CASE REPORT

Brugada phenocopy secondary to extensive inferior wall ischemia induced by coronary steal effect

Carlos Guamán Valdivieso MD¹ | Federico Ferrando-Castagnetto MD¹ | Pablo Marichal TEHN¹ | Federico Acquistapace MD¹ | Pedro Trujillo MD¹ | Adrián Baranchuk MD²

¹Department of Cardiology, Clinical Hospital, School of Medicine, Republic University, Montevideo, Uruguay

²Division of Cardiology, Kingston General Hospital, Queen's University, Kingston, Ontario, Canada

Correspondence

Carlos Guamán Valdivieso, Nuclear Cardiology Unit, Department of Cardiology, Clinical Hospital, School of Medicine, Republic University, Montevideo, C.P 11600, Uruguay.
Email: cgv0792@gmail.com

Abstract

The diagnosis of Brugada syndrome is usually made with a typical ECG pattern. However, different disorders can emulate this pattern (Brugada phenocopies). Pathophysiologic mechanisms underlying this phenomenon remain controversial. We describe the development of type-1 Brugada ECG pattern associated with extensive coronary steal effect during myocardial perfusion scintigraphy. Proximal occlusion of the right coronary artery and severe proximal stenosis in distally occluded left circumflex artery were confirmed by coronary angiogram. Brugada ECG pattern can be a reversible sign associated with inferior left ventricular and right ventricular ischemia. Its presence during acute ischemia deserves appropriate risk stratification.

KEYWORDS

Brugada phenocopy, Brugada syndrome, myocardial ischemia

1 | CASE DESCRIPTION

A 56-year-old man with a history of dyslipidemia and hypertension underwent a neurosurgical preoperative evaluation (spinal arachnoid cyst). He has presented in the emergency department with a 3-week history of minimal exertion angina. The ECG on admission showed nonspecific ST-T changes in the inferior leads and a type-2 Brugada ECG pattern in leads V1-V2 (Figure 1a). Serum troponin I during the first 24 hr was normal (4 samples). Noninvasive preoperative cardiovascular assessment was performed using 99m-Tc-MIBI myocardial perfusion vasodilator-stress imaging, in two consecutive days protocol (stress-rest). Three minutes after ending intravenous infusion of dipyridamole (0.56 mg/kg), a type-1 Brugada ECG pattern was recorded in leads V1-V2, associated with ECG evidence of ischemia (Figure 1b). Type-1 Brugada ECG pattern resolved and electrical evidence of ischemia normalized (Figure 1c) after aminophylline. Gated-single photon emission computed tomography images showed severe and extensive inferior ischemia induced by pharmacological stress (Figure 2a).

Coronary angiogram detected a subacute thrombotic occlusion in the proximal right coronary artery (RCA) and severe proximal stenosis

of a left circumflex artery that was distally occluded (Figure 2b and c). Due to large ischemic burden, percutaneous coronary stenting of proximal and mid RCA was performed (Figure 2d). ECG after coronary intervention showed the same QRS-T morphology as at admission, and the patient was discharged after 48 hr without complications.

2 | DISCUSSION

Characteristic Brugada syndrome (BrS) pattern may be associated with multiple disorders and usually resolves after the resolution of the underlying condition (Brugada phenocopies (BrP)) (Baranchuk et al., 2012; Brugada, Campuzano, Arbelo, Sarquella-Brugada, & Brugada, 2018). The diagnosis of BrS is suspected when the ECG shows a characteristic type-1 Brugada ECG pattern in at least one right precordial lead (Bayés de Luna et al., 2012). This ECG pattern can occur spontaneously or after intravenous administration of sodium channel blocking drugs (ajmaline, flecainide, procainamide, or pilsicainide) (Brugada et al., 2018). However, the ECG alone does not make diagnosis of BrS and all known causes that can cause phenocopies should be excluded (Baranchuk et al., 2012).

FIGURE 1 (a) Baseline electrocardiogram depicting type-2 Brugada ECG pattern. Convex ST-segment elevation > 2 mm in V2 followed by a positive T wave. The β angle is $\geq 58^\circ$, and the duration of the base of the triangle of r' at 5 mm from the high take-off is > 4 mm. (b) Deep descendent ST-segment depression in leads V3–V6 was observed 3 min after dipyridamole infusion (arrows). There is a type-1 Brugada ECG pattern in lead V2 showing concave ST-segment elevation > 2 mm followed by a negative T wave at 40 ms of high take-off (arrowhead), and the decrease in amplitude of ST-segment is ≤ 4 mm. (c) The ECG returns to baseline without signs of ischemia 15 min after aminophylline administration. Note the mismatch between leads V6 and V1 in both type-2 (a and c) and type-1 (b) Brugada ECG patterns

FIGURE 2 (a) Reversible perfusion defect compatible with severe and extensive dipyridamole-induced ischemia in the inferior region. Coronary angiogram (b) detected 70% stenosis of proximal left circumflex artery that occludes distally (black arrowhead). (c) Right coronary artery (RCA) was occluded proximally (black arrow), and inferior wall flow was supplied by hetero-coronary collateral circulation (seen in b). (d) Successful percutaneous coronary intervention

In the case described here, it was mandatory to define whether subendocardial ischemia was responsible for inducing a BrP or, on the other hand, was able of unmask a true BrS. Anselm, Gottschalk, and Baranchuk (2014) developed an online registry of BrP cases that characterize the causes of phenocopies and promote new areas of research that could provide some clues to this clinical question.

We postulate that our patient presented a type-1B BrP of ischemic origin (Anselm & Baranchuk, 2013). The challenge test with sodium channel blockers was rejected by the patient, due to low clinical probability of BrS and the risks of ventricular arrhythmias in this clinical context (Baranchuk et al., 2012; Xu et al., 2019).

The involvement of the right ventricle during myocardial ischemia, acute pulmonary embolism, and mechanical compression can cause ECG changes identical to type-1 BrS pattern (Brugada et al., 2018). We described the appearance of BrP during transmural myocardial ischemia associated with acute thrombosis of the anterior descending artery (Ferrando-Castagnetto, Garibaldi-Remuñan, Vignolo, Ricca-Mallada, & Baranchuk, 2016; Ferrando-Castagnetto, Rivara, Vignolo, Ricca-Mallada, & Baranchuk, 2017). BrP associated with ischemia is manifested mainly through type-1 Brugada ECG

pattern, predominately in men, and the coronary artery most frequently involved was the RCA (65%). However, no significant association was found between the ECG findings and the artery related to ischemia causing BrP (Xu et al., 2019).

To our knowledge, this is the first report to characterize a BrP during coronary steal phenomenon produced by vasodilator-stress subendocardial ischemia. Agrawal, Stevens, Shirani, Garg, and Nanda (2015) described a BrP associated with vasodilator stimulation in a patient with imaging findings suggestive of inferior wall infarction (fixed defect) and moderate obstruction of mid RCA, without evidence of ischemia.

The lack of a sodium channel blocker test represents a limitation reporting this case, so it was not possible to define whether myocardial ischemia induced a BrP or unmasked a true BrS. Although the low clinical probability of BrS, as well as the simultaneous resolution of ischemia and abnormal ECG pattern, an ischemia-induced BrP is more likely.

CONFLICT OF INTEREST

None.

ORCID

Carlos Guamán Valdivieso <https://orcid.org/0000-0002-1065-1988>

Adrián Baranchuk <https://orcid.org/0000-0002-3042-6569>

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How to cite this article: Guamán Valdivieso C, Ferrando-Castagnetto F, Marichal P, Acquistapace F, Trujillo P, Baranchuk A. Brugada phenocopy secondary to extensive inferior wall ischemia induced by coronary steal effect. *Ann Noninvasive Electrocardiol*. 2020;25:e12689. <https://doi.org/10.1111/anec.12689>