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MINI-FOCUS ISSUE ON ELECTROPHYSIOLOGY AND PACING

BEGINNER

IMAGING VIGNETTE: ECG CHALLENGE

The Unpaceable Heart

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ABSTRACT

This is a case of flecainide toxicity in a patient with a permanent pacemaker. This case not only highlights the effects of flecainide toxicity on surface electrocardiography but how toxicity effects pacemaker function and its ability to transvenously pace the heart. The report provides some discussion of the management options for flecainide toxicity. (**Level of Difficulty: Beginner.**) (J Am Coll Cardiol Case Rep 2020;2:595–7) Crown Copyright © 2020 Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

CASE

A 36-year-old woman was referred to our center with pacemaker malfunction (**Figure 1A**) and cardiogenic shock. Her medical history was significant for resolved myocarditis and supraventricular tachycardia (SVT). Years prior, she had radiofrequency ablation of her SVT, which was complicated by complete atrioventricular block and required a dual-chamber permanent pacemaker. Her only prescribed medication was flecainide 100 mg on an as needed basis. Initial biochemistry revealed normal electrolytes, urea and creatinine, normal white cell count and inflammatory markers, and no significant troponemia. Echocardiography demonstrated a mildly dilated right ventricle with gross septal dyssynchrony and a nondilated left ventricle with normal function. Because of her clinical instability and loss of ventricular capture from her pacemaker, transvenous temporary pacing was attempted. Despite high-output pacing in multiple locations in the right ventricle, consistent capture could not be established. An urgent endomyocardial biopsy was performed, which demonstrated normal myocardium. She was placed on extracorporeal membrane oxygenation (ECMO), and all pacing functions were disabled (**Figure 1B**). Within 30 h, her hemodynamics and QRS normalized (**Figure 1C**). How do you interpret these results?

(a) Myocarditis
(b) Flecainide toxicity
(c) Pacemaker lead perforation
(d) Hyperkalemia

DISCUSSION

Of these potential answers, the unifying diagnosis is flecainide toxicity. This patient had a flecainide level >3 times the therapeutic level (3.6 μg/ml; range: 0.4 to 1 μg/ml). Although myocarditis could be a possible cause, the normal endomyocardial biopsies, coupled with an inability to pace the right ventricle at multiple sites, as

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ABBREVIATIONS AND ACRONYMS

ECMO = extracorporeal membrane oxygenation

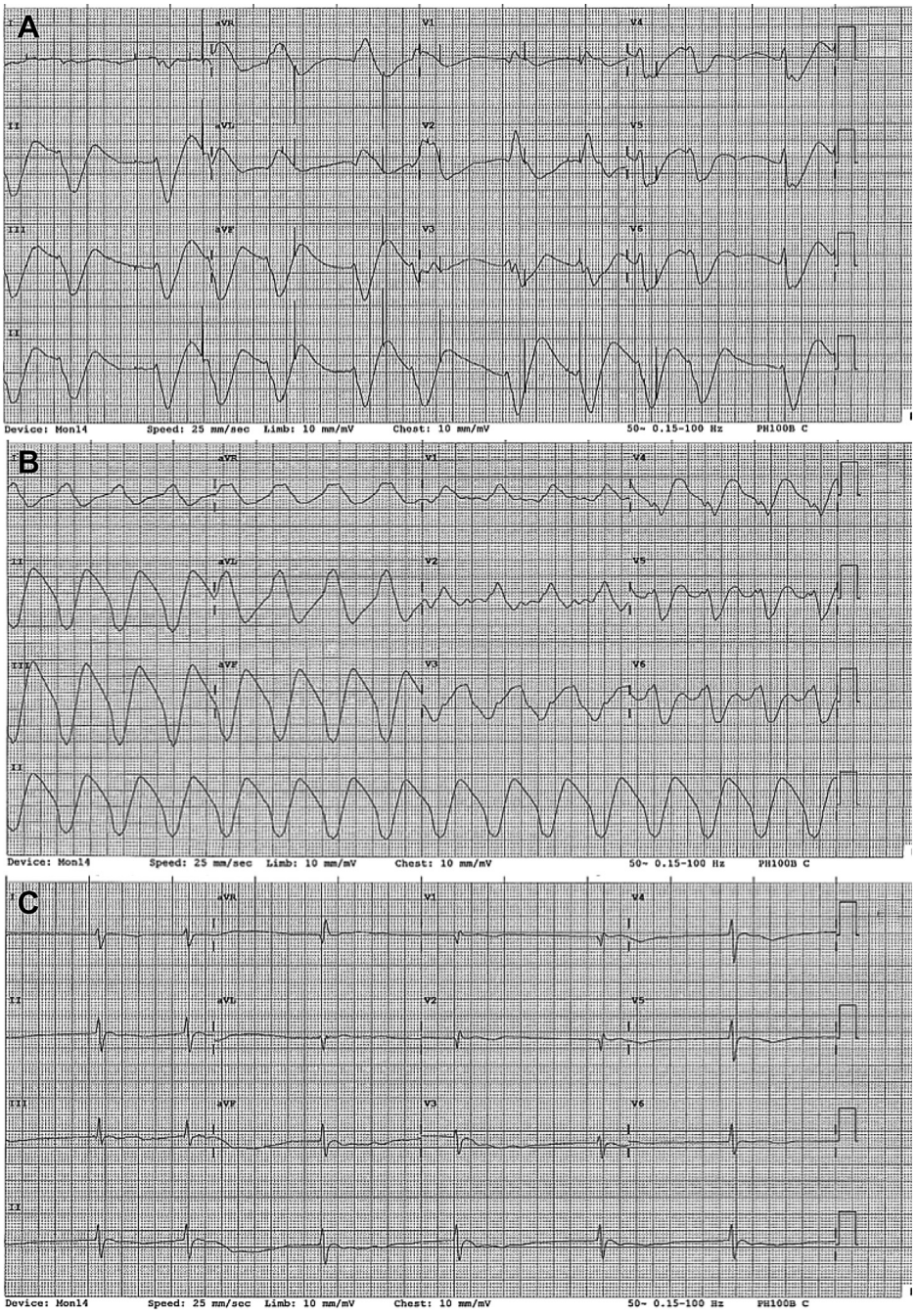
Na+ = sodium

SVT = supraventricular tachycardia

well as the rapid reversal of the electrocardiographic abnormalities makes this diagnosis less likely. Pacemaker lead perforation would not explain the inability to transvenously pace the heart with a temporary wire, and although the sinusoidal electrocardiographic appearance might be consistent with hyperkalemia, no suspicious features were described, and electrolytes were normal.

SEE PAGE 598

FIGURE 1 Electrocardiograms of the Patient



(A) Broad complex irregular rhythm with disordered pacing stimuli. **(B)** Accelerated idioventricular rhythm with sine wave morphology. **(C)** Atrial fibrillation with bradycardia, narrow complex QRS, and prolonged QT interval.

Flecainide is a Class Ic anti arrhythmic agent that depresses the rate of depolarization of cardiac action potentials that produce a membrane stabilizing action. Although contraindicated in patients with previous myocardial infarction and subsequent arrhythmias due to its proarrhythmic effects (1), it is used to treat a range of atrial and ventricular arrhythmias in those with structurally normal hearts. The pro-arrhythmic effect of flecainide may be related to promoting re-entry in ventricular tissue. This phenomenon is due to a rate-dependent blockade of rapid sodium (Na+) channels that slows phase 0 of depolarization and an inhibition of the slow calcium channel (2). Conduction disturbances begin with widening of the QRS complex, which can rapidly progress to ventricular tachycardia and asystole. Early electrocardiography indicates toxicity by demonstrating PR and QRS prolongation. In patients with permanent pacemakers, the ability of flecainide to increase pacing thresholds and cause pacemaker failure has been well established (3,4).

Flecainide is largely excreted by the kidneys and has an elimination half-life of 20 h. In some cases, cardiac monitoring and observation may be the only required action. If intervention is needed, sodium bicarbonate, lidocaine, and ECMO have all been used.

Sodium bicarbonate can be used to correct widening of the QRS. Hypertonic sodium bicarbonate works by increasing the extracellular concentration of Na+, which displaces flecainide from its receptor (2). Lidocaine, another Na+ channel-blocking drug (Class Ib), has been shown to effectively treat flecainide toxicity and restore sinus rhythm (5). The two drugs share a common receptor, but consistent with the modulated receptor hypothesis, these drugs reach this receptor by distinct routes, dictated by the degree of ionization of the drug molecules (5). Lidocaine is believed to compete with the binding potential of flecainide. Lastly, ECMO has been used to support unstable patients (6).

In our case, once the electrocardiogram had normalized, repeat echocardiography demonstrated normal cardiac size and function, and the patient was weaned from ECMO. Following psychological assessment, the patient was discharged without complications.

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