

Letter to the Editor

Brugada-Type ECG Pattern in Patients Treated with Oral Flecainide for Lone Atrial Fibrillation: Is There Any Clinical Implication?

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Key words: **Brugada syndrome, atrial fibrillation, flecainide.**

Manuscript received:
November 15, 2008;
Accepted:
March 31, 2009.

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The Brugada syndrome (BS), a relatively new arrhythmogenic entity, is characterized by ST-segment elevation in right precordial leads (V_1 through V_3) in the absence of structural heart disease, and a high propensity for sudden cardiac death.¹ The clinical phenotype is 8 to 10 times more prevalent in males than in females.^{1,2} Mutations of the *SCN5A* gene encoding for the α subunit of the cardiac sodium channel (I_{Na}) have been reported as the genetic basis of the disease.^{1,2} Three types of repolarization patterns in the right precordial leads have been recognized. Type 1 is diagnostic of BS and is characterized by coved ST-segment elevation, while types 2 and 3 are characterized by a saddleback ST-segment configuration.^{1,2} The ECG features of BS are often concealed, requiring a pharmacological challenge (I_{Na} blocking test) with Class I antiarrhythmic agents (ajmaline, flecainide, procainamide) to unmask the diagnostic pattern in the right precordial leads.^{1,2} The clinical significance of the Brugada type ECG pattern among patients treated with flecainide for atrial fibrillation (AF) remains unknown.

We report on two male patients, 55 and 65 years old, who developed a Brugada-type ECG pattern following one month's treatment with oral flecainide (200 mg daily) for lone AF. Both subjects exhibited a struc-

turally normal heart. None of them displayed a history of syncope or a positive family history of BS or sudden cardiac death. Patient 1 developed a type 1 ECG pattern (Figure 1), while patient 2 developed a type 2 ECG pattern of BS. Withdrawal of flecainide resulted in normalization of the ECG in both cases. Holter recordings failed to demonstrate any ventricular arrhythmic events.

The data regarding flecainide-induced Brugada-type ECG pattern in patients with AF are limited. Beldner et al reported only three cases with ECG changes consistent with BS among 87 patients treated with flecainide (average daily dose 198 ± 61 mg) for AF. No ventricular arrhythmias occurred in any of these patients during follow up.³ The concurrent finding of flecainide-induced Brugada type ECG pattern in subjects with AF may have some clinical implications. Patients with BS exhibit a high incidence (20%) of atrial arrhythmias, mainly AF.⁴ The present report indicates that patients with AF may represent a population with an increased incidence of flecainide-induced Brugada-type ECG changes. A possible genetic link between AF and BS may exist. Over 100 mutations of the *SCN5A* gene have been identified in BS up to now.² *SCN5A* mutations and polymorphisms have also been detected in subjects with lone AF and may increase the

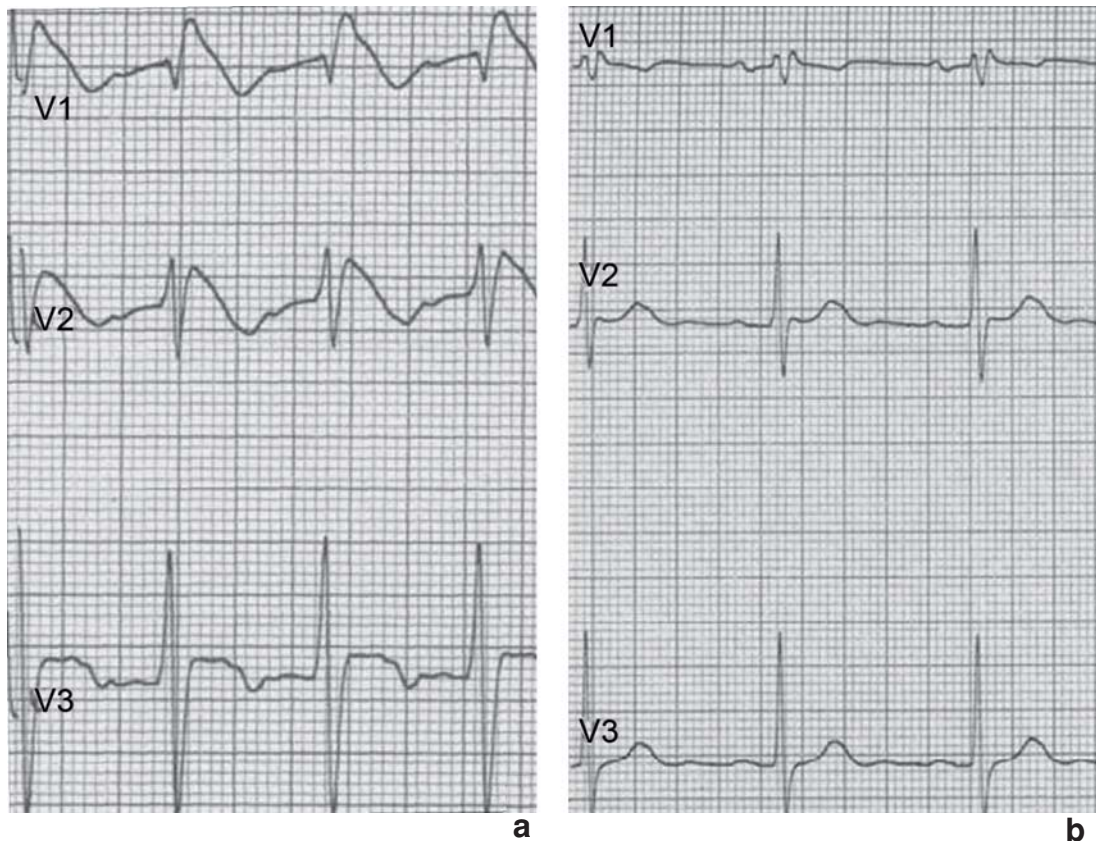


Figure 1. a. ECG recording showing a type 1 ECG pattern of Brugada syndrome during treatment with oral flecainide (200 mg daily). b. Withdrawal of flecainide resulted in normalization of the ECG.

susceptibility to I_{Na} blockade-induced proarrhythmia.^{5,6} It has recently been reported that nearly 6% of AF probands carry heterozygous mutations or rare variants in the *SCN5A* gene.⁶ These mutations and/or polymorphisms may be clinically silent and manifest under certain conditions, including the I_{Na} blocking test. Although our patients had no family history of BS or sudden cardiac death, they could belong to the above group (silent carriers of BS), and therefore be at high risk of developing a ventricular arrhythmic event under flecainide treatment. Moreover, both cases were men, a fact possibly related to the higher prevalence of BS in males than in females. Further studies, including screenings for *SCN5A* gene mutations and/or polymorphisms, are required to evaluate the clinical significance of flecainide-induced Brugada type ECG pattern in patients with AF.

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