Sustained Monomorphic Ventricular Tachycardia Originating from Right Ventricular Outflow Tract in Brugada Syndrome

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Case report

A 28-year-old man was admitted to our hospital for evaluation and treatment of sustained monomorphic ventricular tachycardia (SMVT) which is detected in another hospital. He had occasional episodes of palpitation and fainting for several years. On examination, the 12-lead ECG showed ST segment elevation in the right precordial leads (RPL) and incomplete right bundle branch block (RBBB) were found during the resting ECG without using any drug (Fig.1). Physical examination and laboratory findings showed no abnormality. Echocardiography was normal and coronary angiography showed no stenosis, and no spasm was induced after coronary infusion of acetylcholine. No special remarks were found during the general cardiovascular examination. In the drug loading test with intravenous administration of isoproterenol or epinephrine, neither elevation of the ST segment level in RPL nor induction of premature ventricular contraction or SMVT was observed. However, with intravenous administration of 300 mg procainamide, ST segment elevation in RPL was clearly augmented, followed by induction of spontaneous SMVT (Fig.2). An identical drug loading test after some days has confirmed the reproducibility of this observation. Intravenous administration of 100 mg disopyramide also induced this SMVT. Verapamil or propranolol terminated SMVT.

In an electrophysiological study (both control and isoproterenol loading), neither clear SMVT nor ventricular fibrillation induction was observed by three consecutive extrastimulation or burst stimulation from the right ventricular apex or outflow tract. However, spontaneous SMVT was induced by intravenous administration of 300 mg procainamide. Furtheremore, the following observations have been confirmed; 1) it was possible to induce and terminate SMVT by ventricular pacing, 2) no entrainment phenomenon was observed by ventricular pacing during SMVT, 3) a direct relationship was observed between the coupling interval from the last basic stimulation to the extrastimulation and the interval from the extrastimulation to the first ventricular excitation of SMVT. According to these backgrounds, it was thought that the mechanism of SMVT in our case might be due to triggered activity, and radiofrequency catheter ablation was performed according to the earliest potential site during SMVT and the pace map during the sinus rhythm in the vicinity of RVOT. After ablation, no induction of SMVT was observed even with intravenous administration of 600 mg procainamide.

The characteristics of Brugada syndrome are ST segment elevation in RPL and RBBB.[1] As further characterization of this syndrome, class IA antiarrhythmic drugs augment ST segment elevation in RPL.[2,3,4] However, spontaneous SMVT is rarely induced by intravenous administration of class IA antiarrhythmic drugs (procainamide and disopyramide). In our present case, RVOT tachycardia was never induced by programmed ventricular stimulation during the baseline and isoproterenol loading. However, after the intravenous administration of class IA antiarrhythmic drugs, spontaneous RVOT tachycardia was induced and the electrophysiological characteristics of SMVT indicated triggered activity due to delayed after depolarization. Conventionally, class IA

antiarrhythmic drugs are usually effective for the treatment of idiopathic RVOT tachycardia.[5] To our knowledge, there is no report about spontaneous RVOT tachycardia induced by administration of class IA antiarrhythmic drugs. Accordingly, we considered that there is a relationship between ST segment elevation in RPL and induction of RVOT tachycardia by intravenous administration of class IA antiarrhythmic drugs. Due to the existence of substrates which were similar to Brugada syndrome and combination with RVOT tachycardia with delayed after depolarization, this case may represent a subtype of Brugada syndrome.

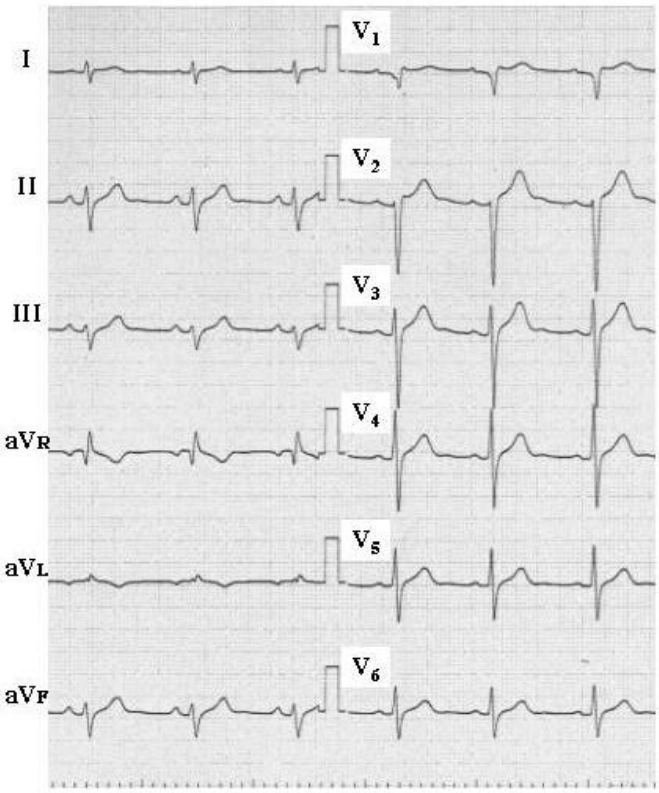

Figure 1

Twelve-lead ECG showed sinus rhythm, the incomplete right bundle branch block and ST segment elevation in the right precordial leads without administration of any drug (25 mm/sec and 1mV/cm).

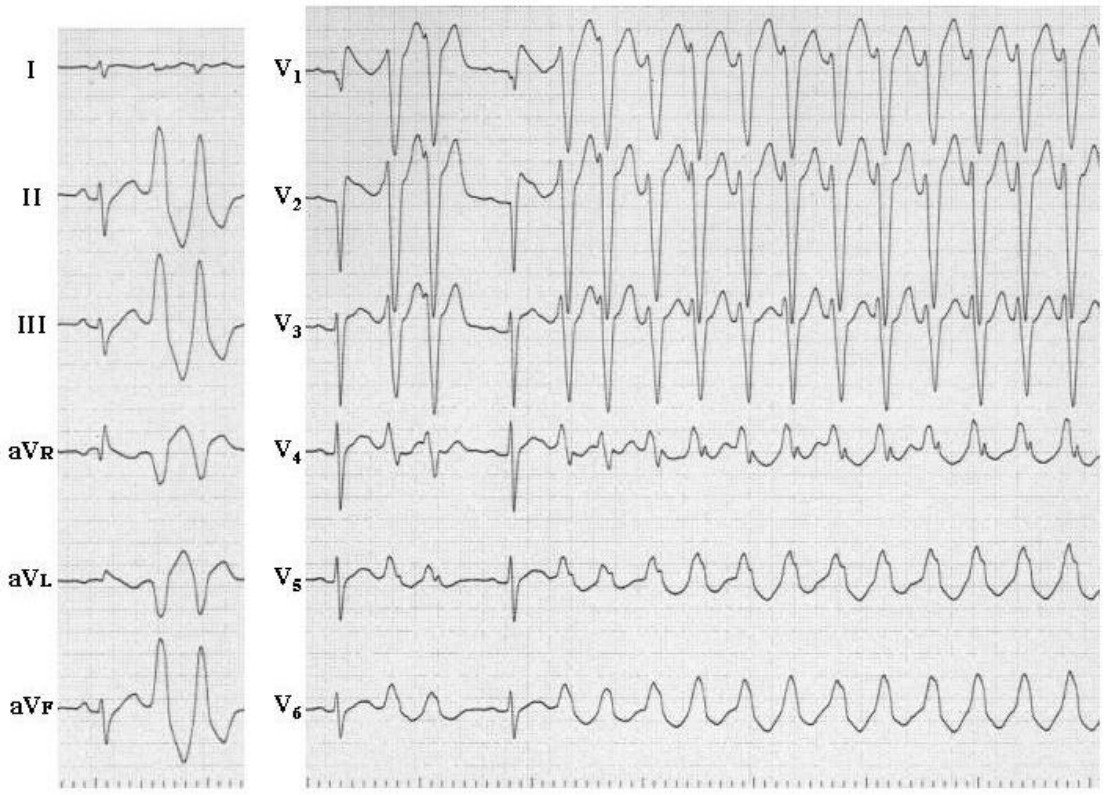

Figure 2

Initiation of right ventricular outflow tract (RVOT) tachycardia by intravenous administration of 300 mg procainamide. ST elevation clearly appeared in the right precordial leads (V_1, V_2: coved type; and V_3: saddle-back type) compared with Figure 1, followed by reproducible induction of sustained RVOT tachycardia (25 mm/sec and 1mV/cm).

References

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