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

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DOI: 10.33963/v.phj.103242

Received:
June 2, 2024

Accepted:
October 24, 2024

Early publication date:
November 5, 2024

Brugada syndrome (BrS) is an inherited primary arrhythmia syndrome distinguished by ST-segment elevation with type-1 morphology ≥2 mm in ≥1 lead in 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 diseases [1]. Limited information is available regarding acute myocardial infarction in BrS [2]. We report a case involving the coexistence of BrS and posterolateral acute myocardial infarction, a scenario that may have led to a diagnostic oversight.

A 64-year-old man had two episodes of syncope within 24 hours before being admitted to the hospital. He had experienced palpitations before the syncope and presented with symptoms consistent with myocardial infarction upon admission to the emergency department. A physical examination revealed hypotension, and an electrocardiogram (ECG) showed sinus rhythm with coved-type ST-segment elevation, inverted T waves in V1 and V2, and ST-segment 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 symptoms, and ST-segment resolution was reduced by more than 50% (Figure 1C). Additionally, echocardiography revealed mildly abnormal LV systolic function and signs of takotsubo cardiomyopathy (Supplementary material, *Figures S1–S3*). A routine early percutaneous coronary intervention 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 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. After receiving treatment, the patient no longer showed signs and symptoms of a myocardial infarction. Despite the resolution of myocardial infarction, ECG still indicated type 1 Brugada morphology. ECG monitoring before discharge and in the outpatient center also showed type 1 Brugada morphology (Supplementary material, *Figure S6*). That did not align with the definition of Brugada phenocopy, stating that ECG should return to normal after the underlying disease is resolved. Other potential causes had been ruled out, including myopericarditis, fever, and electrolyte imbalance [5]. The presence of symptoms accompanied by type 1 Brugada morphology on ECG indicated a high clinical pretest probability of true BrS rather than Brugada phenocopy. The patient declined implantable cardioverter defibrillator or catheter ablation.

The case involved the convergence of electrocardiographic findings, showing both ST-segment depression indicative of posterior myocardial infarction and ST-segment elevation characteristic of BrS. That could have led to potential misdiagnosis. Understanding the counteractive influence of the ST-segment vectors in both conditions is crucial.

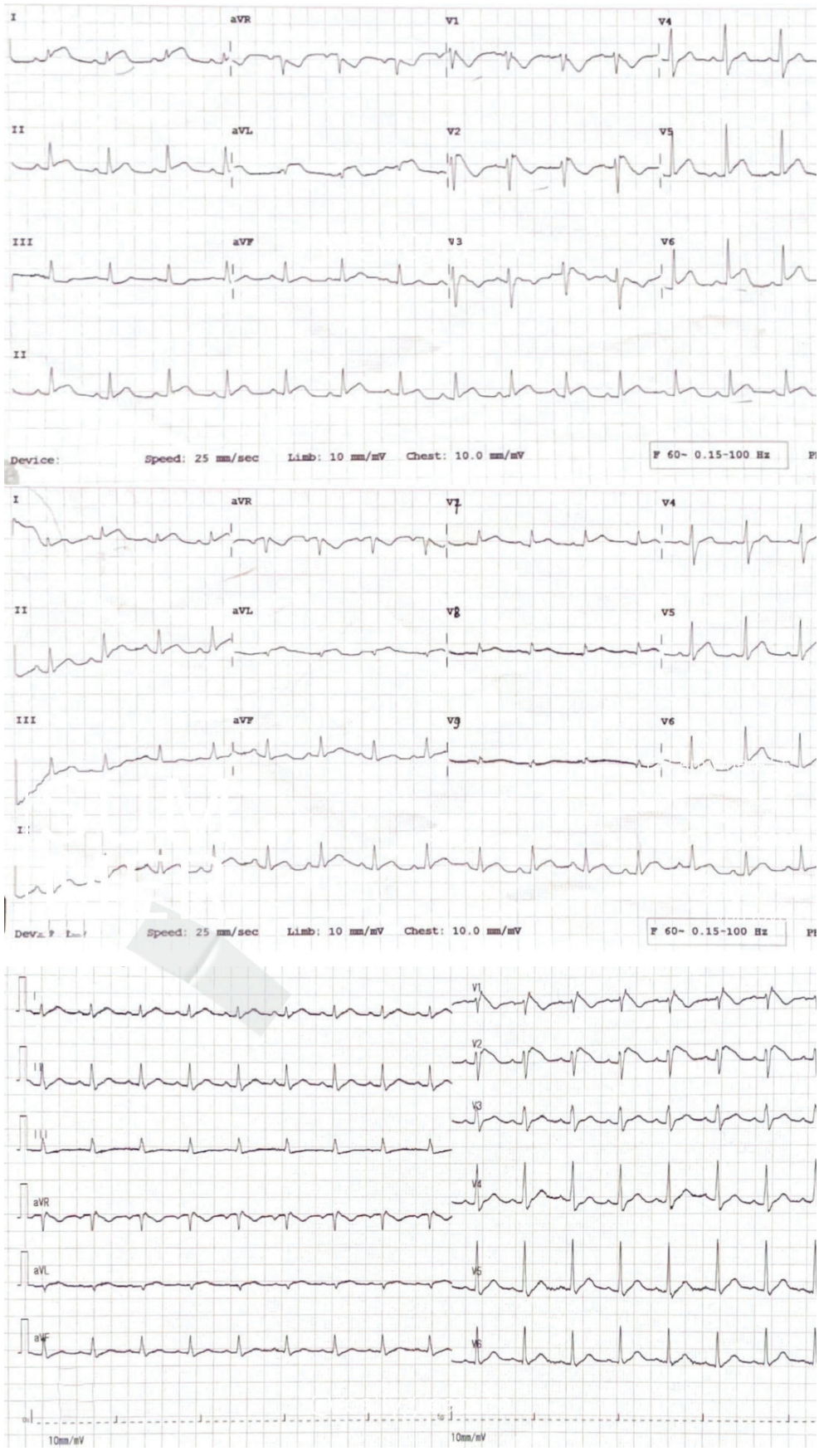

Figure 1. **A**. Electrocardiography on admission showed ST-segment elevation in leads I, AVL, V5–V6, and ST-segment elevation coved-type morphology in leads V1–V2. **B**. Electrocardiography on admission showed ST-segment elevation in leads V7–V9. **C**. Electrocardiography after the fibrinolytic revealed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

Supplementary material

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

Article information

Conflict of interest: None declared.

Funding: None.

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