Sustained ventricular tachycardia and coved-type electrocardiogram in peripheral leads: a particularly malignant phenotype of Brugada syndrome?

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A patient underwent implantable cardioverter-defibrillator (ICD) implantation due to unexplained ventricular fibrillation (VF). During the follow-up, he presented several ICD interventions for both VF and monomorphic ventricular tachycardia. An electrophysiological study failed to induce any ventricular tachycardia, whereas ajmaline administration induced a peculiar Brugada pattern coved-type in V1 and peripheral leads.

A 64-year-old man was admitted at our Hospital for appropriate implantable cardioverter-defibrillator (ICD) interventions. He was hospitalized 7 months before because of out-of-hospital cardiac arrest due to ventricular fibrillation (VF). At that moment, the previous medical history was unremarkable: he had no familiarity of sudden death, no previous syncopal episodes, and he was not taking any

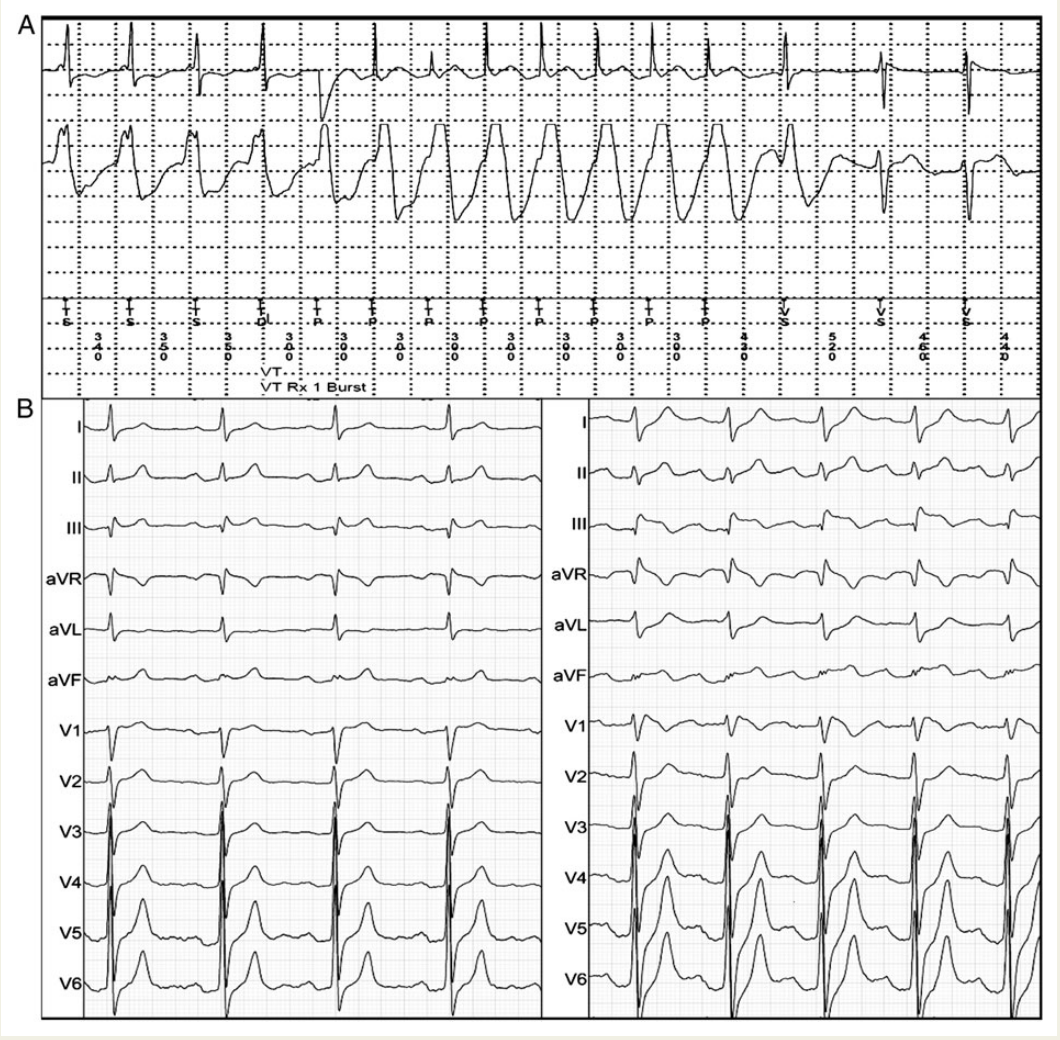

Figure 1 (*A*) Typical episode of MVT successfully treated by ATP. (*B*) Electrocardiogram before and after ajmaline 0.5 mg/kg intravenous administration. Basal ECG showed a first-degree atrioventricular block. After ajmaline challenge, BP developed in V1 and peripheral lead, leading to the diagnosis of BS.

drugs. An ICD was implanted after a negative cardiac evaluation, comprehensive of coronary angiography, echocardiogram, and magnetic resonance imaging (MRI). After 1 month, he received two appropriate shocks for two consecutive episodes of VF (cycle 190–200 ms).

The current hospitalization was caused by 13 repetitive monomorphic ventricular tachycardia (MVT) (mean cycle 178 ms, only endocavitary electrogram available, *Figure 1A*), mostly treated by ICD with efficacious antitachycardia pacing (ATP) and only one shock. The ICD interrogation revealed 2 months before a similar cluster of MVT (12 episodes, mean cycle 195 ms), treated by ATP not adverted by the patient. Both the initial VF and the afterward arrhythmic storm happened during a febrile episode.

A successive echocardiogram confirmed the absence of structural heart disease and the patient underwent an electrophysiological study (EPS). Programmed ventricular stimulation failed to induce any kind of arrhythmia. A more aggressive stimulation, with high rate bursts, induced VF. After administration of intravenous ajmaline (0.5 mg/kg), the electrocardiogram (ECG) showed a Brugada pattern (BP) coved-type in V1 and peripheral leads (*Figure 1B*). The test has been considered diagnostic for Brugada syndrome (BS) and was interrupted. A genetic test was requested.

Brugada syndrome is a primarily arrhythmic disorder that predisposes to malignant ventricular arrhythmias, in the absence of gross structural abnormalities. The diagnosis of BS is based on the presence of coved-type ST elevation (BP) in the right precordial leads (V1–V3).[1]

A spontaneous or drug-induced BP in peripheral ECG leads, mostly concomitant to the presence in precordial leads, seems to be present in up to 10% of BS population, and it has been associated with a worse prognosis, likely as marker of more severe disease.[2]

Brugada syndrome is typically related to polymorphic VT.[1] Rarely cases of MVT have been reported and in one similar case of BS and MVT, arrhythmias were sensitive to ATP and not inducible at the EPS.[3]

Our patient experienced both VF and repetitive MVT in the absence of structural heart disease. An initial form of right ventricular dysplasia is unlikely considering the completely negative MRI. In addition, the lack of VT inducibility, even after isoprenaline infusion, supports the hypothesis of a non-reentry tachycardia mechanism. The ajmaline test suggested a BS diagnosis.

The peculiarity of this case is the contemporary presence of two rare aspects of the BS: MVT and BP in peripheral leads during pharmacological test. The latter could explain the particularly malignant phenotype.

Conflict of interest: none declared.

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