POST-OPERATIVE VENTRICULAR TACHYCARDIA IN AN ASYMPTOMATIC PATIENT WITH BRUGADA SYNDROME: CASE REPORT

GIOVANNI CANTARELLA*, GIUSEPPA LA CAMERA, DANILO CARMELO GRASSO, VALERIA CARNEMOLLA, MIRKO MINERI, BRUNO LANZAFAME, LUCA VITALE, PIERFILIPPO DI MARCO
*Department of Medical and Pediatric Sciences-Section Cardiology; Department of Medical Surgical Specialties-Section Anaesthesia and Intensive Care, University of Catania, Italy

ABSTRACT

Objective: *A patient is described who had Brugada Syndrome but was completely asymptomatic in the pre-operative period, and whose ECG and clinical history did not clearly indicate such pathology.*

Key words*: ventricular tachycardia, Brugada syndrome, cardiac arrhythmia, ECG, anti-arrhythmia drugs.*

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Introduction

Brugada syndrome is a hereditary arrhythmogenic cardiopathy, characterized by a typical ECG in patients with a structurally healthy heart. The clinical exam and history may be completely negative, or there may be such symptoms as syncope, palpitation, tachycardia, or ventricular fibrillation. The family history often includes sudden cardiac death. There are several genetic abnormalities in the literature, but the most significant involves gene SCN5A[1], which codifies the sodium channel responsible for the phase of precocious ventricular muscle re-polarization (Table 1).

GENE	VARIANT	Ionic channel	Percent of patients
SCN5A	SB1	I_{Na}	11-18%
SCN1B	SB5		<1%
SCN3B	SB7		<1%
GPD1-L	SB2		<1%
MOG1	SB8		<1%
CACNA1c	SB3	I_{CaL}	<1%
CACNB2b	SB4		<1%
KCNE3	SB6	I_{Kto}	<1%
KCND3	SB10		<1%
KCNE5	SB9		<1%

Table 1: Genes related to Brugada Syndrome, classified according to the ionic channel involved.

That provokes a different re-polarization between epicardium and endocardium, which is the main cause of arrhythmia in these patients.

Diagnosing Brugada Syndrome may be complicated, not only due to the normal history and clinical exam, but also because of the transitory nature of the ECG (electrocardiogram). There are many factors that alter the characteristic ECG (Type 1 Brudaga ECG pattern), such as body temperature, adrenergic tone and vagus nerve stimulation, and many drugs may directly or indirectly influence the functioning of ionic channels[1,2]. The first description of the ECG pattern was in 1953, before the syndrome was defined by the Brugada brothers in 1992. The ECG is characterized by a persistent elevation of the ST wave in the left precordium independent of myocardial ischemia or other notable causes, associated with a complete or incomplete block of the right branch[1]. The genetic nature of this syndrome renders screening tests of relatives necessary even though there is different penetration in the Brugada syndrome.

Methods

A 67-years-old male, weight 77kg, non-smoker, asymptomatic, normal blood pressure

(135/80mmHg), without any family history of cardiac disease or sudden cardiac death (SCD), nor any previous cardiac events, was admitted to our hospital for a right hemicolectomy due to tumor of the ascending colon.

During the pre-operative period the ECG showed a sinus rhythm with a cardiac frequency of 90bpm, the presence of an incomplete block of the right branch, and a mild elevation of the ST wave (< 1mm) in the right precordial leads V1, V2, and V3 (Figure 1).

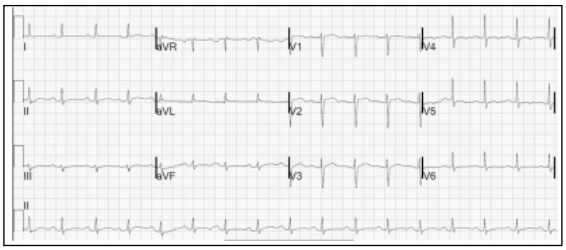

Figure 1: Pre-operative ECG.

The routine blood tests included renal function and blood electrolytes and were normal.

Before general anesthesia a peridural catheter was inserted in the intervertebral disc space L1/L2, to administer a total of 20ml bupivacaine 0.25% in 5 minutes. Once the block was confirmed and we established the number of metameres involved we continued administering local anesthesia at a concentration of 0.125% combined with fentanyl 2.5mcg/ml for a total of 8ml/h.

The induction of anesthesia was achieved by midazolam 2mg, propofol 150mg, fentanyl 200mg, and rocuronium 50mg. The anesthesia was maintained with sevoflurane. During the post-operative period there were frequent ventricular ectopic beats (VEB), which increased until the beginning of ventricular tachycardia (VT). In an attempt to convert the arrhythmia amiodarone was administered, which only decreased the frequency without cardioversion.

The ECG changed compared to the pre-operative period: a convex elevation of the ST wave with a negative T in the V1-V2 lead strongly indicated Type 1 Brugada syndrome. Upon suspicion of the diagnosis the bupivacaine was suspended immediately and the next day the ECG improved with the disappearance of the arrhythmia and normalization of the ST and T wave.

The patient was monitored during the entire post-operative period and no other changes in the ECG occurred. Hence, the patient was discharged with suspected Brugada syndrome, with the prescription to try anti-arrhythmia medication to confirm the diagnosis.

Approximately one month after the surgery the patient returned for testing. Flecainide 150mg was administered intravenously in 10 minutes, and after the third minute a change in the ECG pattern was observed: the appearance of a convex elevation of the ST segment with wave J> 2mm in the right precordial, and the ST wave gradually descending in the terminal portion, and T wave negative in the same precordial, a morphology compatible with the pattern of Type 1 Brugada syndrome (Figure 2).

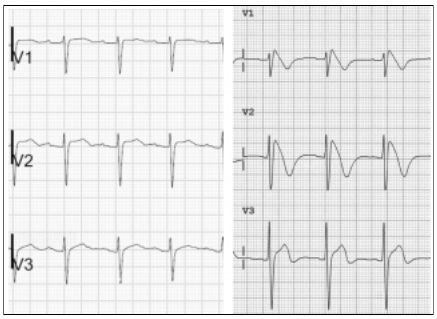

Figure 2: Comparison between the pre-operative ECG and the ECG after administering flecainide.

Once we found the Type 1 pattern, we used a challenge test to see if there were changes through stimulation of the vagus nerve in the recovery period. In the event we noticed a decrease in the elevation during the challenge, which was the result we expected, and which returned to elevation more significantly in the recovery period than during rest.

In our opinion the test confirmed the diagnosis and also informed the anesthesiologist as to how the patient would react to stimulation of the autonomic nervous system[3].

Finally, we decided to conduct a Holter exam to determine the frequency of pathological ECG events, above all during the night when stimulation of the vagus nerve prevails. That test revealed ECG changes over the 24-hours period with the absence of bradycardia events or tachyarrhythmia. After observing the Type 1 pattern, which was not spontaneous but provoked by the Class 1C anti-arrhythmia drugs, and in the absence of symptoms, we considered the patient at low risk of SCD according to the risk classification of guidelines. Hence, we decided against an implant of implantable cardiodefibrillator (ICD). In addition, considering the genetic nature of Brugada syndrome, we performed screening tests on the patient's relatives, and they did not exhibit pathological abnormalities.

Discussion

The anesthesiological management of patients with unusual conditions, such as this patient with Brugada syndrome may be complicated, above all in cases where the patient history and clinical exam are negative. In general there are no guidelines to indicate how the physician should manage a patient with Brugada syndrome, but there are some indications in the literature guided by previous clinical cases[4].

On the basis of our experience we highlight a few aspects that may be of interest in the management of these patients. In the pre-operative diagnosis there is often no clinical or laboratory information to identify a patient with Brugada syndrome.

Recently molecular biology tests have been sought to screen patients in the future, but for now such testing is of marginal value in the pre-operative period. Presently, the use of such testing is appropriate only in those patients whose family history includes sudden cardiac death and a Type 1 ECG.

In our opinion testing by stimulation with anti-arrhythmia drugs (e.g. flecainide) may be useful in doubtful cases to eliminate doubt before bringing the patient to the operating table.

Another aspect is the knowledge of the anesthesiologist and other medical staff of which drugs must be avoided in these patients. The problem here is a lack of clear information, which is being remedied by collecting data gained from literature and by direct clinical experience.

There is a web site www.brugadadrugs.org that may be consulted[5]. (Tables 2 and 3).

The lack of guidelines is due to considerable genetic variability, which results in different morphological aspects in the ECG that are not accompanied by clinical signs.

In such cases a challenge test is useful, above all when there is an open clinical picture and have foresight by avoiding drugs with clear or relative contra-indications whenever Brugada syndrome may be suspected.

Conclusions

When there is a suspicion of Brugada syndrome, the clinical picture and uncertain ECG must be investigated through screening. Using an induction test with anti-arrhythmic drugs and an associated challenge are valid diagnostic methods. In the future genetic testing may take on a primary role in the diagnosis.

Drugs to Definitely Avoid		
Drug	Clinical Use	Comment
Flecainide	Anti-arrhythmia (1C: Na-blocker)	There is evidence or general agreement that these drugs have a potentially arrhythmic effect in patients with Brugada syndrome.
Ajmaline	Anti-arrhythmia (1A: Na-blocker)	
Procainamide		
Propafenone	Anti-arrhythmic (1C: Na-blocker)	There is conflicting evidence on the effects of these drugs, but the data available in the literature and general consensus favor a potential arrhythmic effect in patients with Brugada syndrome.
Bupivacaine	Anesthetic-analgesic.	
Propofol	Anesthetic	
Anti-depressants		
Loxapine	Anti-psychotic	
Acetylcholine	Colinergic	

Table 2: Drugs to probably avoid in patients Brugada syndrome[6].

Drugs to probably avoid.		
Drug	Clinical Use	Comment
Amiodarone	Anti-arrhythmic (Class 3)	There is conflicting evidence and differences of opinion on the effects of these drugs in the literature, and the potential arrhythmic effect in patients with Brugada syndrome has not been well studied.
Lidocaine	Anti-arrhythmic (1A: Na-blocker)	
Propranolol	Anti-arrhythmic (Class 2)	
Verapamil	Anti-arrhythmic (Class 4)	
Ketamine	Anesthetic	
Tramadol	Analgesic-narcotic	
Carbamazepine and phenytoin	Anti-convulsive	
Clotiapine and thioridazine	Anti-psychotic	
Lamotrigine	Anti-epileptic	

Table 3: Drugs to probably avoid in patients Brugada syndrome[6].

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