Complications Related to Laryngeal Mask Airway Use and a Postoperative Diagnosis of Reinke Edema: A Case Study

LCDR Danielle K. Cuevas, DNP, CRNA, NC, USN
CDR Raymond Bonds, DNP, CRNA, CHSE, NC, USN
CDR Angela Powell, MD, MC, USN

This case report describes a partial airway obstruction encountered with the use of a laryngeal mask airway and the inability to deliver adequate tidal volumes despite manipulation and device exchange. The patient later received a diagnosis of Reinke edema, a polypoid degeneration of the true vocal folds, which caused a ball-valve effect intermittently obstructing the glottic aperture with ventilation from excessive bulk and redundancy of the true vocal folds. This condition classically results in dysphonia without airway obstruction demonstrable in awake and spontaneous ventilating patients but became apparent and was readily diagnosed with initiation of positive pressure ventilation, causing an obstruction of flow. Contributing factors to this condition were the preexisting diagnosis of gastroesophageal reflux disease and cigarette smoking.

Keywords: Ball-valve obstruction, cigarette smoking, gastroesophageal reflux disease, polypoid degeneration, Reinke edema.

The use of a supraglottic airway device such as a laryngeal mask airway (LMA) is an alternative to endotracheal (ET) tube intubation for many surgical cases not requiring muscle relaxation. The LMA has many advantages over an ET tube in that LMAs are less invasive, decrease airway trauma, decrease neck mobility requirements, and have a reduced risk of laryngospasm and bronchospasm.1,2 Difficulty with ventilation can often be attributed to malposition in the oropharynx, low occlusive cuff pressure, decreased patient lung compliance, and wrong device size. Anesthesia providers often troubleshoot ventilation issues by adjusting the existing device, adding air in the cuff, and at times removal and reinsertion of a device of varying size.3 This case study will illustrate another cause for airway obstruction and difficult ventilation while using an LMA in a patient with a substantial smoking history and gastroesophageal reflux disease (GERD), in whom Reinke edema was diagnosed postoperatively.

Case Summary
A 54-year-old woman scheduled for a routine open inguinal hernia repair to be performed under general anesthesia reported the morning of the procedure for a preoperative evaluation. The patient complained of lower abdominal pain increasing with activity. Her medical history included well-controlled hypertension, GERD, obesity with a body mass index of 31 kg/m², and a 20 pack per year smoking history. Patient medications were amlodipine and ranitidine. The patient denied any history of obstructive sleep apnea but did admit to experiencing sleep-disordered breathing without apnea or frequent arousals. The patient had a large drug allergy list, which included sulfamethoxazole and trimethoprim, erythromycin, benazepril, tetracycline, penicillin, and nitrofurantoin.

The surgical history contained no anesthetic-related complications, although her last general anesthesia was more than 2 decades ago. On physical examination, the patient appeared to be well nourished, lungs were clear bilaterally, and heart sounds were normal. The patient’s airway was a Mallampati class 2, and thyromental distance was 3 finger breadths. Raspiness to the patient’s voice was appreciated and noted to be longstanding by history. The patient denied any current symptoms of GERD and could lay flat without having reflux. The patient admitted that her last cigarette was just before arriving at the hospital that morning. The vital signs were normal, and the patient had consumed nothing by mouth for 10 hours.

After premedication with 2 mg of midazolam and 50 μg of fentanyl intravenously (IV), the patient was transported to the operating room and connected to monitors with preoperative antibiotics infusing via a secondary line. Oxygen was delivered via mask at FlO₂ of 1.0 for 3 minutes before induction. Induction continued with delivery 100 mg of IV lidocaine and 200 mg of propofol followed by the inhalational agent, sevoflurane, dialed to 3%, ultimately making the patient apneic. A single-use LMA, size 4 (LMA Supreme Airway, Teleflex Medical Europe Ltd, County Westmeath, Ireland), was placed with ease, and positive pressure ventilation was per-
formed noting bilateral chest rise and end-tidal carbon dioxide (ETCO₂). The patient's lungs were auscultated, appearing to be clear bilaterally, but mild stridor was noted in the upper airway. The patient was placed on pressure support ventilation and sevoflurane for maintenance with continued stridor. After incision, the ventilator alarms noted increased peak pressures and decreased tidal volumes, and the patient triggered breaths at a rate of 14/min. The patient was given 6 puffs of albuterol through the LMA with manual positive pressure followed by a dose of narcotic and increasing sevoflurane concentration in an attempt to treat the potential bronchospasm and/or light anesthesia. The patient's lungs appeared to be clear, but the stridor in the upper airway became very audible and increased greatly from baseline. Tidal volumes decreased to less than 150 mL with a required positive pressure of greater than 20 mm Hg, and the ETCO₂ increased to 55 mm Hg. The LMA was adjusted, and manual ventilation did not improve the volumes.

The surgeon was notified of the ventilation difficulty and that an LMA exchange was to occur. The patient was given an additional 150 mg of IV propofol, and the LMA size 4 was removed and replaced with a size 5 LMA (LMA Supreme Airway) without difficulty. After a short time, the patient again started to spontaneously breathe at a rate of 15/min but was unable to take adequate tidal volumes. She was again placed on positive pressure ventilation, although tidal volumes remained below 200 mL and ETCO₂ remained above 60 mm Hg. Communication with the surgeon revealed that the surgery would be complete in 15 minutes so the decision was made to keep the LMA and manually support ventilation for the remainder of the surgery.

On emergence, a nasopharyngeal airway was inserted and the LMA was removed while assisted mask ventilation was continued as the patient transitioned to a more regular breathing pattern. In the postanesthesia recovery area, indirect laryngoscopy was performed using a flexible fiberoptic scope to assess for a possible cause for the obstructed breathing pattern, and excessive laryngeal soft tissue was appreciated. A consultation was requested with the otorhinolaryngology/ear, nose, and throat (ENT) clinic for evaluation.

Six days postoperatively, the patient was evaluated in the ENT clinic with a repeated endoscopic examination of the nares, nasopharynx, oropharynx, hypopharynx, supraglottis, and larynx to include a videostroboscopic evaluation of glottic closure pattern and mucosal wave. The results of this assessment revealed polypoid degeneration of the true vocal folds resulting in ball-valving with phonation (Figure) and moderate diffuse laryngeal edema with severe posterior glottic edema and a thick interarytenoid band. Reinke edema (polypoid degeneration of the true vocal folds) was diagnosed. The patient was given recommendations for smoking cessation, more optimal control of GERD, and endotracheal intubation for securing the airway in future surgeries involving general anesthesia should the laryngeal pathology not reverse with conservative management.

Discussion

Reinke edema is a commonly diagnosed, benign process causing abnormal voice and less commonly glottic incompetence typically presenting in patients aged 40 to 60 years and occurring more frequently in women. The etiology of Reinke edema is unknown; however, it is a possible complication of prolonged endotracheal intubation. It is defined as a pale swelling of the vocal folds filled with a fluidlike substance, making the folds very

Figure. A, Endoscopic/fiberoptic image of postglottic edema with polypoid degeneration of the true vocal folds (TVFs) bilaterally (b/l), from right to left (R>L). B, Endoscopic/fiberoptic image of Reinke edema with ball-valving with phonation.
mobile with phonation. Extracellular matrix production, subepithelial vascularization, and dilated vessels are characteristic of this condition. Sakae et al assessed 20 samples taken from patients with known Reinke edema and found that the normal intertwined network of collagen fibers resembling a wicker basket were deranged and loosely arranged. Rarely does this condition cause problems with spontaneous ventilation, although changes in vocal fold position with positive pressure ventilation can help diagnose the condition. This entity is not associated with an increased risk of malignancy.

Branski et al investigated the effects of cigarette smoke on the oxidative status and fibroblast phenotype in the vocal folds. The authors obtained tissue samples from smokers with Reinke edema and nonsmokers with benign vocal polyps and found that cigarette smoke affected the fibroblast phenotype of the vocal folds. Marcotullio et al retrospectively evaluated 125 patients with an existing diagnosis of Reinke edema, dividing them into 4 groups based on their histologic classification, then further subdivided them into categories based on the (1) number of cigarettes they smoked per day, (2) average exposure to cigarette smoke, (3) occupation, (4) habitual voice use, and (5) diagnosis of GERD. The authors concluded that there was a direct relationship between the duration of exposure to daily cigarette smoke and the severity of the histologic lesion. They also found an association with GERD in 4 of the patients; however, they admitted to the limitation of the patients with GERD having previous treatment, likely being the inconsistency from previous studies having great correlation. Prolonged vocal abuse did not prove to be noteworthy.

Treatment consists of smoking cessation and aggressive management of reflux to arrest the progression of the disease. If dysphonia is a presenting complaint, voice therapy is effective following the initiation of smoking cessation and reflux management, either alone or in combination with surgical drainage of the gelatinous material filling the Reinke space.

The patient presented in this case study is an example of how Reinke edema may cause airway obstruction during general anesthesia. The anticipated primary anesthetic concern in this case was the potential for a reactive airway due to recent smoking history, which prompted the selection of an LMA for the delivery of inhalation agent and oxygenation in an effort to decrease the risk of laryngospasm and bronchospasm. On reflection on this case, it would have been beneficial to evaluate the larynx during the procedure with a fiberoptic scope to guide the decision on whether to proceed with intubation.

In this case, collaboration between the surgeon, anesthetic staff, and ENT physician allowed the patient to be made aware of the tangible effects of her smoking and evidence of inadequately controlled GERD. This case also served as an educational opportunity for the anesthesia staff at the hospital, as some providers verbalized anecdotal experience with LMA partial obstruction and low tidal volumes that could potentially have been attributed to undiagnosed Reinke edema.

REFERENCES

AUTHORS
LCDR Danielle K. Cuevas, DNP, CRNA, NC, USN, was a student at Uniformed Services University of the Health Sciences (USUHS) in Jacksonville, Florida, at the time of writing this article. She is currently a staff CRNA at Naval Medical Center San Diego, California, and assistant adjunct professor at USUHS.

CDR Raymond Bonds, DNP, CRNA, CHSE, NC, USN, is clinical site director for USUHS at Naval Hospital Jacksonville, Jacksonville, Florida.

CDR Angela Powell, MD, MC, USN, is a staff otolaryngologist at Naval Hospital Jacksonville.

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