Anaesthetic Management of a Patient with Brugada Pattern Undergoing Surgery for Cellulitis

Dr Shaheen Khan M[1], Balasubramaniam Gayathri[2], Dr Aravindhan K Y[3], Sai Hrudaya Harshitha Akondi[4]

[1] *Department- Anaesthesiology, designation- MBBS, MD Postgraduate, university name-SRM medical college hospital and research centre country- india. Email id-drshaheenkhan555@gmail.com*
[2] *Department- Anaesthesiology, designation- MBBS, MD (Professor and HOD) university name-SRM medical college hospital and research centre, country- India. Email id- gayathrii.r@gmail.com*
[3] *department- Anaesthesiology, designation- MBBS, MD (Senior resident) university name-SRM medical college hospital and research centre, country- india. Email id-kyaravind@gmail.com*
[4] *Department- Anesthesia Designation- MBBS, MD Postgraduate, University- SRM Medical College and Hospital Research Centre, Country- India. Email id - harshitha0754@gmail.com*
**Corresponding author: Balasubramaniam Gayathri*

KEYWORDS
Brugada syndrome, Electrocardiography, Cardiac event, ST-segment elevation.

ABSTRACT
Brugada syndrome is a genetic condition that has no connection to structural cardiac disease, characterised by an ECG pattern revealing the right bundle branch block, persistent ST-segment elevation in the right precordial leads (V1-V3), and a propensity for malignant. We successfully managed a 60-year-old asymptomatic male patient with Brugada syndrome, identified during pre-anaesthesia evaluation. The patient had a history of early abrupt cardiac death in his family, and his electrocardiogram revealed a Brugada pattern. He was posted for split-thickness skin graft (SSG) under general anaesthesia, following wound debridement for right leg cellulitis. The intraoperative course was uneventful, with the patient remaining haemodynamically stable throughout. The postoperative period was also uneventful. Consequently, early diagnosis and appropriate anaesthetic management require a complete patient history, careful assessment of test data, and ECG.

1. Introduction

The Brugada brothers first described Brugada syndrome in 1992 as a disorder characterised by sudden cardiac mortality or ventricular tachyarrhythmias. Mutations in the SCN5A gene coding for the cardiac sodium ion channels, cause autosomal dominant inheritance.

The prevalence is estimated to be 5-20 per 10,000 patients. It is indigenous to Southeast Asia. The age of onset of the disease is between 20 and 40 years, The prevalence of the illness is roughly 8–10 times higher in men than in women.

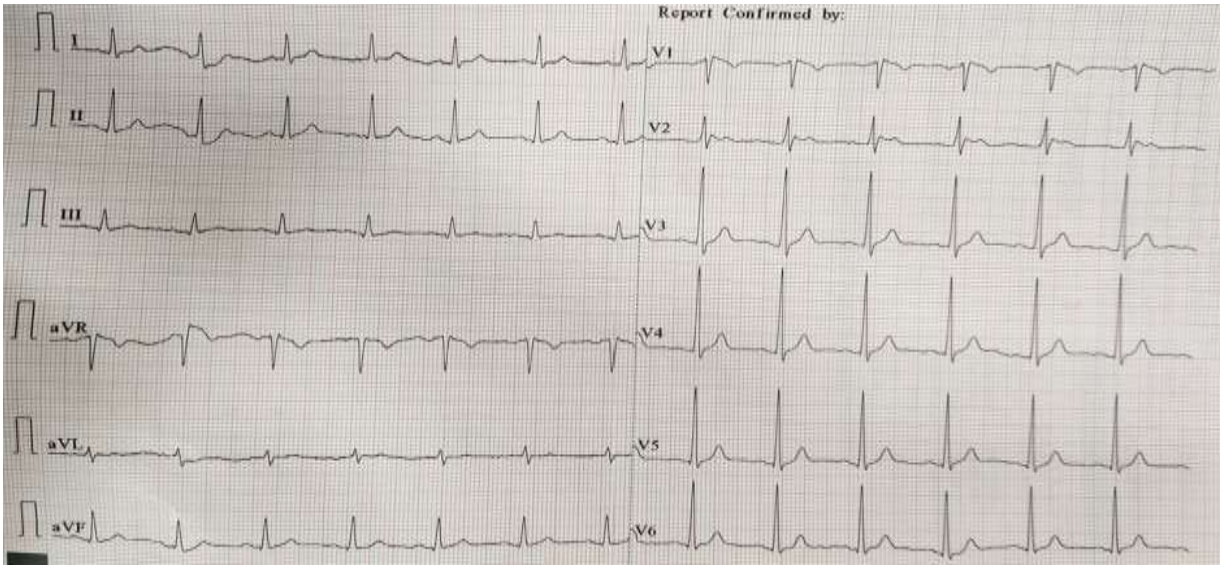
Clinical findings range from asymptomatic to abrupt cardiac death. Initial symptoms may include dizziness, syncope, shortness of breath, palpitations, and convulsions, particularly at night. The most relevant laboratory marker is aberrant electrocardiogram (ECG) findings, which include typical coved-type ST-segment elevation ≥2 mm in leads V1–V3, followed by right bundle branch block (RBBB) and negative T-waves (the most diagnostic pattern) and history of unexplained syncope or sudden cardiac arrest without structural heart disease. If these patients who are incidentally diagnosed with Brugada syndrome are posted for elective or emergency surgical procedures, administering anaesthesia would be a great challenge for any anaesthetist It is an important responsibility of the anaesthesiologist to decide on the anaesthetic techniques such as general anaesthesia, regional anaesthesia, nerve block, intravenous sedation, and monitored anaesthesia care and to choose appropriate drugs.

Certain drugs used in clinical anaesthesia may have proarrhythmic effects and interactions in people with Brugada syndrome. Local anaesthetics and propofol, among other medicines used in anaesthesia and ICU, are known to elicit arrhythmic events in people with Brugada syndrome. This can happen while administering anaesthesia and resolving potential difficulties without an incident. Our goal was to outline the anaesthetic care of a 60-year-old man with Brugada syndrome, who was identified during the pre-anaesthesia assessment since his mother had a history of abrupt cardiac death and exhibited minimal changes in ECG but could be overlooked during preoperative evaluation.

2. Case Report

A male patient, 60 years of age and 65 kg in weight, presented to the General Surgery Department complaining of cellulitis in his right leg and a raw area on his right leg. He had hypertension and type 2 diabetes mellitus for the past 26 years. His family history includes the abrupt cardiac death of his mother. No history of syncope and palpitations.The patient was scheduled for wound debridement followed by a subsequent split-thickness skin graft (SSG) under general anaesthesia. A pre-anaesthetic evaluation was performed, and upon inspection, the patient was oriented and completely aware. Pre-operative vitals showed a non-invasive blood pressure (NIBP) of 110/80 mmHg, a pulse rate of 80 beats per minute, and a SPO2 of 99% in room air. According to laboratory results, the haemoglobin level was 11.5 g/dL, and the leukocyte count, platelet count, coagulation profile, serum electrolyte, and renal parameters were all within normal range.

The ECG revealed a Brugada pattern, which included significant upward coving, a slight right bundle branch block, and T wave inversion in V1–V2. The echocardiography revealed normal left ventricular systolic function with an ejection fraction of 63%, no regional wall motion abnormalities, and Grade 1 diastolic dysfunction (impaired relaxation of the left ventricle). Cardiology consultation recommended close monitoring during anaesthesia, avoiding specific anaesthesia drugs known to trigger arrhythmias in Brugada syndrome. Given the high cardiac risk associated with the Brugada pattern, the patient was assessed as ASA Class III. Informed consent was obtained, and the patient was managed under general anaesthesia with meticulous precautions.



3. Intraoperative Management

After getting informed consent from the patient, he was taken for the surgical procedure. The patient was informed about the significant high risk of the procedure. The patient was placed with standard anaesthetic monitoring equipment, such as a non-invasive blood pressure monitor, a five-lead electrocardiogram, a pulse oximeter, and a capnography.

Intravenous access to the right upper limb was carefully secured with 20G. Preoperative vitals include a Pulse rate of 80/minute, NIBP of 130/80 mm Hg, and SPO2 OF 100% in room air. Antibiotic prophylaxis was given with an injection of Cefazolin 50 mg/kg intravenously 30 minutes before the procedure

The patient was preoxygenated for three minutes before general anaesthesia was administered. Premedication included 0.2 mg of glycopyrrolate, 1 mg of midazolam, and 4 mg of ondansetron. Fentanyl 100 µg was administered to blunt intubation response and the patient was induced with Etomidate 14 mg muscle relaxation was attained with Rocuronium 40 mg following which endotracheal intubation was performed with an endotracheal tube of size 7.5 mm and was checked for bilateral air entry and capnography to confirm the placement of Endotracheal tube. The patient was switched from bag ventilation to volume AC mode with the following ventilatory settings, which include a tidal volume of 500 ml, a respiratory rate of 14 breaths/min, and a positive end-expiratory pressure (PEEP) of 5 mmHg.

Temperature monitoring was done intraoperatively throughout the procedure and normothermia was maintained. Intraoperative vitals remained stable throughout the procedure.

Anaesthesia was maintained with Sevoflurane MAC of 1.5 %, a 50:50 mixture of nitrous oxide and oxygen, and rocuronium top-ups.

There were no intraoperative ST segment changes. The surgical procedure lasted two hours. Crystalloids formulated according to the Holliday-Segar formula were used for fluid control. For pain management, 15 mg/kg of intravenous paracetamol was provided. The total estimated blood loss was around 150 ml.

After the procedure, all volatile medications were stopped, and sugammadex was administered to reverse the neuromuscular blockade. The patient was extubated after his muscle strength and reflexes had fully recovered. Following surgery, postoperative care and pain management were given in PACU for 12 hours. The patient was stable and had no complaints following surgery. Pain management was done with intravenous paracetamol and ketorolac, and appropriate antibiotics started as needed. DVT prophylaxis was started with Enoxaparin 0.4 ml subcutaneously from the postoperative day (POD 1).

In the postoperative period, the patient was hemodynamically stable and there were no ECG changes, and he was shifted to the ward on postoperative day -2 and was discharged from the hospital on postoperative day 5. He was advised to do a further cardiologic evaluation after explaining to the patient about the disease severity.

4. Discussion

Brugada syndrome is characterised by sudden cardiac mortality or ventricular tachyarrhythmias, which occur even in persons who have an absence of structural heart abnormalities. The diagnosis is predominantly based on ECG findings, validated via genetic testing and clinical history. The findings of the case report are represented in Tables 1 and 2. The Table 3 enumerates the list of drugs that precipitates brugada syndrome, which we cautiously avoided using these drugs in our patients

Cuttone G. et al. conducted a systematic review on the pharmacological effects of anaesthetic drugs on patients with BrS, a condition associated with a high risk of sudden cardiac death and arrhythmias which highlighted the need for high-quality care regarding anaesthetic management for these patients, not only in specialized cardiological centres but also in emergency and elective surgical settings.[7]

Ciconte et al [3]. studied how anaesthetic drugs used for general anaesthesia (GA) affect the cardiac activity of patients with Brugada syndrome (BrS) who are at high risk of sudden cardiac death (SCD) and found that using a single bolus dose of propofol and inhalational anaesthetics in these patients may help by reducing the appearance of the type 1 BrS pattern (Table 2) on an ECG. In our study, etomidate used as an induction agent and didn't observe any Brugada syndrome ECG changes in both intraoperative and postoperative periods. Also, sugammadex is used to reverse neuromuscular blockade at the end of the procedure.

Local anaesthetics block sodium channels at varying rates. Several research studies have avoided using regional procedures with local anaesthetics in individuals with BrS due to the potentially increased risk of arrhythmia [4] (Table 3).

Although our patient could have had the procedure done under regional anaesthesia, we chose general anaesthesia to prevent the use of local anaesthesia, which causes Brugada syndrome. several studies have documented the safe and uncomplicated administration of local anaesthetics in individuals with BrS [5].

Table 1: Diagnostic characteristics for Brugada syndrome [6, 7]

A. Major criteria
1. Coved-type ST-segment elevation ≥2 mm in leads V1-V3, followed by negative T-waves (most diagnostic pattern).
2. A family history of cardiac arrest or an immediate family member identified as having Brugada syndrome.
3. History of unexplained hypotension or abrupt cardiac arrest (especially through sleep or at rest) without structural heart disease.
B. Minor criteria
1. Presence of Type 2 or Type 3 Brugada patterns, though these are less specific than Type 1(elevation of < 2 mm or saddleback pattern)
2. Right bundle branch block (RBBB) morphology seen in the ECG, which is often present in

Brugada syndrome.
3. Family history of arrhythmic events, such as sudden unexplained death or family members with unexplained syncope, but not necessarily diagnosed with Brugada syndrome.
4. Mutation in the SCN5A gene that encodes the sodium channel

Table 2: Electrocardiographic patterns of Brugada syndrome (BrS)7

Type-1 (Coved type)	ST-segment elevation of ≥ 2mm in the right precordial leads (V1-V3), typically with a coved (concave) shape.
Type-2 (Saddleback Type)	ST-segment elevation of ≥ 2mm in leads V1-V3, but the ST-segment is more saddle-shaped (it is convex instead of concave)
Type-3(Indeterminate or Minor type)	ST-segment elevation in leads V1-V3, but the elevation is either less than 2mm or may resemble a more subtle variation of Type 1 or Type 2.

Table 3: Drugs triggering Brugada syndrome

Anti-arrhythmic sodium channel blockers	**Anti-convulsant sodium channel blockers**	**Local anaesthetics**
Class 1A-Quinidine, -Procainamide, -Disopyramide	Phenytoin Carbamazepine	Lidocaine Bupivacaine
Class 1B-Lidocaine, -mexiletine	Ox-carbamazepine Lamotrigine	Ropivacaine Mepivacaine
Class 1C-Flecainide, -Propafenone	Lacosamide Rufinamide	Procaine Tetracaine

5. Conclusion

Identifying the Brugada syndrome with thorough history taking and ECG findings will help to meticulously plan the anaesthesia technique to avoid perioperative major cardiac events

6. Conflict of interest

The authors declare no conflict of interest.

7. Acknowledgement

We would like to thank SRM Medical college and Research Centre for their help and cordial support of our study.

8. Funding

This study did not receive any funding

References

[1] Hedley PL, Jørgensen P, Schlamowitz S, et al. The genetic basis of Brugada syndrome: A mutation update. Human Mutation. 2009; 30:1256–66. doi: 10.1002/humu.21066

[2] Pelliccia A, Fagard R, Bjørnstad HH, et al. Recommendations for competitive sports participation in athletes with cardiovascular disease: A consensus document from the Study Group of Sports Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology. European Heart Journal. 2005; 26:1422–45. doi: 10.1093/eurheartj/ehi325

[3] Cuttone G, Martucci G, Napoli R, et al. Anesthesiological management of Brugada syndrome patients: A systematic review. Saudi Journal of Anaesthesia. 2023; 17:394. doi: 10.4103/sja.sja_205_23

[4] Carey SM, Hocking G. Brugada syndrome--a review of the implications for the anaesthetist. Anaesth Intensive Care. 2011;

39:571–7. doi: 10.1177/0310057X1103900406

[5] Kloesel B, Ackerman MJ, Sprung J, et al. Anesthetic management of patients with Brugada syndrome: a case series and literature review. Can J Anaesth. 2011; 58:824–36. doi: 10.1007/s12630-011-9546-y

[6] Brugada P, Brugada J. Right bundle branch block, persistent ST segment elevation and sudden cardiac death: A distinct clinical and electrocardiographic syndrome: A multicenter report. Journal of the American College of Cardiology. 1992; 20:1391–6. doi: 10.1016/0735-1097(92)90253-J

[7] Antzelevitch C, Brugada P, Borggrefe M, et al. Brugada syndrome: report of the second consensus conference: endorsed by the Heart Rhythm Society and the European Heart Rhythm Association. Circulation. 2005; 111:659–70. doi: 10.1161/01.CIR.0000152479.54298.51

[8] de Bliek EC. ST elevation: Differential diagnosis and caveats. A comprehensive review to help distinguish ST elevation myocardial infarction from nonischemic etiologies of ST elevation. Turkish Journal of Emergency Medicine. 2018; 18:1–10. doi: 10.1016/j.tjem.2018.01.008

[9] Brugada Syndrome: Practice Essentials, Background, Pathophysiology. Published Online First: 29 October 2024.

[10] BrugadaDrugs.org | Safe drug use and the Brugada syndrome. https://www.brugadadrugs.org/ (accessed 20 February 2025)