ARCHIVES OF THE TURKISH SOCIETY OF CARDIOLOGY

Official journal of the TURKISH SOCIETY OF CARDIOLOGY

A Rare Manifestation of Brugada ECG Pattern Precipitated by General Anesthesia for Pituitary Surgery

Hipofiz Cerrahisi için Genel Anestezi ile Tetiklenen Brugada EKG Paternin Nadir Tezahürü

ABSTRACT

Brugada Syndrome Type 1 is an arrhythmogenic disorder triggered by various etiologies, including febrile illness, pregnancy, and certain medications. This paper describes the electrocardiographic (ECG) manifestation of the Brugada pattern in a patient who developed ventricular arrhythmia after undergoing general anesthesia for pituitary surgery.

Keywords: Anesthetic management, Brugada Syndrome, pituitary surgery

ÖZET

Brugada tip 1, ateşli hastalıklar, gebelik ve bazı ilaçlar gibi farklı etiyolojiler tarafından tetiklenen aritmojenik bir bozukluktur. Brugada paterninin elektrokardiyografik (EKG) yansıması, hipofiz cerrahisi için genel anestezi sonrası ventriküler aritmi gelişen bir hastada tanımlanmıştır.

Anahtar Kelimeler: Anestezi yönetimi, Brugada sendromu, hipofiz cerrahisi

CASE REPORT
OLGU SUNUMU

The Brugada Type 1 Electrocardiogram (ECG) pattern is crucial for diagnosing Brugada Syndrome (BrS), which can result in sudden cardiac arrest due to ventricular arrhythmias.[1] Brugada Syndrome Type 1 is an inherited arrhythmogenic disease characterized by a specific ECG pattern, which includes coved-type ST elevation of ≥ 2 mm in one or more leads from V1 to V3.[1,2] Clinical manifestations range from an asymptomatic presentation to sudden cardiac death.[2] The most common and significant laboratory marker for diagnosing BrS is abnormal findings on an ECG.[1] Typical ECG features of BrS can fluctuate.[2] The condition is known to be precipitated by various triggers, including febrile illness, pregnancy, and certain medications.[1,2] This case report describes an instance of ventricular fibrillation (VF) in a patient undergoing general anesthesia for pituitary surgery, where the ECG findings demonstrated the Brugada pattern.

Case Report

A 35-year-old male patient was scheduled for transsphenoidal surgery for a pituitary macroadenoma, classified as American Society of Anesthesiologists Physical Status Class I (ASA I). No other medical problems were found in his history and systemic evaluation. Upon further questioning, the patient reported no record of previous surgical procedures, cardiac arrhythmias, or syncope. The preoperative ECG showed a rate of 91 beats/min with a normal sinus rhythm (Figure 1). Although he had no history of drug use, he had quit smoking approximately six months prior. His family history included the sudden death of his 42-year-old father, but details were not available.

Transsphenoidal surgery was performed under general anesthesia. Anesthesia induction and maintenance were achieved with 100 mg of fentanyl, 200 mg of propofol, 40 mg of rocuronium, and placement of a laryngeal mask. Anesthesia was maintained with sevoflurane, and analgesia was maintained with a remifentanil infusion. At the end of the procedure, 4 mg of ondansetron, 200 mg of sugammadex (1x1), and 100 mg of tramadol were administered intravenously. The total operative and anesthesia times were 150 and 175 minutes, respectively. While the patient was being admitted

Aslan Erdoğan
Duygu İnan
Ufuk Yıldız
Taylan Akgün

Department of Cardiology, Başakşehir Çam and Sakura City Hospital, İstanbul, Türkiye

Corresponding author:
Duygu İnan
✉ dr.duyguinan@gmail.com

Received: August 05, 2023
Accepted: October 17, 2023

Cite this article as: Erdoğan A, İnan D, Yıldız U, Akgün T. A rare manifestation of brugada ecg pattern precipitated by general anesthesia for pituitary surgery. *Turk Kardiyol Dern Ars.* 2024;52(5):362-364.

DOI:10.5543/tkda.2023.09145

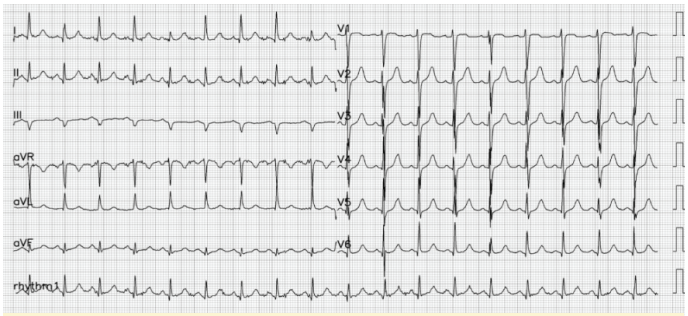

Figure 1. Preoperative ECG of the patient. Heart rate was 85 beats/min with sinus rhythm.

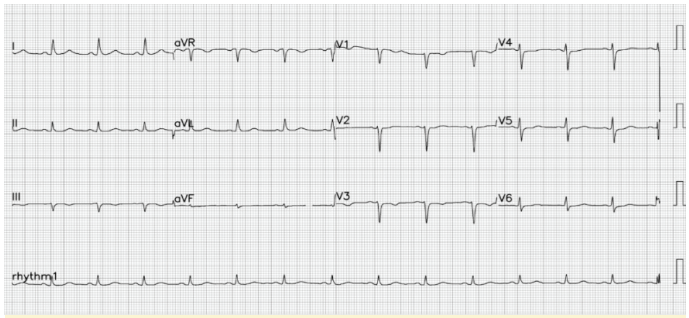

Figure 3. Postoperative 12th-hour ECG of the patient. The Type 1 Brugada pattern disappeared.

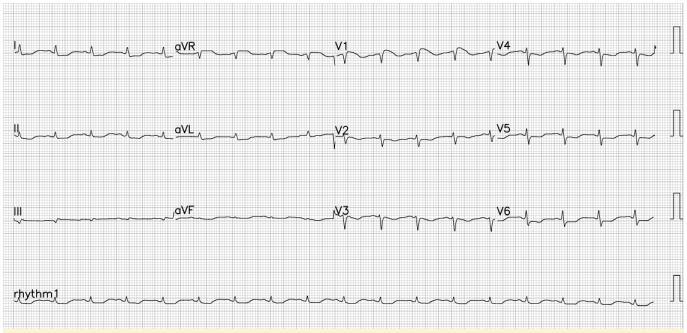

Figure 2. Suspected Type 1 Brugada pattern on ECG (coved-type ST elevation ≥ 2 mm in V1) observed during general anesthesia.

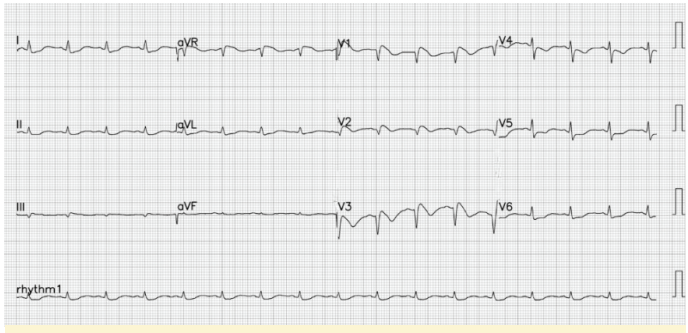

Figure 4. Type 1 Brugada pattern on ECG (coved-type ST elevation ≥ 2 mm from V1 to V3) observed during the Ajmaline test.

to the post-anesthesia care unit for postoperative care, VF developed immediately, and defibrillation was performed repeatedly. Ventricular fibrillation was not resolved by medical therapy and defibrillation but was controlled by overdrive pacing. An indeterminate type 1 Brugada pattern was detected on the ECG taken after defibrillation (Figure 2). Coronary angiography showed no significant coronary stenosis. The type 1 Brugada pattern disappeared on the ECG taken 12 hours after VF (Figure 3). Transthoracic echocardiography revealed a normal ejection fraction and no structurally abnormal pathology. There was no elevation in cardiac enzymes, and no electrolyte abnormalities were detected. The patient underwent the ajmaline test after 96 hours, which revealed a typical type 1 Brugada pattern (Figure 4). The patient was advised to consider implantable cardioverter-defibrillator (ICD) implantation, but he refused. No recurrent cardiac events were observed in the patient, who was followed up for approximately one year.

Discussion

The prevalence of BrS is approximately 1 in 5,000 to 1 in 2,000.[3] While the incidence of the BrS pattern on ECG varies between 0.12% and 0.8%, malignant arrhythmias can be triggered by various conditions (most frequently fever) and medications.[2,3] This case reports on general anesthesia, possibly related to propofol, precipitating a Type 1 ECG pattern in Brugada Syndrome.

The definitive diagnosis of BrS is established by the presence of a Type 1 ECG pattern, either spontaneously or after provocation with a sodium channel blocker, and the presence of one of the following additional criteria: documented VF, spontaneously terminating polymorphic ventricular tachycardia (VT), presence of VT on electrophysiological study, family history of sudden cardiac death under 45 years of age, presence of a Type 1 ECG pattern in a family member, history of unexplained syncope attributed to tachyarrhythmia, or nocturnal agonal respiration.[1,3] The Type 1 Brugada ECG pattern, VF, and family history were diagnostic in this case.

The ECG alterations observed in BrS are based on the theory of depolarization, repolarization, and current-charge mismatch resulting from different molecular mechanisms.[2] In anesthesia, medications that interact with cardiac ion channels and produce changes in depolarization and repolarization are routinely administered. The mechanism by which propofol triggers typical ECG changes and malignant arrhythmias in BrS patients relies on the induction of sodium flow blockade in cardiac myocytes.[4] There are also conflicting articles regarding the use of propofol in patients with BrS.[5] The majority of papers suggest that propofol is associated with malignant arrhythmias in patients with overt BrS or unmasked BrS-like ECG patterns, primarily related to prolonged infusion of propofol.[5,6] Conversely, the use of propofol

ABBREVIATIONS

ASA	American Society of Anesthesiologists
BrS	Brugada Syndrome
ECG	Electrocardiography
ICD	Implantable Cardioverter-Defibrillator
VF	Ventricular Fibrillation
VT	Ventricular Tachycardia

for the induction of anesthesia does not appear to be associated with the emergence of BrS-like ECG patterns, the progression of pre-existing BrS ECG patterns, or the triggering of malignant arrhythmias. However, the level of evidence in the guidelines for the use of propofol in BrS is IIb.[6] It has also been reported that tramadol triggers BrS-related arrhythmias by blocking sodium channels at high doses.[7] In this case, the administration of a low dose of tramadol, the lack of arrhythmic potential of other anesthetic agents, and the propofol-induced BrS and malignant arrhythmia were primarily revealed. The most important point in the preoperative evaluation is to suspect BrS. Since it is a rare syndrome, there are no definitive recommendations for assessing anesthesia risk in Brugada Syndrome patients. In this case, no obvious pattern was observed in the preoperative ECG. A family history of early sudden death may warrant further cardiac evaluation. Sometimes, simple methods such as detailed anamnesis and ECG recordings from the upper ribs may be diagnostic. However, the necessity of provocation testing for BrS in asymptomatic individuals without structural heart disease, but with a family history of sudden death, remains the subject of debate.

The emergency treatment of VF in patients with BrS primarily involves defibrillation and medical management.[8] Overdrive pacing should also be considered for the acute treatment of patients with VF resistant to medical therapy and defibrillation.[8]

Autonomic nervous system activity influences VF and ST elevation in BrS patients.[1] Increased vagal activity is known to trigger VF episodes in BrS, which usually occur at night due to heightened vagal stimulation. This leads to a shortened action potential duration and increased excitability.[1,2] In contrast, beta-adrenergic stimulation through the sympathetic nervous system prevents VF by stabilizing changes in membrane potential.[1,2,9] Isoproterenol has been shown to suppress persistent ventricular arrhythmias in some BrS patients.[9] Additionally, quinidine has been reported to suppress ventricular arrhythmias in these patients by prolonging ventricular refractoriness and restoring electrical homogeneity.[2,3]

BrS is a genetically heterogeneous channelopathy, with disease-causing variants identified in approximately 16 genes.[1-3] It has been reported to be caused by loss-of-function mutations in one allele of the Sodium Channel, Voltage-Gated, Type V, Alpha Subunit (SCN5A) gene in 20-25% of all cases.[2,3] In instances of diagnostic certainty, SCN5A gene testing is recommended to guide family screening cascades.[2,10] SCN5A variant-positive family members should undergo evaluation by a cardiologist shortly after screening.[1,10] During this visit, standard and Brugada ECG recordings should be obtained. It is also advisable to record ECGs during fever and, if no abnormalities are detected, to repeat standard and Brugada ECG recordings every three years.[2,10] Caution is advised with the use of certain QT-prolonging drugs.[6] If the use of these treatments is essential, an ECG recording should be considered to evaluate their effects.[3,6] A baseline echocardiographic evaluation is recommended due to the possible association of BrS with cardiomyopathy.[6,10] 24-hour Holter monitoring is indicated to detect sinus node dysfunction and ventricular arrhythmias, and an exercise test may be considered for these patients.[2,3,10]

Conclusion

This case report highlights the increasing phenomenon of the Brugada ECG pattern being triggered by anesthetic drugs in a subject without any clinical or ECG evidence of BrS. The diagnosis and perioperative management of patients with BrS remain challenges. Further efforts and an in-depth understanding of the physiology of this syndrome are required to devise an ideal anesthesia plan. It is crucial for these patients to have a backup plan for every situation, including emergencies with limited time for pre-assessment.

Informed Consent: Written informed consent was obtained from the patient's first-degree relatives for the publication of this case report and the accompanying images.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – A.E., T.A.; Design – D.İ.; Supervision – T.A.; Resource – U.Y.; Materials – A.E., D.İ.; Data Collection and/or Processing – A.E., D.İ.; Analysis and/or Interpretation – D.İ., U.Y.; Literature Review – A.E.; Writing – A.E., D.İ.; Critical Review – T.A.

Usage of AI for Writing Assistance: Authors declare that AI-assisted technologies were not used in this article.

Conflict of Interests: The authors declare no conflicts of interest.

Funding: The authors declare that no funding was received for this study.

References

1. 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(5):659-670. Erratum in: *Circulation*. 2005;112(4):e74. [CrossRef]
2. Tse G, Liu T, Li KH, et al. Electrophysiological Mechanisms of Brugada Syndrome: Insights from Pre-clinical and Clinical Studies. *Front Physiol*. 2016;7:467. [CrossRef]
3. Brugada J, Campuzano O, Arbelo E, Sarquella-Brugada G, Brugada R. Present Status of Brugada Syndrome: JACC State-of-the-Art Review. *J Am Coll Cardiol*. 2018;72(9):1046-1059. [CrossRef]
4. Saint DA, Tang Y. Propofol block of cardiac sodium currents in rat isolated myocardial cells is increased at depolarized resting potentials. *Clin Exp Pharmacol Physiol*. 1998;25(5):336-340. [CrossRef]
5. Ciconte G, Santinelli V, Brugada J, et al. General Anesthesia Attenuates Brugada Syndrome Phenotype Expression: Clinical Implications From a Prospective Clinical Trial. *JACC Clin Electrophysiol*. 2018;4(4):518-530. [CrossRef]
6. Postema PG, Wolpert C, Amin AS, et al. Drugs and Brugada syndrome patients: review of the literature, recommendations, and an up-to-date website (www.brugadadrugs.org). *Heart Rhythm*. 2009;6(9):1335-1341. [CrossRef]
7. Cole JB, Sattiraju S, Bilden EF, Asinger RW, Bertog SC. Isolated tramadol overdose associated with Brugada ECG pattern. *Pacing Clin Electrophysiol*. 2012;35(8):e219-e221. [CrossRef]
8. Yoshida T, Naito Y, Nishimura K. Temporary ventricular overdrive pacing for electrical storm after coronary artery bypass grafting. *General Thoracic and Cardiovascular Surgery.* 2011;59:756-759. [CrossRef]
9. Watanabe A, Fukushima Kusano K, et al. Low-dose isoproterenol for repetitive ventricular arrhythmia in patients with Brugada syndrome. *Eur Heart J*. 2006;27(13):1579-1583. [CrossRef]
10. Zeppenfeld K, Tfelt-Hansen J, de Riva M, et al.; ESC Scientific Document Group. 2022 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death. *Eur Heart J*. 2022;43(40):3997-4126. [CrossRef]