

# Conversion of supraventricular tachycardia to normal sinus rhythm by dexmedetomidine treatment

Cheol Lee, Yeon-Dong Kim, Dong-Hyuk Seo, Jae-Hun Lee, and Yoon-Kang Song

Department of Anesthesiology and Pain Medicine, Wonkwang University School of Medicine, Iksan, Korea

Cardiac dysrhythmias are an important cause of morbidity and mortality in the perioperative period, and they are more common after thoracic surgery and are most often supraventricular in origin [1]. Dexmedetomidine, a highly selective  $\alpha$ -2 adrenoceptor agonist, is increasingly being used in anesthesia and critical care because it not only produces sedation and analgesia but also decreases sympathetic tone and attenuates stress responses to surgery leading to potential antiarrhythmic effects [2]. Here, we present a case of dexmedetomidine treatment of paroxysmal supraventricular tachycardia (PSVT) that occurred during thoracic surgery.

A 72-year-old man, 168 cm tall and weighing 63 kg, was scheduled for right lower lobectomy suspicious of non-small-cell lung cancer. He has medical history of diabetes mellitus and hypertension for 10 years, which were both well controlled with medications. Preoperative electrocardiography (ECG) and chest radiography was unremarkable. Moreover, laboratory data were within normal limits. The patient was monitored with pulse oximetry, ECG, invasive radial arterial blood pressure, capnography, bispectral index (BIS), esophageal temperature probe. Induction of anesthesia was commenced with a slow (60 second) intravenous bolus dose of remifentanyl (1  $\mu$ g/kg), followed by propofol (1 mg/kg) tracheal intubation was facilitated with rocuronium (0.9 mg/kg). Anesthesia was maintained With  $O_2$  1.5 L/min,  $N_2O$  1.5 L/min and desflurane. Single-lung ventilation using a double-lumen endotracheal tube was the mode of anesthesia. After one-lung ventilation, arterial blood gas analysis showed normal findings. During retraction of the right lung apex, blood pressure abruptly decreased to 70/45 mmHg and

heart rate increased to 185 beats/min. ECG findings showed PSVT. After notifying the surgeon, the surgery was stopped, and sinus rhythm and blood pressure returned to normal within 20–30 seconds after carotid sinus massage. Right lower lobectomy was performed, and during lung retraction for hemostasis, PSVT accompanying a low blood pressure of 70/40 mmHg and high heart rate of 180–190 beats/min occurred again. The dopamine dose (5–10  $\mu$ g/kg/min) was titrated to stabilize vital signs, and carotid sinus massage was performed to terminate the PSVT. However, the PSVT did not revert to normal sinus rhythm even after adenosine was administered using the standard two-stage protocol, i.e., 0.1 mg/kg (6 mg) followed by 0.2 mg/kg (12 mg). The PSVT disappeared temporarily and reappeared later. At that time, arterial blood gas analysis showed the following results: pH, 7.25;  $PaCO_2$ , 53 mmHg;  $PaO_2$ , 139 mmHg;  $Na^+$ , 140 mEq/L;  $K^+$ , 4.5 mEq/L and blood sugar, 186 mg/dl. Dexmedetomidine (0.5  $\mu$ g/kg) was administered as a slow intravenous infusion for over 180 seconds according to blood pressure changes on monitoring arterial blood pressure, followed by a continuous infusion at a dose of 0.7  $\mu$ g/kg/hr. With the return of normal sinus rhythm, the arterial and pulse oximetry waveforms improved (Fig. 1). The dexmedetomidine infusion was then titrated down



Fig. 1. Tachycardia determination after dexmedetomidine administration.

Corresponding author: Yeon-Dong Kim, M.D., Department of Anesthesiology and Pain Medicine, Wonkwang University School of Medicine, 344-2, Sinyong-dong, Iksan 570-711, Korea. Tel: 82-63-859-1560, Fax: 82-63-857-5472, E-mail: kydpain@hanmail.net

© This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (<http://creativecommons.org/licenses/by-nc/3.0/>), which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

to 0.2 µg/kg/hr. After the operation, the patient regained consciousness and was extubated. His postoperative course was uneventful.

Arrhythmia is by far the most common cardiac complication after noncardiac thoracic surgery, with the incidence rate ranging from 10 to 40% after thoracic surgery. Extensive surgical stress, increased sympathetic activity, and hypoxemia and hypercapnia secondary to respiratory depression are all proposed as possible mechanisms of SVTs. Autonomic neural fiber injury may result in sensitization of the atrial myocardium to the circulating catecholamines also known to be cause of SVTs [3].

In our case, PSVT might have resulted from surgical stress that caused by surgical trauma to the atria and to sympathovagal fibers during lobectomy and hypercapnia secondary to one-lung ventilation, which showed a pH value of 7.31 and PaCO<sub>2</sub> value of 49 mmHg on arterial blood gas analysis.

Adenosine has an ultra-short action and half-life, a proarrhythmic effect for the chemical cardioversion of reentrant SVT, followed by calcium channel blockers. However, the use of adenosine can be associated with an unpredictable duration of bradycardia and asystole, even though it is well tolerated in the majority of case and potentially lethal polymorphic ventricular tachycardia reported dose-dependent efficacy between 70 and 95%. These properties make it essentially of no use to patients with multiple recurring SVTs and to those with an SVT recurrence rate of 9–57% [2]. Although limitations and significant adverse events in the use of drugs for treating SVTs have been encountered over the years, an alternative effective and safe agent is not yet available. Dexmedetomidine, an α-2 adrenoceptor agonist with sedative, analgesic properties has recently been shown to have potential antiarrhythmic effects [2]. In most of

the studies, dexmedetomidine served as an agent to prevent tachyarrhythmias [4].

An intravenous infusion of dexmedetomidine, recommended for adults, is commonly initiated with a loading dose of 1 µg/kg, administered for over 10 minutes, followed by a maintenance infusion at a dose of 0.2–1.0 µg/kg/hr. It is reported that a 0.5–1 µg/kg loading dose of dexmedetomidine was a novel antiarrhythmic agent in the acute termination of AV nodal-dependent reentrant SVT [2].

In our case, the patient was successfully treated with a 0.5 µg/kg loading dose of dexmedetomidine, which was administered as a slow intravenous infusion for over 180 seconds according to blood pressure changes on monitoring, followed by a continuous infusion at a dose of 0.7 µg/kg/hr, and the dopamine dose (5–10 µg/kg/min) was titrated to stabilize vital signs.

Although the exact mechanism is not known, a central α-2-adrenoceptor-mediated enhancement of vagal neural activity appears to play a potential antiarrhythmic role. Previous studies have suggested that predominance of sympathetic activity due to injury to cardiac parasympathetic nerves during thoracic surgery is the primary autonomic mechanism triggering postoperative SVTs. Recent studies have also shown that activation of central imidazoline type I receptors has been implicated in the prevention of ventricular arrhythmias, another possible mechanism involves the activation of imidazoline receptors. Dexmedetomidine contains an imidazole ring and has an affinity for these receptors [5].

In conclusion, dexmedetomidine may be used as a second-line drug if adenosine, when used as the first-line drug, fails in the acute treatment of SVTs. Further study is necessary on the dose and dose rate for the acute treatment of SVTs.

## References

1. Thompson A, Balsler JR. Perioperative cardiac arrhythmias. *Br J Anaesth* 2004; 93: 86-94.
2. Chrysostomou C, Morell VO, Wearden P, Sanchez-de-Toledo J, Jooste EH, Beerman L. Dexmedetomidine: therapeutic use for the termination of reentrant supraventricular tachycardia. *Congenit Heart Dis* 2013; 8: 48-56.
3. Amar D, Zhang H, Miodownik S, Kadish AH. Competing autonomic mechanisms precede the onset of postoperative atrial fibrillation. *J Am Coll Cardiol* 2003; 42: 1262-8.
4. LeRiger M, Naguib A, Gallantowicz M, Tobias JD. Dexmedetomidine controls junctional ectopic tachycardia during Tetralogy of Fallot repair in an infant. *Ann Card Anaesth* 2012; 15: 224-8.
5. Wikberg JE, Uhlén S, Chhajlani V. Medetomidine stereoisomers delineate two closely related subtypes of idazoxan (imidazoline) I-receptors in the guinea pig. *Eur J Pharmacol* 1991; 193: 335-40.