J Anesth (2001) 15:182–184

Journal of
Anesthesia

Severe sinoatrial dysfunction after esophageal surgery: a case report

Shinji Takahashi[1], Taro Mizutani[2], Muneaki Watanabe[3], Hiroyuki Iida[3], Kazuo Orii[3], and Hidenori Toyooka[4]

[1] Department of Anesthesia and Critical Care Medicine, Tsukuba Gakuen Hospital, 2573-1 Kamiyokoba, Tsukuba, Ibaraki 305-0854, Japan
[2] Department of Critical Care Medicine, Institute of Clinical Medicine, University of Tsukuba, Tsukuba, Ibaraki 305-8575, Japan
[3] Department of Surgery, Tsukuba Gakuen Hospital, 2573-1 Kamiyokoba, Tsukuba, Ibaraki 305-0854, Japan
[4] Department of Anesthesiology, Institute of Clinical Medicine, University of Tsukuba, Tsukuba, Ibaraki 305-8575, Japan

Key words Arrhythmia · Bradycardia · Sinus arrest · Midazolam · Intensive care

Introduction

Cardiac dysrhythmias are among the most common anesthetic complications and are observed even in patients who have had no previous cardiac disease. Moreover, possible causes of perioperative dysrhythmias are numerous. Although lethal cardiac dysrhythmias are rare in well-managed cases, unexpected dysrhythmias, including sick sinus syndrome (SSS), are potentially dangerous [1,2]. We report a patient in whom temporary sinoatrial dysfunctions (severe bradycardia, sinoatrial blockade, and sinus arrest) were observed during midazolam infusion postoperatively, although he had had no obvious sinoatrial abnormality preoperatively.

Case report

A 61-year-old, 167-cm, 50-kg man with early-stage esophageal cancer was scheduled for esophagectomy and retrosternal reconstruction with the stomach. He had a history of hypertension, diabetes mellitus, and brain infarction. The hypertension was treated with oral nifedipine, and the diabetes was adequately controlled with oral glibenclamide. Moreover, he had not manifested diabetic autonomic neuropathy, such as orthostatic hypotension. All oral intake had been discontinued a week before surgery, and total parental nutrition had been established via a catheter placed in the superior vena cava (SVC). The position of the catheter tip was confirmed by chest X-ray film. His blood glucose level was well controlled with intravenous insulin (blood glucose levels ranged from 100 to 200 mg·dl^{-1}), and his blood pressure (BP) was within the normal limits. A preoperative 12-lead electrocardiogram (ECG) revealed regular sinus rhythm at a rate of 76 beats per minute (bpm) without hypertensive change. His echocardiogram showed normal cardiac function.

After premedication with intravenous famotidine 20 mg, a thoracic epidural catheter was placed at the Th5–6 interspace before induction of anesthesia. General anesthesia was induced by thiopental, fentanyl, and vecuronium intravenously. Then, a double-lumen endobronchial tube (Bronchocath; Mallinckrodt Medical, St. Louis, MO, USA) was placed uneventfully. Anesthesia was maintained with 0.5%–1.0% isoflurane in 40% oxygen-air mixture, with intermittent doses of intravenous fentanyl, and with continuous thoracic epidural anesthesia using 1.5% lidocaine with 1:200000 epinephrine. Dopamine was administered intravenously in doses of 3 to 5 μg·kg^{-1}·min^{-1} to maintain hemodynamic stability and adequate urinary output. The operation was completed without any noticeable changes in ECG monitored by lead II.

The patient was transferred to the intensive care unit (ICU) with ventilatory support after surgery. His vital signs were within normal limits, with BP 150/80 mmHg, and the ECG revealed a normal sinus rhythm with a rate of 80 bpm. He received midazolam 5 mg·h^{-1} and fentanyl 50 μg·h^{-1} intravenously and epidural infusion of 0.125% bupivacaine with morphine (80 μg·ml^{-1}) at a rate of 2 ml·h^{-1}. Intravenous dopamine was continuously administered to maintain hemodynamic stability.

Four hours after admission to the ICU, the ECG revealed junctional rhythm and atrioventricular block

Address correspondence to: S. Takahashi, Department of Anesthesiology, Institute of Clinical Medicine, University of Tsukuba, Tsukuba, Ibaraki 305-8575, Japan
Received: September 8, 2000 / Accepted: March 28, 2001

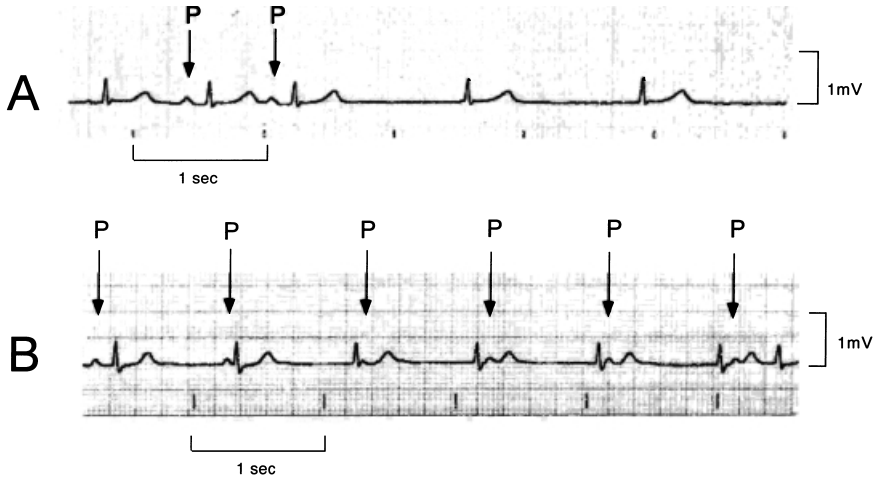

Fig. 1. Dysrhythmias during midazolam infusion. Sinus arrest or sinoatrial block with supraventricular rhythms (**A**) and complete atrioventricular block (**B**). *P* indicates P wave

at a rate of 40–50 bpm (Fig. 1). At that time, systolic BP decreased to 60 mmHg. The dose of intravenous dopamine was increased to 10 μg·kg^{-1}·min^{-1}, and 0.5 mg of atropine and 10 mg of ephedrine were repeatedly given intravenously. Although intravenous fentanyl and continuous epidural block were discontinued, intravenous midazolam was administered uninterruptedly. Neither hypoxia nor hypercarbia was revealed in the blood gas analysis, and the plasma potassium concentration was 4.3 mEq·l^{-1}. Correct position of the central venous catheter tip was verified by postoperative chest X-ray film.

Because bradycardia recurred in spite of repeated intravenous injection of atropine up to 2 mg, intravenous infusion of isoproterenol 0.03 μg·kg^{-1}·min^{-1} was started 13 h after admission. Then, the ECG showed tachycardia at a rate of 150 bpm. Therefore, intravenous isoproterenol was discontinued after an hour and 0.2 mg of deslanoside was given to control HR. After cessation of isoproterenol, midazolam 5 mg·h^{-1} and dopamine 10 μg·kg^{-1}·min^{-1} were continuously infused. The systolic blood pressure gradually decreased to 80 mmHg, and the HR ranged between 125 and 135 bpm. Eleven hours after cessation of isoproterenol, severe bradycardia occurred at a rate of 16 bpm. HR returned to 50 bpm within a short time. An emergency temporary percutaneous pacemaker catheter was inserted via the right femoral vein. The pacemaker produced pacing at a rate of 90 bpm.

After insertion of the pacemaker catheter, intravenous midazolam was discontinued to wean the patient from ventilatory support. Two hours after cessation of intravenous midazolam, HR increased up to 90 bpm without the assistance of pacemaker pacing. The patient's trachea was then extubated uneventfully. Five days after extubation, the pacemaker catheter was removed. Neither bradycardia nor tachycardia was observed by Holter ECG a month after the surgery. The patient had been free from symptoms of sinoatrial dysfunction for 10 months.

Discussion

The important findings of this report were as follows: First, lethal dysrhythmias that included sinus arrest occurred in a patient who had had no previous history of sinoatrial dysfunction. Second, the sinoatrial dysfunction was present during administration of intravenous midazolam and disappeared after cessation of midazolam.

Transitory sinoatrial dysfunction can result from various causes. There were several possible causes of sinoatrial dysfunction in this case. First, sinoatrial function might have been significantly affected by autonomic neuropathy associated with diabetes mellitus. Bradycardia, hypotension, and cardiopulmonary arrest have been reported among diabetics with autonomic neuropathy during the perioperative period [3,4]. Moreover, electrophysiological studies have a low diagnostic value in autonomic sinus node dysfunction [5]. Therefore, although diabetes seemed to be well controlled before the surgery, diabetes might have modified the sinoatrial function in this patient. Second, sympathetic blockade associated with thoracic epidural anesthesia might lead to the vagotonic condition, thereby decreasing sinus node activity. Third, perioperative cardiac complications occur frequently in thoracic and upper abdominal surgery [6], particularly of the esophagus [7]. Direct compression of the heart may induce cardiac muscle damage, thereby causing ischemic changes and sinoatrial dysfunction. Furthermore, arteriosclerosis of the coronary arteries and ischemic heart disease may be associated with sinoatrial dysfunction [8]. The instability of intravascular volume after major surgery might contribute to induction of the cardiac arrhythmia.

Fourth, although malposition and migration of the central venous catheter tip may evoke dysrhythmias and heart block [9], correct position of the catheter tip was radiographically verified in this case pre- and postoperatively. Finally, we speculate that midazolam was involved in the cause of sinoatrial dysfunction observed in this patient, although the dose of midazolam used in this case was not excessively large.

It is unclear whether the diagnosis of SSS can be made in this case. The presentation of SSS is varied, ranging from patients who are asymptomatic to those with sudden cardiac arrest. Therefore, occasionally SSS is not detected by routine preoperative study [1,5]. On the other hand, SSS tends to appear with deep anesthesia and the vagotonic condition [2,10]. Pratila et al. [2] reported that SSS was manifested during general anesthesia. Moreover, Underwood et al. [10] reported that the diagnosis of SSS was made in a patient who developed cardiac arrest on the ward several hours after spinal anesthesia. Since deep anesthesia and sedation take the sympathetic tone off from the sinus node, decreased intrinsic sinus node activity appears to cause a decrease in HR in patients with SSS. In our case, midazolam might disclose the latent sinoatrial dysfunction attenuating sympathetic tone temporarily.

Although midazolam has been widely used for conscious sedation and for induction and maintenance of anesthesia, cardiovascular effects are minimal in general [11–13]. To our knowledge, there has been only one report that described cardiovascular side effects. Hirata et al. [1] reported that 4 mg of midazolam given for anesthetic induction induced severe bradycardia in a 50-year-old man. In their report, the patient had no clinical symptoms before the operation, but he was diagnosed with SSS thereafter. In our case, we were unable to diagnose SSS, since postoperative Holter ECG did not reveal any abnormality.

A case of sinoatrial dysfunction that presented during sedation with midazolam was reported. Because midazolam may be considered a first-line drug for sedation, and may also be useful in a variety of other clinical applications, it will be used frequently in the perioperative settings. Our case report of sinoatrial dysfunction underscores the importance of careful observation of sinoatrial function during continuous midazolam infusion, even if a patient has had no clinical symptoms previously.

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