. Flecainide-induced atrial flutter with 1:1 conduction complicated by ventricular fibrillation after electrical cardioversion. Tex Heart Inst J 2021;48(2):e197099. doi: 10.14503/THIJ-19-7099

Key words:
Anti-arrhythmia agents/adverse effects/therapeutic use; arrhythmias, cardiac/complications/prevention & control; atrial flutter/chemically induced; cardiac complexes, premature; flecainide/adverse effects; heart conduction system/drug effects; treatment outcome; ventricular fibrillation/complications

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Flecainide, a class IC antiarrhythmic agent, can be used to convert atrial fibrillation (AF) to sinus rhythm or to maintain sinus rhythm in patients with AF who have no structural heart disease.[1] Because flecainide can cause atrial flutter with rapid conduction, combined therapy with a β-blocker, verapamil, or diltiazem is recommended to maintain rate control when AF recurs.[1]

Only rarely have patients who are taking flecainide presented with atrial flutter with 1:1 conduction.[2,3] We report this event in a man whose case was complicated by ventricular fibrillation (VF) after synchronized cardioversion.

Case Report

In February 2019, a 59-year-old man arrived at our hospital by ambulance after being found in severe respiratory distress. He had been in normal health until the morning of presentation, when he had sudden-onset dyspnea, palpitations, and a cough that produced pink sputum.

The emergency medical technicians noted wide-complex tachycardia on telemetry (heart rate, 260 beats/min), consistent with atrial flutter with 1:1 conduction (Fig. 1A). The patient was hemodynamically unstable, so the technicians attempted cardioversion with a direct-current, synchronized 100-J shock. However, as a result of the patient's rapid ventricular rate, the synchronized shock immediately precipitated VF secondary to the R-on-T phenomenon—also known as a "shock on T"—in which a premature beat of ventricular origin early after contraction causes persistent ventricular arrhythmia (Fig. 1B). Subsequently, a 360-J shock restored sinus rhythm. The patient needed no cardiopulmonary resuscitation or intubation in the field, and he lost consciousness only temporarily after the first cardioversion.

The patient's electrocardiogram (ECG) on hospital arrival showed sinus rhythm with left bundle branch block (Fig. 2). He had tachypnea, hypoxia, and respiratory distress with bilateral rales at the lung bases. Laboratory values revealed no substantial abnormalities.

The patient's medical history included refractory paroxysmal AF and atrial flutter, which had been diagnosed in 2004. He had undergone 3 electrical cardioversions at 13 years, one year, and one week before the current presentation. After the second

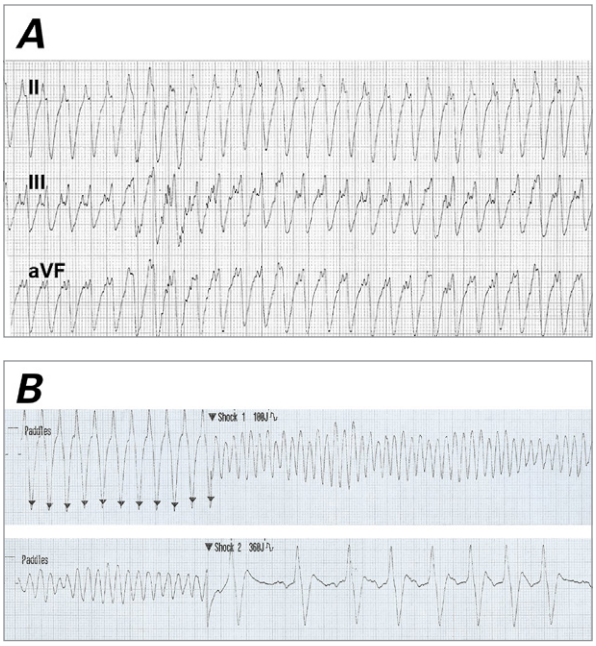

Fig. 1 *Telemetry rhythm strips show* ***A****) wide-complex tachycardia consistent with atrial flutter with 1:1 conduction, and* ***B****) ventricular fibrillation after emergency responders delivered a synchronized 100-J shock on the T wave.*

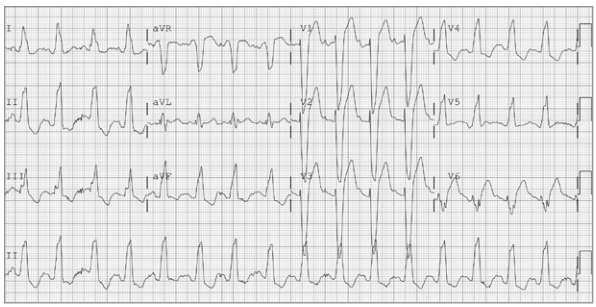

Fig. 2 *The patient's 12-lead electrocardiogram on hospital arrival shows sinus rhythm and left bundle branch block, without ventricular tachycardia.*

treatment, he had begun taking 100 mg of flecainide twice daily to maintain sinus rhythm. His only other prescribed medication was the anticoagulant rivaroxaban. An echocardiogram confirmed the absence of structural heart disease.

The day after hospital admission, the patient underwent radiofrequency catheter ablation across the cavotricuspid isthmus, to treat highly symptomatic atrial flutter. He tolerated the procedure without complication, remained in sinus rhythm, and was discharged from the hospital with instructions for outpatient cardiology follow-up. Flecainide was discontinued. Three months later, he underwent outpatient AF cryoablation. At 3-month follow-up, his ECG showed sinus rhythm, normal QRS duration, and no left bundle branch block (Fig. 3).

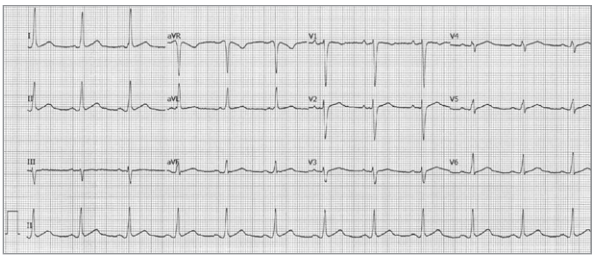

Fig. 3 *At 3-month follow-up after flecainide discontinuation and subsequent cryoablation, the patient's 12-lead electrocardiogram shows no left bundle branch block.*

Discussion

The first published ECG tracing of atrial flutter, in 1915, was from a patient whose ventricular rate was 270 beats/min.[4] Flecainide is well studied and is a safe therapy for AF.[5-8] Like other class IC antiarrhythmic agents, it selectively blocks sodium currents and inhibits potassium channels, thus prolonging cardiac conduction velocity and refractoriness.[9] The extent to which flecainide delays cardiac conduction varies in different cardiac tissue; its effects are more prominent in the atrial myocardium and the His-Purkinje system than in atrioventricular (AV) nodal tissue, thus giving rise to its antiarrhythmic actions and its proarrhythmic side effects.[10] Because flecainide can cause life-threatening ventricular proarrhythmias in individuals who have structural heart disease, its use is now contraindicated in this patient population.[8]

More notable, as in our patient's case, are the atrial arrhythmogenic properties of flecainide. Atrial proarrhythmia has been described as the development of a persistent atrial arrhythmia that is either new or was previously paroxysmal.[11] Flecainide slows conduction through the atrial tissue more than it prolongs refractoriness, which enables organized AF activity and promotes atrial flutter.[11] Hemodynamic compromise results when the atrial rate slows to enable 1:1 conduction through the AV node,[12] in the presence of rapid ventricular response. The estimated incidence of atrial proarrhythmic effects from class IC agents is 3.5% to 5%.[12,13] Therefore, patients taking flecainide may also need an AV nodal-blocking agent to mitigate this risk if AF recurs.[1] Our patient was not taking one, and he had 1:1 atrial flutter with a rapid ventricular response.

Some ECG features predict the development of this condition in patients who take flecainide. One proposed marker, a shortened PR interval (<0.13 ms) on the surface ECG, may identify existing rapid AV nodal conduction, which predisposes patients to atrial flutter with 1:1 conduction when they take a class IC antiarrhythmic drug.[14] Flecainide also causes a use-dependent prolongation of the QRS interval that becomes more

pronounced during faster heart rates. One patient who had an underlying conduction block showed an 80% prolongation in QRS, suggesting that flecainide-induced conduction delay is more likely and more pronounced in the presence of existing disease.[10] After flecainide was discontinued, our patient's left bundle branch block resolved, and his QRS duration returned to normal, highlighting the drug's adverse effect on the His-Purkinje system.

When the QRS interval widens, the ECG pattern may reveal an aberrantly conducted, regular, wide-complex tachycardia that mimics ventricular tachycardia.[15] In our patient, the attempted synchronized cardioversion precipitated VF consequent to the R-on-T phenomenon. However, VF induction after a 100-J synchronized shock is unusual, because synchronization should prevent a shock on the T wave.

Although the atrial and ventricular proarrhythmic properties of flecainide are well documented, this case provides the opportunity to review an infrequent and severe adverse effect of this widely used medication. Furthermore, it suggests that an AV nodal-blocking agent should also be prescribed when a patient who has AF is taking flecainide to maintain sinus rhythm.

Published: 4 June 2021

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