

Brugada Syndrome: Management and Anaesthetic Implications: A Case Report

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Abstract

Brugada syndrome (BrS) is a genetically determined disorder and has an autosomal dominant pattern of transmission, with incomplete penetrance and variable expression. These alterations may predispose to the development of reentrant arrhythmias, and therefore cause ventricular tachycardia and ventricular fibrillation. Patients may be asymptomatic, but they are prone to develop ventricular tachycardia or ventricular fibrillation and sudden death. Many events that occur during general anesthesia, such as drugs, temperature changes, and heart rate variations, may precipitate lethal arrhythmias in these patients. Thus, an individual anesthetic plan, taking into consideration the drugs that may induce arrhythmias, must be arranged before the surgery. Furthermore, it is essential to provide an exhaustive monitoring during the entire perioperative period. We describe a case of general anesthesia in a patient with Brugada syndrome who was diagnosed with right colon neoplasia and programmed for scheduled right hemicolectomy by laparotomy.

Keywords: Brugada syndrome; General anesthesia; Perioperative management

Introduction

First described in 1992, Brugada syndrome (BrS) is a disorder characterized by a specific electrocardiographic pattern in right precordial leads, which may cause ventricular arrhythmias and sudden death. Brugada syndrome is included among the channelopathies, primary electrical disorders that are not associated with structural cardiac defects [1]. The most specific ECG pattern for BrS is characterized by ST segment elevations in leads V1-V2, followed by a negative T wave, and complete or incomplete right bundle-branch block. BrS is genetically determined and has an autosomal dominant pattern of transmission, with incomplete penetrance and variable expressivity. In most cases of BrS, mutations in the *SCN5A* gene have been described. This syndrome has been associated with various alterations in the transmembrane ion channels that form part of the cardiac conduction system, in absence of structural abnormalities. It consists in a dysfunction of sodium channels which causes an increase of the refractory period. These alterations may predispose to the development of reentrant arrhythmias, and therefore cause ventricular tachycardia and ventricular fibrillation. Patients may be asymptomatic, but they are prone to develop ventricular tachycardia (VT) or ventricular fibrillation and sudden death. It is estimated that the prevalence of BrS is around 5/10,000 [1]. Many events that take place during general anesthesia, such as drugs, temperature changes, and heart rate variations, may precipitate lethal arrhythmias in these patients. Thus, an individual anesthetic plan, taking into account the drugs that may induce arrhythmias, must be arranged before the surgery. Furthermore, it is essential to provide an exhaustive monitoring during the entire perioperative period.

Case Report

A case of general anesthesia is described in a patient with Brugada syndrome. A 68-year-old man was diagnosed with right colon neoplasia and programmed for scheduled right hemicolectomy by laparotomy. His medical history included allergic reaction to chloramphenicol and hypertension. In the preoperative examination, type I Brugada syndrome was diagnosed by positive provocation test with flecainide performed by the Department of Cardiology of our hospital. Surgery was performed for 3 hours under balanced general anesthesia. Premedication was carried out with midazolam (2 mg IV); induction with etomidate (20 mg IV), low dose of propofol (50 mg IV),

fentanyl (200 mcg) and rocuronium (100 mg). Intubation was done with Cormack-Lehane grade II without incident. For maintenance was used sevoflurane (MAC 0.7 to 1.7), fentanyl (150 mcg IV/h), NSAIDs and paracetamol. Monitoring was standard (pulse oximetry, NIBP, continuous ECG in II, V1, V2), connected external defibrillator, eye and dependent parts protection, serum heater system, continuous measurement of esophageal temperature sensor, anesthetic depth (Sedline®), hemodynamic optimization through left radial artery with FloTrac® system and two peripheral venous lines 16G [2,3]. At the end of the surgery, neuromuscular blockade was reversed with sugammadex (400 mg IV) and the patient was extubated without incident. He was transferred to Reanimation Unit for postoperative control and continuous monitoring of ECG for 24 hours.

Discussion

It is difficult to formulate evidence-based guidelines for anesthetic management of these patients due to the absence of prospective studies combined with the low prevalence of Brugada syndrome [2,3]. In patients with BrS, both pharmacological and physiological factors can trigger malignant arrhythmias, including commonly used anesthetic drugs, electrolyte disorders, temperature variations, physiological stress and increased vagal activity [4,5]. All these factors should be considered by the anesthesiologist for the management of these patients. It is necessary to keep a complete intraoperative monitoring, including heart rate, ECG tracing (especially right precordial leads and ST segment), anesthetic depth, body temperature, neuromuscular blockade and blood pressure. Furthermore, it is essential to have an external defibrillator in the operating room, and automatic implantable cardioverter defibrillators (ICD) must be disconnected before the surgery.

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Local anesthetics

Local anesthetics are class Ib antiarrhythmics and thus block sodium channels. Accordingly, they should be used cautiously, and if used, the dose should be minimized [3]. Bupivacaine constitutes a special case, as it remains linked to sodium channels for a long time, and it increases the depression of the rapid phase of ventricular muscle depolarization [6,7]. Although local anesthetics work by blocking sodium channels, the use of those with lower cardiotoxicity than bupivacaine may be considered [3]. We rejected the use of epidural analgesia in our patient, as owing to literature consulted usual local anesthetics are considered possible triggers of Brugada syndrome.

Propofol

An *in vitro* experiment on cardiac myocytes demonstrated that propofol exerts a dose-dependent blockade of whole cell sodium current and induces a hyperpolarizing shift in the voltage-dependence of the inactivation of sodium currents [8]. Clinical experience does not support the recommendation of avoiding bolus dosing for induction in Brugada syndrome patients. Caution is advised for continuous infusions. Propofol has been associated with the development of ventricular arrhythmias in long time infusions. This fact suggests that the causal mechanism may be similar to the one involved in arrhythmogenesis in propofol infusion syndrome, and it is not related to bolus induction dose [9]. Propofol has also been found to inhibit cardiac L-type calcium channels [10], attenuate beta-adrenergic signal transduction [11], and augment acetylcholine receptor activity [12].

Inhalational anesthetics

Among volatile anesthetics, sevoflurane is the one of choice, as it provides greater QT interval stability [13, 14]. There are described cases in which isoflurane and desflurane have been used without incident [15,16]. From our point of view, the fact that isoflurane is associated with “coronary steal” would require further study and caution when using it.

Neuromuscular-blocking drugs and anticholinergics

Regarding neuromuscular-blocking drugs, both depolarizing (succinylcholine) and non-depolarizing agents (generally steroids as vecuronium and rocuronium) have been used without incident. As succinylcholine is related to hyperkalemia phenomena, it should be used with caution.

In terms of blockade reversal, neostigmine has been used safely in many cases [13], however it should be avoided because it can increase ST segment elevation [17]. In our case, we used sugammadex, a γ -cyclodextrin with a lipophilic core that encapsulates rocuronium and vecuronium molecules, forging water-soluble complexes that are excreted in urine. That way we obtained a quick and effective reversal of neuromuscular blockade, reducing the risk of residual paralysis which is particularly relevant in cases of major abdominal surgery. Additionally, we avoided side effects associated with neostigmine and antimuscarinic agents, which may be very significant in patients with BrS.

Antiemetic agents

Use of antiemetic agents has also been questioned, although there are reported cases on use of ondansetron, granisetron, dexamethasone and metoclopramide without incident. Ondansetron bolus should be used cautiously, because it has been associated with prolongation of the QT interval [3]. Metoclopramide should also be used with caution, since as a 5-HT₄ receptors agonist it has potential cardiotoxic effects.

These receptors are found in many locations, for instance at atrial level where their stimulation can trigger supraventricular arrhythmias [18].

Vasoactive agents

Modulation of adrenergic receptors can modify ECG tracing in BrS patients by disturbing ST segment [19]. Alpha-receptor agonists and beta-receptor antagonists can disturb ECG patterns by increasing the magnitude of ST segment elevation or by revealing a Brugada ECG pattern. On the other hand, alpha-receptor antagonists and beta-receptor agonist improve ECG patterns by returning elevated ST segments back to baseline or by decreasing the magnitude of ST segment elevations. The β_1 and β_2 receptor agonist activity of isoproterenol increases calcium current, and it has been used to reduce ST segment elevation [20] and suppress arrhythmic events in patients with BrS [21].

However, data regarding the safety of other commonly used vasopressors is almost non-existent. Ideally, the predominant β -agonist action of dobutamine could be beneficial, while other vasopressors with dual α -agonist and β -agonist effect may have unpredictable results.

Brugada syndrome supposes a challenge for the anesthesiologist, due to the possible appearance of fatal arrhythmias induced by common anesthetic factors such as drugs, temperature variations or hemodynamic and electrolytic changes. It is essential to plan an individual management of these patients by avoiding potential risk factors, as well as to provide an adequate perioperative monitoring.

Limitations

Due to Brugada syndrome's low prevalence and type of studies published, existing literature on this topic is low scientific evidence. There is a webpage (www.brugadadrugs.org), based in case series and expert consensus, that standardizes the use and safety of common drugs used in anesthesia. In any case, owing to the low prevalence of this syndrome and the low volume of publications, further studies are necessary.

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