Correspondence

Letter by Casado-Arroyo et al Regarding Article, “Electrocardiographic Characteristics and SCN5A Mutations in Idiopathic Ventricular Fibrillation Associated With Early Repolarization”

To the Editor:

We have read “Electrocardiographic Characteristics and SCN5A Mutations in Idiopathic Ventricular Fibrillation Associated With Early Repolarization” by Watanabe and colleagues1 with great interest and congratulate the authors for their publication. However, there are some aspects that in our opinion should be clarified.

The authors state in the “Methods” section, “Patients with Brugada type ST-segment elevations at baseline or after sodium channel blocker challenge were excluded.”1 As has been previously published, the diagnosis of Brugada syndrome (BS) should be strongly considered if there is appearance of Type 1 ST segment elevation (coved type) in at least 1 of the precordial leads or in the inferior leads in the presence or absence of a sodium channel blocker plus at least 1 clinical symptom.2,3

We disagree with the electrocardiographic interpretation in Patient 1 (Figure 2B): the basal electrocardiogram shows a spontaneous Type 1 Brugada pattern in lead V2. After pilosicainide, the patient presents an inferior–early lateral repolarization pattern. This pattern and the response to sodium channel blockers are specific of patients with BS and interestingly have been associated with a more severe phenotype.4 Patient 2 (Figure 2C) illustrates a classical coved-type electrocardiographic pattern after the infusion of the drug in lead V2. These 2 patients presented spontaneous ventricular fibrillation after the infusion of the drug and also during programmed stimulation, because it has been previously described in patients with BS.2,3 On the other hand, the third patient presents a typical case of spontaneous Type 1 at baseline in lead V2 (Figure 2E). Accordingly, it is important to mention that the SCN5A mutation (R367H) had been previously described in patients with BS, specifically in a large European family with 18 members4 and is internationally recognized as a mutation causative of BS.5 This amino acid substitution is associated with high incidence of spontaneous Type 1, as is the case in the third patient.

Although differentiation between idiopathic ventricular fibrillation and BS may be difficult, frequent overlap between these 2 entities is possible. From our point of view, BS has not been properly excluded. These patients discussed by Watanabe et al fit all phenotype and genotype characteristics of BS.

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