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ANESTHETIC MANAGEMENT IN A PATIENT WITH TYPE 1 BRUGADA SYNDROME UNDERGOING INCISION AND DRAINAGE OF A NECK ABSCESS: A CASE REPORT

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Abstract

Background: Brugada Syndrome is a rare inherited arrhythmia characterized by distinct ECG patterns and a risk of sudden cardiac death from ventricular arrhythmias. Anesthetic management is challenging due to the potential for perioperative triggers to precipitate malignant arrhythmias.

Case Presentation: A 27-year-old male with an anterior neck abscess was scheduled for incision and drainage under general anesthesia. Routine ECG revealed a Type 1 Brugada pattern, confirmed on repeated recordings and by cardiology consultation. A supraglottic airway (i-gel) was used to minimize intubation-related sympathetic stimulation. Anesthesia was maintained with spontaneous ventilation on a low-flow 50% oxygen–50% air mixture, avoiding volatile anesthetics and muscle relaxants. The procedure was uneventful except for a brief self-resolving episode of bradycardia. Postoperative cardiac monitoring in the ICU revealed no arrhythmias.

Conclusion: This case highlights the importance of meticulous preoperative planning, arrhythmia trigger avoidance, and vigilant perioperative monitoring in Brugada Syndrome patients. Supraglottic airway use can be a safe alternative to endotracheal intubation in select cases.

Keywords: Brugada Syndrome, anesthesia, supraglottic airway, arrhythmia, case report.

INTRODUCTION

Brugada Syndrome (BrS) is an inherited arrhythmogenic disorder caused primarily by sodium channel mutations, particularly in the SCN5A gene, and is associated with a high risk of sudden cardiac death due to polymorphic ventricular tachycardia or ventricular fibrillation [1,2]. First described in 1992 by Brugada et al., it is diagnosed based on characteristic ECG findings—particularly coved ST-segment elevation in leads V1–V3—either spontaneously or after sodium channel blocker challenge [3,4].

The prevalence of BrS is estimated at 0.05–0.2% globally, with higher rates reported in Southeast Asia [5]. Many patients are asymptomatic at diagnosis, but triggers such as fever, electrolyte disturbances, vagal stimulation, and certain medications can precipitate life-threatening arrhythmias [6,7].

Anesthesia in BrS poses specific challenges due to potential arrhythmogenic effects of anesthetic agents, perioperative autonomic fluctuations, and airway manipulation [8,9]. We present a case of a young male with

Type 1 Brugada Syndrome undergoing incision and drainage of a neck abscess under general anesthesia with a supraglottic airway to avoid intubation-related sympathetic stimulation.

Case Presentation

Patient Profile

A 27-year-old male presented with a pus-point abscess measuring 3 × 4 cm in the anterior neck, scheduled for elective incision and drainage.

Preoperative assessment:

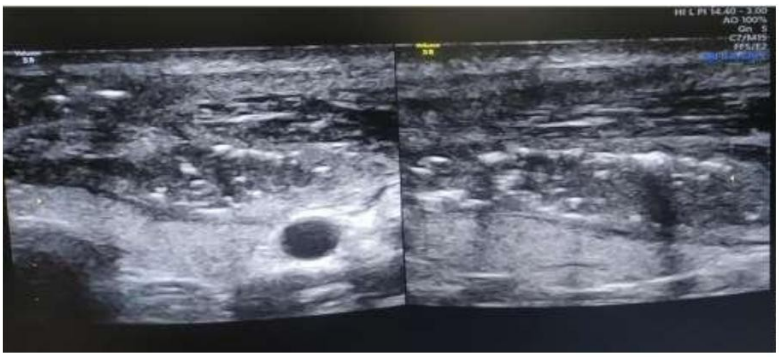
- Vitals: HR – 68 bpm, BP – 130/80 mmHg, SpO_2 – 98% (room air)
- Airway: Mallampati grade II, normal neck mobility, adequate extension, poor oral hygiene
- Respiratory system: Bilateral equal air entry, no added sounds
- Cardiovascular system: S1 and S2 present, no murmurs

Investigations:

- Hemogram: Hb – 14.6 g/dL; WBC count and platelets within normal range
- LFT, RFT: Within normal limits
- Cardiac markers: Troponin I and CK-MB – Normal
- ECG: Normal sinus rhythm, pseudo-RBBB in V1, coved ST elevation in V2—consistent with Type 1 Brugada pattern
- Echocardiography: Normal LV systolic function, EF – 60%, no regional wall motion abnormality

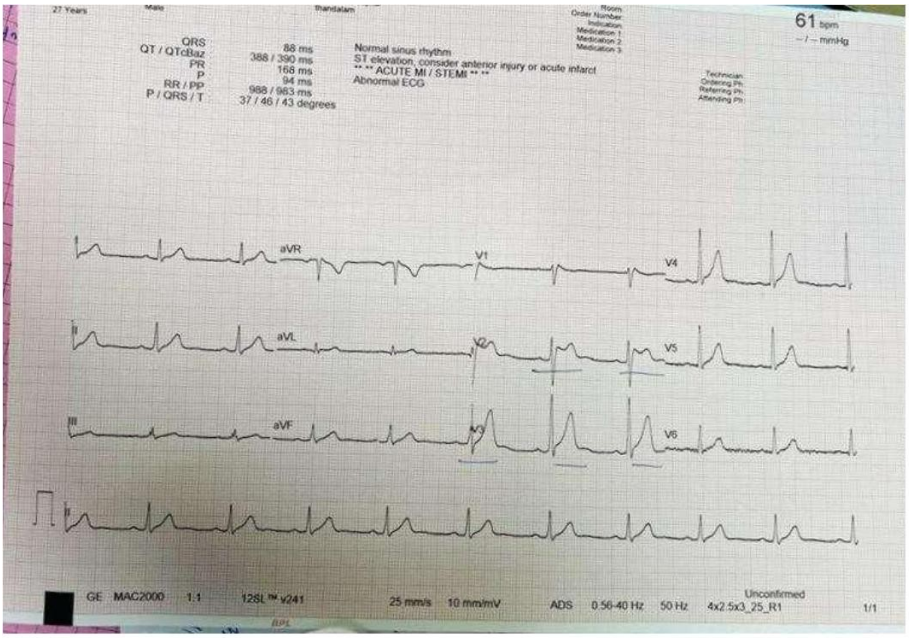
Cardiology consultation confirmed Type 1 Brugada Syndrome. The patient had no history of syncope, palpitations, or family history of sudden cardiac death.

Neck abscess usg Image



ECG with ST elevation

Brugada syndrome type 1



Anesthetic Plan

The primary goals were to avoid arrhythmogenic medications, maintain normothermia and electrolyte balance, minimize sympathetic and vagal triggers, and ensure immediate availability of defibrillation and pacing equipment. A supraglottic airway was planned to avoid laryngoscopy-induced sympathetic surges.

Intraoperative Course

Following adequate NPO status and application of standard ASA monitors, the patient was preoxygenated with 100% oxygen. Intravenous fentanyl 100 mcg was given to provide analgesia and blunt the sympathetic response, followed by induction with intravenous propofol 130 mg. Muscle relaxants were deliberately avoided to maintain spontaneous ventilation and minimize the risk of bradyarrhythmia.

Airway management was achieved with the insertion of an i-gel size 3 supraglottic airway device, chosen to avoid the arrhythmia risk associated with endotracheal intubation. Correct placement was confirmed by equal bilateral air entry on 5-point auscultation and an appropriate capnographic waveform. After securing the airway, the patient was maintained on spontaneous ventilation using a 50% oxygen–50% air mixture delivered through a low-flow anesthesia circuit. No volatile anesthetics were used during the procedure to reduce arrhythmogenic potential.

All sizes of supraglottic airway devices, endotracheal tubes, a defibrillator, and antiarrhythmic drugs were readily available. Intraoperatively, the patient remained stable except for a single transient bradycardia episode, with HR dropping to 47 bpm, which spontaneously returned to 65 bpm within a few seconds without intervention.

Fluid management consisted of 300 mL crystalloids, and estimated blood loss was less than 20 mL. No urinary catheterization was performed. The surgical procedure lasted approximately 20 minutes. At the conclusion, after ensuring the patient was awake, breathing adequately, and hemodynamically stable, the i-gel was removed smoothly, and the patient was transferred to the ICU for postoperative cardiac monitoring.

Postoperative Course

Continuous ECG monitoring in the ICU for 24 hours showed no arrhythmias or hemodynamic instability. The patient remained stable and was discharged after an uneventful recovery.

DISCUSSION

Brugada Syndrome (BrS) is an inherited arrhythmogenic disorder primarily caused by loss-of-function mutations in the SCN5A gene, which encodes the α-subunit of the cardiac sodium channel. This defect impairs sodium influx during phase 0 of the cardiac action potential, resulting in heterogeneous depolarization and repolarization across the right ventricular epicardium. The consequent transmural dispersion of repolarization predisposes affected individuals to phase 2 re-entry and malignant ventricular arrhythmias, including ventricular fibrillation and polymorphic ventricular tachycardia, which can lead to sudden cardiac death [10,11].

The diagnosis is based on characteristic electrocardiographic patterns in the right precordial leads. Among the three described ECG types, the Type 1 pattern—a coved ST-segment elevation ≥2 mm followed by a negative T wave in leads V1–V3—is considered diagnostic and pathognomonic for BrS [12]. The Type 2 (saddleback ST elevation with ≥2 mm elevation) and Type 3 (either coved or saddleback with <2 mm elevation) patterns are not diagnostic but may warrant further evaluation with provocative drug testing.

Perioperative management of BrS requires meticulous attention to the avoidance of factors known to exacerbate the arrhythmogenic substrate. These include electrolyte disturbances such as hyperkalemia, hypokalemia, and hypercalcemia; hypothermia; increased vagal tone; bradycardia; and specific pharmacologic agents known to inhibit sodium channels [13]. Commonly avoided drugs in BrS include certain volatile anesthetics (particularly sevoflurane and halothane), ketamine, high-dose propofol infusions, bupivacaine, and agents with strong vagotonic or sodium-channel-blocking properties [14].

In the present case, induction was achieved with propofol in a single bolus dose and fentanyl, both of which have been reported to be used safely in BrS when administered judiciously in the absence of prolonged infusion. Volatile anesthetics, ketamine, and bupivacaine were deliberately avoided to minimize the risk of unmasking ECG changes or provoking arrhythmias. Furthermore, the use of a supraglottic airway (SGA) rather than direct laryngoscopy with endotracheal intubation significantly reduced sympathetic stimulation during airway management. Sympathetic surges are recognized triggers for arrhythmias in BrS, and avoiding such stimulation is considered a key preventive strategy [15].

A notable intraoperative event was a brief episode of bradycardia (heart rate decreased to 47 bpm) that resolved spontaneously without intervention. Transient bradycardia is not uncommon in BrS, as increased vagal tone—whether from surgical manipulation, anesthetic depth, or airway instrumentation—can accentuate the ST-segment elevation and potentially precipitate malignant arrhythmias [16]. In this case, the maintenance of spontaneous ventilation, avoidance of high-dose vagotonic drugs, and a gentle emergence likely contributed to the rapid spontaneous resolution of the event without hemodynamic compromise. Continuous vigilance, immediate availability of resuscitation equipment, and a pre-formulated crisis management plan were integral to ensuring a safe outcome.

CONCLUSION

With careful drug selection, avoidance of known triggers, and vigilant intraoperative and postoperative monitoring, anesthesia can be administered safely in patients with Brugada Syndrome. Supraglottic airways are a viable alternative to endotracheal intubation in suitable cases, particularly when minimizing sympathetic stimulation is a priority.

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