

EP CASE REPORT

Optimizing the detection of macroscopic T-wave alternans using high precordial leads in a patient with Brugada syndrome

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Case presentation

Type 1 Brugada electrocardiogram (ECG) becomes more clear when using higher precordial leads.¹ T-wave alternans (TWA) is associated with arrhythmia risk in Brugada syndrome (BrS).^{2,3} However, manifested and accentuated TWA in an ECG recorded at a higher intercostal space has not been reported.

A 31-year-old man was resuscitated after cardiopulmonary arrest due to VF. He had no family history of sudden cardiac death. Physical examination, laboratory tests, echocardiography, brain computed tomography, coronary angiography, and left ventriculography did not show any abnormalities. Five days after resuscitation, standard 12-lead ECG showed spontaneous macroscopic TWA in the V2 lead. In addition, QT interval alternans (480–520 ms) with changes in the degree of a deeply negative T wave were observed (Figure 1A, arrows). At the same time, an ECG recorded with the V1–V6 leads placed one intercostal space higher showed pronounced TWA in the V2 lead. Furthermore, TWA manifested in the V1 and V3 leads (Figure 1B, arrows). The patient was diagnosed with BrS and received an implantable

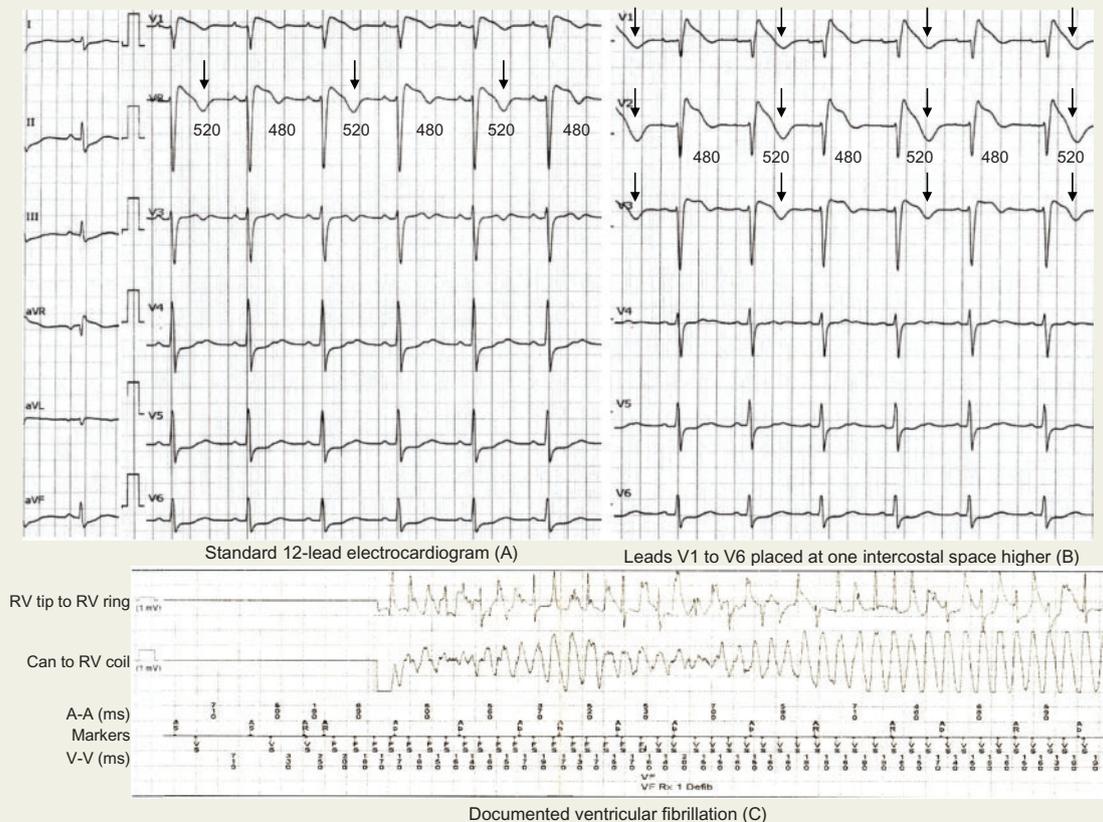


Figure 1 (A) Note the spontaneous macroscopic TWA with beat-to-beat augmentation of the negative T-wave in the V2 lead. Note the QT interval alternans (480–520 ms), with changes in the degree of the deeply negative T-wave (arrows). (B) Note that macroscopic TWA was more pronounced in the V2 lead and that a macroscopic TWA was confirmed with the accentuation of ST-segment elevation and the following negative T-wave in the V1 and V3 leads. No remarkable ST-segment elevation or TWA was observed in the V4–V6 leads. (C) Documented VF from the patient's implantable cardioverter defibrillator.

cardioverter defibrillator (ICD). During the year after implantation, he received 18 appropriate ICD shocks for episodes of documented VF accompanied by occasional loss of consciousness (12 episodes occurred during the night) (Figure 1C). No appropriate ICD shocks have been delivered in the 2 years since he received oral quinidine 1200 mg/day and cilostazol 200 mg/day (body weight 54 kg).

Fish and Antzelevitch suggested that TWA in BrS could be generated by loss of the epicardium action potential dome in the right ventricular outflow tract or/and concealed phase 2 reentry on alternate beats. They also showed that the degree of TWA with a following deeply negative T-wave is predictive of developing VF.² Regional distribution of TWA in BrS has been reported.³ ECG recording with the V1 and V2 leads placed at a higher intercostal space increases the sensitivity for detecting a type 1 Brugada ECG.¹ Therefore, a less obvious TWA recorded with a standard ECG may be more obvious when using higher precordial leads.

This is the first report in the literature suggesting that ECG using higher intercostal spaces may not only optimize the detection of ST-segment elevation but also augment the degree of the deeply negative T-wave, leading to verification of macroscopic TWA and ultimately the identification of patients with BrS who are at a high risk for VF.

Acknowledgements

We are grateful to Tomoki Kubota and Nobuhiro Takasugi for drafting this report.

Conflict of interest: none declared.

References

1. Shimizu W, Matsuo K, Takagi M, Tanabe Y, Aiba T, Taguchi A et al. Body surface distribution and response to drugs of ST segment elevation in Brugada syndrome: clinical implication of eighty-seven-lead body surface potential map-ping and its application to twelve-lead electrocardiograms. *J Cardiovasc Electrophysiol* 2000;**11**:396–404.
2. Fish JM, Antzelevitch C. Cellular mechanism and arrhythmogenic potential of T-wave alternans in the Brugada syndrome. *J Cardiovasc Electrophysiol* 2008;**19**:301–8.
3. Uchimura-Makita Y, Nakano Y, Tokuyama T, Fujiwara M, Watanabe Y, Sairaku A et al. Time-domain T-wave alternans is strongly associated with a history of ventricular fibrillation in patients with Brugada syndrome. *J Cardiovasc Electrophysiol* 2014;**25**:1021–7.