

Letters to the Editor

Brugada Phenocopy Emerging as a New Concept

Fenocopia de Brugada: surgimiento de un nuevo concepto

To the Editor,

We read the recent case report "Hyperkalemia Mimicking a Pattern of Brugada Syndrome" by Recasens et al.¹ with great interest. The paper is important because it contributes to the growing body of literature describing Brugada phenocopies (BrP).^{2,3} BrP are characterized by electrocardiographic (ECG) patterns that are identical to type 1 or type 2 Brugada patterns despite the absence of the true congenital Brugada syndrome. BrP are caused by various clinical circumstances including: hyperkalemia, adrenal insufficiency, hypothermia, mechanical cardiac compression, myocarditis, pericarditis, and ischemia.² Currently, 6 etiological categories of BrP exist: *a)* metabolic conditions; *b)* mechanical compression; *c)* ischemia and pulmonary embolism; *d)* myocardial and pericardial disease; *e)* ECG modulation; and *f)* miscellaneous.⁴ The case presented by Recasens et al.¹ qualifies under the category of metabolic conditions. We suggest to the authors that future reports use the established term "Brugada phenocopy" to facilitate research and consistency within the literature.

In the presented case, we would like to clarify the appropriate morphological terminology pertaining to the description of the Brugada patterns. The authors state that leads V₁ to V₃ are consistent with a "saddle back-type ST segment elevation in V₁ to V₃". However, our interpretation is slightly different. Leads V₁ and V₂ have a type 1 "coved" Brugada pattern while lead V₃ has a type 2 "saddle back" Brugada pattern.⁵ Additionally, the authors state that hyperkalemia-producing Brugada patterns are very unusual; however, there have been several case reports of hyperkalemia associated with both type 1 and type 2 Brugada patterns.^{2,6–8} Furthermore, the etiology of the Brugada pattern was solely attributed to hyperkalemia (K, 7.53 mEq/L); however, this patient had concurrent hyponatremia (Na 127 mEq/L) and acidosis (pH, 7.22). Prior case reports have noted Brugada patterns in the context of hyponatremia⁹ and also with concurrent hyperkalemia, hyponatremia, and acidosis.¹⁰ As such, in the present case, the Brugada pattern cannot be attributed solely to the potassium abnormality.

Finally, we must congratulate the authors on ruling out sodium channel dysfunction with a flecainide test. This is an important step in excluding Brugada syndrome and diagnosing BrP. We have previously established and applied systematic diagnostic criteria to ascertain BrP.¹¹ The current BrP diagnostic criteria are: *a)* the ECG pattern has a type 1 or type 2 Brugada morphology; *b)* the patient has an underlying condition that is identifiable; *c)* the ECG pattern resolves after resolution of the underlying condition; *d)* there is a low clinical pretest probability of true Brugada syndrome, determined by lack of symptoms, medical history, and family history; *e)* negative provocative testing with sodium channel blockers such as ajmaline, flecainide, or procainamide; *f)* provocative testing not mandatory if surgical right ventricular outflow tract manipulation has occurred within the last 96 hours;

and *g)* the results of genetic testing are negative (desirable but not mandatory because the SCN5A mutation is identified in only 20% to 30% of probands affected by true Brugada syndrome).^{3,11} We suggest to future authors that when diagnosing BrP, this systematic approach including provocative testing with a sodium channel blocker, be rigorously applied.

We are in the process of developing an international registry database of BrP¹² and invite the authors to submit this case along with future cases to the registry.³ The goal of this registry is to provide long-term follow-up and insight into the pathophysiology of BrP and their distinct clinical evolution from Brugada syndrome.

Daniel D. Anselm* and Adrian Baranchuk

Department of Cardiology, Electrophysiology and Pacing, Queen's University, Kingston General Hospital, Kingston, Ontario, Canada

* Corresponding author:

E-mail address: danselm@rogers.com (D.D. Anselm).

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