Letter to the Editor

Brugada phenocopy in acute pulmonary embolism

Zhong-Qun Zhan a,⁎, Chong-Quan Wang a, Kjell C. Nikus b, Andrés Ricardo Pérez-Riera c, Adrian Baranchuk d

a Department of Cardiology, Shiyan Taihe Hospital, Hubei University of Medicine, Shiyan City, Hubei Province, China
b Department of Cardiology, Heart Center, Tampere University Hospital, Tampere University, Faculty of Medicine, Tampere, Finland
c ABC Faculty of Medicine (FMABC), ABC Foundation (FUABC) Cardiology Discipline Electro-Vectorcardiology Sector, Santo André, São Paulo, Brazil
d Heart Rhythm Service, Kingston General Hospital, Queen’s University, Kingston, Ontario, Canada

ARTICLE INFO

Article history:
Received 23 August 2014
Accepted 16 September 2014
Available online xxxx

Keywords:
Acute pulmonary embolism
Brugada syndrome
Brugada phenocopy
Myocardial ischemia

The Brugada electrocardiographic (ECG) pattern presents in leads V1–V3 as: (i) Type-1 ECG pattern (“coved”) with ST-segment elevation (STE) ≥ 0.2 mV and T-wave inversion (TWI); and (ii) Type-2 ECG pattern (“saddle-back”) with STE and usually positive T-wave [1]. Acute pulmonary embolism (APE) can present as a Brugada phenocopy (BrP) with ECG changes identical to the Brugada pattern. The pattern disappears upon resolution of the APE and the test with sodium channel blockers is negative [2,3]. APE associated BrP is a rare phenomenon and has been reported by only three previous articles [4–6]. Further characterization of BrP during the course of APE is needed to avoid misdiagnosis. Here we report two new patient cases of BrP during APE and briefly review the existing literature and discuss possible underlying mechanisms.

Case 1 was a 43-year-old woman, who presented to the emergency department with progressive dyspnea and repetitive syncope for the last two days. On arrival, she was sweating, pale and unconscious. Blood pressure (BP) was 60/40 mm Hg. ECG on arrival (Fig. 1, left) showed sinus tachycardia, prominent “coved” STE in leads V1–V2 and ST-segment depression (STD) in leads I, II, III, aVF, V4 to V6, minor terminal TWI in lead V2. A Type-1 Brugada ECG pattern was detected. Cardiopulmonary resuscitation was initiated and an urgent echocardiogram revealed a large thrombus in the main pulmonary artery, enlarged right ventricle (RV), severe tricuspid regurgitation and hypokinesia of the RV free wall. Massive APE was diagnosed and a urokinase infusion was initiated with acceptable hemodynamic response after 1 h of thrombolytic therapy. The second ECG (Fig. 1, right) showed a resolution of the STE in leads V1–V2 and TWI in lead V1. The patient recovered.

Fig. 1. The electrocardiogram before thrombolysis (left) showing Type-1 Brugada pattern in V1–V2. The electrocardiogram after thrombolysis (right) showing resolution of the Brugada pattern and T-wave inversion in V1.

⁎ Corresponding author.
E-mail addresses: zzqun21@126.com (Z.-Q. Zhan), sythwqc@medmail.com.cn (C.-Q. Wang), Kjell.Nikus@sydansairaala.fi (K.C. Nikus), riera@uol.com.br (A.R. Pérez-Riera), barancha@kgh.kari.net (A. Baranchuk).

http://dx.doi.org/10.1016/j.ijcard.2014.09.046
0167-5273/© 2014 Elsevier Ireland Ltd. All rights reserved.
favorably and was discharged home with anticoagulation after eight days.

Case 2 was a 48-year-old man, admitted in Cardiology department due to palpitation and dyspnea for 6 h. On admission, he was hemodynamically stable. Shortly after admission, the patient presented with sweating and severe abdominal pain. The BP dropped to 85/55 mm Hg. The ECG (Fig. 2, left) showed S1–Q3, a prominent “saddle-back” STE in leads V1–V3, and STD in leads I, aVL, and V6. The “saddle-back” STE in leads V1–V3 was identical to a Type-2 Brugada ECG pattern. Acute coronary syndrome was suspected. Emergent coronary angiography showed no significant coronary artery disease. Pulmonary angiography showed massive embolism in both pulmonary arteries. A urokinase infusion was started. After thrombolysis, the ECG (Fig. 2, right) showed a disappearance of the S1–Q3 pattern, and ST-segment deviation, with new TWI in the inferior and precordial leads. The patient was discharged home in good health after 10 days.

The ECG manifestations of the three previously published [4–6] and the two new cases from our series are listed in Table 1. The ECGs from the acute phase of the five cases with the BrP during APE have several ECG commonalities. Based on these cases, the BrP associated with APE may manifest as: (i) STE ≥ 0.2 mV in leads V1–V3, usually in the context of cardiogenic shock or severe hypotension; (ii) if STE with concomitant TWI was present, the manifestation would be identical to a Type-1 Brugada ECG pattern – a prerequisite for the diagnosis of BrP – along with the disappearance of the pattern upon resolution of APE; (iii) if the STE with concomitant positive T-waves higher than ST-segment was present, the manifestation would be identical to a Type-2 Brugada ECG pattern. The other observed ECG manifestations were sinus tachycardia, S1–Q3, STD in leads I and V4/V5 to V6.

Grand et al. [7] found that STE in the right precordial leads is a transient sign of moderate to severe APE. This phenomenon indicates RV transmural ischemia due to hypotension, hypoxemia, right ventricular strain and catecholamine surge [8,9]. Although STE in the right precordial leads (<2 mm) is not a rare phenomenon [7–9], prominent STE (≥0.2 mV) confined to leads V1–V3 is not frequently seen. The majority of the patients, also our two new cases, with prominent STE in the right precordial leads presented with hypotension or cardiogenic shock. TWI in the right precordial leads is a frequent manifestation of APE and may represent an evolutionary “post-ischemic” stage following STE [8,9]. In the case of a new episode of transmural RV ischemia, usually during hypotension or cardiogenic shock, TWI will decrease and the STE may re-appear [9].

Anselm et al. [10] reported a case of acute inferior STE myocardial infarction presenting as a Type-1 BrP induced by RV involvement. Our opinion is that the cases of BrP during APE may be induced by transmural myocardial ischemia, mainly secondary to RV stretch.

Although the ECG findings of the BrP during APE are identical to those of the Brugada syndrome, the differences between the two entities should be highlighted and recognized. The Brugada syndrome occurs in patients with an apparently structurally normal heart and predisposes patients to malignant ventricular arrhythmias and sudden cardiac death. Patients with APE present a myriad of symptoms including acute onset of dyspnea, tachypnea, palpitations, syncope, hypotension or cardiogenic shock, especially in patients with STE in the right precordial leads. Once the patient’s condition resolves, there is a prompt resolution of the STE in leads V1–V3.

Table 1

<table>
<thead>
<tr>
<th>Case</th>
<th>G/A</th>
<th>Clinical condition during BrP</th>
<th>ECG characteristics during BrP in APE</th>
<th>Other ECG manifestation during BrP in APE</th>
<th>ECG after treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Case 1</td>
<td>F/43</td>
<td>Cardiogenic shock</td>
<td>Type-1: prominent “coved” STE in V1–V2, terminal TWI in V2</td>
<td>ST, STV2S3, STE in aVR, STD in I, II, III, aVF, V4–V6</td>
<td>TWI in V1, resolution of STE in V1–V2</td>
</tr>
<tr>
<td>Case 2</td>
<td>M/48</td>
<td>Hypotension</td>
<td>Type-2: prominent “saddle-back” STE in V1–V4</td>
<td>S1–Q3, STD in I, aVL, V6, STE in III, aVF</td>
<td>TWI in V1 to V3, resolution of STE in V1–V4</td>
</tr>
<tr>
<td>Varricka [5]</td>
<td>M/50</td>
<td>The patient was found unresponsive</td>
<td>Type-1 or 2: STE in V1–V2</td>
<td>RBBB, other manifestations not described</td>
<td>RBBB resolution and STE in V1–V2</td>
</tr>
</tbody>
</table>

G/A = gender/age; F = female; M = male; BrP = Brugada phenocopy; APE = acute pulmonary embolism; STE = ST-segment elevation; TWI = T-wave inversion; ST = sinus tachycardia; RBBB = right bundle branch block; STD = ST-segment depression.

Please cite this article as: Zhan Z-Q et al, Brugada phenocopy in acute pulmonary embolism, Int J Cardiol (2014), http://dx.doi.org/10.1016/j.ijcard.2014.09.046
The two new cases should be classified as Type-1 (Fig. 1 before thrombolysis) and Type 2 (Fig. 2 before thrombolysis) BrP, respectively. However, they should be regarded as Class B, indicating that not all criteria for the diagnosis of BrP have been met (no sodium blocker testing was performed).

In conclusion, BrP associated with APE is apparently a rare phenomenon. Prompt recognition helps in avoiding false diagnosis leading to unnecessary medical tests and treatments.

The authors of this manuscript certify that they comply with the Principles of Ethical Publishing in the International Journal of Cardiology.

Conflict of interest

The authors declare no conflicts of interest or financial disclosures.

References


