

Postextrasystolic unmasking of Brugada electrocardiogram

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A 50-year-old patient presented to our emergency department due to syncope without prodromi during sitting with subsequent head injury. Medical history was inconspicuous except one exertional syncope during childhood with unremarkable medical evaluation. Family history for sudden death was negated, but the father was reported to suffer from epilepsy. The patient did not take any medications.

A 12-lead electrocardiogram (ECG) on admission (*Figure 1A*) showed sinus rhythm with right axis deviation, borderline PQ interval, and a broad fractionated QRS complex with right bundle branch block (RBBB) appearance. In order to seek for transient repolarization or depolarization abnormalities, repeat ECGs with rhythm strips were recorded and revealed several broad monomorphic premature ventricular complexes. In a single ECG, one postextrasystolic beat with Type 1 Brugada ECG pattern could be recorded during extended rhythm ECG strip (*Figure 1A*, asterisk).

Due to high risk ECG features the patient was admitted to the telemetry ward and serial 12-lead rhythm strips were assessed. Transthoracic echocardiography as well as coronary angiography were inconspicuous. Furthermore a cardiac magnetic resonance imaging was performed confirming normal right and left ventricular volumes and function without signs of late gadolinium enhancement.

Serial ECGs revealed exceptional dynamics with intermittent Type 1 Brugada ECG during slower heart rates in the morning (*Figure 1B*), changing into the most prevailing ECG with complete RBBB during faster heart rates in terms of a frequency dependent bundle branch block completely masking the Brugada ECG (*Figure 1C*, right side). Due to spontaneous Type 1 Brugada ECG and syncope without prodromi the diagnosis of Brugada syndrome was made and a dual chamber implantable cardioverter-defibrillator (ICD) was implanted. Drug challenge with sodium channel blockers was not performed due to lack of consequence.

The challenge of diagnosing Brugada syndrome in patients with complete RBBB has been described before.¹ Besides repeated ECG recordings and pacing from the right ventricle for resolution of complete RBBB, performance of pharmacological tests has been reported to be useful to demonstrate typical ST-segment elevation.¹ However, caution should be advised when sodium channel blockers are administered to patients in whom complete RBBB completely masks Type 1 ECG as this may induce further conduction disturbance or ventricular fibrillation storm.² As patients with Brugada syndrome masked by complete RBBB have been reported to carry the same risk of fatal ventricular tachyarrhythmia as other patients with Brugada syndrome unmasking is important to exclude potential high-risk patients.²

Six months follow-up was unremarkable without syncope or ICD interventions. Genetic counselling was performed, but genetic testing was refused by the patient and his family.

This case highlights the importance of performing repeat 12-lead rhythm ECG strips in patients with syncope and again demonstrates the exceptional ECG dynamics in channelopathies such as the Brugada syndrome.

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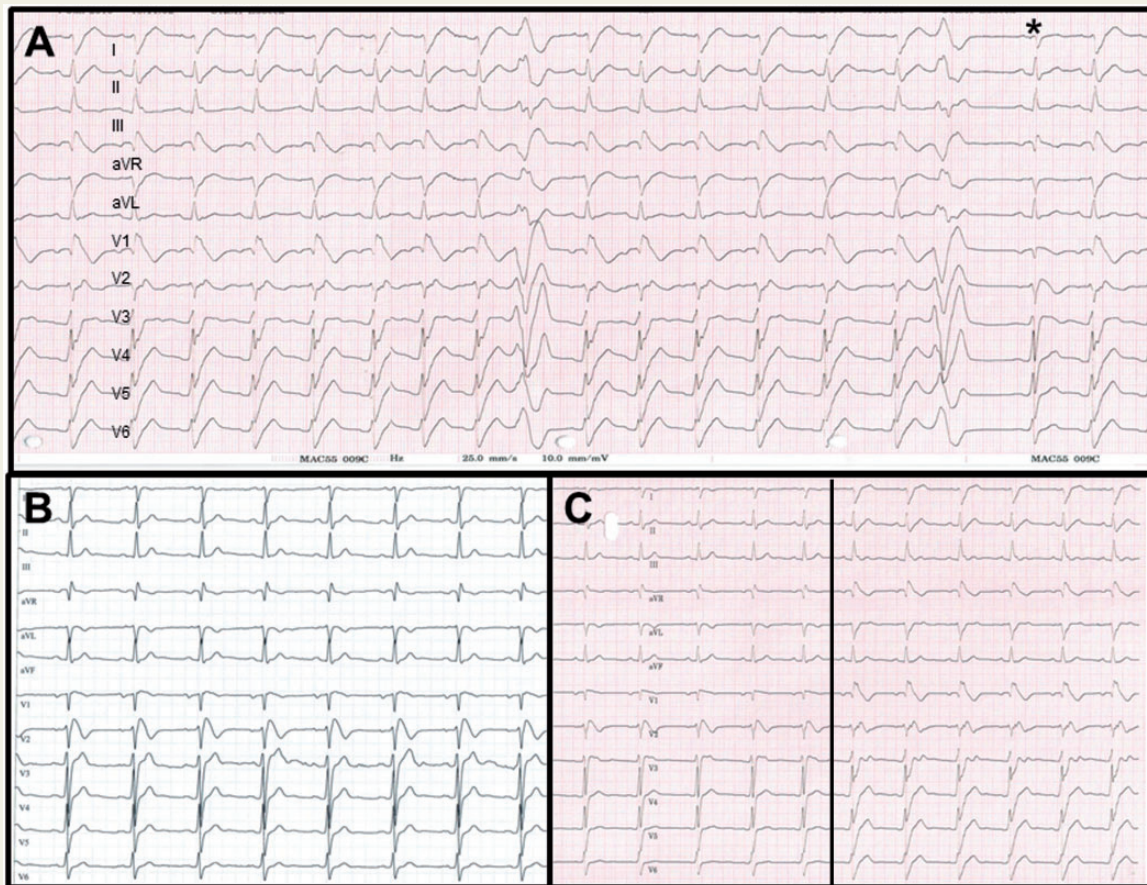


Figure 1 12-lead electrocardiogram (ECG) on admission (Panel A) showing sinus rhythm with right axis deviation, borderline PQ interval and a broad fractionated QRS complex with right bundle branch block (RBBB) appearance with several broad monomorphic premature ventricular complexes. During extended rhythm ECG strip one postextrasystolic beat with type 1 Brugada ECG pattern could be recorded (Panel A, asterisk). 12-lead ECG with type 1 Brugada ECG pattern during slower heart rates in the morning (Panel B), changing into the most prevailing ECG with complete RBBB during faster heart rates in terms of a frequency dependent bundle branch block completely masking the Brugada ECG (Panel C, right side).

References

1. Aizawa Y, Takatsuki S, Sano M, Kimura T, Nishiyama N, Fukumoto K *et al*. Brugada syndrome behind complete right bundle-branch block. *Circulation* 2013;**128**:1048–54.
2. Wada T, Nagase S, Morita H, Nakagawa K, Nishii N, Nakamura K *et al*. Incidence and clinical significance of Brugada syndrome masked by complete right bundle-branch block. *Circ J* 2015;**79**:2568–75.