INTRODUCTION

Brugada syndrome (BrS) was first described as a separate clinical entity in 1992 by P. Brugada and J. Brugada.¹ These patients have an abnormality of cardiac voltage-gated sodium ion channels that increases the delay in depolarization in the right ventricular outflow tract leading to the risk of fatal arrhythmia and sudden cardiac death. Mutations in calcium and potassium channels, associated channel proteins, and desmosomal proteins have also been linked with the disease. It is an autosomal dominant genetic disease,² the prevalence is 3-5 per 10,000 people more prevalent in middle-aged South East Asian males.

Symptoms may include palpitations, chest discomfort, and syncope.³ However, as many patients are asymptomatic, Brugada syndromes is frequently an incidental diagnosis on ECG. Initially, it was described to have a structurally normal heart, however, newer research revealed right ventricular outflow tract abnormalities, such as an increase in adipose tissue and fibrosis. Depending on ECG findings Brs has been classified into three types,⁴ as shown in [Figure 1]. Apart from 12 lead ECG, provocative drug testing, invasive cardiac electrophysiology, and genetic testing are other modes of diagnosis.

The potential triggers are the increased vagal tone, fever, or sodium channel-blocking drugs, alcohol, and electrolyte disorders; these can all accentuate the ST elevation noted in a Brugada ECG.⁶ Many events that take place during general anaesthesia, such as drugs, temperature changes, and heart rate variations, may precipitate lethal arrhythmias in these patients. Thus, an individual anaesthetic plan, taking into account the drugs that may induce arrhythmias, must be arranged before the surgery. Furthermore, intensive perioperative monitoring is essential.

CASE SERIES

CASE 1

A 38-year-old female with a right supraspinatus tear was posted for endoscopic repair under General anaesthesia with interscalene block. The patient was American Society Anaesthesia (ASA) II with a history of hypothyroidism for 2 years for which Tablet Thyonorm 25 mcg was regular treatment. The patient’s vitals were within normal limits (WNL) except ECG showed RBBB with coved ST-T, and 2-Dimensional Echocardiograph(2DEcho) was in the normal range. A cardiologist reference was done and the patient was diagnosed to have Brugada syndrome.
 Syndrome type II. Preoperatively ultrasound-guided right interscalene block was administered using 10 ml of 0.2 % Ropivacaine followed by General anaesthesia using a standard dose of the injection of midazolam, fentanyl, propofol, and rocuronium. The Patient was reversed using an injection Suggamadex (2 mg /kg). The perioperative patient was haemodynamically stable without ST-T exaggeration and arrhythmia. Postoperatively the patient was monitored in the recovery room till recovery from anaesthesia.

**CASE 2**

An 80-year-old female was posted for cystocele repair under SAB. The patient was a known case of Hypertension for 5 years and was controlled by Tablet Cilacar T 10/40 mg twice a day. The patient had a history of Dyspnoea of grade II. ECG showed a Brugada type I pattern with RBBB, 2DEcho showing left ventricular hypertrophy, left ventricular diastolic dysfunction, and ejection fraction 55 %. Pulmonary function test showed severe small airway obstruction with poor bronchodilator therapy. On general examination, there were missed beats 10 per minute with a pulse rate of 80/min and BP of 130/80 mmHg. The patient received SAB with 2 ml of hyperbaric Ropivacaine 0.75%. Adequate sensory and motor level was achieved and the patient was stable perioperatively without any fatal arrhythmias. The patient was monitored in ICU for 24 hrs and shifted to the ward.

**CASE 3**

A 48-year-old male had a right-side distal end radius fracture with a right bicondylar humerus fracture scheduled for open reduction and internal fixation of the radius and humerus. His medical history was insignificant. ECG showed J point elevation and early repolarisation in V1, V2, and V3 leads along with T wave inversion in I, II, and aVL leads which was diagnosed as Brugadas syndrome type I. 2DEcho was normal. This patient was administered an Ultrasound-guided right supraclavicular block using 15 ml of 0.5 % ropivacaine. The perioperative patient was comfortable with the sedation of midazolam and fentanyl. Vital parameters were stable periopeatively without any fatal arrhythmias. The patient was shifted to the recovery room for further monitoring.

**CASE 4**

A forty-five-year-old female was posted for hysteroscopic removal of a cervical polyp. The patient presented with menorrhagia and weakness. She had recent history of upper respiratory tract infection which was treated with antibiotics and nebulization. The patient’s ECG reported having Brugadas syndrome type II with saddle-shaped ST-T segment elevation in lead V1, V2 with RBBB. The patient didn’t have any significant medical or surgical history. Her 2D Echo study was normal. Spinal anaesthesia was given using 2.5 ml of hyperbaric Ropivacaine 0.75%. Adequate sensory and motor level was achieved and the patient was stable periopeatively. The patient was monitored in the recovery room till the wearing of anaesthesia.

**CASE 5**

A forty-one-year-old male was posted for open reduction with internal fixation of right distal humerus fracture. The patient was known to have a case of Diabetes mellitus type II and was controlled by an oral hypoglycaemic agent. On ECG, the patient had J point elevation in V1, V2 with RBBB. 2DEcho was normal. This patient was administered an Ultrasound-guided right supraclavicular block using 15 ml of 0.5 % ropivacaine. The perioperative patient was comfortable with the sedation of midazolam and fentanyl. Vital parameters were stable periopeatively without any fatal arrhythmias. The patient was shifted to the recovery room for further monitoring.

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**Table 1: List of safe /unsafe drugs in Brugada syndrome.**

<table>
<thead>
<tr>
<th>Class I</th>
<th>The drug is potentially arrhythmic</th>
<th>Class IIa conflicting evidence about the drug, being potentially arrhythmic</th>
<th>Class IIb less well-established evidence/opinion being potentially arrhythmic</th>
<th>Class III no or very little evidence about the drug being potentially arrhythmic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plecanide</td>
<td>Propafenone</td>
<td>Ethacizin</td>
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<tr>
<td>Pilsicainide</td>
<td>Aminopyrine</td>
<td>Allapinn</td>
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<td>Procanamide</td>
<td>Desipramine</td>
<td>Lithium</td>
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<td>Isoproterenol</td>
<td>Loxapine</td>
<td>Amiodarone</td>
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<td>Quinidine</td>
<td>Nortryptiline</td>
<td>Lidocaine</td>
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<td>Oxcarbazepine</td>
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<td>Bupivacaine</td>
<td>Carbamazepine</td>
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<td>Propofol</td>
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<td>Veerapamil</td>
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<td>Neostigmine</td>
<td>Phenytion</td>
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<td>Succinylscoline</td>
<td>Ketamine</td>
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<td>Noradrenaline</td>
<td>Tramadol</td>
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<tr>
<td>Metoclopramide</td>
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<td>Fentanyl</td>
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International Journal of Academic Medicine and Pharmacy (www.academicmed.org)  
ISSN (O): 2687-5365; ISSN (P): 2753-6556  
127
DISCUSSION

Although Brugada is uncommon, due to its nature of disease leading to sudden death in the middle-aged population,[1] it is principal for health workers, to be aware of this genetic disease,[2] As anesthetists run across these patients in their practice, it is mandatory to be aware of dos and don’ts while anesthetizing such patients for various procedures. Patients who have survived cardiac arrest have been treated with an implantable cardioverter defibrillator (ICD). The new emerging, promising treatment is radiofrequency ablation of the anterior part of the right ventricular outflow tract.[4-8] Even, asymptomatic patients with Brugada coming for surgical procedures demand intensive perioperative monitoring. There is a list of drugs by Brugada drug organization, categorized as safe to use, precautions while using, or contraindicated as shown in [Table 1]. The safe management of anaesthesia of Brugada patients involves avoiding trigger factors,[9] using safe drugs and methods, and managing arrhythmias perioperatively by in-depth monitoring. In our case series, we managed asymptomatic, Brugada syndrome patients posted for the different surgical processes with the appropriate type of anaesthesia and choosing available safe drugs, avoiding trigger factors and drugs. We didn’t encounter fatal arrhythmias perioperatively in our patients.[10]

CONCLUSION

It is paramount to manage Brugada syndrome patients with safe anaesthesia techniques with available safe drugs, avoiding trigger factors and drugs along with rigorous perioperative monitoring for fatal dysrhythmia.

REFERENCES

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