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CORRESPONDENCE

Perioperative isoproterenol infusion in a patient with Brugada syndrome

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To the Editor,

We report the novel use of perioperative isoproterenol in a 38-yr-old 65-kg man undergoing emergency laparoscopy for an abdominal stab wound. He was suspected to have Brugada syndrome (BrS) after his preoperative electrocardiogram (ECG) demonstrated pathognomonic characteristics, including J elevation $\geq$ 0.2 mV and a coved-type ST segment followed by a negative T wave in leads V2 and V3 (Figure A). He reported no other clinical features of BrS (i.e., unexplained syncope, nocturnal agonal respiration, family history of sudden cardiac death at $<$ 45 yr), nor did he have any known history of ventricular fibrillation or polymorphic ventricular tachycardia.[1] His laboratory investigations and chest radiography were otherwise normal.

On arrival to the operating room, he was alert and oriented. Standard monitors[2] revealed a pulse rate of 100 beats·min^{-1}, blood pressure 100/62 mmHg, room air pulse oximetry 100%, and body temperature 36°C. A defibrillator was readily available if a malignant arrhythmia occurred.

He was induced with midazolam 2.5 mg, fentanyl 150 μg, propofol 120 mg, and rocuronium 50 mg. After securing his airway, and with an abundance of caution (considering that the CO_2 pneumoperitoneum might enhance his arrythmogenicity), we prophylactically initiated a continuous isoproterenol[5] infusion 0.004 to 0.005 μg·kg^{-1}·min^{-1} General anesthesia was maintained with sevoflurane, with additional rocuronium and fentanyl as needed. A liver laceration was subsequently repaired, after which the neuromuscular blocking agent was reversed by neostigmine 2.5 mg and glycopyrrolate 0.5 mg, and he was uneventfully extubated. The estimated blood loss was 350 mL, and total fluid (crystalloid) given was 660 mL. His heart rate throughout the procedure was 60-100 beats·min^{-1} without any atrial or ventricular arrhythmia.

An ECG obtained in the postanesthesia care unit (while the isoproterenol was still infusing) showed significantly reduced ST elevation (Figure B). He was subsequently transferred to the intensive care unit for continued postoperative monitoring. The isoproterenol was discontinued seven hours later, after normalization of the ST segment (Figure C). Thirty-one hours after discontinuation, typical J point and coved ST segment elevation reappeared on lead V2 (Figure D). One month later, the patient underwent an electrophysiological study (EPS) during which sustained ventricular tachycardia and ventricular fibrillation were induced. The diagnosis of BrS was confirmed, and an intracardiac defibrillator (ICD) was implanted to prevent sudden cardiac death.

Brugada syndrome, an inherited autosomal dominant cardiac disease, is one of the leading causes of sudden death in young men without structural cardiac defects.[3] Patients with BrS are vulnerable to life-threatening ventricular arrhythmia.[4] Symptomatic BrS patients with a spontaneous type 1 ECG pattern (described above and in the figure) should undergo ICD implantation. In addition, an ICD should be considered for asymptomatic BrS patients if an EPS induces ventricular tachyarrhythmia.

When a patient with a suspected type 1 Brugada ECG pattern who has not been fully evaluated and is not receiving any therapy presents in need of an emergent operation, medications that interact with cardiac electrical

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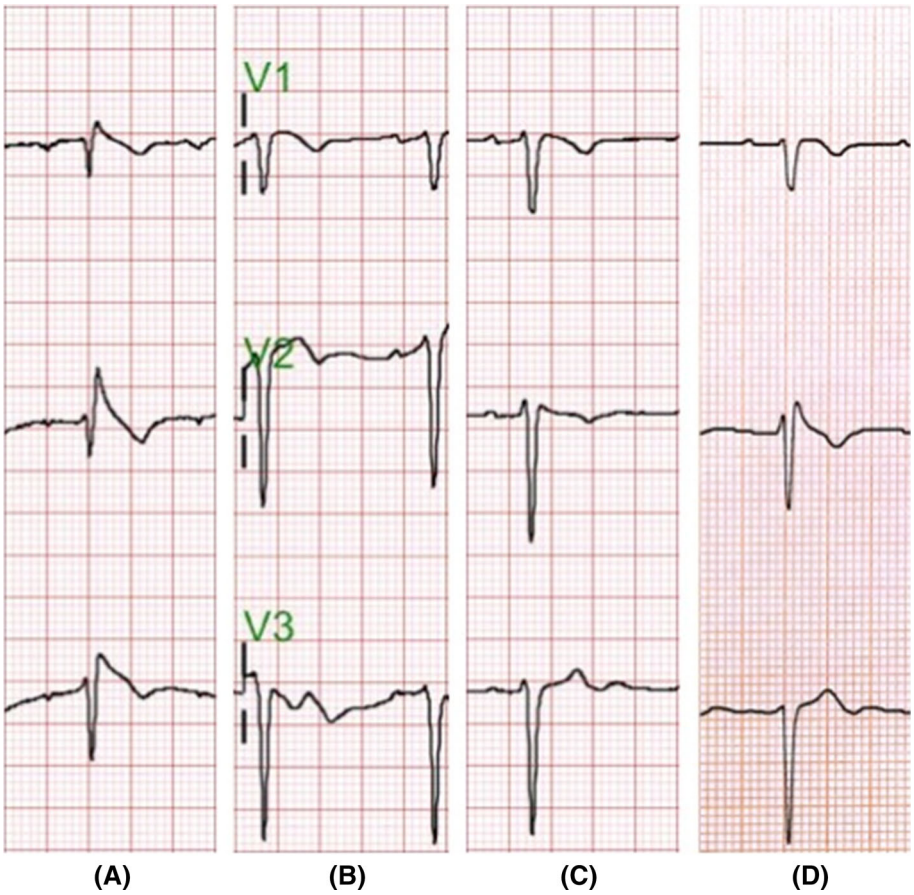

Figure Sequential electrocardiographic changes in a patient with Brugada syndrome given a prophylactic continuous isoproterenol infusion. (A) Preoperative electrocardiogram (ECG) shows a typical type 1 Brugada pattern, with J elevation $\geq$ 0.2 mV and a coved-type ST segment followed by a negative T wave in leads V2 and V3. (B) After two hours of isoproterenol infusion, the ST elevation in ECG had reduced significantly. (C) After seven hours of isoproterenol infusion, the ECG was normal. (D) At 31 hr after discontinuing the isoproterenol, typical J point and coved ST segment elevation reappeared on lead V2

activities may be the only preventive therapeutic option. Sodium channelopathies linked with BrS result in the J point elevation and ST segment changes because of a prominent, transient, outward current-mediated notch and loss of the epicardial action potential dome.[5] By increasing the calcium current, the β-agonist isoproterenol may restore the action potential dome, thus normalizing the ST segment.[5]

Perioperative infusion of isoproterenol for preventing malignant ventricular arrhythmias has not been previously reported. In our patient, low-dose isoproterenol temporarily diminished the ST segment abnormalities. Although anesthesia is generally considered relatively safe in these patients,[3] this novel low-dose isoproterenol infusion may offer further safety for those with uncontrolled BrS who require emergent operations. Whether this approach prevents ventricular arrhythmias requires further study.

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References

1. *Vohra J*, *Rajagopalan S*, *CSANZ Genetics Council Writing Group*. Update on the diagnosis and management of Brugada syndrome. Heart Lung Circ 2015; 24: 1141-8.
2. *Merchant R, Chartrand D, Dain S, et al.* Guidelines to the practice of anesthesia – revised edition 2016. Can J Anesth 2016; 63: 86-112.
3. *Smith D*, *Martz DG*. Brugada syndrome: a review of peri-operative management for the anesthesiologist. Int J Clin Anesthesiol 2014; 2: 1019.
4. *Postema PG*, *van Dessel PF*, *Kors JA*, *et al*. Local depolarization abnormalities are the dominant pathophysiologic mechanism for type 1 electrocardiogram in Brugada syndrome a study of electrocardiograms, vectorcardiograms, and body surface potential maps during ajmaline provocation. J Am Coll Cardiol 2010; 55: 789-97.
5. *Jongman JK*, *Jepkes-Bruin N*, *Ramdat Misier AR*, *et al*. Electrical storms in Brugada syndrome successfully treated with isoproterenol infusion and quinidine orally. Neth Heart J 2007; 15: 151-5.