

Dexmedetomidine for Rigid Bronchoscopy in an Infant with Tracheal Web after Ventricular Septal Defect Patch Repair

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The authors have no financial conflicts of interest.

We report herein successful rigid bronchoscopy with preserved spontaneous breathing of a 54-day-old infant with tracheal web associated with previous ventricular septal defect (VSD) repair. We considered the use of dexmedetomidine in conjunction with intermittent ketamine from the following three clinical aspects. First, this infant was suffering from respiratory distress with chest retraction, the cause of which was not revealed by a computerized scan of the neck and chest. Second, the patient was scheduled for rigid bronchoscopy, which is accompanied by brief but strong stimulation. Third, this infant underwent congenital VSD heart repair approximately 1 month earlier.

Key Words: Bronchoscopy, dexmedetimidine, infant, tracheal web

INTRODUCTION

Successful anesthetic management of infant patients suffering from respiratory distress is one of the most challenging scenarios for anesthesiologists.

In this case, we used dexmedetomidine and ketamine for rigid bronchoscopy while maintaining spontaneous ventilation in an infant suffering from respiratory difficulties, because of tracheal web associated with previous ventricular septal defect (VSD) repair.

CASE REPORT

Prior to the publication of this report, parental informed consent was obtained. A 54-day-old, 3.41-kg infant presented for tracheal evaluation with bronchoscope. At full-term birth, he was diagnosed with a large VSD by echocardiography. He underwent a VSD patch repair when he was 14 days old. Three days before admission, the patient presented with symptoms of upper respiratory infection like coughing with scanty sputum accompanied by dyspnea with chest retraction and wheezing. Computerized tomography of the chest and neck revealed no visible airway anomaly.

The infant was brought to the operating room with oxygen of 3 L min⁻¹ via nasal cannula. After applying electrocardiogram, non-invasive blood pressure, and pulse

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oximetry (SpO₂) instruments, he was administered 14 µg of glycopyrrolate. As an anesthetic regimen to maintain spontaneous respiratory efforts and minimize possible hemodynamic instabilities, 0.5 µg kg⁻¹ dexmedetomidine was loaded over 15 min and infused at 1-1.5 µg kg⁻¹ h⁻¹ thereafter. In addition, 1 mg kg⁻¹ ketamine was given intravenously. After the patient was sedated adequately and preoxygenated via face mask, rigid bronchoscopy began. Additional ketamine doses totaling 2 mg were administered intravenously during rigid bronchoscopy advancement, which revealed tracheal web formation with an opening of 2.5 mm at 1.0 cm below the level of vocal cord. Dexamethasone (1 mg) was administered to prevent tracheal and laryngeal edema. Oxygen was administered via the ventilatory port of the bronchoscope during the procedure without desaturation of SpO₂ below 95%. The infusion of dexmedetomidine was stopped at the end of the procedure. In total, 1.25 µg kg⁻¹ dexmedetomidine was used during the 45-minute procedure. The patient experienced no coughing episodes. After loading of dexmedetomidine, blood pressure and heart rate decreased temporarily (Fig. 1). The patient was transferred back to the pediatric cardiac care unit, where he remained sedated for 1 hour and 25 minutes and recovered without agitation.

DISCUSSION

An anesthetic technique to counter strong stimuli while preserving spontaneous respiration is a challenge for anesthesiologists, especially in patients with suspected airway anomalies. In this case, an infant suffering from respiratory difficulty

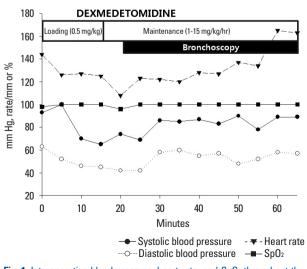


Fig. 1. Intraoperative blood pressure, heart rate, and SpO_2 throughout the procedure.

showing chest retraction was brought to the operating room for rigid bronchoscopic evaluation.

The pharmacological properties and clinical application of dexmedetomidine, especially for sedation for surgical procedures, has been well reviewed previously.1 Dexmedetomidine has a distinctive advantage of preserving spontaneous ventilatory ability over conventional intravenous sedatives/ anesthetics or inhalational anesthetics. With an aid of these properties, dexmedetomidine has been suggested in infants and children as a main or adjuvant sedative agent during intraoperative care where spontaneous ventilation is required, such as procedures on the airway¹ or postoperative evaluation following airway reconstruction.² Dexmedetomidine has been mostly applied to children in order to prevent airway catastrophe rather than due to airway distress.3 Unfortunately, dexmedetomidine is currently used off-label for pediatric patients because of its unproven safety, especially with regard to cardiovascular responses. Its deleterious cardiovascular responses include hypotension and bradycardia. Transient hypertension may also occur, especially during the loading dose or high maintenance dose infusion, and the incidence of hypertension has reached 8.5% in infants of less than 6 months of age.³ A case series of direct laryngoscopies and bronchoscopies, in 4 infants aged between 2 weeks and 11 months, with upper airway abnormalities but likely with healthy hearts, reported successful use of dexmedetomidine either as a sole agent or in conjunction with propofol.⁴ In that report, the total doses of dexmedetomidine ranged from 2 to 5 µg kg⁻¹, mean blood pressure increased 20-30% in three infants, and heart rates decreased 20-30% in all patients.

In the present case, we considered the use of dexmedetomidine in conjunction with intermittent ketamine from the following three clinical aspects. First, this infant was suffering from respiratory distress with chest retraction, the cause of which was not revealed by a computerized scan of the neck and chest. Second, the patient was scheduled for rigid bronchoscopy, which is accompanied by brief but strong stimulation. Third, this infant underwent congenital VSD heart repair approximately 1 month earlier.

Therefore, we loaded dexmedetomidine at a dose of 0.5 μ g kg⁻¹ for 15 minutes to minimize, if possible, transient hypertension and thereafter maintained an infusion rate of 1-1.5 μ g kg⁻¹ hr⁻¹ in conjunction with intermittent small doses of ketamine (in total, 3 mg) on movement of the bronchoscope. Because dexmedetomidine was successfully used in cardiac catheterization for infants and toddlers with con-

genital heart disease,⁵ dexmedetomidine of 1.25 µg kg⁻¹ in total was used during the 45-minute procedure. Blood pressure and heart rate decreased temporarily during entire procedure (Fig. 1). While both dexmedetomidine and ketamine have advantages in maintaining ventilatory status, they also have complementary effects. Bradycardia and hypotension during dexmedetomidine infusion can be counteracted in part by sympathomimetic effect of an intermittent ketamine bolus injection. In addition, the undesirable feature of increased airway secretions with ketamine, especially during airway manipulation, may be attenuated by xerostomia induced by dexmedetomidine.

One of the choices of anesthetics for pediatric patients suffering from airway distress is a combination of propofol and sevoflurane. However, the combination of propofol and sevoflurane enhances the function of γ -aminobutyric acid type A receptors and results in respiratory depression.⁶⁻⁸

Oxygen with FiO₂ of 1.0 was administered via ventilatory port of bronchoscopy, and SpO₂ never fell below 95%. However, we could not confirm ventilatory status because we did not analyze arterial blood gas status. Bronchoscopy revealed the tracheal web at the subglottic level, perhaps resulting from prolonged endotracheal intubation after the VSD patch repair. The patient tolerated the procedure well and remained sedated without agitation for 1 hour and 25 minutes during the anesthetic emergence period.

In conclusion, dexmedetomidine in conjunction with ketamine provided adequate sedation without any respiratory or significant hemodynamic compromise during rigid bronchoscopy in an infant suffering from respiratory distress due to tracheal web associated with surgically corrected congenital heart disease.

REFERENCES

- Shukry M, Miller JA. Update on dexmedetomidine: use in nonintubated patients requiring sedation for surgical procedures. Ther Clin Risk Manag 2010;6:111-21.
- Silver AL, Yager P, Purohit P, Noviski N, Hartnick CJ. Dexmedetomidine use in pediatric airway reconstruction. Otolaryngol Head Neck Surg 2011;144:262-7.
- Mason KP, Lerman J. Review article: Dexmedetomidine in children: current knowledge and future applications. Anesth Analg 2011;113:1129-42.
- Shukry M, Kennedy K. Dexmedetomidine as a total intravenous anesthetic in infants. Paediatr Anaesth 2007;17:581-3.
- Barton KP, Munoz R, Morell VO, Chrysostomou C. Dexmedetomidine as the primary sedative during invasive procedures in infants and toddlers with congenital heart disease. Pediatr Crit Care Med 2008;9:612-5.
- Loria CJ, Stevens AM, Crummy E, Casadesus G, Jacono FJ, Dick TE, et al. Respiratory and behavioral dysfunction following loss of the GABAA receptor α4 subunit. Brain Behav 2013;3:104-13.
- Jenkins A, Franks NP, Lieb WR. Effects of temperature and volatile anesthetics on GABA(A) receptors. Anesthesiology 1999;90: 484-91.
- Bai D, Pennefather PS, MacDonald JF, Orser BA. The general anesthetic propofol slows deactivation and desensitization of GABA (A) receptors. J Neurosci 1999;19:10635-46.