

Unusual ST-Segment Elevation in the Anterolateral Precordial Leads

Ischemia, Brugada Phenocopy, Brugada Syndrome, All, or None?

ECG CHALLENGE

The patient is a 65-year-old white male with history of type 2 diabetes mellitus, hypertension, chronic smoking, and prior stroke with residual left hemiparesis and aphasia. His medication included losartan, furosemide, simvastatin, and metformin.

He was admitted to the emergency room in cardiac arrest and was quickly resuscitated with cardiopulmonary resuscitation and external electric cardioversion maneuvers. Immediately after return of spontaneous circulation, a 12-lead ECG was performed (Figure 1).

Based on the ECG, what is the most likely etiology of his cardiac arrest?
Please turn the page to read the diagnosis.

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Figure 1. ECG performed immediately after cardiopulmonary arrest reversion.

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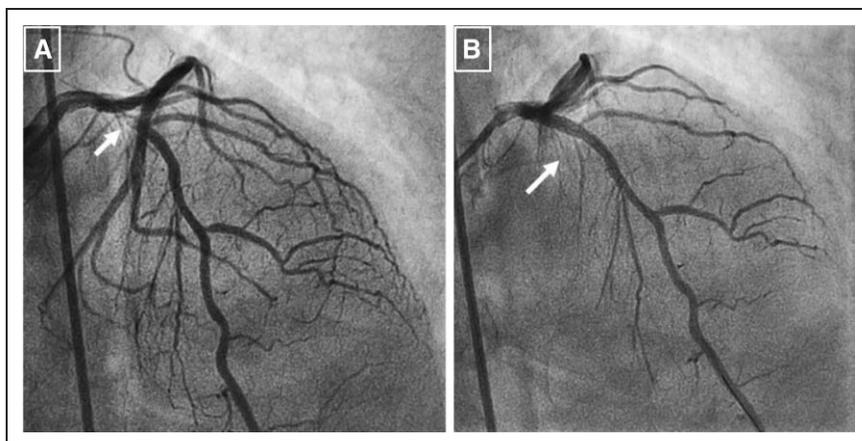


Figure 2. Coronariography with and without obstruction.

Left anterior oblique: left anterior descending coronary artery with 80% proximal obstruction with thrombus (A) and after drug-eluting stent placement (B).

RESPONSE TO ECG CHALLENGE

The first ECG shows accelerated junctional rhythm and a heart rate of 94 bpm, with J point and anterior ST elevation >2 mm. The correct diagnosis was acute ST elevation myocardial infarction. The patient underwent coronary angiography, which revealed proximal subocclusion of the left anterior descending coronary artery, with significant thrombus (Figure 2) successfully revascularized after placement of a drug-eluting stent. After the coronary intervention, we performed another ECG shown in Figure 3.

The initial ECG on presentation was compatible with ischemia-induced atypical Brugada phenocopy (BrP),¹ confirmed later by negative provocative ajmaline test, indicating low probability of true Brugada syndrome (BrS).

Brugada Phenocopy Emerging as a New Concept

BrP is a clinical entity in which patients present with an ECG pattern identical to either type 1 or 2 Brugada ECG patterns, yet it differs etiologically from true BrS. The defining feature of BrP is the absence of true congenital BrS. Therefore, a provocative testing with a sodium channel blocking agent such as ajmaline, flecainide, or procainamide will not reproduce the typical type 1 Brugada ECG pattern. The diagnostic criteria we have suggested for BrPs are the following (I–V are mandatory)^{1,2}:

- I. Type 1 or 2 Brugada ECG pattern
- II. Underlying identifiable condition to explain the Brugada-like pattern on ECG

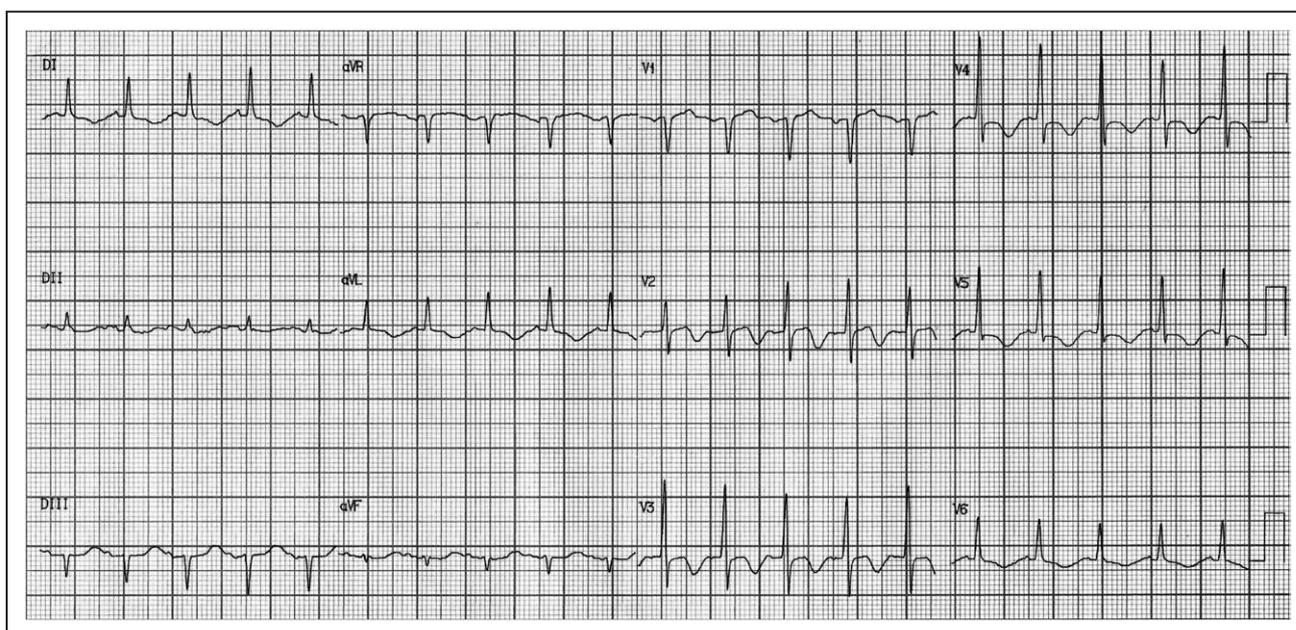


Figure 3. ECG performed after percutaneous coronary intervention.

Absence of Q wave infarction initially, loss of ST elevation with QRS narrowing. These changes are likely representative of reperfusion T wave inversions afterward. The postischemic diffuse T wave inversion is observed in the anterolateral wall, coincident with left anterior descending (LAD) territory, including its first diagonal branch (proximal LAD territory obstruction).

- III. The ECG pattern immediately resolves on resolution of the underlying condition
- IV. Low clinical pretest probability of true BrS determined by a lack of symptoms and medical and family history
- V. Negative provocative testing with ajmaline, flecainide, or procainamide
- VI. Provocative testing is not mandatory if surgical right ventricular outflow tract manipulation has occurred within the last 96 hours
- VII. Negative genetic screening (mutations are identifiable in only 20% to 30% of cases affected by true BrS)

Recently, Alper et al¹ showed the first case of BrP with atypical type 1 Brugada ECG pattern located in inferior leads, emphasizing that Brugada-like syndromes have been reported to present as ST elevation in inferior leads.³

The potential mechanisms and pathophysiology underlying BrP remain unclear. BrPs have been reported under a multitude of clinical circumstances in the following distinct etiologic categories^{1,2}: metabolic conditions, endocrine disease (ie, hypopituitarism), electrolyte imbalances, mechanical compression (mediastinal tumors, pectus excavatum), ischemia-induced (the present case), myocardial and pericardial disease, acute pulmonary embolism, and others. For a detailed list of conditions, please refer to the Educational Portal and International Registry on Brugada Phenocopies (www.brugadaphenocopy.com).

DISCLOSURES

None.

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FOOTNOTES

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