

# Anaesthetic issues in Brugada electrocardiogram / syndrome undergoing laparoscopic common bile duct exploration and laparoscopic primary common bile duct closure

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## Abstract

Here we report a rare case of Brugada Syndrome in a young Nepalese male undergoing laparoscopic common bile duct exploration and laparoscopic primary common bile duct closure. Recognition of the disease, preparedness to tackle the developing arrhythmias, avoidance of precipitating drugs, adopting policy of anaesthetic interventions to balance the autonomic system, maintenance of normothermia and electrolyte balance, avoidance of hypoxia, hypercarbia and acidosis are the key to management of this disease in anaesthetic pursuit.

**Key words:** Anaesthetic, Brugada Syndrome, Common Bile duct, Laparoscopy

## INTRODUCTION

In 1992, Pedro and Joseph Brugada described a series of eight patients successfully resuscitated from ventricular fibrillation. They all had common features of Right bundle branch block (RBBB) with ST elevation in right precordial leads V<sub>1</sub> to V<sub>3</sub> and this new clinical entity was syndromised as Brugada syndrome (BS) the same year.

BS is one of the causes of sudden cardiac death following ventricular tachycardia (VT) and Ventricular fibrillation (VF) in young healthy men in absence of demonstrable morphological cardiac pathology. We report a case with preoperative electrocardiogram (ECG) typical of Brugada type. Though the given case cannot be syndromised on the background of absence of strong clinical features, preoperative anaesthetic issues warrant complete preparation ahead of time due to possible sudden lethal arrhythmias it can cause leading to sudden cardiac death. This clinical morbidity has attracted the attention of clinicians all over the world.

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## CASE REPORT

A 35 year male had an episode of chest pain, palpitation and shortness of breath while awaiting laparoscopic common bile duct (CBD) exploration. Electrocardiogram was suspicious of BS and he was referred to Sahid Gangalal National Heart Centre (SGNHC) where, after further cardiac evaluation, the diagnosis of BS was confirmed. He denied previous syncope attacks or sudden cardiac death in his family. Echocardiography showed normal systolic (Ejection Fraction 60%), diastolic and valvular functions. Twelve lead ECG showed RBBB and ST elevation in right precordial leads V<sub>1</sub>-V<sub>3</sub>.

He was anxious and his anxiety was exacerbated after the nature of the syndrome and the risk of sudden death was explained to him.

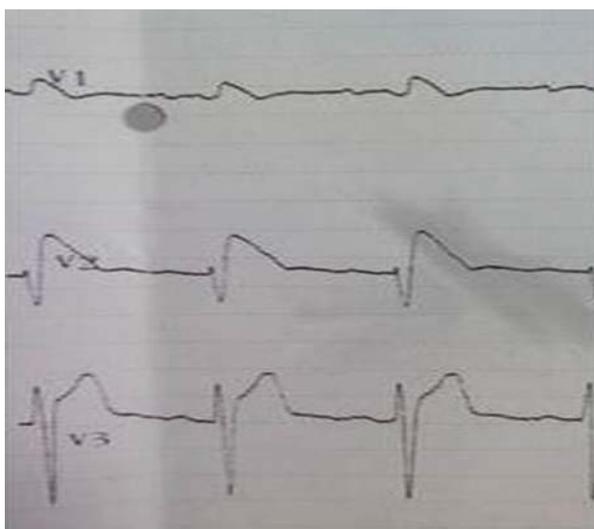
Preoperative anaesthetic evaluation was carried out. Airway evaluation revealed grade I Mallampati Grading. Cardiovascular and respiratory system examination were normal. Heart rate was 85 per minute and regular. Blood pressure was 140/80 mmHg. His repeat ECG was unchanged. Laboratory investigations were within normal limits. He was premedicated with tablet Diazepam 10 mg and Pantoprazole 40 mg the night before surgery.

On arrival to theatre peripheral intravenous access was secured. Routine monitoring was applied comprising

ECG, pulse oximetry, capnography and invasive blood pressure monitoring. In view of the increased cardiac risk additional preparations made were application of defibrillator pads to the chest, insertion of temporary transvenous pacing via right internal jugular vein and loading of Isoproterenol for infusion.

He was induced with Propofol (2 mg/kg). Analgesia was provided with Fentanyl (2.5 mcg/kg) and muscle relaxation was achieved with Vecuronium (0.1 mg / kg). Anaesthesia was maintained with oxygen-air and Propofol infusion. Prior to creation of Carbon dioxide pneumoperitoneum, Inj Atropine 0.6 mg was administered intravenously and intra-abdominal pressure of 11 mmHg maintained during the procedure. Ventilatory parameters were adjusted to have End Tidal carbon dioxide (ETCO<sub>2</sub>) of 36 to 38 mmHg. Intraoperative ECG monitoring showed no significant deviation from that of preoperative pattern. Patient remained haemodynamically stable with blood pressure ranging from 110 to 140 mmHg systolic, 70 - 85 mmHg diastolic and heart rate 68 to 114 per minute. Analgesia was achieved with addition of Fentanyl and Non-steroidal anti-inflammatory drug. Total consumption of Fentanyl was 375 mcg. The surgery lasted for about 3 hours and 15 minutes.

He had perforated and sealed gall bladder wall. Common bile duct was dilated proximally with impacted distal stones. Cholecystectomy, choledocholithotomy and the primary closure of the duct was carried out laparoscopically. T tube was not used.



**Figure 1:** Coved type ECG showing ST elevation in V<sub>1</sub>-V<sub>3</sub> and RBBB in the patient

Intraoperative electrolytes, blood glucose and arterial blood gas were within normal limits. The effect of muscle relaxant was assessed with Train of Four (TOF) stimulation and following spontaneous restoration of muscular activity and respiration, trachea was extubated without use of anticholinesterase.

Postoperative analgesia was covered with Inj Diclofenac, Paracetamol and Fentanyl. No ECG changes other than he had preoperatively were encountered during serial investigations. He had persistent ST elevation in the same ECG leads like before. Cardiac enzymes, electrolytes, echocardiography were found to be normal. He was then stepped down to intermediate surgical intensive care unit (ICU) after three days. His stay in the hospital was uneventful and he was discharged home six days after the surgery.

## DISCUSSION

The patient had no structural cardiac defects in echocardiography. He neither had attacks of seizure nor agonal breathing at night time. The ECG encountered was typical of BS: coved type which persisted throughout the perioperative period, Figure 1. The characteristic ECG pattern is an RBBB with right precordial ST segment elevation (V<sub>1</sub>-V<sub>3</sub>). The QT interval is normal and the ST elevation may be "coved" or "saddle shaped." The syndrome affects patients of all age groups, including infants, but it peaks in the fourth decade<sup>2</sup>. He was not under any medications which could precipitate the syndrome nor had he fever during the episode. Functioning of cardiac sodium channels is body temperature dependent<sup>3</sup>. Hyperthermia or febrile episodes have been claimed to depress the sodium channels which are already low in BS. BS is one of the "channelopathies" and autosomal dominant transmission is usual. Mutations of the SCN5A gene responsible for the alpha subunit of the cardiac sodium channel have been claimed in approximately 20 to 25% of individuals<sup>3,4</sup>.

Hyper/hypokalaemia or hypocalcaemia may be the precipitating factors in generating ECG characteristic of BS<sup>4</sup>. Serial electrolyte reports showed he had normal electrolytes. Presence of coved type ECG in right precordial leads, history of syncope or documented VT/VF or survived cardiac arrest with family history of sudden cardiac death confirm the diagnosis of BS. In concealed BS provocative test with sodium channel blocker is carried out in laboratory. Above mentioned are the diagnostic criteria devised by consensus conference<sup>5</sup>.

The diagnosis requires a high level of suspicion as resting ECG is often borderline or even looks normal. Genetic testing facility may not be easily available and everywhere. Pharmacological and/or exercise testing manipulates the cardiac action potential and provokes a characteristic arrhythmia. This test may be useful in unmasking the syndrome in more than 50% cases where diagnosis is more difficult<sup>4</sup>.

Provocative testing is performed employing either of the drugs like Flecainide, Ajmaline or Procainamide with continuous cardiac monitoring of the patient where advanced resuscitation equipments are instantly available<sup>3,5</sup>.

Our patient did not undergo pharmacological stimulation test with sodium channel blocker. Provocative test with Flecainide is diagnostic, but carries risks too. In presence of coved type ECG the test is not recommended at present. Moreover the test carries no prognostic value<sup>6</sup>. The definitive gold standard treatment is placement of implantable cardioverter defibrillator (ICD) to prevent sudden cardiac death. Though ICD has been strongly recommended in such patients, availability and cost make it an unrealistic option to most of our population.

Arrhythmias and symptoms usually occur at times of rest or sleep when vagal activity predominates. Avoidance of parasympathetic dominance, beta blockers, halothane, calcium channel blockers and anticholinesterase were the precautionary measures used perioperatively<sup>7-9</sup>. Isoproterenol was loaded ready for infusion in case of VT/VF. The L-type calcium channels which are down regulated in this syndrome are activated by Isoproterenol. Polymorphic VT has been corrected by the infusion of Isoproterenol in such patients<sup>10</sup>. In hypotensive conditions mixed beta and alpha agonists like Ephedrine has been found to be safe in this syndrome<sup>7</sup>. Titrated dose of Dopamine is also recommended for this purpose in case of hypotension not correctable with fluid therapy<sup>11</sup>. Provision was made for temporary transvenous pacing in this patient in order to provide an effective therapy against bradycardia-related VT/VF in BS<sup>12</sup>.

The mechanism of VT/VF in this syndrome is thought to be due to phase two re-entry type arrhythmia<sup>13</sup>. ST-segment elevation in BS is associated with an imbalance in action potential gradients between the right ventricular epicardium and endocardium<sup>14</sup>. The functional loss of sodium channel causes predominance of temporary outgoing potassium channel which are usually balanced in phase 1 action potential. This results in shortening of action potential and loss of plateau phase in epicardium of right ventricle.

Amiodarone and  $\beta$ -blockers have been shown to be ineffective, rather detrimental in BS<sup>15</sup>. This makes VT and VF in BS different and complex from that of other origin. Propofol, Fentanyl and Vecuronium have good safety profile in BS from reports of previous documentations of cases in the literature<sup>8,9</sup>. Hypotension and bradycardia are frequently encountered with concomitant use of these three drugs. This may not be favourable in BS where slow heart rate may precipitate VT/VF. The use of atropine in our case might be the reason why the combination of these drugs did not accentuate the vagolytic effects. Neostigmine may augment ST segment elevation and increases acetylcholine level which may be counterproductive in BS<sup>8</sup>. Reports in laparoscopic surgery in patients with Brugada syndrome are yet sparse in the world literature. Furthermore laparoscopic primary CBD closure itself in a patient with BS holds academic interest. It is our common practice to provide analgesia through thoracic epidural in CBD exploration, but epidural line was not placed in this case as bupivacaine being a sodium channel blocker has been reported to trigger VT in BS<sup>16</sup>.

## CONCLUSION

Anaesthesiologists should have high index of suspicion if such pattern of ECG is encountered in surgical patients. Understanding of BS is necessary as it may impose sudden cardiac death. Perioperative beta blockers and parasympathetic stimulation are not allowable. Isoproterenol may be a key agent in case of lethal ventricular arrhythmias. Safe execution of Ephedrine and cautious use of Dopamine have been advised in case of perioperative hypotensive episodes.

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