Acute uvular edema after regional anesthesia and sedation: A Case Report

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ABSTRACT

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Acute uvular edema is a rare complication that can be presented in the postoperative period, usually after general anesthesia. If severe enough, it can even result in a compromised airway. In this case report, we present a case of postoperative acute uvular edema occurred in an orthopedic patient, after spinal anesthesia and infraclavicular brachial plexus nerve block combined with intravenous sedation.

INTRODUCTION

Postoperative acute uvular edema has been reported as a rare complication of endotracheal intubation[1,2] or following the use of a laryngeal mask (LMA) after general anesthesia[3]. It can be presented as an exceedingly rare cause of sore throat postoperatively, that might lead to respiratory compromise and airway obstruction. It may even also occur in the absence of endotracheal tube or LMA and in these cases it has been most frequently reported following angioedema (iatrogenic, hereditary or acquired), trauma, infections and toxic reactions[4].

Neither regional nor monitored anesthesia care is usually associated with complications of the airway. However, a few cases of postoperative uvular edema have been reported in patients after regional anesthesia with deep intravenous sedation[5,6]. This present article is a case study and discussion of a postoperative acute uvular edema, presenting after performing spinal anesthesia and infraclavicular brachial plexus nerve block (BPNB) together with intravenous sedation, in a patient undergoing orthopedic surgery.

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CASE REPORT

A written and informed consent was obtained from the patient for publication of his clinical information in a medical journal.

A 42-year-old male patient with a body mass index of 27,8 was scheduled to undergo a left tibial and left brachial fracture repair. The surgical treatment included the use of an intramedullary nail for the left tibial fracture and an open reduction internal fixation for the left brachial fracture. Patient's co-morbidities included dyslipidemia, arterial hypertension and a generalized anxiety disorder, while his medications included simvastatin, amlodipine, ramipril, nebivolol, and bromazepam (American Society of Anesthesiologist physical status II). He was a non-smoker and did drink occasionally alcohol. He reported no known drug allergies and he had not previously received anesthesia as an adult. There was no positive history of obstructive sleep apnea at the time of pre-anesthetic visit. On physical examination he had a Mallampati class 1 airway with a completely visible and normal sized uvula. His laboratory studies were unremarkable and there was no contraindication for regional anesthesia. The patient made an informed choice of regional anesthesia.

On arrival to the operating theater, an intravenous (IV) access was established and standard monitoring {electrocardiogram, noninvasive blood pressure, peripheral oxygen saturation (SpO_2)} was applied. Left tibial fracture procedure took place first, followed by the left brachial fracture procedure. So, spinal anesthesia was performed, for the intramedullary nail of left tibial fracture, using 3.6ml of hyperbaric solution of 0.5% levobupivacaine. The duration for this procedure was 130 min. Then, an infraclavicular BPNB was performed, for the open reduction internal fixation of left brachial fracture, under ultrasound guided technique, using 20ml of Ropivacaine 0.5%. This last procedure lasted 115 min. For intraoperative sedation patient received a total of 2 mg of midazolam, 100 μg of fentanyl and a propofol infusion at a rate 10 mcg/kg/minute. Perioperatively supplemental oxygen at 6 L/min via an adult-size capnometry-type oxygen mask was administrated. Teicoplanin 400mg and Amikacin 500mg were given as antibiotic prophylaxis 30 min prior to surgery. The patient received a total of 3000 ml of crystalloid fluid and his total urine output was 700 ml. The intraoperative period lasted 4 hours and 5 minutes. It must be noted that during surgery the patient was able to breathe without assistance but snored loudly throughout the procedures and did experience airway obstructions, which lasted 2-3 seconds each time. There were no episodes of oxygen desaturation as measured by pulse oximetry and also there was no need at any point to insert an oral or nasal airway.

The operation proceeded otherwise uneventfully and after its completion the patient was transferred to the post-anesthesia care unit (PACU). At this time, he complained of discomfort and a feeling of a painless mass in the back of his

throat. The patient denied dyspnea or any other complaints. There was no hoarseness of his voice and he did not experience any respiratory distress. He was afebrile and his vitals were normal: blood pressure of 130/78 mm Hg, heart rate of 86 beats/min, respiratory rate of 14 breaths/min, and an oxygen saturation of 98% on room air. Physical examination was performed which revealed an enlarged, elongated and edematous uvula. The uvula had no discharge or exudate and the remainder of the oropharyngeal tissue was normal. There was no cervical lymphadenopathy. Also, there was no evidence of a hematoma, necrosis or a generalized allergic reaction.

The patient was treated with 8 mg dexamethasone and 4 mg dimetindene intravenously. There was no response after this intervention and patient received also 2mg nebulized epinephrine and 500 mg methylprednisolone intravenously. After 30 minutes of close monitoring the uvula remained completely unchanged so 5 μg of epinephrine were administered intravenously. The patient responded shortly after this intervention and the uvular edema was completely resolved after 10 minutes. After the uvular edema had resolved the patient stayed at PACU for one hour under close monitoring. He was reevaluated and then transferred to an orthopedic ward.

On further questioning, he again denied any drug or food allergies and any use of recreational drugs. However, he did recount experiencing a similar episode about 5 years ago that it was not revealed by the patient at the time of preanesthetic visit. Back then he had consumed high amount of alcohol and fell asleep. Next morning his friends found him asleep and snoring loudly. When he woke up he had the same feeling of a painless mass in the back of his throat and he was admitted to the hospital. There he received various medications intravenously and he responded to one of them. He was unable to provide a more detailed history regarding this prior event. He did, however, mention that during that day the uvular edema reappeared twice again and both times he had to receive intravenously the same medications.

Indeed, 2 hours after his transfer to the orthopedic ward, our patient complained of having the same feeling. He was reevaluated and the reoccurrence of the uvular edema was confirmed. ENT consultation was suggested by the anesthesiologist. ENT registrar confirmed that apart from uvula the remainder oropharyngeal tissue was normal and without erythema. Phonation remained also normal, which excluded any acute pathology of the vocal cords. ENT registrar prescribed 125 mg methylprednisolone three times daily for 2 days followed by 125mg methylprednisolone twice daily for another 2 days and then 125 mg methylprednisolone once per day for 6 days. Half an hour later there was no reduction of the edema. The patient started to get distressed so another 5 μg of adrenaline were administered intravenously by the anesthesiologist under close monitoring. Few minutes later the edema had

resolved, and the patient was feeling comfortable and relaxed. However, two hours later the patient complained once again, he was reevaluated, and reappearance of the uvular edema was confirmed for a third time. The anesthesiologist administered the same dose of adrenaline and the edema resolved. The patient was under close monitoring and he did not complain again. He was feeling comfortable and relaxed for the rest of his hospital stay. He was discharged from the hospital on the 4th postoperative day.

DISCUSSION

Acute uvular edema is a rare condition and should be considered in the differential diagnosis of postoperative airway-related complications. It can be accompanied by difficulty in breathing and, since it affects the vocal cords, dysphonia, that may lead to patient discomfort and in severe cases in respiratory compromise[7]. We present a case where acute uvular edema presented after regional anesthesia without any airway manipulation or known pharyngeal trauma.

The differential diagnosis of this rare complication includes allergic reactions, infectious diseases, trauma, adverse reactions to drugs, genetic diseases and intense snoring[4].According to literature in more than half of the patients presenting with uvular edema the cause isnever identified (idiopathic uvular edema)[4,7].

Anesthesiologists must be aware and very careful during head positioning, airway manipulation and insertion of airway devices during anesthesia management in order to avoid any uvular injury[5]. In our case there was no airway manipulation (e.g. endotracheal intubation, nasal/oral airway device insertion, nasogastric tube) and no suctioning was performed, so trauma is not likely to have occurred. Also, our patient did not mention any drug allergy, so such a complication resulting from immune mechanisms does not also seem likely in this case. It is known that uvular edema is commonly associated with drug reactions. It has been clearly documented after marijuana use[8]. However, our patient denied any use of recreational drugs. Alcohol addiction can also result in edema of the uvula[5], which is not a possible cause in this case as our patient denied such an addiction. In addition, according to the literature, ACE inhibitors have been considered a predisposing factor for the development of uvular edema[5,7]. Banerji A. et al in a multicenter study noted that ACE inhibitor-induced angioedema accounts for almost one-third of angioedema treated in emergency departments[4,9]. One of our patient's medications was indeed an ACE inhibitor which might have a role as a predisposing factor in our case. Also, it is known that respiratory compromise, caused by phrenic nerve paresis, is a severe complication of interscalene BPNB and it is less often seen after an infraclavicular BPNB[10]. It is unlikely that the infraclavicular BPNB led to the complication observed in our case because edema of the uvula is not associated with this anesthetic technique.

However, it is possible that, the rest of the anesthetic management and more specifically the

sedation and the duration of surgery did contribute to the airway complication of our patient.

Also, intraoperative snoring and recurrent episodes of apneas that were noted in our patient must be considered as prominent signs of an undiagnosed obstructive sleep apnea (OSA) [5,11]. It is known that during OSA starling forces determining capillary fluid shifts facilitate the development of airway edema. Under normal conditions starling forces create a small pressure gradient that leads to fluid transudation from the capillary space to the interstitial space. Return of the interstitial fluid then ensues via lymphatic system. Interestingly, in patients with OSA highly negative pressures develop that lead to much more increased transudative forces[5,11]. In OSA patients' uvular edema could result due to synergistic respiratory depression caused by propofol when administered with midazolam and fentanyl for sedation in conjunction with a preexisting dysfunctional airway[11].

In our case, an undiagnosed OSA and the high negative mechanical pressure developed seems to be the most likely explanation for the induction of postoperative uvular edema. The administration of sedation and its duration have probably contributed to the development of this airway complication by allowing an exacerbation of airway collapse and intense snoring. The patient's medication which included ACE inhibitors also should be considered as a predisposing factor for the development of uvular edema. Any other possibility from the differential diagnosis was excluded because there was no clinical evidence available to support it.

Treatment options in acute uvular edema as mentioned in case reports include observation, iv. steroids, antihistamines, topical epinephrine administration, nebulized racemic epinephrine andhumidified air[3,7,12] It is however unclear how effective these treatment options are. It is important to note that vigilance is the key factor to avoid possible airway obstruction and respiratory compromise.

CONCLUSION

Acute uvular edema should be included in the differential diagnosis of postoperative airway complications. Anesthesiologists should be aware of the possibility of its occurrence as a result of negative pressure trauma caused by deep snoring during IV sedation. Therefore, preventive strategies should be employed concerning mainly the careful manipulation of the airway. Prompt diagnosis and management are of utmost importance in order to avoid severe adverse outcomes.

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Key words: uvular edema, spinal anesthesia, infraclavicular brachial plexus nerve block, sedation.

Author Disclosures:

Authors Serchan P, Papanikolaou D, Toliopoulou K, Papagiannopoulou P, Kostoglou Ch and Zachariadou Ch have no conflicts of interest or financial ties to disclose.

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