

SUDDEN CARDIAC ARREST DURING GENERAL ANAESTHESIA IN AN UNDIAGNOSED BRUGADA SYNDROMEJyothisna M¹, G. V. Rao², Vinutha Patil³, Ramesh K⁴**HOW TO CITE THIS ARTICLE:**

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ABSTRACT: The clinical presentation of BS varies from patients being asymptomatic, to having a history of syncope, seizures, palpitations, nocturnal agonal respiration, and aborted sudden death and the majority of patients have a family history of sudden death or malignant arrhythmias. It is important for the anesthesiologist to be familiar with Brugada syndrome since known and unknown BS patients may present for surgery. Investigations revealed an haemoglobin of 9.3gm/dl, serum creat-0.73, normal electrocardiogram (ECG) and serum electrolytes and blood sugar level were within normal range. ECG showed ventricular fibrillation followed by asystole. Immediate Cardiopulmonary resuscitation (CPR) was initiated and inj adrenaline 1 in 10000 1mg iv given. Rest of the period of ICU stay was uneventful while adequate analgesia/and sedation along with close monitoring was done

KEYWORDS: Brugada syndrome, LSCS, CPR.

INTRODUCTION: Brugada syndrome (BS) was first identified by Brugada brothers in 1992, is an autosomal dominant disorder with variable penetrance,^[1] and describes patients at risk of ventricular arrhythmias and sudden death who have a structurally normal heart.^[2]

The clinical presentation of BS varies from patients being asymptomatic, to having a history of syncope, seizures, palpitations, nocturnal agonal respiration, and aborted sudden death,^[3] and the majority of patients have a family history of sudden death or malignant arrhythmias.^[4]

It is important for the anesthesiologist to be familiar with Brugada syndrome (BS) since known and unknown BS patients may present for surgery. BS accounts for four percent of all sudden deaths and up to twenty percent of sudden deaths in patients without structural cardiac defects.^[1]

Proper identification of BS is important as the two year mortality approaches 30 percent.^[4] The prevalence of BS differs based on population and is more common in Southeast Asia (12/10,000) than in Europe (1-5/10,000).^[1] The average age of diagnosis is 40-45 years and 80% of patients are men.^[1]

METHODOLOGY & RESULTS: A 35 year old lady weighing 50 kg was scheduled to undergo laparotomy in our hospital. She had presented to the hospital with history of lower abdominal pain and purulent discharge from previous Lower Segment Caesarian Section (LSCS) wound site. On preoperative assessment, she had no history of syncopal attacks or palpitations, had stable vitals, systemic examination was normal and airway was adequate. Investigations revealed haemoglobin of 9.3gm/dl, serum creat-0.73, normal electrocardiogram (ECG) and serum electrolytes and blood sugar level were within normal range. X-ray erect abdomen showed large dilated intestinal coils. Ultrasonography (USG) revealed intestinal obstruction with mild loculated intraperitoneal collection seen in pelvis. Contrast Enhanced Computed Tomography (CECT) showed features suggestive of gossypiboma.

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Standard Nil per oral (NPO) guidelines were followed and was adequately preloaded with 10 ml/kg of Ringer's lactate. She was monitored with electrocardiogram (ECG), Non Invasive Blood Pressure and pulse oxymetry. Under full aseptic precautions, subarachnoid block in L3-4 interspace, using 23g Quincke's needle, inj bupivacaine (Heavy) 14mg and 60microg buprenorphine administered. Sensory level was achieved till T6 dermatome. Segmental resection, recto sigmoid anastomosis, sigmoid colostomy was done for multiple small bowel perforations and a 20*10 mop found. Following recovery from subarachnoid block, general anaesthesia was induced. Patient was administered inj glycopyrrolate 0.2mg iv, inj pethidine 50 mg iv after preoxygenation with 100% oxygen for 3 mins. Induced with inj thiopentone 200mg, inj succinylcholine 100mg and then intubated with 7.5mm cuffed Endotracheal (ET) tube. After 5 mins of induction, patient went into sudden cardiac arrest. Pulse was not felt, BP was not recordable, ECG showed ventricular fibrillation followed by asystole.

Immediate Cardiopulmonary resuscitation (CPR) was initiated and inj adrenaline 1 in 10000 1mg iv given. After 2 cycles of CPR patient was revived. Blood samples were sent for serum electrolytes, sugars. Patient was maintained with 100% oxygen and inj vecuronium 1 mg intermittently. Vasoactive support was initiated with appropriate doses of Inj dopamine and noradrenaline, while blood pressure improved and surgery was allowed to continue. At the end of surgery (which lasted for 6 hours) patient was moved to intensive care unit (ICU) with ET tube in situ and without reversing neuromuscular block. In the ICU patient was put on mechanical ventilatory support and connected to monitors. A 12 lead ECG was done, which showed ST elevation of 2mm in lead V2. Meanwhile the electrolytes and sugars were within normal limits after one hour in the ICU patient had ventricular tachycardia for which patient was treated with inj amiodarone 300mg.

Rest of the period of ICU stay was uneventful while adequate analgesia/and sedation along with close monitoring was done. Patient was gradually weaned and extubated on 4th postoperative day. After a further 24 hr observation that was devoid of any arrhythmia, she was transferred to the ward.

However next day in the ward patient developed sudden onset of breathlessness and desaturation. Even with oxygen supplementation via face mask at 10lt/min her saturations were only 80%. On auscultation of chest she had bilateral coarse crepitations. She was immediately intubated and ventilation was supported with ambu bag and moved to the ICU. In the ICU she was put on ventilator support and connected to monitors. She was adequately sedated and analgesia given. Meanwhile the ECG showed rSR pattern with ST segment elevation in leads V1, V2, and V3. A 2D echo showed no structural abnormality and systolic and diastolic functions were adequate. Arterial blood Gas estimation showed metabolic acidosis which was corrected. Electrolytes were within normal limits. Next day she was started on nasogastric tube feeds. Her general condition improved and on third day of ICU stay she was extubated. On fourth day she was transferred to ward from where she was discharged later with advice to undergo further cardiac evaluation at higher centre. She was followed up after two weeks and was doing well.

DISCUSSION: Brugada syndrome is more common in young men without any underlying cardiac disease and most common arrhythmia is ventricular fibrillation (VF) and it usually presents with fainting, syncope and sudden death without any warning signs.^[1] The pathology is due to gene mutation SCN5A located on 3p,^[5] which codes for sodium ion channel on cell membranes of myocytes of heart. Mutation of this gene leads to loss of action potential dome of some epicardial and

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transmural areas in right ventricle. This results in dispersion of repolarisation in epicardial and transmural areas. In transmural areas, due to dispersion of repolarisation, there is ST segment elevation, whereas due to dispersion in epicardial areas there is generation of extra asystole due to phase 2 reentry leading to Ventricular tachycardia or Ventricular Fibrillation which may lead to sudden cardiac death. Adrenergic stimulation decreases ST elevation whereas vagal stimulation worsens it.

The diagnostic criteria of BS consist of 2 parts: (A) detection of the typical ECG abnormality and (B) clinical characteristics.^[6] Coved-type ST-segment elevation and negative T wave in the right precordial leads (Fig. 1) with or without a drug challenge test in the 12-lead ECG is the hallmark of diagnosis. The ECG in our patient is depicted below (Fig. 2) which correlates with type 2 saddle-back type segment elevation. In conjunction with the ECG abnormality, 1 of the following criteria is necessary: (a) a history of VT/VF, (b) a family history of SCD, (c) a family history of coved-type ECG, (d) agonal respiration during sleep, or (e) inducibility of VT/VF during electrophysiological study.

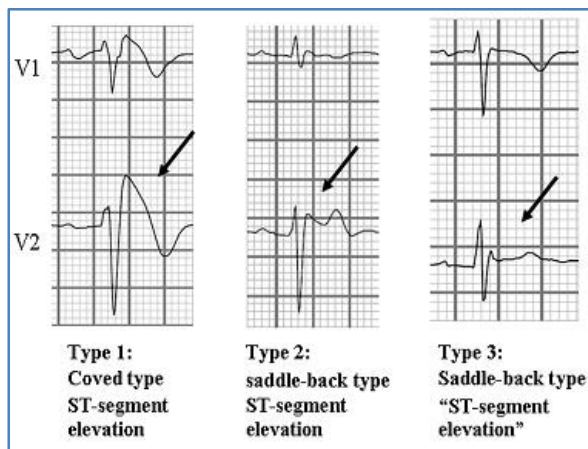


Fig. 1



Fig. 2: ECG after

Anaesthesiologists sometimes encounter patients with brugada syndrome who most often than not have already been diagnosed preoperatively. Patients with BS who have survived a ventricular fibrillation arrest are recommended to receive an implantable cardioverter-defibrillator (ICD) in light of the significant risk of recurrent events.^[1]

The present case was not diagnosed preoperatively as BS. Pre-operatively she had no history of palpitations or syncopal attacks and her ECG was normal. In this case it was diagnosed postoperatively by 12 lead ECG which showed saddle back type ST elevation of 2mm in lead V2 and on doing echocardiography no structural abnormality was found. She had two episodes of arrhythmia postoperatively – in the ICU and later in the ward (Probably she could have had another episode of ventricular arrhythmia which was not witnessed – write in discussion). Existing literature says that ECG in a patient with BS is dynamic and ECG changes are often concealed such that it is revealed only by challenge test with class 1a, 1c (flecainide) antiarrhythmics.^[7] It also says that before and during Ventricular Tachycardia and Ventricular Fibrillation, dynamic appearance of prominent J wave will be seen in ECG.

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Also in our patient the ECG changes developed after induction of general anaesthesia. As autonomic changes can precipitate arrhythmias, adequate depth of anaesthesia should be ensured and postural changes should be minimized.^[8] Increased vagal tone has been implicated in the development of Brugada ECG changes.^[9]

CONCLUSION: Brugada syndrome - Prediagnosed or undiagnosed BS patients present for various surgeries and if not diagnosed presents great challenge to anaesthesiologist. They manifest sudden cardiac arrest intraoperatively. Hence anaesthesiologist should be aware of such occurrences and proper preoperative evaluation and optimisation along with appropriate intraoperative management will prevent unnecessary morbidity and mortality. The documentation of such events will help in improved care of the patient when they present for any other surgeries in future.

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