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A rare case of posterolateral ST-elevation myocardial infarction in Brugada syndrome: A double trouble beyond mimicking

Short title: Posterolateral ST-elevation myocardial infarction in Brugada syndrome

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Brugada syndrome (BrS) is an inherited primary arrhythmia syndrome distinguished by ST-segment elevation with type-1 morphology ≥2 mm in ≥1 lead in the right precordial leads V1 and V2 positioned in the 2nd, 3rd, or 4th intercostal space. It significantly increases the risk of sudden cardiac death, even in the absence of major structural heart disease [1]. Limited information is available regarding acute myocardial infarction in BrS [2]. Our report highlights a case involving the coexistence of BrS and posterolateral acute myocardial infarction, a scenario that may have led to a diagnostic oversight.

The patient is a 64-year-old man who had two episodes of syncope within 24 hours before being admitted to the hospital. He experienced palpitations before the syncope and presented with symptoms consistent with a myocardial infarction upon admission to the emergency department. The physical examination revealed hypotension, and

electrocardiogram (ECG) showed sinus rhythm with coved-type ST elevation, inverted T waves in V1 and V2, and ST elevation in leads I, aVL, V5–V6, V7–V9 (Figure 1 A–B).

After assessing the patient, a working diagnosis of posterolateral acute myocardial infarction and BrS was established. Additionally, there was a consideration of Brugada phenocopy in the differential diagnosis. Fibrinolysis with streptokinase was administered, and the patient's hemodynamic parameters improved significantly. After the procedure, the patient reported no further complaints, and ST-segment resolution was reduced by more than 50% (Figure 1C). Bedside echocardiography revealed mildly abnormal LV systolic function and signs of takotsubo cardiomyopathy (Supplementary material, *Figures S1–S3*). A routine early PCI strategy showed no stenosis in the coronary arteries, leading to a diagnosis of myocardial infarction with non-obstructive coronary arteries (MINOCA) (Supplementary material, *Figures S4–S5*) [3]. Coronary spasm was considered as a plausible differential diagnosis [4].

The patient was managed in the intensive care unit with dual antiplatelet therapy, unfractionated heparin, high-intensity statin, and norepinephrine. The patient no longer showed signs and symptoms of a myocardial infarction after receiving treatment. Despite the resolution of the myocardial infarction, the ECG still indicates Brugada morphology type 1. The ECG monitoring before discharge and at the outpatient clinic also shows Brugada morphology type-1 (Supplementary material, *Figure S6*).

The ECG still shows Brugada morphology type 1 despite the myocardial infarction being resolved. This does not align with the definition of Brugada phenocopy, which states that the ECG should return to normal after the underlying disease is resolved. Other potential causes have been ruled out including myopericarditis, fever, and electrolyte imbalance [5]. The presence of symptoms accompanied by a type 1 Brugada morphology in the ECG indicates a high clinical pretest probability of true BrS than Brugada phenocopy. The patient declined implantable cardioverter defibrillator insertion or catheter ablation.

The case involves the convergence of electrocardiographic findings, showing both ST depression indicative of posterior myocardial infarction and ST-elevation characteristic of BrS. This can lead to potential misdiagnosis. Understanding the counteractive influence of the ST vectors in both conditions is crucial.

Supplementary material

Supplementary material is available at https://journals.viamedica.pl/polish_heart_journal.

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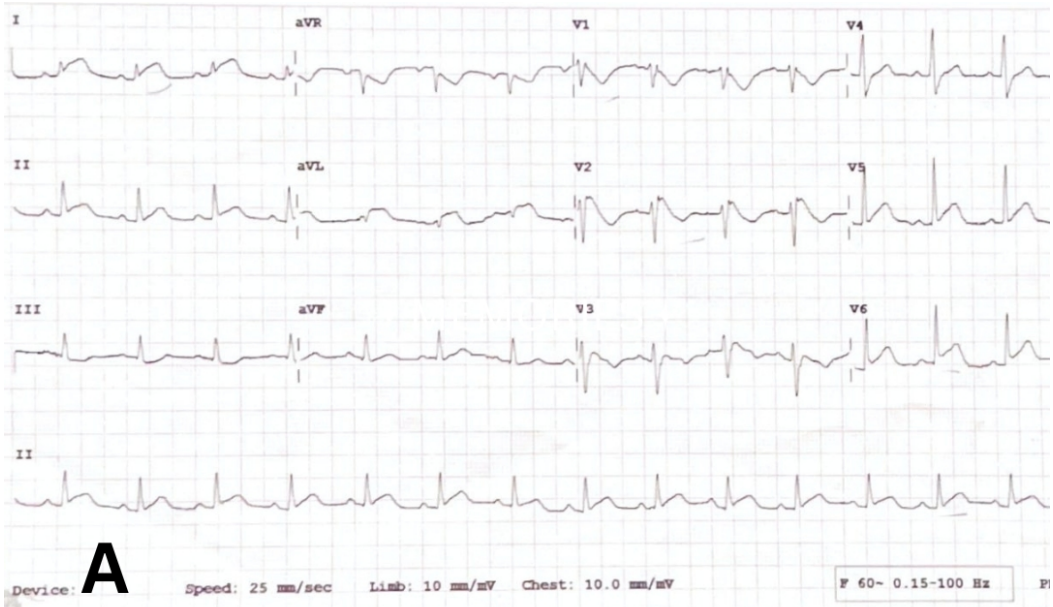
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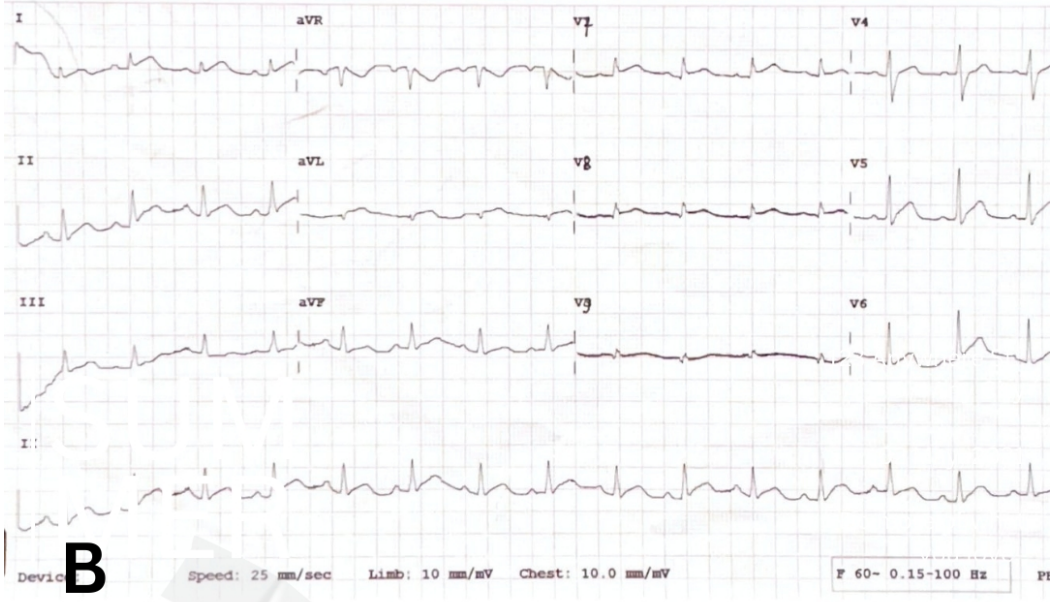
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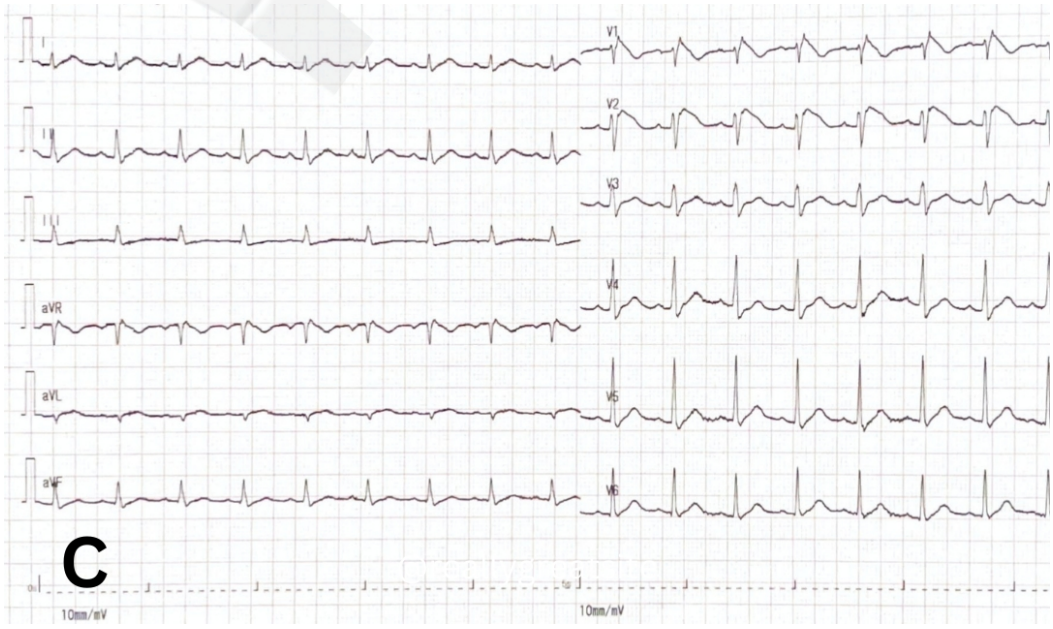
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Figure 1. A. Electrocardiography at admission showed ST-segment elevation I, AVL, V5–V6, and ST-segment elevation coved-type morphology leads V1–V2. **B**. Electrocardiography at admission showed ST-segment elevation leads V7–V9. **C**. Electrocardiography after fibrinolytic revealed resolution ST-segment elevation lead 1, AVL, V5–V6, and ST-segment elevation coved-type morphology leads V1–V2