Transient Bilateral Vocal Cord Paralysis after Insertion of a Laryngeal Mask Airway

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A laryngeal mask airway (LMA) is used as an alternative to tracheal intubation and has been used safely in anesthetized adults and children during spontaneous breathing or controlled ventilation. Compared with an endotracheal tube, airway-related complications such as sore throat occur much less frequently with the LMA. Additionally, tracheal intubation has been followed by transient or persistent vocal cord paralysis.

To our knowledge, there has been no clinical report describing vocal cord paralysis associated with insertion of an LMA. In this case report, we describe a patient who experienced transient bilateral vocal cord paralysis following insertion of an LMA.

Case Report

A 45-year-old, 149-cm, 41-kg woman underwent elective abdominal total hysterectomy for uterine cancer. Preoperative examination disclosed no airway malformation or any sign of upper respiratory infection. She received 0.5 mg intramuscular atropine and 20 mg intravenous ranitidine 1 and 6 h, respectively, before arrival in the operating room.

Routine monitoring (blood pressure, electrocardiogram, precordial stethoscope, and pulse oximeter) was established in the operating room. She was placed in the lateral decubitus position, and a catheter was inserted and advanced cephalad approximately 4 cm into the epidural space through a 16-G Tuohy needle at the L2–L3 intervertebral space. She then was placed supine with her head in a neutral position using a donut-shaped pillow. The level of epidural anesthesia (determined by the pinprick) extended to the T5 dermatome after epidural injection of 12 ml 1.5% lidocaine with 1:200,000 epinephrine.

Thereafter, general anesthesia was induced via a face mask with 5% sevoflurane inspired in oxygen. After end-tidal sevoflurane concentration had been maintained at 4% for 5 min, anesthetic depth was judged sufficient for insertion of an LMA. We applied 2% clear viscous lidocaine jelly to the back of the LMA (size 3) and deflated its cuff, which was folded back away from the aperture before placement. The LMA was inserted using a laryngoscope to lift the tongue anteriorly without administration of neuromuscular relaxants. We did not detect airway malformation, tonsillar hypertrophy, or edema of the hypopharynx during laryngoscopy. The tip of the laryngoscope had not touched the vocal cords or the posterior wall of the pharynx during the procedure. At the first attempt, the LMA was correctly placed without the patient coughing. A good seal was obtained with the LMA after 15 ml of air was injected into the LMA cuff, but LMA intracuff pressure was not monitored during anesthesia. Air leakage around the LMA cuff occurred when inspiratory airway pressure exceeded 19 cmH2O. Neither a nasogastric tube nor an esophageal stethoscope was inserted. Anesthesia was maintained with end-tidal sevoflurane concentration of 0.5% and 67% N2O in oxygen plus epidural anesthesia. Although systolic blood pressure transiently decreased to 86 mmHg at 10 min after the epidural injection, it immediately returned to 124/61 mmHg after 5 min intravenous ephedrine. Thereafter, her systolic blood pressure and pulse oximeter readings were maintained above 102 mmHg and 98%, respectively. Blood loss was approximately 200 ml, and the operative course was uneventful.

After the end of surgery, she spontaneously breathed oxygen via the LMA for 11 min. During emergence, she did not cough, even when the airway was suctioned before removal of the LMA. Because she opened her eyes and mouth spontaneously and regained consciousness, the LMA was gently removed. The duration of LMA insertion was 97 min. Although she maintained a patent airway, she could neither utter a sound nor cough.

During visual examination of her airway was performed with a fiberoptic bronchoscope (Olympus PF Type 27 MTM, Olympus, Japan). We found signs of bilateral vocal cord paralysis with edema of the posterior wall of the hypopharynx and arytenoid region. The vocal cords were located in the intermediate position and did not move with inspiration and expiration. Neither airway distortion nor malformations were noted.

Eight minutes after removal of the LMA, she could vocalize an "Ah" with effort. Forty minutes after surgery, she complained of a
slight sore throat and could cough and utter sounds. Her family confirmed that her voice was almost the same as that before the surgery.

Discussion

As far as we know, this is the first case report showing the occurrence of transient vocal cord paralysis after the use of an LMA. Among several causes for vocal cord paralysis after tracheal intubation, the most likely one is compression of the anterior branch of the recurrent laryngeal nerve by the inflated cuff of the endotracheal tube.11–14 Another possible mechanism is hyperextension of the neck that can result in stretching and paralysis of the vagus nerve, which is anchored by the recurrent laryngeal nerve in the mediastinum.15 Other less likely mechanisms include toxic neuritis and hereditary neuropathy.12,14

It is unlikely that direct laryngoscopy before LMA insertion had caused vocal cords dysfunction in this patient, because the tip of laryngoscope touched neither the vocal cords nor the posterior wall of pharynx during the procedure. In our case, we detected erythema of the posterior wall of hypopharynx and arytenoid region with a fiberoptic bronchoscope. In addition, because she had no significant personal or family history and she was placed supine with the head in a neutral position using a donut-shaped pillow during the entire period of the surgery, it may be that this transient bilateral vocal cord paralysis resulted from compression of anterior branch of the recurrent laryngeal nerve between the inflated LMA cuff in the midline of hypopharynx and thyroid cartilage. However, from the viewpoint that restoration of normal vocal cord function occurred approximately 105 min after the application of lidocaine, it is possible that 2% viscous lidocaine jelly applied to the LMA cuff might have contributed, at least partly, to this temporary vocal cord paralysis, because viscous lidocaine jelly tends not to dissolve easily and can be effective for a prolonged period.

Furthermore, a recent report described unilateral hypoglossal nerve paralysis caused by an LMA.15 In our case, because we detected erythema in the arytenoid region, this transient bilateral vocal cord paralysis is likely to be caused by direct compression of both the arytenoid region and interarytenoid muscles by the LMA. Likewise, because nitrous oxide can diffuse across the semipermeable membrane of the LMA cuff and increase the intracuff pressure by 38% in a 30-min interval,16 such an increase in the LMA cuff pressure might result in expansion of the cuff into the hypopharynx, possibly compressing the recurrent laryngeal nerve. Nevertheless, there has been no evidence that prolonged insertion of the LMA and/or expansion of the LMA cuff damages the pharyngeal mucosa or that cuff pressure monitoring is beneficial during anesthesia.17,18

In conclusion, it may be advisable to ascertain that a patient can cough and/or speak after removal of an LMA, because these simple examinations may permit an early detection of this rare but potentially hazardous complication.

References