CASE REPORT

Anesthesia for tracheostomy for huge maxillofacial tumor

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ABSTRACT

Providing sedation for patients with compromised upper airway is challenging. A 19-year-old female patient with huge maxillofacial tumor invading the whole pharynx scheduled for elective tracheostomy under local anesthesia due to compromised airway. The patient had gastrostomy tube for feeding. Venous cannulation was totally refused by the patient after repeated trials for exhausted sclerosed veins. Pre-operative mixture of dexmedetomidine with ketamine was administered through the gastrostomy tube with eutectic mixture of local anesthetics cream application over the planned tracheostomy site. The patient was sedated with eye opening to command. Local infiltration followed by tracheostomy was performed without patient complaints or recall of operative events.

Key words: *Airway, oral dexmedetomidine, oral ketamine, sedation*

INTRODUCTION

Lesions such as nasopharyngeal tumors, huge tonsils, oropharyngeal tumors and edema or tumors of the tongue are examples of pathological factors that predispose to upper airway compromization. Narrow airway resists collapse by generating an increase in both transmural pressure and pharyngeal wall tension. Also, this narrow airway, with high resistance to airflow, creates more negative intramural pressures with inspiration thus making it vulnerable for collapse. With progressive depth of sedation, there is loss of the compensatory pharyngeal muscles tone leading to deterioration in the upper airway obstruction with its detrimental ventilatory consequences. Maintenance of the airway during procedural sedation in patients with compromised airway is quite a challenging task for the anesthesiologist. We report a pre-operative enteral intake of ketamine-dexmedetomidine mixture to induce sedation with eutectic mixture of local anesthetics (EMLA) cream application over the planned tracheostomy site in poor compliant patient with huge craniofacial tumor compromising the airway.

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CASE REPORT

A 19-year-old female patient, weighted 40 kg, with a huge craniofacial tumor was scheduled for elective tracheostomy due to airway compromization. The tumor invaded the hypo and oropharynx causing displacement of the eyes, nasal obstruction with limited mouth opening and neck extension [Figure 1]. The patient had obstructive sleep apnea, shortness of breath, visual impairment and dysphagia for which gastrostomy was inserted for enteral feeding. The patient had a recurrence of the tumor after surgical intervention and multiple courses of chemo and radiotherapy. The patient absolutely refused any trial to insert an intravenous (IV) cannula as she had a low threshold for pain and on examination, her veins were apparently exhausted and sclerosed from chemotherapy and no vein could not be chosen to apply, EMLA cream. The patient was categorized as impossible to both intubate and ventilate due to severely distorted airway anatomy in

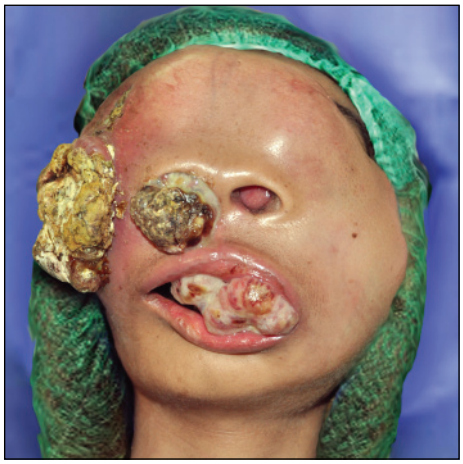

Figure 1: Huge tumor disfiguring facial and airway structures

uncooperative patient. Communication with the patient was challenging due to dysphonia. With the help of her mother, she reported that her daughter is absolutely refusing IV cannulation and she expressed her fear of pain perception during the tracheostomy procedure. Consent was taken from the patient to take a photo for her. In the high dependency unit, 2 h before surgery, sedation was initiated under full monitoring with baseline non-invasive blood pressure (BP) 111/74 mmHg, heart rate (HR) 92/min, oxygen hemoglobin saturation 98% and respiratory rate (RR) 17/min.

Ketamine (Tekam, Hikma, Jordan) with a dose of 120 mg (3 mg/kg) diluted in 10 ml normal saline was administered through the gastrostomy tube. That was followed by administration of 80 mcg (2 mcg/kg) dexmedetomidine (Precedex; Hospira, Inc., Lake Forest, IL, USA) diluted in 10 ml normal saline. EMLA 5 g cream 5% (Astra Pharma, Inc., Mississauga, Ontario, Canada) was applied over the planned tracheostomy site about 10 cm^2 and covered by transparent film dressing (Tegaderm Film, 3M Health-Care, USA).

In OR, the patient was sedated yet awake with eye opening to verbal commands. Non-invasive monitoring was applied for the patient (BP, electrocardiogram, pulse oximetry). BP was 105/70 mmHg, HR: 89/min, RR: 16/min and O_2 Sat: 98%. A 24G IV cannula was inserted in the left antecubital vein guided by ultrasound without complaint. 5 ml lidocaine 1% + adrenaline 1/200,000 were infiltrated over tracheostomy site by the surgeon after IV was secured with full cooperation by the patient. Cuffed tracheostomy tube size 6.0 mm tube was easily inserted. No changes in vital signs were observed during the procedure. On the following day, the cuffed tracheostomy tube was replaced by a fenestrated one; to allow the patient to speak. The mother informed the team that her daughter was highly satisfied and had no recall of the procedure.

DISCUSSION

Our patient was a candidate for surgical airway due to progressive airway obstruction that renders endotracheal intubation impossible. Awake fiber-optic intubation was an option, but the patient was uncooperative and also the tumor was invading the nose and even protruding from the mouth opening making topicalization with local anesthesia difficult. Our aim was to utilize the gastrostomy as enteral route to provide sedation and analgesia with neither respiratory depression nor airway obstruction during the procedure.

Proper dermal anesthesia by EMLA cream was achieved in our patient revealed by no changes in HR, BP or patient movements on pinching the skin by toothed forceps and during local anesthesia infiltration.

EMLA cream (eutectic mixture of lidocaine 2.5% and prilocaine 2.5%) provides dermal anesthesia/analgesia by penetrating the dermo-epidermal layers and accumulation around the skin's nerve endings. EMLA cream has been studied for pain management associated with venipuncture where it had equivalent efficacy to lidocaine infiltration and ethyl chloride spray.[1] EMLA cream significantly decreased the response associated with the painful phases of circumcision also, it offers significant pain reduction and improved patients' co-operation who underwent surgical management of molluscum contagiosum, split thickness skin grafting and superficial lymph node biopsy.[2-4]

Dexmedetomidine is a α_2 adrenoceptor agonist that is 8 times more specific than clonidine with shorter half-life. Activation of the pre- and post-synaptic α_2 receptors in the central nervous system leads to inhibition of norepinephrine release and neuronal firing. A decrease in sympathetic activity in locus coeruleus leads to sedation and hypnosis.[5] Furthermore, activation of α_2 receptors in medulla leads to bradycardia and hypotension while α_2 receptor stimulation at the supraspinal level and substantia gelatinosa in spinal cord leads to analgesia without respiratory depression.[6]

Pre-operative administration of dexmedetomidine, lead to reduction in intraoperative opioid consumption with a description of having narcotic-sparing effect.[7]

For sedation and analgesia, IV dexmedetomidine has been used successfully for patients undergoing awake fiber-optic intubation due to its unique form of sedation in which patients are co-operative and communicative. Secondly, it has analgesic and antisialagogue effects and moreover it does not affect the respiration.[8]

Zub *et al*. study concluded that oral dexmedetomidine with a mean dose of 2.5 mcg/kg was effective premedication before induction of anesthesia or procedural sedation. Parenteral separation, acceptance of the mask and insertion of IV cannula were achieved without complaint within 30-50 min.[9] Bioavailability of dexmedetomidine with an oral dose 2 mcg/kg is 15.6% with maximum serum concentration achieved at 2.2 ± 0.5 h.[10]

Ketamine is phencyclidine derivative that has analgesic effect in a wide range of pain settings. It blocks N-methyl-D-aspartate receptor with subsequent prevention of central sensitization, which plays a major role in the pathogenesis of pain.[11] Ketamine can be administered through several routes, e.g., IV, intramuscular (IM), subcutaneous, intraarticular, sublingual, oral and nasal.[12] Oral ketamine undergoes extensive the first pass metabolism by N-demethylation in the liver by cytochrome P450 system to form norketamine. Although bioavailability of oral ketamine is approximately 16%, it is associated with high serum level of norketamine, which contribute to prolonged analgesic effect after administration.[13] When ketamine administered in a racemic mixture, both the R-norketamine and S-norketamine are formed. S-norketamine is weaker than S-ketamine by 5 times.[14] Following IM or IV racemic ketamine administration, analgesia was achieved at ketamine serum level 100-200 ng/ml while after oral ketamine intake, analgesia was achieved at ketamine serum level 40 ng/ml but norketamine level was higher (200 ng/ml) after oral administration than parenteral one, which pointed to contribution of norketamine to the analgesic effect of oral ketamine.[13] Turhanoğlu *et al*. evaluated the sedative effects of different doses of orally administered ketamine (4, 6, 8 mg/kg). They concluded that sedation started after 30 min in patients, who received 4 mg/kg, but patients showed more reaction to parenteral separation, IV cannulation and mask application in comparison with patients who received 6, 8 mg/kg.[15]

Furthermore, another study concluded that oral ketamine 3 mg/kg did not produce effective sedation or lessen parenteral separation.[16] On the other hand, Sekerci *et al*. study revealed that oral ketamine 3 mg/kg was effective as 6 mg/kg in pediatric premedication with a lower incidence of side-effects as nystagmus.[17] Tobias *et al*. study concluded that oral ketamine 10 mg/kg was effective sedative and analgesic dose for children underwent pediatric oncology procedures without cardiorespiratory depression.[18] Oral ketamine 4 mg/kg combined with oral midazolam 0.4 mg/kg produced effective sedation in children compared with oral ketamine 6 mg/kg.[19] IV bolus dexmedetomidine 1 mcg/kg followed by infusion rate 0.7 mcg/kg/h combined with IV ketamine bolus 15 mg followed by infusion at rate 20 mg/h provided sedation and analgesia to the patient who required awake fiber-optic intubation. The patient did not experience bradycardia or hypotension associated with dexmedetomidine due to cardiostimulatory properties of ketamine. Also, there was no change in mental status associated with ketamine.[20] EMLA cream is recommended to be applied 45-120 min before minor procedures.[21] Elimination half-life of oral ketamine is 3 h and norketamine 4 h, which plays a major role in anesthesia induced by ketamine.[13] Maximum serum concentration after oral dexmedetomidine in a dose of 2 mcg/kg is achieved at 2.2 ± 0.5 h.[10] Hence, the timing of EMLA cream application and ketamine-dexmedetomidine administration had been chosen to be 2 h before surgery. Cardiostimulatory effects of ketamine attenuated the hypotensive and bradycardic effects commonly obtained by dexmedetomidine.

CONCLUSION

Pre-operative minimum effective enteral dose of both dexmedetomidine-ketamine provided adequate depth of sedation and analgesia with neither airway obstruction nor obtaining side-effects of both drugs.

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