Case report

Dexmedetomidine for conscious sedation in difficult awake fiberoptic intubation cases

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Abstract
Currently used methods of sedation for fiberoptic intubation such as benzodiazepines, propofol, or opioids have their limitations. Dexmedetomidine (DEX) is a selective α-2 adrenergic agonist that has been used clinically for its sympatholytic, analgesic, and sedative properties. We report on 4 patients with particularly difficult airways who underwent successful awake fiberoptic intubation with DEX. Dexmedetomidine was used to provide a moderate level of conscious sedation without causing respiratory distress or hemodynamic instability during fiberoptic intubation.

1. Introduction
Awake fiberoptic intubation (AFOI) is indicated in a variety of clinical situations. An integral part of AFOI is preparing the patient for optimal intubating conditions. One challenge associated with this procedure is providing adequate anxiolysis while maintaining patient safety with respect to a patent airway and adequate ventilation. Currently used methods of conscious sedation such as benzodiazepines, propofol, or opioids have their limitations with regard to respiratory depression and loss of a patent airway. The ideal sedative would allow the patient to maintain spontaneous ventilation, protect their own airway, be cooperative or at least rousable, and tolerate passage of the fiberoptic scope. We describe 4 cases of challenging AFOI using dexmedetomidine (DEX) for conscious sedation. We administered one μg/kg intravenously (IV) over 10 minutes followed by a continuous infusion at 0.5 μg/kg/hr. Dexmedetomidine is a selective α-2 adrenergic agonist [1] that has been used clinically for its sympatholytic, analgesic, and sedative properties. By promoting natural sleep pathways [1], DEX creates a conscious, sedated patient who is rousable with minimal respiratory depression. There have been few published reports of DEX for use in the management of the airway [2,3]. This series of case reports presents diverse clinical scenarios in which the use of more...
traditional methods to provide adequate intubating conditions for AFOI would have been difficult.

2. Case reports

2.1. Case 1

An 18-year-old, otherwise healthy man presented with a crush injury to the head, a C1 fracture, and a cranial epidural hematoma. His initial Glasgow Coma Scale (GCS) score in the field was 15, but deteriorated to a GCS score of 10 after his arrival at the hospital. In the operating room (OR), the patient became agitated and combative to tactile stimuli. The decision was made to use DEX to prepare the patient for AFOI. The patient was uncooperative for the application of topical anesthetics to the airway or for traditional nerve blocks. We administered one $\text{mg} \cdot \text{kg}^{-1}$ DEX IV over 10 minutes followed by a continuous infusion at $0.5 \text{mg} \cdot \text{kg}^{-1} \cdot \text{hr}^{-1}$. During administration of the loading dose of DEX, a Williams airway was placed to facilitate AFOI and to serve as a bite block. The patient did not tolerate this action and he vomited. He was log-rolled and suctioned. The patient did not show any signs of aspiration and his oxygen saturation via pulse oximetry ($\text{SpO}_2$) remained above 97%. Auscultation of the chest showed clear lung sounds. In addition, no particulate material was observed during AFOI, there were no pulmonary symptoms during his surgery, and no radiologic indications of aspiration postoperatively. The DEX loading dose was followed by a DEX infusion. This action provided excellent intubating conditions without derangement in hemodynamics. Dexmedetomidine provided adequate sedation to perform successful AFOI on the first attempt, without change in hemodynamic parameters or signs of airway stimulation (i.e., coughing). The patient was responsive and followed commands to assure intact neurologic function after intubation. During and after intubation, his blood pressure (BP) remained within 15% of baseline, despite the lack of topical or airway nerve block anesthesia. This action was deemed important in the presence of increased intracranial pressure.

2.2. Case 2

A 54-year-old man presented with a 3.3 × 4-cm cystic thyroid mass (Fig. 1). Computed tomography scan of the neck showed left lobe involvement, with resultant tracheal compression and deviation. The patient was prepared for oral AFOI with midazolam two mg, glycopyrrolate 0.2 mg, and topical lidocaine 5% ointment applied to the posterior pharynx via the nasal route. We administered one $\text{mg} \cdot \text{kg}^{-1}$ DEX IV over 10 minutes followed by a continuous infusion at $0.5 \text{mg} \cdot \text{kg}^{-1} \cdot \text{hr}^{-1}$. Dexmedetomidine was used as the primary conscious sedative because of our concern for respiratory depression and/or loss of a patent airway, which has been associated with other sedatives. Glycopyrrolate was administered as an adjunct antispasmodic. On insertion of the fiberoptic bronchoscope, the extent of the tracheal deviation was such that only the right vocal cord could be visualized. Insertion of the endotracheal tube (ETT) was successfully achieved orally after moderately difficult positioning of the fiberoptic bronchoscope. The patient appeared to be comfortable and cooperative throughout the extended duration of the fiberoptic bronchoscopy, and his hemodynamics remained stable.

2.3. Case 3

A 44-year-old man with a herniated cervical disk and radiculopathy who was scheduled for coronary artery bypass graft surgery (CABG) required AFOI. A neurologic examination was performed. He was prepared for oral AFOI with glycopyrrolate 0.3 mg, midazolam two mg, viscous 2% lidocaine, oral Cetacaine spray, and two mL 4% lidocaine transtracheally. Dexmedetomidine one $\text{mg} \cdot \text{kg}^{-1}$ IV was administered over 10 minutes followed by a continuous infusion at $0.5 \text{mg} \cdot \text{kg}^{-1} \cdot \text{hr}^{-1}$. The combination of glycopyrrolate and DEX resulted in a dry airway. Awake fiberoptic intubation and passage of the ETT were completed without complication. After intubation, the patient was cooperative and was given a second neurologic examination, which was unchanged. The patient’s hemodynamics remained stable during the entire process.

2.4. Case 4

A 44-year-old man, who had been involved in a motorcycle trauma three days earlier, presented in a C-collar with fractures of his fifth and sixth cervical vertebrae, and was in active delirium tremens. The patient was scheduled for surgical repair of an open tibial and fibula fracture. Although he was uncooperative, aerosolized 4% lidocaine was administered via an acorn nebulizer for three minutes before his arrival in the OR. Dexmedetomidine one $\text{mg} \cdot \text{kg}^{-1}$ IV was administered over 10 minutes.
followed by a continuous infusion at 0.5 µg/kg/hr. After successful conscious sedation, the signs of delirium tremens were abated, then three mL 4% lidocaine was locally injected adjacent to the hyoid bone for a bilateral superior laryngeal nerve block. Bilateral glossopharyngeal nerve blocks were then administered via intraoral infiltration of the tonsillar pillar with three mL of 4% lidocaine. The patient underwent oral AFOI. Excellent intubating conditions were achieved, and no hemodynamic response or patient movement was seen on passage of the ETT, although the recurrent laryngeal nerves were not blocked. After intubation of the trachea, the patient was calm and able to follow commands. His hemodynamics remained stable throughout the procedure.

3. Discussion

One of the challenges of AFOI is preparing the patient for fiberoptic bronchoscopy and tracheal intubation. Several accepted methods of decreasing airway sensation include the use of injected local anesthetics to block the individual upper airway nerves and/or application of topical anesthetics to the upper airway. Anxiolytics and antiallrogues are also used to achieve optimal conditions. However, it may not always be possible to block the nerves of the upper airway. As illustrated in Case 1, often the patient’s condition does not allow the use of nerve blocks. For some patients, such as those with Ludwig’s angina, the respiratory depression of sedating medications can be deleterious. In our series of case reports, we used DEX to facilitate AFOI. In Case 1, it was the only medication used to facilitate AFOI. In Cases 2, 3, and 4, it was used as an adjunct to traditional conscious sedation and the use of airway nerve blocks and/or topical anesthesia.

Dexmedetomidine is a selective α-2 agonist with approximately 8 times the affinity of clonidine for the α-2 receptor [1]. It is also 1620 times more potent as an α-2 agonist than as an α-1 agonist [1]. Dexmedetomidine is pharmacologically unique in that the induced conscious sedation involves activation of the endogenous sleep-promoting pathway [1]. It activates the postsynaptic α-2 receptors in the locus coeruleus, which is an important modulator of wakefulness. Dexmedetomidine has analgesic, anxiolytic, and antiallrogue properties [4-6]. It does not depress respiration, and SpO2 remains within normal limits if DEX is used in the dose range of 0.2 to 0.7 µg/kg/hr [6]. We typically use 0.5 µg/kg/hr because it is a mid-range dose for conscious intubation. As expected from DEX pharmacokinetics, the pharmacologic effects during intubation result from the loading dose.

Case 1 was done without the aid of nerve blocks or topical anesthesia for a patient who was combative and agitated to tactile stimuli. The sole medication used to facilitate AFOI was DEX. This observation is consistent with our clinical experience, in which we have noted that patients frequently tolerate noxious stimuli after sedation with DEX. We have successfully performed AFOI in more than 20 patients using DEX as the sole sedative without placement of airway nerve blocks and have achieved acceptable intubating conditions. In some patients, DEX may provide adequate sedation and blunting of airway reflexes without additional airway nerve blocks. Placing nerve blocks in a combative patient with a C1 fracture in a C-collar can be quite challenging and risks injury to the patient. We did not feel that the patient should be consciously sedated with any medication that would blunt his ventilatory drive and increase his CO2 levels. Ketamine was also ruled out in this patient owing to its potential to increase intracranial pressure.

Case 2 presented with a large thyroid mass that had caused a deviation of the trachea (Fig. 1). We felt this would be a difficult AFOI due to the distortion of the anatomy caused by the mass. Distortion would have rendered nerve blocks of the upper airway challenging, so only topical lidocaine was used. The extent of the tracheal deviation was such that we were able only to visualize the right vocal cord during AFOI. Dexmedetomidine provided excellent intubating conditions in this patient.

In Case 3, a patient with known cervical disk pathology and coronary artery disease (CAD) was brought for CABG. In this patient, we used DEX for its ability to suppress increases in heart rate and BP in response to intubation. Furthermore, DEX provides myocardial protection in patients with cardiac risk factors [7,8]. Because of his CAD, it was essential in this patient to blunt the hemodynamic response to intubation. We also felt that it would be prudent to assess the patient’s neurologic function after intubation and positioning, given his cervical pathology, for which DEX was successful.

Case 4 involved a patient with acute cervical pathology and poor cooperative skills secondary to active delirium tremens. α-2 Agonists such as clonidine have long been known to help alleviate the signs and symptoms associated with delirium tremens and other types of withdrawal reactions [3]. Using DEX as an adjunct in this situation, we were able to provide stable conscious sedation and thus cervical protection for AFOI in an obviously difficult patient.

A few areas of concern exist when using DEX for AFOI. Bradycardia or hypotension is a potential side effect of α-2 agonist administration. In our series, we did not experience these problems. However, two of 4 patients were pretreated with glycopyrrolate, an action that may have decreased the frequency of bradycardia and hypotension in these patients. The loading dose for DEX requires 10 minutes to infuse. Titration by infusion then may be required to provide adequate conscious sedation. If used along with topical anesthesia or other modes of conscious sedation, this period may not fit into the anesthesiologist’s time constraints in an emergency situation.
In summary, our experience with these 4 cases shows that DEX can be used in a variety of challenging situations for AFOI. Dexmedetomidine may be used as an adjunct to nerve blocks or topical anesthesia. Some may argue that with good topical anesthesia and nerve blocks, DEX does not add much benefit. However, when these two adjuncts cannot be reliably accomplished, DEX appears to be beneficial. We believe that each case demonstrated the ability of DEX to act as an adjunct to traditional methods of preparing patients for AFOI. Case 1 suggests that DEX may be used as the sole agent for AFOI, even in the absence of topical anesthesia, and provides stable hemodynamics to facilitate ETT passage.

References


