BRUGADA SYNDROME

Editorial

The yet unresolved dilemma of syncope in Brugada syndrome

In their case report[1] in this issue of Europace, Samniah et al. conclude as follows: ‘Inasmuch as current recommendations favour implantable defibrillators in symptomatic Brugada syndrome, the identification of other causes of syncope in such patients poses an uncomfortable and currently unsettled dilemma’. We cannot agree more with this statement.

In 1996, we were approached by a 40-year-old female with recurrent episodes of syncope. Her clinical history was consistent with vasovagal-induced loss of consciousness. Her tilt test was positive, thus supportive of the diagnosis of vasovagal syncope. An electrophysiological study showed no abnormalities, but ventricular fibrillation was induced during pacing of the right ventricular apex at 100 bpm with two ventricular premature beats. Due to the positive tilt test, a lack of family history of sudden cardiac death and the totally normal electrocardiogram, we considered the induced ventricular fibrillation a non-specific finding. She was discharged on oral propranolol.

Two weeks later, the patient requested the immediate help of her general practitioner because of recurrent syncope. An electrocardiogram taken at that time showed isolated ventricular premature beats, and the general practitioner started treatment with oral flecainide. Two days later, she presented to our emergency room after multiple recurrent episodes of syncope. Her electrocardiogram showed the typical findings of Brugada syndrome. After discontinuation of flecainide, the electrocardiogram became normal. A repeat electrophysiological study proved the reproducibility of induction of ventricular fibrillation by two ventricular premature beats. Administration of intravenous procainamide reproduced the electrocardiographic pattern of Brugada syndrome. A permanent cardioverter-defibrillator was implanted and all medication discontinued. The first appropriate shock to treat ventricular fibrillation was delivered by the device a few months later.

This patient, a case report like the case report presented by Samniah et al., illustrates the same dilemma in the opposite way. Samniah et al. were cautious in suspecting Brugada syndrome and performed the appropriate pharmacological tests to confirm the diagnosis. Later on, doubts arose about the exact cause of the syncopal spells and they carefully considered what the best therapeutic approach could be. The patient has been treated with midodrine for 16 months without recurrence of syncope suggesting that the correct decision, as far as vasovagal syncope is concerned, was taken. However, the patient undisputedly suffers also from Brugada syndrome and that raises many questions on how to approach that part of his problem.

In 1996, we were less lucky. We did not know too much about pharmacological challenges in individuals with the concealed form of the Brugada syndrome. Our patient seemed to suffer from two diseases, and vasovagal syncope dominated the scene at the time of the first encounter. It was only after an involuntary pharmacologic challenge that the Brugada electrocardiogram became evident.

We are totally convinced that these two patients suffer from two conditions: vasovagal syncope and Brugada syndrome. They are examples of the many diagnostic and therapeutic challenges that we face in everyday practice of medicine. Our patient survived to the implantation of a cardioverter-defibrillator that was later appropriately used. Whether Dr Samniah’s patient needs a defibrillator is not definitively proven, but most data would support that[2,3]. We will all face the same dilemma until studies with larger patient series and with a longer follow-up become available. Meanwhile, any decision that a
physician takes should continue to respect the principle ‘do no harm’. You may harm by abuse, but also by not doing something. When the physician does not know the answer, the only one who should not pay for the consequences is the patient.

Thus, overprotecting a patient with vasovagal syncope and a Brugada electrocardiogram by implanting a cardioverter-defibrillator may be better than just accepting a vasovagal origin of all syncopal episodes. That is the most important lesson that we have learnt from our patient.

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References

