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Syncope in a patient with acute pulmonary embolism and Brugada Type-2 ECG pattern: Brugada phenocopy or Brugada syndrome?☆

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ABSTRACT

Brugada phenocopies are clinical entities characterized by electrocardiographic patterns that are identical to true Brugada syndrome, but are elicited by a number of clinical circumstances. ECG normalizes upon resolution of underlying condition, family history of arrhythmic syncope or ventricular arrhythmias is strictly absent and provocative tests with sodium channel blockers have to be negative. We describe herein the case of type-2 ECG Brugada pattern in a patient with acute pulmonary embolism presenting with recurrent syncope but negative provocative test with sodium channel blockers. Transthoracic echocardiography and transcranial Doppler did not show atrial septal defect. In conclusion, to the best of our knowledge no other cases excluded atrial septal defect and paradoxical embolism as a possible cause of acute pulmonary embolism related Type-2 Brugada ECG pattern. Therefore in our case right ventricle transmural myocardial ischemia due to acute pulmonary embolism, mainly secondary to right ventricle stretch, could explain Brugada ECG pattern.

Introduction

Brugada syndrome (BrS) is an autosomal dominant genetic disease electrocardiographically characterized by marked ST-segment elevation in the right precordial ECG leads and is considered a primary electrical heart disease associated with a high incidence of sudden and unexpected arrhythmic death. Brugada phenocopy (BrP) encopasses a number of clinical entities characterized by ECG patterns that, while identical to true BrS, are elicited by various clinical conditions. Moreover, ECG normalization upon resolution of underlying condition, absence of family history of arrhythmic syncope or ventricular arrhythmias, and negative provocative tests with sodium channel blockers, are typical [1]. We describe and subsequently discuss the case of Type-2 ECG Brugada pattern likely due to acute pulmonary embolism-related mycordial ischemia.

Case report

A 60-year-old male without a history of cardiac disease was admitted to the Emergency Department (ED) due to episodes of recurrent syncope. He reported repetitive syncope for the last two days but denied chest pain, dyspnea, cough or hemoptysis. On arrival, he was hemodynamically stable, his blood pressure was 120/75 mmHg. Cardiac auscultation revealed a normal S1, loud and split S2. A 12 lead electrocardiogram showed sinus rhythm, Brugada Type-2 ECG pattern ("saddle-back") with ST-segment elevation (STE) ≥2 mm from the isoelectric baseline without T-wave inversion in the right precordial leads V1–V2 (Fig. 1A). Blood tests revealed raised levels of troponin (0.086 ng/ml, normal range: 0–0.02 ng/ml), brain natriuretic peptide (2496.0 pg/ml, normal range: 0–100 pg/ml) and D-dimer (14.0 mg/l, normal range: 0–0.5 mg/l). Blood gas showed pO2 of 66 mmHg, pCO2 of 27 mmHg and pH of 7.45. Echocardiography revealed enlarged right heart and mild tricuspid regurgitation. Systolic pulmonary artery pressure, by tricuspid regurgitation pressure gradient, was 48 mmHg and left ventricular ejection fraction was 58%. Twenty-four hours Holter monitoring did not show any evidence of ventricular arrhythmias. Acute pulmonary embolism (APE) was highly suspected and this was confirmed by computed tomographic pulmonary angiogram. Oxygen and subcutaneous fondaparinux were promptly started. Seven days after admission, he recovered fully and was discharged with rivaroxaban. ECG at discarge showed resolution of the Type-2 Brugada pattern (Fig. 1B). ECG changes did not recur on follow up and an ECG Holter performed three months later showed no arrhythmia.

Transthoracic echocardiography bubble study and transcranial Doppler did not show atrial septal defect (ASD) (Fig. 2A and B). The ajmaline test planned six months after the acute event was negative (Fig. 2C and D).

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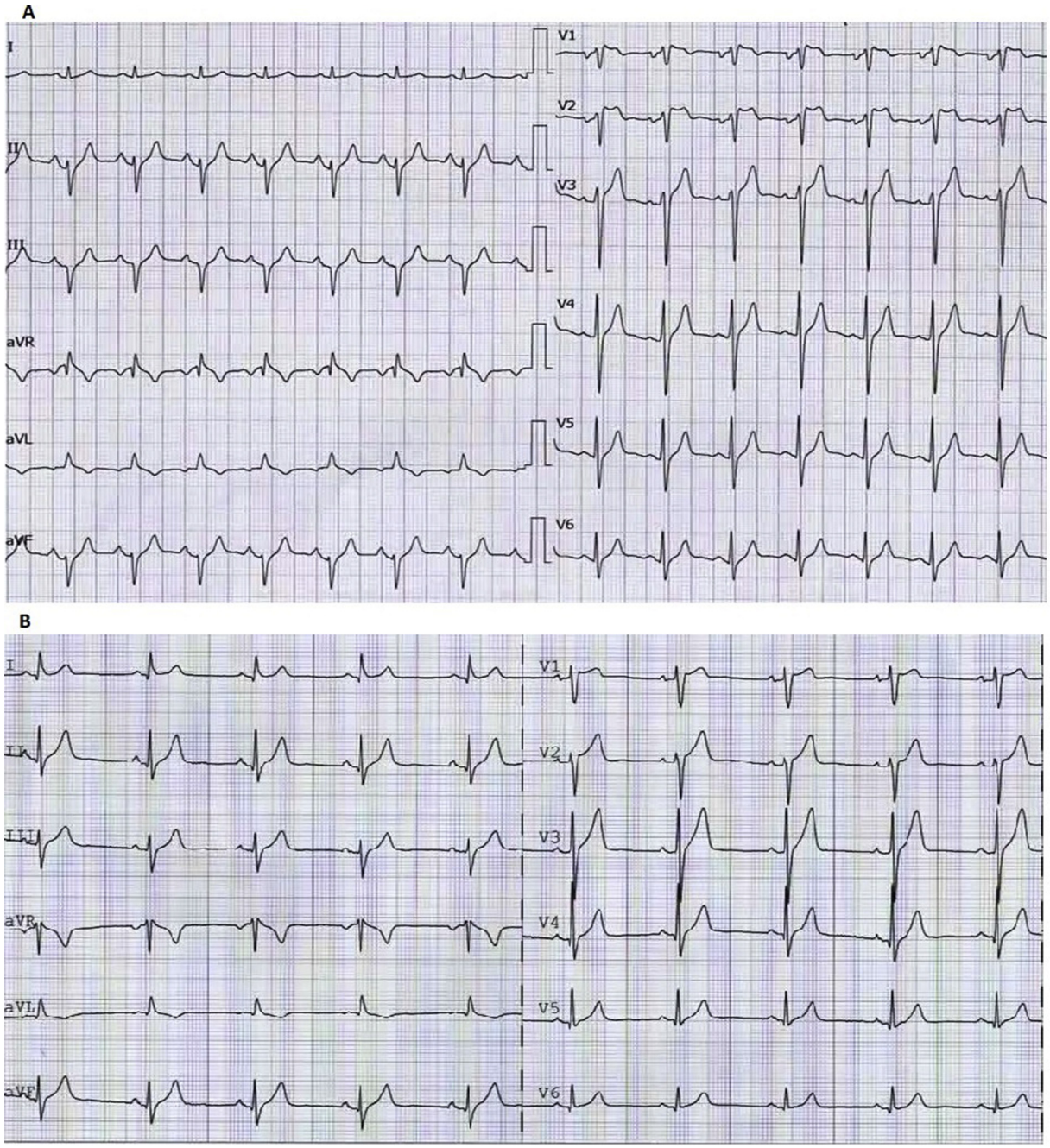

Fig. 1. (A) ECG at presentation to the Emergency Department showes Brugada Type-2 ECG pattern ("saddle-back") with ST-segment elevation ≥2 mm from the isoelectric baseline in the right precordial leads V1–V2. (B) ECG at discarge showes resolution of the Type-2 Brugada pattern.

Discussion

BrP form a group of heterogeneous conditions that are the most difficult to differentiate from true congenital BrS due to identical ECG patterns. A systematic diagnostic approach is crucial to avoid diagnostic and therapeutic paths that are sometimes burdened with considerable risks for the patient. Few clinical descriptions of BrP observed during APE are reported in the literature [2] and are properly summarized in the International Registry and Educational Portal on BrP [3]. As in our case, two other cases presented in ED with recurrent syncope. Based on current diagnostic and morphological criteria proposed by Anselm et al. [4], these two cases should be classified as Type-1 and Type 2 BrP respectively, but should also be regarded to as Class B, indicating that not all criteria for the diagnosis of BrP were met. Indeed, there was a prompt resolution of the STE in leads V1–V3 once the patient's condition resolved, but no sodium blocker testing was performed. Wynne et al. [5] reported a case of APE associated with a typical type 1 Brugada ECG pattern and suggested that the most likely mechanism for APE-related anterior ST-segment elevation was paradoxical embolization to the left anterior descending coronary artery through a patent foramen ovale. This would suggest that APE associated with a Brugada ECG pattern is due to ischemia in the context of a pre-existing atrial septal defect (ASD). In this case no diagnostic testing (i.e. transthoracic echocardiography and transcranial Doppler) was performed in order to show a pre-existing ASD. In our case Type-2 Brugada ECG patterns was found in the context of APE but ultrasonographic assessment of right-to-left shunting, either by transthoracic echocardiography or using transcranial Doppler, excluded ASD. Therefore in our case paradoxical embolism causing ischemia is unlikely. As suggested by Anselm et al. [4], a drug-challenge using sodium channel blockers such as

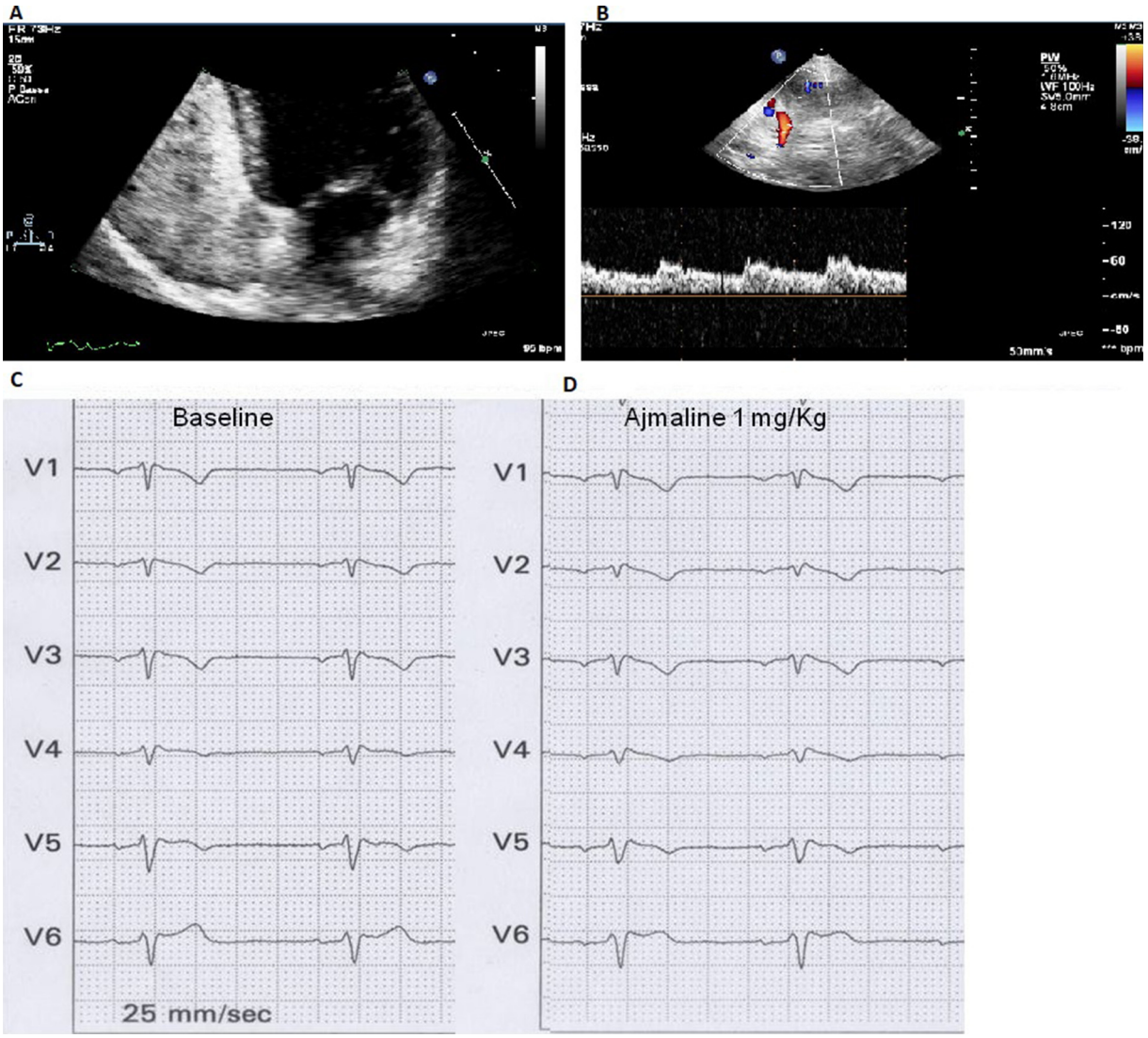

Fig. 2. (A) Apical 4-chamber view after intravenous contrast administration: microbubbles are not visualised in the left side of the heart; (B) Transcranial Doppler study of intracranial middle cerebral artery after intravenous contrast admistration do not show microembolic signals; (C) Right precordial leads placed in the II° (V1–V2), III° (V3–V4) and IV° (V5–V6) intercostal space at baseline and (D) after infusion of ajmaline 1 mg/kg in 5 min.

ajmaline, procainamide or flecainide should be used to differentiate BrP from BrS. Therefore, six months after the acute event, we performed ajmaline test which was negative. As reported in literature, right ventricle (RV) ischemic involvement could explain STE presenting as a Type-1 or 2 BrP during an acute ischemic cardiac event [6,7]. Transmural myocardial ischemia during APE, mainly secondary to RV stretch, could cause Brugada ECG pattern. Elevated right ventricle pressures causing transient sodium channel dysfunction in the right ventricular outflow tract remains speculative. In conclusion our case meets all criterias for the clinical diagnosis of Type-2, Class A BrP: (1) clinical factors (APE as identifiable underlying condition that triggers the ECG pattern and complete absence of family history of syncope or malignant ventricular arrhythmias); (2) ECG morphologic characteristics (identical to true BrS but disappearing on resolution of the triggering underlying condition), (3) negative results on provocative testing with sodium channel blockers. To the best of our knowledge no other cases excluded ASD and a paradoxical embolism as a possible cause of Type-2 Brugada ECG pattern. Therefore in our case APE-related transmural myocardial ischemia, mainly secondary to RV stretch, could explain Brugada ECG pattern.

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