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CLINICAL REPORT

Dexmedetomidine as sole agent for awake fiberoptic intubation in a patient with local anesthetic allergy

Maxime Madhere · David Vangura · Alik Saidov

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Abstract A series of case reports acknowledges the efficacy of dexmedetomidine as a sole sedative for awake intubations in managing a critical airway. However, most case reports documented in the literature used topicalization of the oropharynx either via nebulized lidocaine or the spray-as-you-go technique with either 2% or 4% lidocaine spray to achieve successful intubation. The following case report presents an intensive care unit (ICU) patient with a critical airway who had a true documented allergy to local anesthetics. This case report demonstrates that dexmedetomidine appears to be useful for sedation during awake intubations in critical airways, without the need for airway topicalization. The ability of dexmedetomidine to act as a sedative, anxiolytic, analgesic, and antisialagogue without causing respiratory depression is promising to the field of anesthesiology. Additional studies are needed to elucidate its potential role as the sole agent for awake fiberoptic intubation.

Keywords Dexmedetomidine · Awake fiberoptic intubation · Local anesthetic · Allergy

Introduction

Awake fiberoptic intubations provide an excellent alternative for patients with a difficult airway. However, there are limitations, especially if adequate anxiolysis has not been established, which include gag, cough, hypersalivation, and laryngospasm. Other concerns are centered around minimizing the deleterious effects that opioids, benzodiazepines, and induction agents cause. Such effects include respiratory depression and hemodynamic instability [1]. Dexmedetomidine (Precedex) is a viable option because patients are able to maintain a normal respiratory pattern without significant respiratory depression and a relatively normal respiratory pattern to blood carbon dioxide tension [2, 3]. It is a short-acting selective alpha-2 agonist, which has already been approved by the US Federal Drug Administration (FDA) for short-term sedation in intubated ICU patients [3]. In addition, a series of case reports acknowledged its efficacy as a sole sedative for awake intubations in managing a critical airway [1, 4, 5]. However, most case reports documented in the literature used topicalization of the oropharynx either via nebulized lidocaine or the spray-as-you-go technique with either 2% or 4% lidocaine spray to achieve successful intubation [1, 5, 6]. The following case report presents an ICU patient with a critical airway who had a true documented allergy to local anesthetics.

M. Madhere (✉) · D. Vangura · A. Saidov
Department of Anesthesiology, Henry Ford Hospital, 2799 West Grand Boulevard, Detroit, MI 48202, USA
e-mail: drmax08@aim.com

Case description

The patient was a 45-year-old woman with a history of documented allergic reaction to local anesthetics (causing angioedema), end-stage renal disease on hemodialysis, hypertension, and seizure disorder who initially presented to the hospital with confusion and a suspected submandibular/retropharyngeal infection. She was ultimately transferred to the MICU with increased effort in breathing, stridor, and deterioration in mental status secondary to hypertensive emergency. She also had a recent prior hospital admission with ICU monitoring after undergoing an elective vascular procedure for catheter exchange whereby

she developed angioedema after exposure to IV lidocaine. At the time she was evaluated by otolaryngology, a concern for tracheomalacia was noted. However, she was lost to follow-up (secondary to noncompliance) prior to readmission. On arrival at the MICU, she was agitated, tachypneic, and had stridor, with an oxygen (O_2) saturation of 93% on 50% Ventimask in a sitting position. Our department was called to intubate for airway protection and impending respiratory failure. On physical examination, an anterior neck scar from a previous tracheostomy was noted. Because of her submental abscess, documented history of an allergic reaction to local anesthetics, and a tracheostomy scar with concern for tracheomalacia, the decision was made to perform an awake nasal fiberoptic intubation without the use of topical lidocaine. Therefore, dexmedetomidine was chosen for conscious sedation at the bedside.

Standard American Society of Anesthesiologists (ASA) monitors were applied. As the usual administration program, a loading dose of 1 mcg/kg dexmedetomidine was given over 10 min, followed by 0.6 mcg/kg per hour as a continuous infusion. There was no topicalization of the naso-oropharynx with lidocaine spray or nebulization because of her documented allergy. During the dexmedetomidine infusion, the patient maintained a spontaneous respiratory pattern, and her level of excitability decreased to a point of no acute distress (equivalent Ramsay Sedation Scale score of 2). Twelve minutes later, a flexible fiberoptic nasopharyngoscope was inserted into the oropharynx to further evaluate her airway. There was some mild edema noted at the base of the tongue, but vocal cords were easily visible and mobile bilaterally. The scope was then removed and placed into the patient's right nares with a size 6.5 endotracheal tube attached. It was advanced slightly above the carina to check for anatomical obstruction. There was no direct visualization of any tracheal stenosis noted. The size endotracheal tube was then advanced over the scope on one attempt without difficulty, and the patient tolerated the procedure well with minimal discomfort. Correct tracheal tube placement was confirmed by fiberoptic visualization, positive end-tidal carbon dioxide (CO_2) detector, and bilateral breath sounds via auscultation. Oxygen saturation improved to 100%, and the patient remained hemodynamically stable throughout the entire procedure.

Discussion

This case demonstrates successful use of dexmedetomidine to provide adequate sedation and analgesia without airway topicalization for an awake fiberoptic tracheal intubation. The drug's sedative effects are well documented, with a mechanism of action mediated via postsynaptic alpha-2 adrenergic receptors, subsequently leading to alterations in ion channel conduction and decreased neuronal activation [7, 8]. Central acting presynaptic alpha-2a adrenergic agonists also activate receptors in the medullary vasomotor center, which causes central nervous system stimulation of parasympathetic outflow and inhibition of sympathetic outflow from the locus ceruleus [7, 9]. The decreased noradrenergic output from the locus ceruleus allows increased firing of inhibitory neurons, mainly γ-aminobutyric acid (GABA), which also play a major role in sedation and anxiolysis manifested by patients on dexmedetomidine [7]. The disadvantage of using benzodiazepines in a case scenario such as this is that they are well known to cause dose-dependent respiratory depression (decreasing both respiratory rate and tidal volume) and hypotension. Opioids were avoided in this case because of the risk of decreasing the respiratory rate, minute ventilation, and the sensitivity of the medullary center to CO_2 [10]. Opioid administration may also result in histamine release leading to bronchoconstriction, further compromising the airway. Because sedation is not considered an adequate substitute for regional anesthetic preparation of the airway, awake fiberoptics are generally done with topicalization. However, as our patient had a documented drug allergy to local anesthetics in addition to suspected tracheomalacia, we opted to avoid airway nebulization. Dexmedetomidine is also a moderate antisialagogue and causes minimal respiratory impairment, making it an ideal agent for critical airways [11].

A major reason for our successful intubation may be secondary to dexmedetomidine's underestimated analgesic properties, which exerts its effects through alpha-2 adrenergic receptors (located in the locus ceruleus and dorsal horn of the spinal cord) and inhibition of substance P release [9]. The specific subtype is the α_{2A} receptor, which apparently couples in an inhibitory fashion to the L-type calcium channel in the locus ceruleus [12]. The result is inhibition of norepinephrine release from the locus ceruleus, which aids in terminating the propagation of pain signals [10, 12]. The precise mechanisms of antinociception during intubation have not been clearly delineated. However, the analgesic effects of dexmedetomidine are well documented in the literature. Arain et al. [13] showed that dexmedetomidine administration before completion of major inpatient surgical procedures reduced early postoperative need for morphine by 66%. Ebert et al. [14] showed that increasing doses of dexmedetomidine lead to linearly decreasing pain sensation and mean arterial pressure response to cold pressor testing.

One potential limitation of this report is that this patient had end-stage renal disease and was dialysis dependent, which may have played a role in her Ramsay Sedation Scale Score (which was 2 throughout the entire procedure) [15, 16]. De Wolf et al. [17] evaluated the

pharmacokinetics of six adult patients with severe renal disease and showed there was prolonged sedation in patients taking dexmedetomidine compared with those with normal renal function. The authors speculated that the increased sedation duration with renal failure resulted from decreased protein binding and increased free fraction of the drug.

Although dexmedetomidine can cause bradycardia and hypotension, our patient did not suffer from any hemodynamic instability throughout the procedure. She also did not encounter any initial hypertensive response, which was also a concern given the fact that one reason for her transfer to the unit was hypertensive emergency. The literature shows that this hypertensive response usually occurs when high doses of dexmedetomidine are administered at a rate beyond the standard loading dose (which consists of 0.5–1 μg/kg over a period of 10–20 min followed by continuous infusion at a rate of 0.2–0.7 μg/kg per hour) and is secondary to stimulation of peripheral alpha-2 receptors [18]. Other antihypertensive medications administered to the patient prior to admission at our institution are another limitation in this case report. However, there have been numerous reports of attenuation of the sympathoadrenal response to tracheal intubation after dexmedetomidine administration [19].

In summary, we demonstrated that dexmedetomidine appears to be useful for sedation during awake intubations in critical airways without the need for airway topicalization. The drug's ability to act as a sedative, anxiolytic, analgesic, and antisialagogue without causing respiratory depression is promising to the field of anesthesiology. Additional studies are needed to elucidate its potential role as the sole agent for awake fiberoptic intubation.

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