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General anesthesia in patient with Brugada syndrome

María de Loreto Gómez-Martínez, MD;* Pilar Fernández-Garijo, MD;* Francisco Rodríguez-Cabo, MD;** Juan Bustamante-Munguira, MD;*** María del Rocío Gómez-Martínez, MD;* Margarita Vegas-Pavesio, MD;* César Aldecoa-Santullano, MD;* José Ignacio Gómez-Herreras, MD*

* Service of Anesthesiology and Resuscitation. Hospital Universitario Río Hortega. Valladolid.
** Medical Health Emergencies of Castilla y León.
*** Service of Cardiac Surgery. Instituto de Ciencias del Corazón (ICICOR). Hospital Clínico Universitario de Valladolid. España.

Reprint request:
María de Loreto Gómez Martínez
Camino a Arroyo Núm. 27. Urbanización los Palomares. 47610. Zaratán. Valladolid. España.
Teléfono 606169304
Correo electrónico: mariadeloreto@hotmail.com

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Abbreviations:
CSD. Cardiac sudden death
ADI. Automatic defibrilator implantation.

No existe conflicto de intereses en relación con el manuscrito.

SUMMARY

A man with Brugada syndrome underwent an otologic surgery delivery. The operation with general anesthesia was uneventful. Brugada syndrome is characterized by a right bundle branch blockade, ST segment elevation in right precordial leads and a high risk of presenting malignant ventricular arrhythmias (polymorphic ventricular tachycardia and/or ventricular fibrillation) and sudden cardiac death in patients with structurally normal hearts. From 20 to 60 percent of idiopathic ventricular fibrillation could be associated with this syndrome. There are several precipitating factors. General anesthesia in these cases needs continuous ECG recording. The monitoring of the bispectral index, temperature, neuromuscular block and arterial blood pressure is important. In conclusion, we need to know the ECG manifestations of Brugada syndrome, the recommendations for automatic defibrillator implantation (ADI) and the pathophysiological mechanisms to avoid agents or conditions that may induce arrhythmias. During the first 24 hours after the surgery, these patients must be closely monitored by right precordial ECG leads.

Key words: Brugada syndrome, ventricular fibrillation, cardiac arrest, general anesthesia.

RESUMEN

Varón con síndrome de Brugada programado para cirugía otológica. La intervención se realizó bajo anestesia general y transcurrió sin incidencias clínicas destacables. El síndrome de Brugada es una entidad caracterizada por un patrón electrocardiográfico con bloqueo de rama derecha, elevación del segmento ST en precordiales derechas y riesgo aumentado de presentar arritmias ventriculares malignas (taquicardia ventricular polimórfica y/o fibrilación ventricular) y/o muerte súbita cardíaca (MSC) sin pródromos, en pacientes previamente sanos y con corazón estructuralmente normal. Entre el 20 y el 60% de las fibrilaciones ventriculares idiopáticas que tienen lugar durante la anestesia podrían estar asociadas a este síndrome. Varios factores pueden precipitar episodios de arritmias. La anestesia general de estos pacientes precisa de una vigilancia y monitorización continua de la frecuencia cardíaca y trazado electrocardiográfico, importante es también la monitorización de la profundidad anestésica, temperatura corporal, bloqueo neuromuscular y tensión arterial. En conclusión, debemos reconocer los hallazgos electrocardiográficos del síndrome, las indicaciones del desfibrilador automático implantable (DAI) y la patogenia a fin de evitar el uso de fármacos y condiciones que pudieran desencadenar arritmias malignas, además de garantizar una monitorización electrocardiográfica continua durante la intervención quirúrgica y durante las primeras 24 horas postoperatorias.

Palabras clave: *Síndrome de Brugada, fibrilación ventricular, muerte súbita, anestesia general.*

INTRODUCTION

The Brugada syndrome is an entity characterized by malignant ventricular arrhythmias (polymorphic ventricular tachycardia and/or ventricular fibrillation) that can derive in cardiac sudden death (CSD) without prodomes, in previously healthy patients with a structurally normal heart[1,2].

The type 1 electrocardiographic pattern is a diagnosis and it corresponds to a right branch block with convex ST segment elevation greater or equal to 2 mm (0,2 mV) in more than a right precordial lead (V1, V2, V3), followed by a negative wave T. The type 2 pattern shows ST-segment elevation (saddle-back type) greater than or equal to 2 mm with the terminal portion of the segment greater than or equal to 1 mm and positive or biphasic T wave. The type 3 electrocardiographic pattern shows ST-segment elevation (saddle-back type) with final portion with less than 1 mm and positive T wave. Types 2 and 3 are not diagnosis of Brugada syndrome but they should be considered before a type 2 or 3 that expresses the type 1 pattern after administration of sodium channel blockers. The electrocardiographic pattern is dynamic and may be masked or modulated with spontaneous and temporal variations of the ST segment.

It is genetically transmitted with autosomal dominant pattern and incomplete penetrance, being most common in males (8:1). It is a heterogeneous syndrome in which various types of mutations have been described; the genetic defect is on chromosome 3 affecting in a different way the sodium channel functioning in approximately 20% of cases such as alteration or absence of functioning. The cause of ventricular fibrillation may be in the presence of a same tissue of normal or mutated sodium channels, with heterogeneous refractory periods or in the reduction of functional sodium channels that may develop reentry arrhythmias. These findings reinforce the theory that it is a primary electrical disease of the heart[3].

The incidence is difficult to determine because the typical electrocardiographic pattern may be present, intermittent, hidden or inducible with class IA and IC antiarrhythmics, vagotonic agents, alpha adrenergic agonists, beta adrenergic blockers, tricyclic antidepressants, and combinations of glucose and insulin, hypocalcaemia, hypokalemia, hyperkalemia, alcohol, cocaine ... [4]

There are patients in whom bradycardia can trigger the arrhythmia, which explains the higher incidence of SCD overnight. In other patients, arrhythmias are most frequent during adrenergic stimulation as occurs in fever or stress states. Challenge test with flecainide or ajmaline is used as pharmacological test of induction. Brugada syndrome could be responsible for 50% of deaths in young people with structurally normal hearts and without previous heart disease. It is more common in Asian countries where the disease occurs between 0.05 and 0.6% of the adult population. Between 20 and 60% of idiopathic ventricular fibrillations that occur during anesthesia could be associated to this syndrome[5]. In symptomatic patients,

The recurrence or possibility of syncopal episode or SCD is 30% in two years. The diagnosis is performed in patients with or without symptoms, by detecting abnormal baseline ECG or through pharmacological challenge tests[1,2]. Sometimes the diagnosis is secondary to the death of a relative by SCD.

Until now, the only effective treatment is the implantation of an implantable automatic defibrillator (IAD). According Antzelevitch et al[4], the recommendations of IAD implantation are:

- Patients with type 1 electrocardiographic pattern (spontaneous or after sodium channel blockers) that have suffered an recovered episode of sudden death or with cardiac origin symptoms (syncope, nocturnal agonal respiration, dizziness) of cardiac origin.
- Asymptomatic type 1 patients (spontaneous or after sodium channel blockers) with a family history of SCD, in whom a polymorphic ventricular tachycardia or ventricular fibrillation can induced during the electrophysiological study.
- Spontaneous asymptomatic type 1 patient with no family history of SCD and positive electrophysiological study.

We present the case of a patient with type 2 Brugada syndrome scheduled for otologic surgery.

CASE REPORT

Male aged 46 years, 1.80 meters in height and 75 kilos in weight, without drug allergies or known toxic habits. The patient admitted to our center to perform an exploratory tympanostomy and perilymphatic fistula closure in his right ear. A meningitis during infancy and asymptomatic Brugada syndrome are highlighted as personal background. Among the family background, a brother died of SCD.

In the preoperative examination, the electrocardiogram showed mild abnormalities of ventricular repolarization, incomplete right branch block and ST elevation (bundle type) in V1-V2 (Figure 1). Pharmacological challenge test with flecainide at a 2 mg/kg dose showed characteristic changes of Brugada syndrome and ventricular extrasystoles. The electrophysiological study did not induce ventricular arrhythmias, so it was not indicated the IAD implantation. The other preoperative tests (biochemistry, hemogram, coagulation and chest plate) were normal.

Before starting the surgery procedure, we placed an external defibrillator. Patient was premedicated with antiemetics. Electrocardiogram, heart rate, pulse, bloodless blood

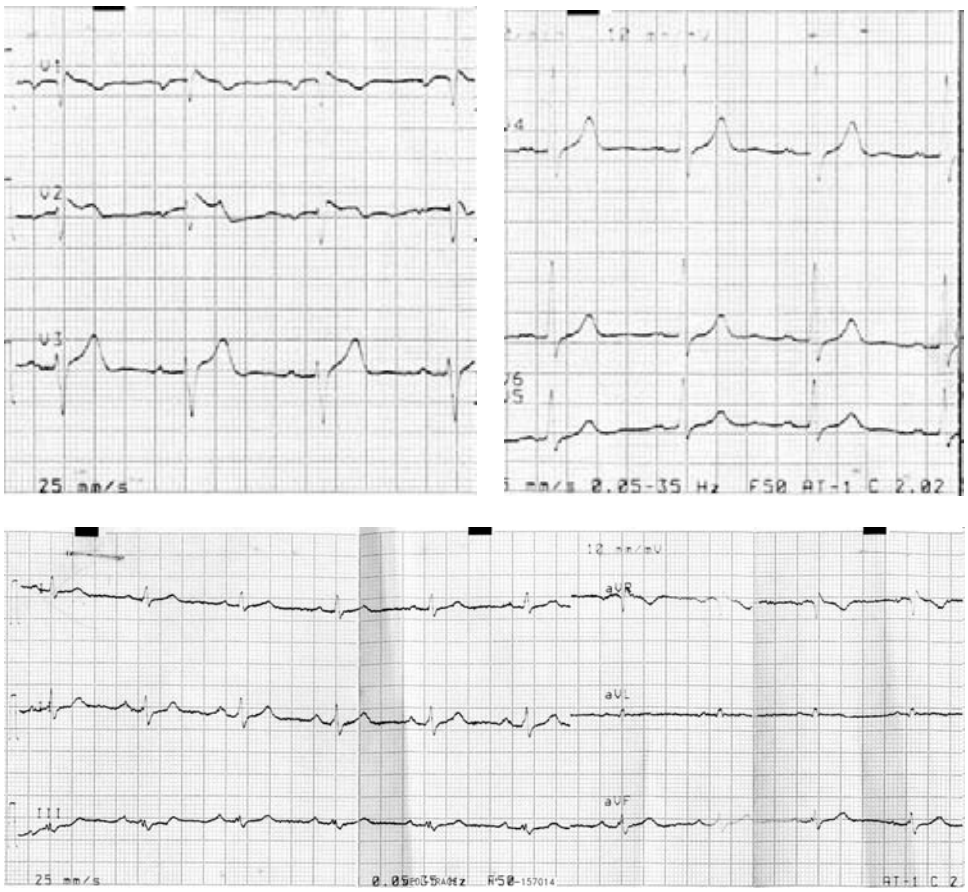

Figure 1. Preoperative electrocardiogram in patients with Brugada syndrome type 2.

pressure, neuromuscular block and exhaled CO2 were monitored. The induction is performed with remifentanyl at 0.15 mg/kg/min dose, 100 mg propofol and 40 mg rocuronium. After a minute of manual ventilation, patient's tracheal intubation was performed and controlled ventilation was started with oxygen-air (40/60) and tidal volume of 600 mL at 12 breaths per minute. Maintenance of anesthesia was performed with 1.5% sevoflurane and remifentanyl by continuous infusion at doses between 0.05 and 0.1 mg/kg/min. For postoperative analgesia, we used 50 mg dexketoprofen and 1 g paracetamol administered 40 min before the end of the surgery procedure. The patient was extubated once spontaneous ventilation recovered. The surgery procedure lasted 125 minutes during which the patient remained hemodynamically stable with a heart rate between 65 and 70 bpm, systolic blood pressure between 100 and 120 mmHg and diastolic blood pressure between 60 and 80 mmHg.

DISCUSSION

In these patients, episodes of arrhythmias can be induced during anesthesia by the following factors: vagotonic agents, episodes of bradycardia, beta-adrenergic blockers, alpha-adrenergic agonists, bradycardia, thermal variations, hyperkalemia, hypokalemia, combinations of glucose and insulin, hypercalcemia(6 -8) so they must be precisely indicated and potential adverse reactions should be monitored. General anesthesia for these patients requires vigilance and continuous monitoring of heart rate, electrocardiographic tracing including the right precordial leads and ST segment, anesthetic depth, body temperature, neuromuscular block and blood pressure[8,9] . Before the onset of any intervention, it is necessary that the operating room has an external defibrillator with the blade attached, provided do not interfere with the surgical field.

Changes in heart rate are factors that contribute to the onset of malignant ventricular arrhythmias in patients with Brugada syndrome. For this reason, BIS monitoring of anesthetic depth is useful in preventing very deep anesthesia which increase vagal tone by suppression of sympathetic system[7,9].

The thermal variations can also induce tachyarrhythmias, so the core temperature should be monitored, especially in surgery of long duration. Monitoring of muscle relaxation

in a patient with Brugada syndrome is useful for extubation in optimal conditions and in order to avoid anti-acetylcholinesterase drugs such as neostigmine which could cause ST-segment elevation. Halogenated anesthetics may interfere with or alter the QT interval. Niyazi et al found an increase in this interval during induction with isoflurane, no changes were found with sevoflurane but a modest reduction using halothane was observed[10].

We have not found literature data on general anesthesia using remifentanyl in patients with Brugada syndrome. In our patient who underwent an ear surgery procedure, we used remifentanyl due to our experience in the quality of the surgical field and in order to adapt the surgery needs provided by this drug. Remifentanyl administration did not cause any problems, but requires a thorough monitoring of heart rate to prevent bradycardia which may be produced by the drug and that could trigger an episode of arrhythmia.

It is very important to provide adequate analgesia in order to prevent potential arrhythmias that may be triggered by changes in the tone of the autonomic nervous system.

Surveillance of these patients should continue in the immediate postoperative period, monitoring the heart rate and electrocardiogram during the first 24 hours after surgery procedure, as arrhythmias may occur during this period.

In conclusion, we must recognize the electrocardiographic findings of the syndrome, the IAD indications and pathogenesis in order to avoid the use of drugs and conditions that may trigger malignant arrhythmias, and for ensuring a continuous electrocardiographic monitoring during surgery procedure and the first 24 postoperative hours.

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