CASE REPORT

Lateral “Coved” ST-Segment Elevation: Exceptional but Malignant Electrocardiographic Sign in a Patient with Brugada Syndrome

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Ann Noninvasive Electrocardiol 2017;22(3):e12221, <https://doi.org/10.1111/anec.12221>

lateral ST-segment elevation; atypical Brugada ECG pattern; Brugada syndrome

CASE REPORT

We report a case of a healthy 32-year-old male, with no family history of sudden cardiac death, who was admitted because of syncope and palpitations. A surface 12-lead ECG showed typical, “coved” type ST-segment elevation in the right precordial leads V₁–V₂. A 12-lead Holter monitoring revealed spontaneous, dynamic “coved” ST-segment changes in leads I, aVL, and V₁–V₃ (Fig. 1). Echocardiography excluded structural heart disease. A stress-test did not induce ischemia or arrhythmias. An electrophysiology study did not induce arrhythmias. An implantable cardioverter-defibrillator (ICD) was implanted due to syncope and spontaneous type 1 Brugada ECG pattern. Three weeks later, the patient presented an electrical storm (6 appropriate ICD therapies –5 ATP for VT and 1 shock for VF). One month later, an episode of VF was successfully treated by the ICD. Quinidine therapy at 600 mg daily was started. No arrhythmia recurrences were observed in a follow-up period of 6 months.

DISCUSSION

The typical ECG pattern in patients with Brugada syndrome (BrS) includes “coved” ST-segment elevation (≥ 2 mm J point elevation) in at least one of the right precordial leads.¹ However, sporadic cases have been reported in which the “coved-type” Brugada ECG pattern was also observed in the inferior or lateral ECG leads.^{2–5}

Sarkozy et al. reported in a large cohort of patients with BrS (280 patients) that the “coved” Brugada pattern did not occur spontaneously in the inferior or lateral leads but rather after challenging with sodium blockers.⁶ These data suggest that the presence of spontaneous “coved” ST elevation in the inferior–lateral leads is an exceptional finding in BrS. In contrast, class I antiarrhythmic drugs administration provoked “coved” Brugada pattern in the inferior or lateral leads in 4.6% of their study population. The localization of the “coved” Brugada pattern was the lateral leads in only one patient, corresponding to 0.4% of their database.

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Figure 1. (A) shows “coved” ST segment elevation in leads V_1 , V_2 and malignant repolarization pattern (terminal QRS slurring-lambda like) in leads aVL and I (black arrows). (B) shows more prominent “coved” ST segment elevation in leads V_2 , V_3 , and more prominent terminal QRS slurring in leads I, aVL. (C) shows discrete changes observed only in lead I, as comparing to Panels A and B, with disappearance of negative T wave. (D) black arrows indicate QRS fragmentation in lead V_2 . (E) shows “parietal block,” there is a mismatch in QRS duration between leads V_2 and V_6 .

This finding suggests that “coved” Brugada pattern in the lateral leads is a very rare variant. In turn, “coved” Brugada pattern was provoked in the inferior leads in 4.3%.⁶

There have been also two case reports, by Bonakdar et al. and by van den Berg et al., on patients with Brugada syndrome who had alternating (daily variations) ST-segment elevation in the right precordial leads and the high lateral leads (I and aVL).^{4,7} Letsas et al. reported a patient with Brugada “saddle-back” or type 2 Brugada pattern in the right precordial leads and early repolarization pattern (spontaneous; with J wave and ST segment elevation but not “coved” type pattern) in the high lateral leads (I, aVL).⁸

Here we describe a unique and interesting ECG variant of BrS with simultaneous and spontaneous combination of the typical “coved” type ST-segment elevation in the right precordial and high lateral leads. This rare electrocardiographic variant seems

to be a malignant form of BrS (history of frequent syncope and electrical storm). The presented case has also other previously described ECG markers of risk:

- (1) aVR sign: prominent final positive “aVR sign” ($R \geq 0.3$ mV or 3 mm or R/q ratio ≥ 0.75).⁹
- (2) Fragmented QRS (fQRS)—seen transiently in lead V_2 (Fig. 1D).
- (3) Malignant early repolarization pattern in lateral leads resembling the Greek letter lambda (Fig. 1A).¹⁰
- (4) Parietal block¹ (Fig. 1E).

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