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Case Report Open Access

A Case Report on Brugada Syndrome and Anesthesia Considerations

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Introduction

Brugada Syndrome (BrS) is a rare autosomal disease caused by a genetic mutation affecting the ion channels of the cardiac conduction system. The administration of some routinely used anesthetic agents have shown to trigger spontaneous ventricular arrhythmias on BrS patients leading to sudden cardiac arrest. This is a case report on a 20-year-old male patient with a history of cocaine abuse presenting to the emergency room (ER) with palpitations, chest discomfort, and syncope. Electrocardiogram (ECG) changes led to the diagnosis of BrS. The patient underwent an automatic implantable cardioverter-defibrillator (AICD) under general anesthesia without any complications, however routine anesthetic agents may induce lethal arrhythmias in BrS. This case report focuses on increasing the safety awareness for anesthesia care of patients with BrS.

Case Report

A 20-year-old male patient, 65 inches tall and weighing 85 kg, with a history of cocaine abuse, presented to the ER with discomfort and fluttering in the chest and syncopal episodes. All blood tests done were within normal limits including cardiac enzymes. The patient tested positive for cocaine in urine toxicology drug screen. Cardiovascular examination revealed ongoing complaints of palpitations and syncope. Rest of the systems were found to be within normal limits. The 12 lead ECG performed on this patient showed a partial right bundle branch block (RBBB) with a concave pattern of ST elevation in leads V1 to V3 consistent with the BrS pattern [1,2]. Echocardiogram showed no structural or valvular heart disease (Figure 1).

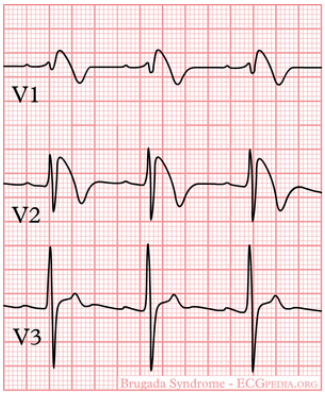

Figure 1: Typical ECG pattern in Brugada syndrome is ST elevation in V1-V3 without ischemia. Diagnosis often missed unless the patient becomes symptomatic as a result of life threatening arrhythmias.

EF was 60%. Surgical history included a brain tumor removal 2 years ago. According to the American society of anesthesiologists (ASA) physical status, the patient was classified as an 'ASA 4' since BrS put him at a high risk for life-threatening ventricular arrhythmias. Therefore, the patient underwent an AICD implant under general anesthesia.

Anxiolysis was achieved with the intravenous administration of midazolam 2 mg at the start of anesthesia. General anesthesia was then induced using intravenous doses of fentanyl 150 mcg, lidocaine 100 mg, propofol 200 mg, and succinylcholine 200 mg. After intubating the trachea, anesthesia was maintained using sevoflurane 2.5%, fentanyl 100 mcg and rocuronium 50 mg. A single chamber AICD was implanted uneventfully by the surgeon. Ventricular fibrillation (VF) was induced and AICD placement confirmed prior to the surgical closure. Neostigmine 4 mg and glycopyrrolate 0.8 mg was administered intravenously at the end of procedure to antagonize the neuromuscular blockade. Continuous monitoring of the ECG showed no abnormal findings as a result of surgery or anesthesia. Patient was transferred to ICU for 24 hour continuous cardiac monitoring. He recovered well and was discharged home the following day.

Discussion

Induced VF intra-operatively converted to sinus rhythm, however, if the VF required immediate emergency management, the use of a class I antiarrhythmic agent such as lidocaine could worsen this patient's condition due to its effect on the sodium ion channels affecting cardiac conduction [2]. The administration of propofol and neostigmine has also shown to induce arrhythmias on some BrS patients according to the evidence from the literature [2,3]. Therefore, this case report focuses on the challenges faced by the anesthesia providers in administering safe anesthesia to BrS patients and make recommendations for alternative anesthetic agents that could be considered on BrS patients in order to minimize their risks under anesthesia.

BrS is a hereditary cardiac disease, which was first described in 1992 by P. Brugada and J. Brugada. The physiology behind BrS is thought to be related with a genetic mutation in SCN5A gene, coding for the human cardiac sodium channels (hH1) insensitive to tetrodotoxin [2]. In BrS the sodium channels do not function normally leading to an enhanced inactivation of these channels causing fatal ventricular arrhythmias [2]. The mechanism of ventricular arrhythmias is not clear and thought to be related to calmodulin, a ubiquitous calcium-sensing protein that binds to the carboxy-terminal IQ domain of the hH1 channel in a calcium-dependent manner [2]. A naturally occurring mutation in the IQ domain alters hH1 function in a manner characteristic of BrS [2].

Although BrS is rare, it is considered the most prevalent cause of sudden cardiac death in younger population [1,2]. BrS raises specific

concerns because anesthesia providers routinely administer drugs that interact with cardiac ion channels, potentially triggering the development of malignant arrhythmias [1-4]. History of previous surgeries done uneventfully under general anesthesia do not lower the risks of subsequent adverse events in future [1]. This patient had a brain tumor removed two years ago without any major complications. BrS was left undiagnosed which put the patient at a potential risk for arrhythmias under general anesthesia with the anesthesia providers unaware of his condition and not anticipating any risks with the anesthetic agents normally used.

BrS is characterized by an ECG pattern of RBBB and ST segment elevation in the right precordial leads (V1 to V3), without evidence of underlying structural heart disease [2] (Figure 2).

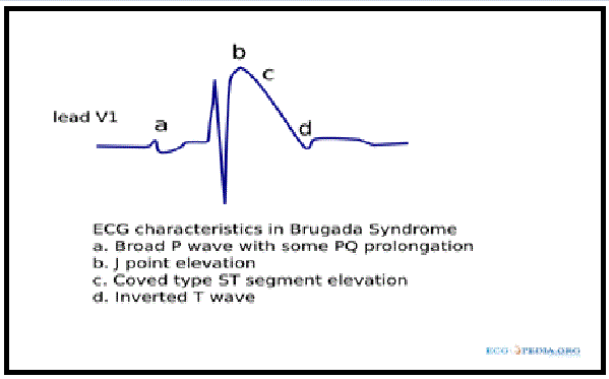

Figure 2: ECG characteristics in Brugada syndrome.

It is associated with a high risk of ventricular tachyarrhythmias and sudden death [2]. This patient showed classic presentation of the BrS ECG pattern with no underlying structural or valvular disease. Establishing the diagnosis of BrS can be difficult since the electrocardiographic signature is concealed in about 30% of the affected patients [2]. These changes are usually seen after the administration of sodium channel blockers, lidocaine being the most commonly used [2].

Cocaine ingestion can also unveil the BrS pattern ECG, which is how this young patient was diagnosed [4]. Because of the ability of cocaine to inhibit the generation and conduction of action potentials related to its sodium channel blocking effects, it acts in a manner similar to that of a Class I antiarrhythmic agent and its use could be detrimental in a patient with BrS [4].

Propofol, the commonly used hypnotic agent has the potential to alter ion channel function inducing Brugada-like ECG abnormalities, which can exacerbate the risk of malignant arrhythmias in BrS patients [1,3]. While the literature review supports the avoidance of propofol on induction and maintenance, no ST changes were noted on induction with its use on this patient.

Thus, the evidence from case reports cautions the use of class I antiarrhythmic agents, propofol, and neostigmine on BrS patients due to its tendencies to induce fatal arrhythmias and augment ST segment changes [1,3]. Therefore, it is best to avoid these agents on BrS patients. While propofol and lidocaine were safely administered and neuromuscular blockade reversed using neostigmine without any problems in this patient, an awareness of the associated risks will alert the anesthesia providers to exercise more caution while considering to administer these agents on BrS patients.

The proposed anesthetic plan should consider safer alternatives during induction, maintenance and emergence. BrS induction may be performed with midazolam, fentanyl, etomidate; general anesthesia maintained with volatile anesthetics, opioids, and muscle relaxants. Edrophonium may be considered over neostigmine as a possible choice to antagonize the neuromuscular blockade. Thus, anesthetic agents that might exacerbate ST segment elevations and lead to dysrhythmias such as lidocaine, propofol, and neostigmine should be avoided and replaced with safer alternatives while achieving amnesia, analgesia, sedation, and muscle relaxation [1-3].

While the use of lidocaine on this patient was concerning to the anesthesia providers, the risks associated with propofol, and neostigmine were not considered significant since BrS was thought to be exacerbated with sodium channel blockers alone. Although these drugs were safely used on this patient, an awareness about the cardiovascular risks involved would enable the anesthesia providers to remain more vigilant and consider alternative agents in maintaining stable hemodynamics under anesthesia. Reported experience of general anesthesia in BrS is limited at present, and more research is warranted towards formulating an ideal anesthetic plan.

BrS is being recognized increasingly resulting in a greater exposure of the anesthesia providers in their clinical practice to this relatively rare syndrome affecting the cardiac sodium channels [2]. Close cooperation of the anesthetist with the cardiologist and surgeon is essential both before and after surgery. Recovery of any post-operative patient with BrS should take place in an intensive care unit. This will permit detection and treatment of cardiac arrhythmias most likely to occur in the postoperative period [1,2]. This calls for a multidisciplinary approach that is tailored to individual patient's needs in order to successfully implement a well-planned perioperative care [1]. The diverse anesthetic management, prompt recognition, and therapeutic interventions required in BrS makes the perioperative management a challenge for the anesthesia providers [1,5].

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