INTERESTING ELECTROCARDIOGRAM

COMPLEX CASE STUDY

Brugada Phenocopy Induced by Acute Inferior ST-segment Elevation Myocardial Infarction with Right Ventricular Involvement

1DANIEL D. ANSELM, MD, 2RAIMUNDO BARBOSA-BARROS, MD, 2LUCIA DE SOUSA BELÉM, MD, 2RAFAEL NOGUEIRA DE MACEDO, MD, 3ANDRÉS RICARDO PÉREZ-RIERA, MD, PhD and 1ADRIAN BARANCHUK, MD, FACC, FRCPC

1Division of Cardiology, Kingston General Hospital, Queen’s University, Kingston, Ontario, Canada; 2Hospital de Messejana Dr. Carlos Alberto Studart Gomes, Fortaleza, Ceará, Brazil; 3Cardiology Discipline, ABC Medical Faculty, ABC Foundation, Santo André, São Paulo, Brazil

ABSTRACT. The first case in the literature on Brugada phenocopy induced by acute inferior myocardial infarction with a systematic approach to rule out sodium channel dysfunction is presented here.

KEYWORDS. acute myocardial infarction, Brugada phenocopy, Brugada syndrome, right ventricular infarction.

ECG description

A 70-year-old male presented with acute inferior ST-segment elevation myocardial infarction with right ventricular involvement. His comorbidities included hypertension and type 2 diabetes. The patient was treated with aspirin, enalapril, simvastatin, carvedilol, amlodipine, and metformin. The 12-lead electrocardiogram (ECG) (Figure 1a) shows a heart rate of 78 bpm, normal axis, PR interval of 260 ms, and QRS duration of 90 ms. The rhythm strip (II) shows junctional bigeminy with retrograde atrial activation. There is ST-segment elevation in the inferior leads (II, III, aVF) with reciprocal changes in the high lateral leads (I, aVL) consistent with acute inferior ST-segment elevation myocardial infarction. Lead V1 shows a typical “coved” type-1 Brugada pattern. The right-sided precordial leads (V3R, V4R) show ST-segment elevation indicative of right ventricular involvement (Figure 1b). The patient was treated with streptokinase, and a subsequent ECG showed resolution of the ST-segment elevation in the inferior leads. The “coved” type 1 Brugada pattern in lead V1 can no longer be seen (Figure 2).

Points to ponder

Brugada phenocopies (BrP) are characterized by ECG patterns that are indistinguishable from true Brugada syndrome (BrS). Recently, criteria for differentiating BrP from BrS have been established (Table 1).1,2 The patient in this case presented with a type 1 Brugada ECG pattern (criterion i) in the context of acute myocardial infarction (criterion ii). Resolution of the type 1 Brugada pattern was observed immediately after resolving ischemia (criterion iii, Figure 2). There was no known family history of sudden cardiac death and therefore low clinical pretest probability of true BrS (criterion iv). The patient underwent ajmaline provocative testing, with a negative result (Figure 3) confirming the diagnosis of BrP in the context of acute inferior myocardial infarction with right ventricular involvement (criterion v).

Prior case reports3–6 have been published indicating the association of ischemia producing type 1 and type 2
Figure 1: (a, b) Electrocardiogram on admission.

Figure 2: Electrocardiogram post streptokinase showing resolution of ST-segment elevation in the inferior leads (II, III, aVF) along with resolution of type-1 Brugada pattern (V1). HR 90, normal axis, normal sinus rhythm, PR 188ms, QRS 100 ms, QTc 411 ms.
Table 1: Criteria for defining Brugada phenocopy*

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<thead>
<tr>
<th>i</th>
<th>The ECG pattern has a type 1 or type 2 Brugada morphology</th>
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<td>ii</td>
<td>The patient has an underlying condition that is identifiable</td>
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<td>iii</td>
<td>The ECG pattern resolves after resolution of the underlying condition</td>
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<td>iv</td>
<td>There is a low clinical pretest probability of true Brugada syndrome determined by lack of symptoms, medical history, and family history</td>
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<td>v</td>
<td>Negative provocative testing with sodium channel blockers such as ajmaline, flecainide, or procainamide</td>
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<td>vi</td>
<td>Provocative testing not mandatory if surgical right ventriculocardio outflow tract (RVOT) manipulation has occurred within the last 96 h</td>
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<td>vii</td>
<td>The results of genetic testing are negative (desirable but not mandatory because the SCN5A mutation is identified in only 20–30% of probands affected by true Brugada Syndrome)</td>
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*Adapted from Anselm et al. 2

Brugada ECG patterns; however, none was able to clearly differentiate BrP from BrS. This is the first report to systematically prove, by excluding the presence of sodium channel dysfunction, the presence of BrP in the context of acute ischemia using clearly defined current diagnostic criteria.

References