

Case Report

Association of Syncope and Atrioventricular Nodal Reentrant Tachycardia in a Patient with Brugada-type Electrocardiogram—Importance of Electrophysiologic Study in Differential Diagnosis of Wide QRS Tachycardia—

Hidemori Hayashi MD, Masataka Sumiyoshi MD,
Satoru Suwa MD, Hidehiko Sakurai MD

Department of Cardiology, Juntendo University Shizuoka Hospital

A 65-year-old man developed syncope following palpitation during an outdoor activity in the daytime. The 12-lead electrocardiogram (ECG) showed Brugada-type ST segment elevation. Holter ECG monitoring documented an episode of regular wide QRS tachycardia. During an electrophysiologic study (EPS), ventricular tachyarrhythmia was not induced. However, a common (slow-fast) type atrioventricular nodal reentrant tachycardia with aberrant ventricular conduction, which was the same configuration as the wide QRS tachycardia recorded by the Holter ECG monitoring, was induced. The patient has been asymptomatic for the 12-month follow-up after the successful slow pathway ablation. This patient reminds us of the importance of EPS in the differential diagnosis of a wide QRS tachycardia.

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Key words: Syncope, Brugada-type electrocardiogram, Paroxysmal supraventricular tachycardia, Aberrant ventricular conduction, Slow pathway ablation

Case Report

A 65-year-old man developed syncope during an outdoor activity in the daytime following palpitation. His brother died suddenly at 66 years old, but cause is unknown. The 12-lead electrocardiogram (ECG) showed the Brugada-type ST segment elevation with coved-type in V₁ and saddleback-type in V₂ (**Figure 1**, left). Daily fluctuation of ST segment

elevation was not observed. He had normal chest X-rays, echocardiograms, and coronary angiograms. The intravenous administration of a pure sodium channel blocker, pilsicainide (1 mg/kg), exaggerated the ST segment elevation in V₁ and V₂ (**Figure 1**, right). Signal-averaged ECG was positive for late potential (f-QRS 117msec, LAS40 48msec, RMS40 11 μ V). Holter ECG monitoring documented an episode of a wide QRS tachycardia with a rate of

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Address for correspondence: Masataka Sumiyoshi MD, Department of Cardiology, Juntendo University Nerima Hospital, 3-1-10 Takanodai, Nerima-ku, Tokyo 177-8521, Japan. Phone: 03-5923-3111 Fax: 03-5923-3217 E-mail: sumi@med.juntendo.ac.jp

181 beats/min sustained for 10 seconds (Figure 2). Therefore we performed an electrophysiologic study (EPS) for differential diagnosis of the tachycardia. Ventricular tachyarrhythmia was not induced by programmed ventricular stimulation (PVS) from the right ventricular apex and outflow tract with a

maximum of 3 ventricular premature stimuli, to the shortest coupling interval of 180 ms, given at 2 different basic cycle lengths (600 ms and 500 ms). However, a narrow QRS tachycardia was easily induced, with dual atrioventricular (AV) nodal physiology, by both atrial and ventricular extra-

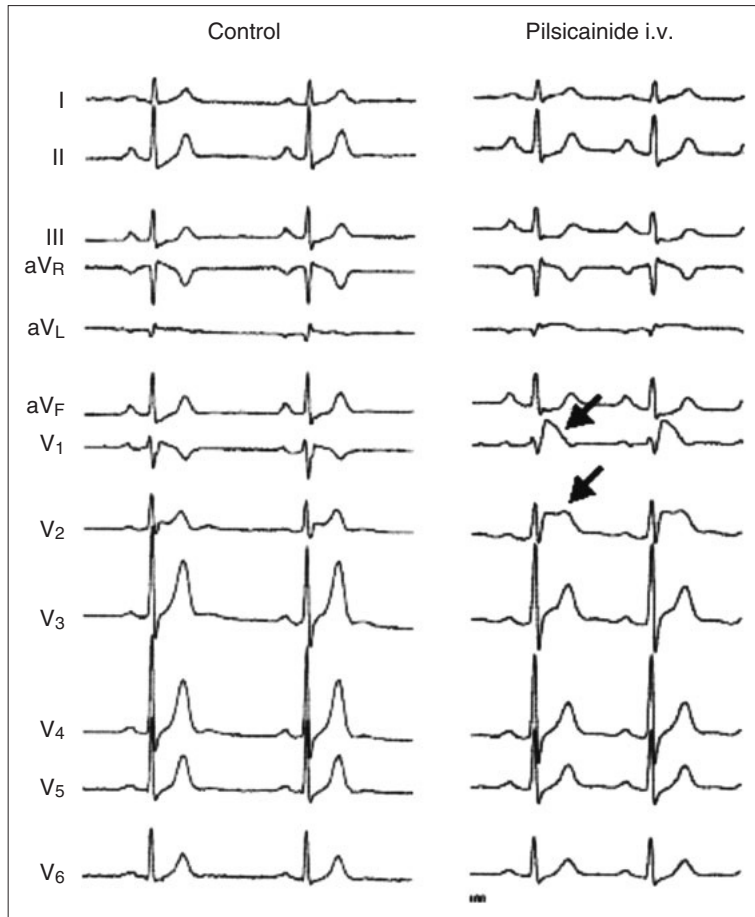


Figure 1 The 12-lead ECG showed the Brugada-type ST segment elevation with coved-type in V₁ and saddleback-type in V₂ (left). The intravenous administration of a sodium channel blocker, pilsicainide (1 mg/kg), exaggerated the ST segment elevation in V₁ and V₂ (right).

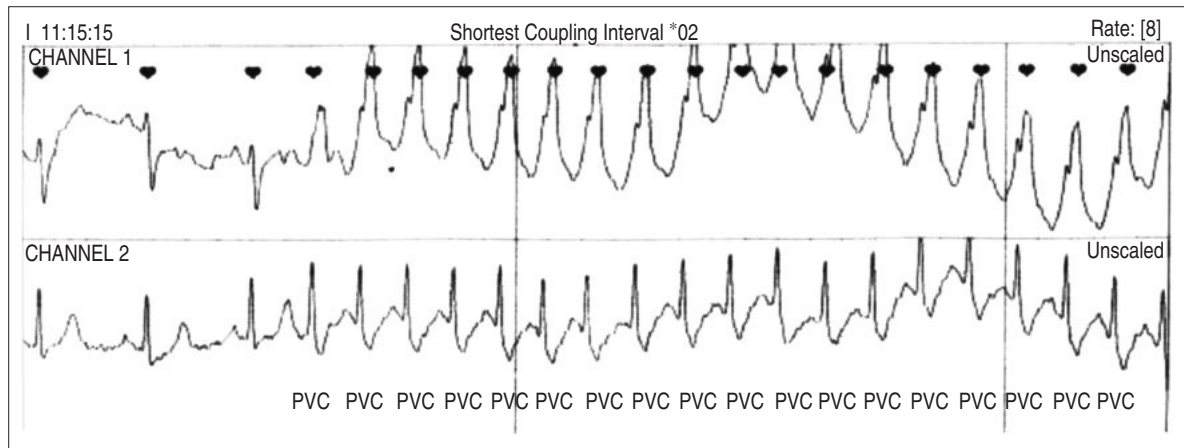


Figure 2 A wide QRS tachycardia documented during Holter ECG monitoring: NASA (top) and CM5 (bottom) recordings.

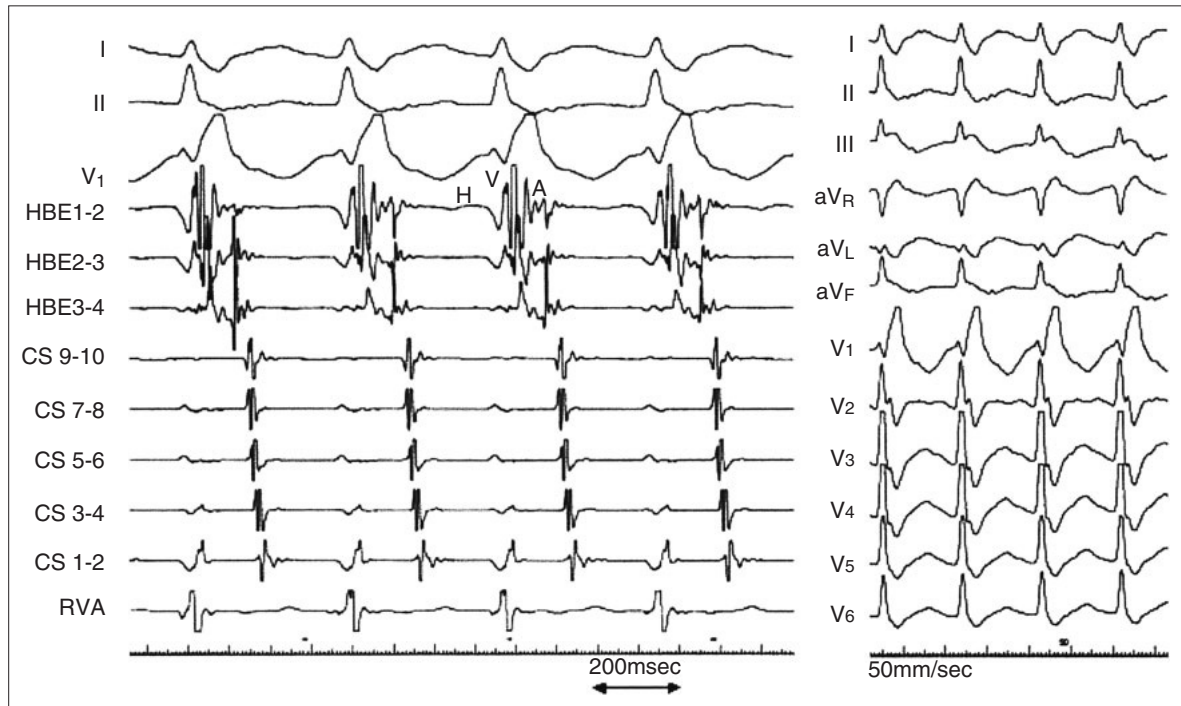


Figure 3 Intracardiac recording (left) and 12 lead ECG (right) during the induced wide QRS tachycardia with right bundle branch block type morphology.

HBE: His bundle electrogram, CS: coronary sinus, RVA: right ventricular apex

stimulation. During the tachycardia, the QRS morphology was changed to a right bundle branch block (RBBB) type wide QRS complex with the same atrial activation sequence (**Figure 3**). The earliest atrial activation was noted at the His-bundle region. Premature ventricular complexes delivered during tachycardia at the time of His-bundle refractoriness did not advance atrial activation nor terminate tachycardia. The finding of para-Hisian pacing was suggestive of retrograde conduction over an AV node. Therefore, the wide QRS tachycardia was diagnosed as a common (slow-fast) type AV nodal reentrant tachycardia (AVNRT) with RBBB due to aberrant ventricular conduction. It was the same configuration (RBBB type) as a non-sustained wide QRS tachycardia recorded by the Holter ECG monitoring, especially the QRS morphology in CM5 on Holter ECG and that in V₅ lead on 12-lead ECG were very similar. His slow pathway was successfully ablated and AVNRT could not be induced after the procedure. Despite the typical Brugada-ECG findings, we decided to not implant an implantable cardioverter defibrillator (ICD) in this patient. Genetic evaluation for SCN5A was not performed. He has been asymptomatic for the 12-month follow-up after the slow pathway ablation.

Discussion

Brugada syndrome is a primary electrical disorder characterized by RBBB and right precordial ST segment elevation and a high incidence of sudden cardiac death due to ventricular tachyarrhythmia. In our patient, a regular wide QRS tachycardia was recorded by Holter ECG monitoring (**Figure 2**). During an EPS, the wide QRS tachycardia was diagnosed as a common (slow-fast) type AVNRT with RBBB due to aberrant ventricular conduction. Paroxysmal supraventricular tachycardia (PSVT) associated with Brugada syndrome or the Brugada-type ECG has been reported recently.^{1,2)} Eckardt et al.¹⁾ showed that 10 out of 35 consecutive patients with Brugada syndrome, including 9 asymptomatic patients, had inducible PSVT on EPS. In addition, PSVT was found to be AVNRT in 6 of the 10 patients. Mok and Chan²⁾ also reported a case of AVNRT associated with the Brugada-type ECG. Increased atrial vulnerability in Brugada syndrome has been reported.³⁻⁵⁾ As Mok and Chan²⁾ suggested, the arrhythmogenic substrate might be present in the perinodal and atrial tissue in patients with the Brugada-type ECG findings.

We speculate that the cause of syncope was AVNRT that occurred during physical activity in the

upright posture and may be associated with vaso-vagal reaction leading to hypotension and syncope.^{6,7)} Finally, we did not implant an ICD in this patient because his only syncopal episode occurred during an outdoor physical activity in the daytime. It is atypical as ventricular fibrillation (VF) episodes in Brugada syndrome, which usually occur at rest or during sleep in the nighttime.⁸⁾ However, careful follow-up is mandatory to find future events because the cause of syncope and wide QRS tachycardia recorded by Holter ECG or induced on EPS might be irrelevant. This patient reminds us of the importance of EPS in the differential diagnosis of a wide QRS tachycardia.

References

- 1) Eckardt L, Kirchhof P, Loh P, et al: Brugada syndrome and supraventricular tachyarrhythmias: A novel association? *J Cardiovasc Electrophysiol* 2001; 12: 680–685
- 2) Mok N-S, Chan N-Y: Supraventricular tachycardia with a baseline ECG pattern of Brugada syndrome. *PACE* 2005; 28: 602–603
- 3) Itoh H, Shimizu M, Ino H, et al: Arrhythmias in patients with Brugada-type electrocardiographic findings. *Jpn Circ J* 2001; 65: 483–486
- 4) Morita H, Kusano-Fukushima K, Nagase S, et al: Atrial fibrillation and atrial vulnerability in patients with Brugada syndrome. *J Am Coll Cardiol* 2002; 40: 1437–1444
- 5) Bordachar P, Reuter S, Garrigue S, et al: Incidence, clinical implication and prognosis of atrial arrhythmias in Brugada syndrome. *Eur Heart J* 2004; 25: 879–884
- 6) Leitch JW, Klein GJ, Yee R, et al: Syncope associated with supraventricular tachycardia. An expression of tachycardia rate or vasomotor response? *Circulation* 1992; 85: 1064–1071
- 7) Brembilla-Perrot B, Beurrier D, Houriez P, et al: Incidence and mechanism of presyncope and/or syncope associated with paroxysmal junctional tachycardia. *Am J Cardiol* 2001; 88: 134–138
- 8) Matsuo K, Kurita T, Inagaki M, et al: The circadian pattern of the development of ventricular fibrillation in patients with Brugada syndrome. *Eur Heart J* 1999; 20: 465–470