Atypical Cause of Syncope in Patients with Brugada Syndrome

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Examination of a 32 year old male with chest pain and suggestive Brugada ECG pattern was sent to our outpatient clinic for cardiac evaluation. The patient denied having suffered any previous syncpe. He had a positive history of intravenous drug abuse and was HBV+. There were no sudden deaths, although there was important consanguinity in the family. A complete cardiac study was carried out, including a drug challenge test with a sodium channel blocker (flecainide) that confirmed the diagnosis (Figure 1). The echocardiogram, 24 hours ECG holter monitoring, and electrophysiological study (ventricular programmed stimulation with up to three extrastimuli and three basic cycle lengths from right ventricular apex and outflow tract), were normal. A blood sample was taken for genetic testing, clinical check-up were recommended and a family study was started.

Three months after the diagnosis, the patient presented two presyncopal episodes and an isolated syncope in unclear circumstances. The syncope occurred at rest, at night and was related to alcohol abuse. Due to the negative results of the cardiac examinations, and the context of the syncope, an subcutaneous loop recorder (Reveal Plus® Medtronic) was implanted.

Four months later, the patient was admitted to the emergency room 24 hours after having had a new syncopal episode. The syncope occurred during a party where the patient took alcohol and drugs (cannabis and cocaine). The device was checked and a progressive sinus bradycardia, followed by a 4.5 seconds sinus arrest were evidenced (Figure 2) at the time of the syncope. The patient was discharged with the following diagnosis: neurocardiogenic syncope and secondary bradicardia/sinus arrest. No other syncopes or further brady-tachyarrhythmias were recorded during the 15 months of device duration, which was finally explanted. The patient remains alive and visits the clinic regularly.

Brugada syndrome was first described in 1992 by Brugada and Brugada. The sodium channel malfunctioning observed in the syndrome, can trigger ventricular arrhythmias, that often occur at night or during enhanced vagal activity. It can clinically express through syncope or sudden death. Implantable defibrillators are the only therapy that has proved to be effective in such conditions.

The incidence rate of benign syncopes (vasovagal or orthostatic) in the general population is not low, and some authors have estimated it 20% in the course of a lifetime with two peaks both at early and late age. It is of importance to appropriately identify syncopal episodes in Brugada ECG patients. The clinical profile of the syncope can predict prognosis and will lead to different management and treatment options. A detailed interview is essential for this purpose in order to differentiate the type of syncope. Nevertheless, when the characteristics are not typical or the cause of the syncope is uncertain, additional examinations or devices can provide essential information.

The implantable ECG loop recorder has proved to be useful in selected populations with suspicions of bradycardia-related syncope. Similarly, the device has been helpful in the study of mechanisms of vasovagal syncope. However, its value for patients with a high risk of tachyarrhythmic syncope has not been evaluated appropriately. Most of these patients are recommended to undergo ICD implantation due to the high morbi-mortality risk. Some ethical issues in this setting have been raised.

In the present case, the device implantation was very useful for appropriate diagnosis in a patient with syncope of uncertain cause. Our patient was diagnosed with Brugada ECG pattern and had a toxic history of drug and alcohol abuse, both known as triggers for arrhythmic syncopal episodes.
The use of Reveal in Brugada patients is infrequent and controversial and one case of NSVT, diagnosed by the insertion of an implantable device, has been described previously. The clinical suspicion that the syncope in our patient could not be related to ventricular tachycardia, led to the indication of device implantation. Nevertheless, the conclusion drawn from this case should not be that an implantable loop recorder should be indicated for the evaluation of syncope in Brugada patients, but only to remark that this device can be helpful in selected cases. Besides, neurocardiogenic syncope in Brugada patients is not necessarily benign, as high vagal tone could induce ventricular arrhythmias.

Additionally, we aim to highlight the possibility that Brugada patients are susceptible to having non-arrhythmic syncopes, and that a careful interview is essential before considering aggressive therapeutic options such as the implantation of a defibrillator.

REFERENCES


