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A 32-Year-Old Man Diagnosed with Type II Brugada Syndrome on Preoperative Electrocardiogram 1 Week Before Elective Tympanoplasty

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Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Conflict of interest: None declared

Patient: Male, 32-year-old
Final Diagnosis: Brugada syndrome
Symptoms: No specific symptoms
Medication: —
Clinical Procedure: —
Specialty: Anesthesiology • Cardiology

Objective: Rare disease

Background: Brugada syndrome is a potentially fatal cardiac arrhythmia characterized by incomplete right bundle-branch block (RBB) and characteristic ST-segment elevation in the anterior electrocardiogram (ECG) leads. This report is of a case of type 2 Brugada syndrome, and discusses the importance of preoperative history and ECG evaluation.

Case Report: A 32-year-old man was scheduled for tympanoplasty. His preoperative ECG revealed saddleback-type J waves in V_2 (>2 mm) and ST increase (>1 mm) detected 1 week before elective surgery, but the ECG 1 year before showed normal. He had no notable past history.

Anesthesia was induced with remifentanil and propofol, and maintained with sevoflurane in combination with remifentanil. Routine monitoring of vital signs was supplemented with V_2 monitoring on the ECG. The heart rate was maintained at above 60 beats/min using ephedrine. The course of the operation was uneventful.

Conclusions: We managed anesthesia for a patient with a type 2 Brugada syndrome ECG without events, probably because he had no notable past history such as syncope. Type 2 and type 3 Brugada syndrome ECGs are difficult to recognize, and patients with them are considered to be less risky than a patient with a type I ECG. However, as Brugada syndrome ECG is dynamic and changeable, a type 2 or 3 Brugada syndrome ECG can change to a type I ECG under some conditions, and thus should not be overlooked, and the patient's past history or symptoms, such as syncope, should be carefully investigated.

Keywords: Brugada Syndrome • Syncope • Anesthesia • Ventricular Fibrillation

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Background

Brugada syndrome, characterized by refractory ventricular fibrillation (VF) and sudden death, was reported in Brugada and Brugada in 1991 [1]. Brugada syndrome is classified into 3 types according to the electrocardiographic patterns: type 1 (coved type), type 2 (saddle-back type with ST increase ≥1 mm), and type 3 (saddle-back type with ST increase <1 mm) [2]. In the latest criteria, observation of a coved-type electrocardiogram (ECG) is necessary, either spontaneously or after intravenous administration of a sodium channel-blocking agent, for a diagnosis of Brugada syndrome: (1) BrS is diagnosed in patients with ST-segment elevation with type I morphology ≥2 mm in ≥1 lead among the right precordial leads V1 and V2 positioned in the 2nd, 3rd, or 4th intercostal space occurring either spontaneously or after provocative drug test with intravenous administration of class I antiarrhythmic drugs. (2) BrS is diagnosed in patients with type 2 or type 3 ST-segment elevation in ≥1 lead among the right precordial leads V1 and V2 positioned in the 2nd, 3rd, or 4th intercostal space when a provocative drug test with intravenous administration of class I antiarrhythmic drugs induces a type I ECG morphology [3]. However, the coved-type ECG does not persist and can change to the saddle-back type, which is more frequently detected in clinical settings [4], or a normal ECG. Brugada syndrome patients with a history of cardiac arrest, sustained ventricular tachycardia, or syncope caused by ventricular arrhythmias are at high risk and qualify for implantable cardioverter defibrillator (ICD), but asymptomatic Brugada syndrome patients do not [3].

We previously reported the case of a patient with type 2 Brugada syndrome ECG, who had not been diagnosed with Brugada syndrome but had a history of syncope, who underwent cardiac arrest during general anesthesia [5]. The present case report is of a case of type 2 Brugada syndrome, detected 1 week before elective surgery, and discusses the importance of preoperative patient ECG evaluation and the need for careful preoperative history-taking, including any history of syncope, in addition to a literature review [6-10].

Case Report

A 32-year-old man (172 cm, 59 kg) was scheduled for tympanoplasty. He underwent mastoidectomy 1 year before. The ECG taken at that time was normal (**Figure 1A**). The preoperative ECG at this time revealed saddle-back-type J waves in V_2 (>2 mm) and ST increase (> 1 mm), which is a type 2 Brugada syndrome ECG (**Figure 1B**). He had no notable past history including syncope, or family history of fatal arrhythmias or sudden death. He had never been diagnosed with Brugada syndrome. No abnormalities were found in physiological examinations and laboratory analyses. His American Society of Anesthesiologists (ASA) physical status was 1 [11].

Anesthesia was induced with 100 mg of propofol and 0.3 $\mu g^{-1} \cdot kg^{-1} \cdot min$ of remifentanil, and the trachea was intubated with a cuffed tube with the aid of 50 mg of rocuronium. Anesthesia was maintained with sevoflurane, remifentanil, and intermittent administration of rocuronium. In addition to routine monitoring (ECG: Lead II), V2 on the ECG and invasive radial artery pressure were supplemented. Defibrillation pads were placed over the praecordium and on the back in the left infrascapular region. The heart rate (HR) was maintained at above 60 beats/min using ephedrine. His body temperature was kept at 36.5-37.5°C. No adverse events happened during the surgery.

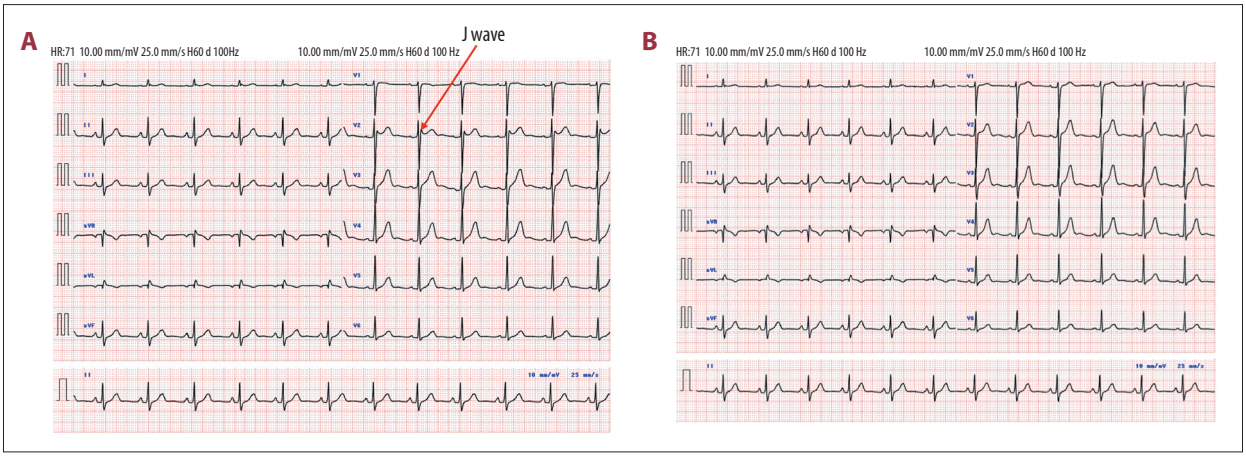

Figure 1. (**A**) Twelve-lead electrocardiogram (ECG) of the patient taken approximately 1 year before the operation. The ECG is normal: There are no J waves in V_2. (**B**) Twelve-lead electrocardiogram (ECG) of the patient taken 1 week before the operation. The ECG changes clearly from the ECG 1 year ago. The arrow shows the saddle-back-type J waves in V_2 (>2 mm) and ST increase (>1 mm), indicating that the patient has a type II Brugada syndrome ECG.

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Discussion

Patients with a Brugada syndrome ECG who were not diagnosed preoperatively are sometimes encountered by anesthesiologists, because the prevalence of type I Brugada syndrome ECG patterns is approximately 0.15% in Asia and less than 0.02% in Western countries [12], and that of all types of Brugada syndrome in Japan is 0.7% [13]. The Brugada syndrome ECG is dynamic and changeable, and the characteristics of a type I Brugada syndrome ECG are often concealed, resembling a normal, type 2, or type 3 ECG. Therefore, a type I Brugada syndrome ECG can sometimes be only be confirmed by challenge with sodium channel-blocking drugs such as flecainide, or pilsicainide, or procainamide [2]. However, it is impractical to perform the drug challenge test on all patients with a type 2 or type 3 ECG. Furthermore, a type 2 or type 3 Brugada syndrome ECG is difficult to recognize, as demonstrated in the previous case [5], because it may be considered an early repolarization pattern. Bernardo and Tiyyagura reported a syncopal case of type I or type II Brugada syndrome phenocopy, which was revealed by high fever in a patient with a normal baseline ECG [14]. In the present case, the 12-lead ECG recorded 1 week before the preoperative anesthesia consultation revealed type 2 Brugada syndrome, but the ECG 1 year before was normal. We previously reported a case of VF during surgery in a patient with a type 2 Brugada syndrome ECG. The patient's preoperative 12-lead ECG revealed saddle-back type J waves in V_2, but there were no saddle-back-type changes 1 year before the event or 2 days after the event, similar to the present case. However, the previous patient had a history of short-term syncope occurring 3 times, but we neglected this before anesthesia because the duration was short and it occurred after bathing [5]. On the other hand, the present patient had no history of symptoms such as syncope. The FINGER (France, Italy, Netherlands, Germany) registry, which included 1029 individuals with a Brugada syndrome ECG, demonstrated that symptoms and a spontaneous type I Brugada syndrome ECG are predictors of arrhythmic events, whereas sex and a familial history of sudden cardiac death are not [15]. However, as Brugada syndrome ECGs are dynamic and changeable, a type I Brugada syndrome ECG cannot always be detected. Kamakura et al reported that the long-term prognosis in the non-type I Brugada syndrome group was similar to that in the type I Brugada syndrome group, and the long-term prognosis of asymptomatic Brugada syndrome patients was better than that of the symptomatic patients [16]. Thus, when we encounter a patient with a Brugada syndrome ECG preoperatively, we should carefully check a past history or symptoms of the patient. The ASA physical status of the present patient was 1 according to the ASA physical status classification system [11]. If the patient had had some symptoms, such as syncope, his ASA physical status would have become 2. Thus, the ASA physical status classification system could be helpful in predicting anesthesia risk even in a patient with a Brugada syndrome ECG.

Anesthesiologists sometimes have to manage anesthesia for a patient with Brugada syndrome who has an implantable cardioverter-defibrillator (ICD) [17]. As the ICD should be off during surgery to avoid influence on an electric scalpel, knowledge of methods to prevent fatal arrhythmias during surgery is needed. There are many factors and drugs that can cause Brugada-like ST-segment increases, such as high body temperature, bradycardia, sodium channel blockers, and antidepressants [6,10]. Indeed, ventricular fibrillation was induced by severe bradycardia in our previous case [5]. Therefore, we controlled the HR at above 60 beats/min with ephedrine in the present case. Furthermore, isoproterenol (which increases the L-type calcium current) proves to be effective for the treatment of electrical storms and quinidine (a class Ia antiarrhythmic drug with transient outward potassium current and rapid delayed rectifier current blocker effects) has been shown to prevent the induction of lethal arrhythmias in Brugada syndrome patients [3]. As for anesthetics and anesthesia-related drugs, volatile anesthetics, opioids, benzodiazepines, and non-depolarizing neuromuscular muscle relaxants are safely used for patients with Brugada syndrome [6,7,9,10,17]. Local anesthetics used are sodium channel blockers; those with rapid dissociation properties such as lidocaine appear to be safe, but those with slow dissociation properties such as bupivacaine, ropivacaine, and levobupivacaine are still controversial for use in patients with Brugada syndrome [6,7,10,17]. Propofol can induce Brugada-like ST increases and the relationship of the ECG changes and propofol infusion syndrome (PRIS) was suspected [10,17-20]. Its use in patients with Brugada syndrome was previously advised against [17,18]; however, a recent study demonstrated that propofol has no specific risks in patients with Brugada syndrome [9,20]. We used propofol for anesthesia induction in the present case and the previous cases [5] with no adverse events.

Conclusions

We managed anesthesia for a patient with a type II Brugada syndrome ECG without adverse events, probably because he had no notable past history such as syncope. Type II and type III Brugada syndrome ECGs are difficult to recognize, and patients with them are considered to be less risky than patients with a type I ECG. However, as Brugada syndrome ECGs are dynamic and changeable, a type II or III Brugada syndrome ECG can change to a type I ECG under some conditions and thus should not be overlooked, and the patient's past history, such as syncope, should be carefully investigated.

Conflict of Interest

None declared.

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