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CASE REPORT

Bilateral vocal cord paralysis and airway obstruction during postoperative period after partial glossectomy

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Abstract

Bilateral vocal cord paralysis is a rare complication of endotracheal intubation, causing serious problems. We report a case of bilateral vocal cord paralysis and airway obstruction in the postoperative period after partial glossectomy. A 61-year-old male patient with diabetes mellitus underwent partial glossectomy under general anesthesia. The anesthesia during surgery was uneventful. After extubation, the patient appeared to have airway obstruction and complained of dyspnea. Flexible fiberoptic laryngoscopy was performed immediately, and the patient was diagnosed with bilateral vocal cord paralysis. Therefore, reintubation was performed. The patient was extubated the following day and discharged with incomplete recovery of vocal cord paralysis. The study findings indicate that inappropriate endotracheal tube location and cuff pressure can cause bilateral vocal cord paralysis. The involvement of head position and tongue traction during glossectomy was suggested in this case. Anesthesiologists should be able to consider and manage bilateral vocal cord paralysis in patients with airway obstruction in the postoperative period.

Keywords

Airway obstruction; Extubation; Glossectomy; Vocal cord paralysis

1. Introduction

Complications of endotracheal intubation include dental damage, sore throat, upper airway injury, laryngospasm, bronchospasm and vocal cord injury [1, 2]. Vocal cord paralysis is a rare but serious complication of endotracheal intubation, particularly in cases of surgeries that do not involve the recurrent laryngeal nerves, with a reported incidence of 0.4–0.8% [3–5]. In most cases, vocal cord paralysis occurs on one side; however, bilateral vocal cord paralysis can cause catastrophic respiratory problems during awakening and postoperative care. This can result in vocal disability and aspiration [6], leading to severe postoperative discomfort. In severe cases, ventilation may be inadequate because of airway obstruction caused by unopposed vocal cord adduction [7]. We report a case of airway obstruction due to bilateral vocal cord paralysis following partial glossectomy.

2. Case presentation

A 61-year-old man, 166.7 cm in height and 69.9 kg in weight, visited the dental clinic for removal of a whitish mass on his tongue. The patient was taking medication for diabetes mellitus and dyslipidemia. The patient's blood glucose levels were well controlled. Preoperative blood tests, chest radiography and electrocardiography revealed no abnormalities. The patient had no respiratory or speech symptoms.

Pre-anesthetic vital signs were normal, and pre-oxygenation was performed at 5 L/min of 100% oxygen for 2 min. General anesthesia was intravenously induced with 60 mg lidocaine, 120 mg propofol, 50 µg fentanyl and 50 mg rocuronium. Mask ventilation was performed for 2 min, followed by nasotracheal intubation, with a 7.0-mm internal diameter cuffed preformed tube (Polar Preformed Tracheal Tube, Smiths Medical, Minneapolis, MN, USA) and the aid of a blade size 4 video laryngoscope (McGRATH^{TM} MAC Video Laryngoscope, Medtronic, Minneapolis, MN, USA). The patient's Cormack-Lehane score was evaluated as grade 2, and he was intubated without difficulty. Anesthesia was maintained using 6-vol% desflurane. During the operation, ventilation was provided with volume control mode with 450 mL tidal volume, respiratory rate of 11 breaths/min, and 5 cmH_2O of positive end-expiratory pressure (PEEP). Fraction of inspired oxygen (FiO_2) was maintained at 0.5, and the peak airway pressure was maintained at <17 cmH_2O. Partial glossectomy was completed without any specific problems. The operative time was 40 min, and 200 mg sugammadex was administered to reverse muscle relaxation. A peripheral nerve stimulator was used to monitor the train-of-four ratio (T_1/T_4), which was over 0.9, indicating sufficient neuromuscular recovery. The patient showed spontaneous respiration with a tidal volume of >300 mL, and he responded to verbal command; thus, extubation was performed. A leak test was not performed in the patient, as the intubation was performed without any difficulty.

After extubation, we noticed difficulty in ventilation with a face mask, and the patient complained of severe inspiratory dyspnea. Fortunately, the patient's oxygen saturation remained at 95–96%. A nasal airway device was inserted immediately to secure the airway. Thereafter, 5 mg dexamethasone was administered intravenously to alleviate any possible airway edema or swelling that could occur in the future. Moreover, a mixture of epinephrine (2 mg) and normal saline was nebulized to ease airway congestion. However, the patient constantly complained of dyspnea and showed signs of airway obstruction. Therefore, we suspected vocal cord paralysis and contacted an otolaryngologist to perform flexible fiberoptic laryngoscopy (ENF-P4, Olympus, Tokyo, Japan). Flexible fiberoptic laryngoscopy was performed only to examine the vocal cords; therefore, additional local anesthetics were not used. Laryngoscopy showed both vocal cords in a fixed paramedian position, with a narrow glottic opening of only 1–2 mm, and the patient was diagnosed with bilateral vocal cord paralysis.

Although the oxygen saturation remained above 95%, manually assisted ventilation with 100% Oxygen (O_2) was not sufficient for the patient. Therefore, our team decided to observe the postoperative progression of vocal cord paralysis in the intensive care unit after reintubating the patient. Nasal reintubation with 120 mg propofol and 3.0 vol% sevoflurane, without the use of any muscle relaxant, was performed using a 6.5-mm internal diameter reinforced tube (Lo-Contour Oral/Nasal Tracheal Tube Cuffed Reinforced, Murphy Eye, Medtronic, Minneapolis, MN, USA), with the aid of a blade size 4 video laryngoscope. After reintubation, oxygen saturation reached 100%, and the patient was transferred to the intensive care unit.

In the intensive care unit, the patient was mechanically ventilated in volume control mode with tidal volume of 450 mL, respiratory rate of 12 breaths/min, PEEP of 5 cmH_2O, FiO_2 maintained at 0.4, and sedation with dexmedetomidine continuous infusion. Twelve hours later, the mode was changed to continuous positive airway pressure mode with 10 cmH_2O pressure support above 5 cmH_2O PEEP. FiO_2 was set at 0.3, and the patient's oxygen saturation was maintained at 95–100%. The following morning, the patient showed clear consciousness and spontaneous respiration. Before extubation, the anesthesiologist performed a cuff leak test. Leaks were present when the tube cuff deflated; therefore, we assumed the risk of post-extubation airway obstruction was low. Thereafter, extubation was performed, and 2 L/min of 100% oxygen was administered *via* the nasal prong. Oxygen saturation was maintained at 95–100%, and the patient had no dyspnea. After 4 hours, the patient was transferred to a general ward.

An otolaryngologist conducted video-laryngoscopy 3 days after surgery, and a small erosion was identified in the left vocal cord and partially improved bilateral vocal cord paralysis (Fig. 1). The patient did not complain of any respiratory discomfort, was discharged the next day, and a short-term follow-up was planned. At the 1-month and 3-month follow-ups, paralysis of both vocal cords had not fully resolved, but the patient had no specific complaints at the outpatient clinic's follow-up (Fig. 2).

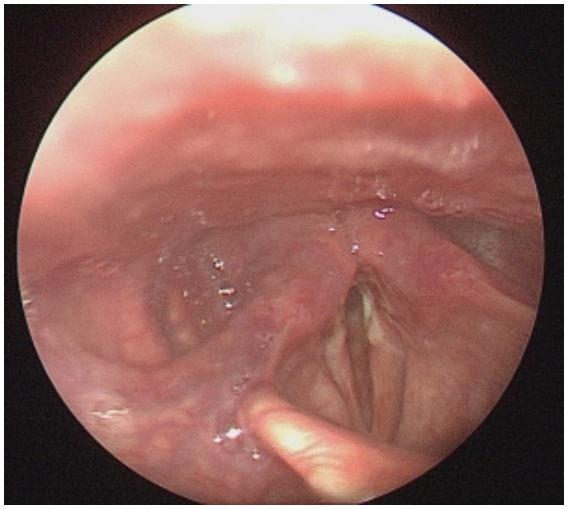

FIGURE 1. A laryngoscope image of the patient obtained 3 days after surgery. Small erosion is seen in the patient's left vocal cord, and bilateral vocal cord paralysis is partially improved.

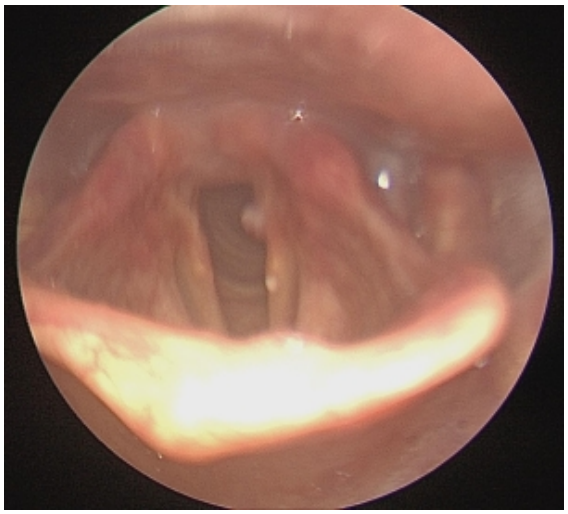

FIGURE 2. A laryngoscope image of the patient obtained 3 months after surgery. The patient's vocal cord movement is recovered and symmetric. This image was taken at the outpatient clinic's follow-up 3 months after the surgery.

3. Discussion

Vocal cord paralysis can be caused by mechanical or neurogenic injury [4, 6]. Mechanical damage, such as cricoarytenoid joint dislocation or subluxation, may result from traumatic intubation [6]. In our patient, immediate postoperative fiberoptic laryngoscopy revealed no evidence of cricoarytenoid dislocation or subluxation.

Vocal cord paralysis can also be caused by neurogenic injury to the recurrent laryngeal nerves. Neurogenic injury can be caused by surgery or less commonly, by endotracheal intubation [2, 6]. Furthermore, thyroid surgery is the most common cause of recurrent laryngeal nerve injury [4, 8]. Recurrent laryngeal nerve injuries are less commonly caused by endotracheal intubation, with the risk of vocal cord paralysis being endotracheal tube lumen size, cuff location and cuff inflation

pressure [3, 6, 8, 9].

First, the size of the endotracheal tube lumen is an important factor that can cause laryngeal damage. In particular, intubation with a tube lumen size larger than 7.5 mm is a major risk factor [6, 8]. However, in our case, nasotracheal intubation was performed using a preformed tube with an internal diameter of 7.0 mm. Consequently, it is less probable that vocal cord paralysis is attributable to the endotracheal tube size. Second, bilateral vocal cord paralysis may be related to the position during surgery. Specifically, hyperextension of the neck pulls the endotracheal tube cuff towards the vocal cords, changing the initial tube cuff location from 3.8 to 6.4 cm [4, 8, 10]. Furthermore, an endotracheal tube cuff placed at or below the vocal cords compresses the recurrent laryngeal nerve, and the ideal position of the cuff is at least 2 cm distal to the vocal cords to prevent subglottic inflation [8]. In this case, neck extension was performed by placing a roll under the patient's shoulder to facilitate surgical access. With the patient's neck hyperextended, the tube may have migrated toward the vocal cords and compressed the recurrent laryngeal nerve, causing vocal cord paralysis. Third, the recurrent laryngeal nerve compression by a hyperinflated endotracheal tube cuff may have caused vocal cord paralysis [7, 8, 11]. When an endotracheal tube with a hyperinflated cuff is placed in the larynx, the anterior branch of the recurrent laryngeal nerve is compressed between the cuff of the endotracheal tube and the posterior part of the thyroid cartilage [7, 8]. Cuffs inflated by palpation often exceed 30 mmHg, contributing to the recurrent laryngeal nerve compression and injury [8]. Therefore, verification of the initial cuff pressure using a manometer is recommended. In our patient, the tube cuff pressure was not measured accurately using a manometer. Thus, a hyperinflated tube cuff may have caused neuropraxia, resulting in immobile vocal cords.

Previous studies have reported on vocal cord paralysis after surgery of the upper limbs, thorax or abdomen, but with a small incidence [3, 4, 8]. Thus, surgery-related causes of vocal cord paralysis should be considered. Herein, we speculate that head extension and tongue traction with gauze packing to the posterior pharynx for glossectomy could injure the recurrent laryngeal nerve.

Kikura *et al.* [3] reported that old age, comorbidities, such as diabetes mellitus or hypertension, duration of tracheal intubation and tracheal tube size may increase the risk and incidence of vocal cord paralysis. Notably, the risk and incidence are three times higher in patients aged >50 years and two times higher in those with diabetes mellitus or hypertension [3]. In our case, the risk factors were old age and diabetes mellitus. Diabetes mellitus is associated with peripheral neuropathy, which makes the recurrent laryngeal nerve more susceptible to neuropraxia and increases the risk of recurrent laryngeal nerve palsy [3]. With intubation and surgery as stimuli, pre-existing neuropathy may have worsened intraoperatively. Second, patient age is a risk factor for vocal cord paralysis. However, the exact mechanism of action is not well understood. However, we can assume that as laryngeal tissues degenerate with age, they become more vulnerable to inflammation or microcirculatory insufficiency following endotracheal intubation [3].

Vocal cord paralysis can be temporary and spontaneously recover within 6 months [4, 8]. However, bilateral vocal cord paralysis can cause a reduction in the glottic area, resulting in respiratory compromise in the early onset period [12]. Therefore, anesthesiologists should manage airway obstruction due to bilateral vocal cord paralysis during the acute phase. Notably, endotracheal intubation is required in most cases. If intubation is impossible, tracheostomy is one of the most common surgical interventions for bilateral vocal cord paralysis, representing an effective, emergent, and initial method in the short term [12].

4. Conclusions

Our patient was diagnosed with bilateral vocal cord paralysis and managed with endotracheal reintubation during the postoperative period. The patient was extubated the next day and discharged without any respiratory symptoms. To prevent vocal cord paralysis, anesthesiologists should always verify factor such as cuff pressure, tube cuff location and surgical factors in head and neck surgery. If a patient complains of dyspnea in the postoperative period, vocal cord paralysis should be suspected, and prompt evaluation must be performed. We should always be ready to reintubate and prepare for tracheostomy when needed.

AVAILABILITY OF DATA AND MATERIALS

The datasets used and/or analyzed during the current study are available from the corresponding author upon reasonable request.

AUTHOR CONTRIBUTIONS

SK—examined the patient and diagnosed the case. JS and HS—wrote the first version of the manuscript. HS and SMJ—contributed to the revision and editing of the draft. SK and MAK—approved the final version of the paper and edited it. All authors contributed to editorial changes in the manuscript. All authors read and approved the final version of the manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

This study protocol was approved by the Institutional Review Board of Dankook University Hospital (IRB file no. 2023-03-027). The IRB waived the requirement for written consent for the publication of this case. This clinical case was written in compliance with the CARE guideline.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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