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Brugada Syndrome and Vasovagal Syncope

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CASE REPORT

A 56-year-old male presented to the emergency room (ER) complaining of left upper quadrant abdominal pain. Among other examinations, a cardiology consultation was requested because of a significant ST-segment elevation in the precordial lead V_2.

The ECG was repeated showing a morphology of type-2 (saddleback) Brugada syndrome (Fig. 1). A modified ECG recording, obtained by placing the right precordial leads in higher positions at the second and third intercostal spaces, revealed a type-1 (coved) Brugada syndrome morphology (Fig. 2).

While the patient still in the ER, he started feeling anxious and within seconds he sustained a syncopal episode in sitting position, lasting about 30 sec with prompt and full recovery. At this time he was not connected to an external monitor and the underlying rhythm could not be witnessed. The ER physician reported that the patient had no pulse during the episode and that he was frightened when he woke up but not disoriented.

The patient was then transferred to the cardiac care unit (CCU) where he was placed on rhythm monitoring for 24 hours without further events. Cardiac enzymes remained normal, including three consecutive cTnI measurements, over the first 24 hours. Blood counts and biochemistry revealed no pathological values.

The next day the patient had an echocardiogram that was interpreted as normal for his age, noting that the right ventricle was of normal dimensions and contraction without any wall motion abnormality suspect of right ventricular cardiomyopathy. Subsequently he was submitted to a treadmill stress test. Using the Bruce protocol, he achieved a heart rate of 87% of his predicted for his age target rate and performed a satisfactory workload of 11 METS for 9.5 min. The Brugada type 1 morphology on the ECG remained unchanged during the whole process including the recovery period after the exercise test.

After completing the stress test and leaving the exercise room, while standing in the waiting room, before being transferred back to the cardiac ward, the patient suddenly developed another syncopal episode. He was immediately connected to a cardiac monitor which revealed sinus bradycardia of about 30/min. His recovery was again rapid and full. As vasovagal syncope was suspected as the cause of these episodes, the next day the patient underwent a tilt-table test. During tilting the patient remained asymptomatic for the first 30 min in the standing position. It took 8 min

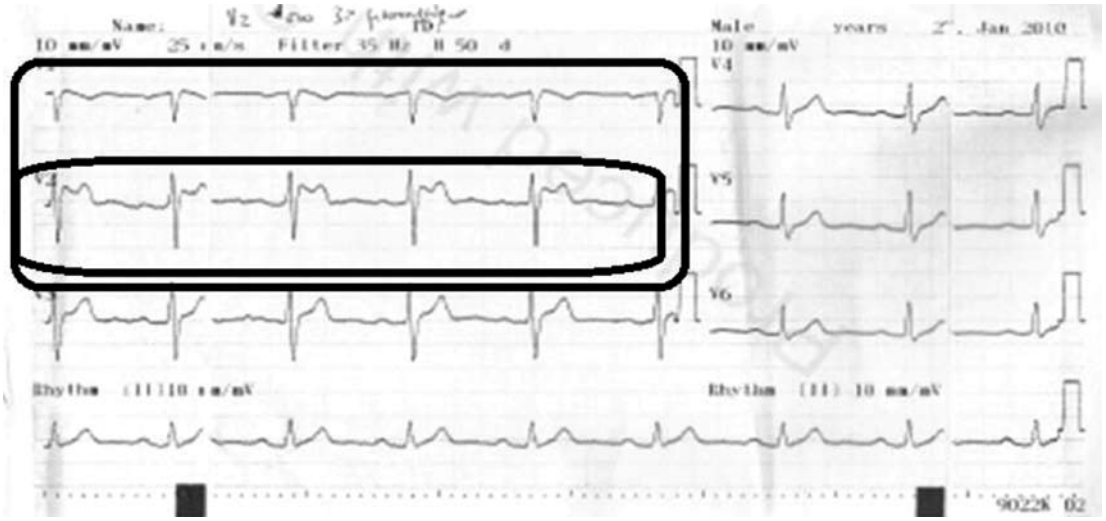

FIGURE 1. ECG showing a morphology of type-2 (saddleback) Brugada pattern in lead V2.

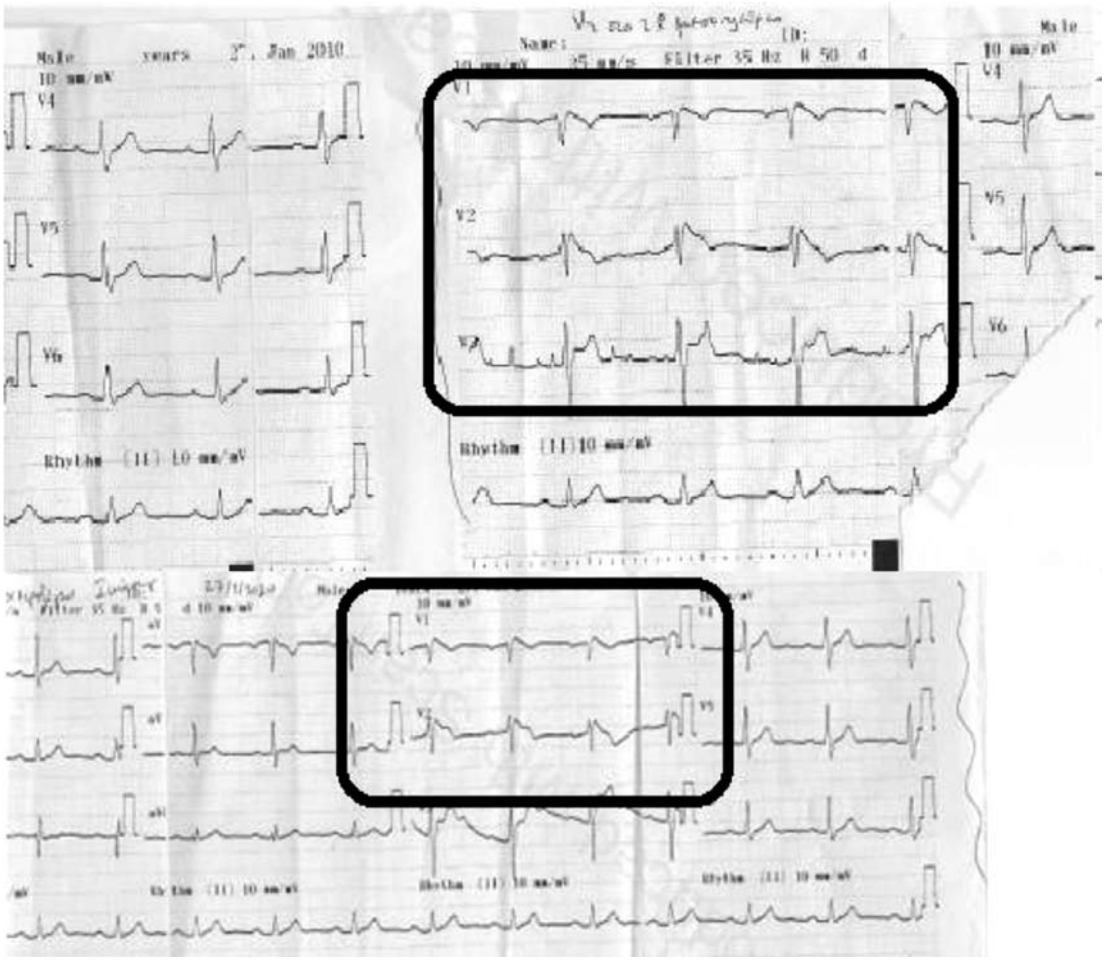

FIGURE 2. A modified ECG recording was obtained by placing the right precordial leads at the second and third intercostal spaces, revealing a type-1 (coved) Brugada pattern

into the isoproterenol infusion period and while titrating the dose up to 3 mcg/min, when his heart rate increased from 87/min to 115/min. At this time he started having prodromal symptoms like sweating, getting pale and his blood pressure fell rapidly, while he became relatively bradycardic. The test was terminated by putting the patient in a supine position, to prevent another syncopal spell. Thus, a diagnosis of vasovagal syncope of mixed (cardioinhibitory & vasodepressive) type was reached. Due to the history of syncopal episodes, and the Brugada type I ECG pattern, the patient was referred for an electrophysiology study. Polymorphic ventricular tachycardia was induced and the patient subsequently had an automatic defibrillator (ICD) implanted for prophylaxis from sudden cardiac death.

DISCUSSION

The Brugada syndrome is characterized by ST-segment elevation in the right precordial leads V1 through V3 and an episode of venricular tachyarrythmia in the absence of structural heart disease.[1-3] Patients with spontaneously or inducible Brugada type-1 morphology are at high risk of polymorphic ventricular tachycardia (and less frequently for monomorphic ventricular or supraventricular tachycardias), which when it is self-terminating can present as syncope. According to the 2009 guidelines of the European Society of Cardiology (ESC) on syncope, neurally-mediated syncope (NMS) is the most common cause of all syncopal episodes,[4] having a quite favorable prognosis. Individuals who fulfill the ECG criteria of the Brugada syndrome and have episodes of syncope of cardiac origin should proceed to ICD implantation without the need of an EPS study.[3] However, it behooves us to determine the precise cause of syncope even in patients with the Brugada syndrome, as they are also entitled to have syncope of other more common causes, like NMS, and not necessarily suffer from ventricular tachyarrhythmias.

Two recent reports studying the incidence of NMS in a patient population with the Brugarda-type ECG pattern,[5,6] reported a similar incidence of NMS with that noted in the general population. This observation might suggest that the most common cause of syncope in Brugada patients is NMS. Of course, vasovagal syncope is considered benign in the general population in the absence of underlying cardiac pathology, but in patients with the Brugada syndrome things may be different since a vagally-induced episode of bradycardia could be a potential trigger factor for lethal ventricular arrhythmias. This dilemma seems to exist since 2001[7,8] and todate it remains unresolved. As the Brugada brothers have proposed,[8] it may be better to overprotect the patient with Brugada syndrome and vasovagal syncope by implanting an ICD than just accepting the vasovagal origin of all syncopal episodes in this particular patient population.

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