Transient unilateral vocal cord paralysis following endotracheal intubation in elderly patient with the abdominal surgery
–A case report–

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Vocal cord paralysis is one of the most serious complications, which, in most situations, is preventable, associated with tracheal intubation. Unilateral vocal cord paralysis following tracheal intubation usually causes hoarseness. Postoperative vocal cord paralysis may be due to mechanical or neurogenic factors. The patient complained of hoarseness one day after operation and coughing on swallowing water ten days after operation. The vocal cords were examined with a fiberoptic nasopharyngolaryngoscopy and the right vocal cord was fixed in the paramedian position. We present a case of unilateral vocal cord paralysis following endotracheal intubation in a 71-year-old male patient with descending colon carcinoma and left renal cell carcinoma. (Anesth Pain Med 2012; 7: 67~70)

Key Words: Coughing, Hoarseness, Tracheal intubation, Vocal cord paralysis.

Complications of tracheal intubation are well known and vocal cord paralysis is one of the most serious complications among them, resulting in severe vocal disability and aspiration [1]. Unilateral vocal cord paralysis following surgical procedures unrelated to the neck has been reported rare [2,3]. Because of an increase in elderly patients and pre-existing morbidities in surgical patients, patients remain in danger of vocal cord paralysis and vocal cord dysfunction despite advance in intubation techniques and devices [4]. We present a case of unilateral vocal cord paralysis ten days after abdominal surgery in a 71-year-old male patient with descending colon carcinoma and left renal cell carcinoma.

CASE REPORT

A 71-year-old man, 74 kg in weight, and 168 cm in height, entered the hospital via emergency room with a history of abdominal distension and vomiting. He was diagnosed with descending colon carcinoma and left renal cell carcinoma and admitted for left hemicolectomy and left nephrectomy. There was no significant past medical history except for lumbar discectomy thirteen years before. The preoperative physical examination, blood tests, electrocardiogram, pulmonary function test, and echocardiography were normal and airway assessment revealed no abnormalities. His chest X-ray revealed minimal pleural effusion on both side of the lung. He had a normal speaking voice without evidence of respiratory obstruction. The patient was arrived conscious in the operating room without premedication. The patient was positioned with the supine position for general anesthesia and operation. The preanesthetic vital signs were normal and peripheral oxygen saturation (SpO2) was 97% before induction of general anesthesia. Patient received 0.2 mg glycopyrrolate and 100 µg fentanyl intravenously just before induction of general anesthesia. After preoxygenation, the general anesthesia was induced with propofol 140 mg intravenously, rocuronium 50 mg was administered and the trachea was intubated without difficulty using an 8-mm cuffed endotracheal tube (Tracheal tube, Mallinckrodt Medical, Athelone, Ireland) with a No. 3 Macintosh style of fiber optic blade (fiber optic laryngoscope blades, Welch Allyn®, NY, USA). With laryngoscopy, a laryngeal view score by Cormack and Lehane was grade one in this patient. The tracheal cuff was inflated until gas failed to leak around the cuff. It was confirmed that breathing sound was equal on both sides of the lung by stethoscope. The
endotracheal tube was fixed at 22 cm from incisor and taped into place. General anesthesia was maintained with 2 vol% sevoflurane, 2 L/min O₂ and 2 L/min N₂O. Respirations were controlled by mechanical ventilator during general anesthesia. The operation was terminated uneventfully, and during the eight hour surgical procedure neither the head nor the endotracheal tube was moved. At the end of surgery, the lungs were ventilated with 100% oxygen and when spontaneous ventilation appeared adequate, the cuff was deflated and extubation was performed. The patient did not demonstrated evidence of airway obstruction and transported to the intensive care unit with stable vital signs.

The patient complained of hoarseness one day after operation and coughing on swallowing water ten days after operation. His voice remained hoarse. The vocal cords were examined with a fiberoptic nasopharyngolaryngoscopy by the otolaryngologist, who found the right vocal cord paralysis. The left vocal cord was normal and the right vocal cord was fixed in the paramedian position (Fig. 1). The vocal cords showed no edema, hematoma, or ulceration, and no sign of chemical damage. The videofluoroscopic scoring by the physiatrist revealed the pattern of penetration and delayed aspiration on liquid during pharyngeal phase. Therefore, the patient was treated with rehabilitative dysphagia therapy and vital stimulation therapy using vital stim® (PJO LCC, USA) for dysphagia rehabilitation by rehabilitation therapist. After four weeks, the vocal cords were again examined by an otolaryngologist and movement of the right vocal cord was recovered. The patient was discharged after thirty-two days when movement of both vocal cords was adequate and his voice had recovered completely and he could tolerate liquids orally.

DISCUSSION

Injuries to the airway are well-recognized complications of anesthesia. The most frequent types of laryngeal injury are vocal cord paralysis, which is one of the most serious anesthetic complications related to endotracheal intubation, and hematoma or granuloma of the vocal cords [1]. Mun et al. [5] reported that unilateral vocal cord paralysis occurred after difficult endotracheal intubation using intubating laryngeal mask airway.

Unilateral or bilateral vocal cord paralysis has been reported frequently following neck surgery, whereas unilateral vocal cord paralysis such as this case following surgical procedures unrelated to the neck and recurrent laryngeal nerve has been reported rare [2,3]. The clinical symptoms of unilateral vocal cord paralysis were hoarseness and aspiration but complete bilateral recurrent laryngeal nerve paralysis usually causes acute airway obstruction due to unopposed vocal cord adduction [6]. In this case, the clinical pictures of unilateral vocal cord paralysis were hoarseness, coughing and aspiration on liquid postoperatively. The usual cause of unilateral or bilateral vocal cord paralysis is a direct injury to one or both recurrent laryngeal nerves, either directly by severing the nerve or pressure during dissection following neck surgery or thoracotomies [2]. Therefore, we could rule out the cause of unilateral vocal cord paralysis was the surgery-related injury because of abdominal operation in this case. Yamashita et al. [7] reported nine cases of vocal cord paralysis after endotracheal anesthesia for abdominal operation like this case. In this case, we could suspect that laryngeal damage may not only occur during intubation but also be the result of some intraoperative factors.

Postoperative vocal cord paralysis may be due to mechanical or neurogenic factors. Ellis and Pallister [8] reported that when an endotracheal tube with the inflated cuff was passed within the larynx, it compressed the anterior branch of the recurrent laryngeal nerve, which passed medial to the lamina of the thyroid cartilage to supply the lateral cricoarytenoid and thyroarytenoid muscles, between the cuff and the interior surface of the thyroid lamina. Mechanical injury such as dislocation or subluxation of the cricoarytenoid joints may result from traumatic endotracheal intubation or extubation.

Fig. 1. The white arrow demonstrates the right paralyzed vocal fold, which is characteristically foreshortened, lateralized, and flaccid.
Unilateral vocal cord paralysis following tracheal intubation is associated with discernible damage to tracheal or laryngeal mucosa, or visible evidence of trauma to the arytenoid cartilage or joint. Injuries of the arytenoid cartilage or joint may be caused by excessive extension of the neck, driving the tube back against the arytenoids with or without dislocation of the cricoarytenoid joint [1]. This complication may occur if the cuff is placed at too high a level so that it lies at, or just below, the level of the cords. The need to avoid intubation of the right main bronchus or the use of too short a tube may encourage this complication [8]. In this case, intubation and extubation were performed without difficulty and we confirmed the inflated cuff was passed within the larynx during intubation and fixed the endotracheal tube at 22 cm from the incisor. Then the correct placement of the endotracheal tube was confirmed and the neck was not overextended. Also, the head and endotracheal tube were not moved during surgery. Because of no hematoma, laceration or granuloma of the laryngeal mucosa, we could rule out traumatic intubation and extubation in this case. However, a mechanical injury to the laryngeal nerve could not be excluded.

Several risk factors for vocal cord injury and postoperative hoarseness include endotracheal tube size, cuff design, and cuff pressure, and demographic factors such as sex or age [1]. Gibbin and Egginton [9] reported bilateral vocal cord paralysis following endotracheal intubation with too large endotracheal tube in relation to the subglottic region. In this case, we could rule out the cause of vocal cord paralysis was the use of the large endotracheal tube because that the trachea was intubated with ease with 8-mm cuffed endotracheal tube. Insufficient microcirculatory supply to the recurrent laryngeal nerve and its peripheral branches in the larynx due to the over-expanded endotracheal cuff pressure may cause ischemic neuronal degeneration and subsequent recurrent nerve paralysis and vocal cord immobility [4]. Irregular inflation of a cuff, due to a defect in manufacture or following many sterilizations, may also exert excessively high pressure localized to the anterior branch of the recurrent laryngeal nerve and damage from gas sterilization, like ethylene oxide, may be a cause of vocal cord paralysis if the sterilized material has not been well aerated [2,3]. The more worsening of the nerve injury could be induced by the cuff, since it diffuses rapidly into the cuff increasing cuff pressure when nitrous oxide is used during general anesthesia [3]. For preventing these problems, we used the disposable endotracheal tube with low pressure, high residual volume cuffs and eliminated the use of endotracheal tubes with cuffs that, on testing, inflated unevenly before induction of general anesthesia in this case. Also, the cuff was inflated with air to the level just necessary to prevent gas leakage but measurements of the cuff pressure were not performed. Therefore, we could not definitely exclude the possibility of a misplaced endotracheal tube, possibly combined with a hyperinflated cuff which caused the vocal cords paralysis. Kikura et al. [4] reported that the risk of vocal cord paralysis was increased in patients with old age, hypertension, or diabetes mellitus, and increased intubation time. They suggested that degenerated tissues in the laryngeal system in patients with old age are more susceptible to acute inflammation and microcirculatory insufficiency due to cuff pressure and mechanical damage by the tracheal tube. In case of unilateral vocal cord paralysis, the left vocal cord is more frequently injured than the right vocal cord because the laryngoscope is typically held in the left hand with the endotracheal tube being inserted from the right side [1,10]. Males are affected more frequently than females [10]. In this case, the patient was old aged male with no underlying disease such as hypertension or diabetes mellitus and especially, vocal cord paralysis involved right vocal cord. The other causes of recurrent laryngeal nerve damage are a toxic neuritis which may occur during influenza, diphtheria, malaria, or exposure to cold. Usually, these produce unilateral lesion [2].

In spite of known risk factors, the cause of the vocal cord paralysis remains unknown in some cases. Faabrog-Anderson [11] reported recurrent laryngeal paralysis of unknown etiology. Salem et al. [12] reported a number of cases of unilateral and bilateral vocal cord paralysis following the use of non-cuffed tubes in pediatric patients. Vocal cord paralysis after spinal anesthesia was reported [10]. The diagnosis is relatively easy and is obtained by direct laryngoscopy and rigid or flexible bronchoscopy and tracheography, if necessary. The movement of the vocal cords is observed by a small, flexible fiberoptic bronchoscope. With a complete unilateral paralysis of the recurrent laryngeal nerve, abductor and adductor fibers are affected and the vocal cord assumes a paramedian position with the vocal cord neither abducted nor adducted. In this situation, on phonation the unaffected vocal cord crosses the midline to meet the affected vocal cord, and on inspiration the unaffected vocal cord moves to full abduction [13]. In this case, we also suspected a complete unilateral paralysis of the recurrent laryngeal nerve due to a paramedian position with the right vocal cord neither abducted nor adducted.
Vocal cord paralysis recovers spontaneously under most circumstances. The duration of recovery varies from days to months [14]. Wason et al. [6] reported the symptoms of hoarseness and coughing on swallowing subsided in about six weeks without any active intervention. Mostly, the unilateral vocal cord paralysis returns to normal within three months and sometimes, the voice returns to normal within the first year after damage in some cases [3]. Therefore, the initial treatment includes observation, and if indicated, speech therapy. But routine tracheal assessment should be performed 4-6 weeks after extubation for every patients exposed to factors predisposing to laryngotracheal injury [15]. Whereas, if bilateral vocal cord paralysis is diagnosed, tracheostomy or transient endotracheal intubation would be the correct treatment despite the possibility of quick recovery. In this case, the symptoms of hoarseness and coughing on swallowing subsided in about six weeks with rehabilitative dysphagia therapy and vital stimulation therapy for dysphagia rehabilitation.

In conclusion, unilateral or bilateral vocal cord paralysis following endotracheal intubation is rare but the most serious complications in patients with surgical procedures unrelated to the neck. In spite of the known risk factors of the vocal cord paralysis, the cause of the vocal cord paralysis remains undetermined such as this case. For preventing the vocal cord paralysis following endotracheal intubation, we have to bear in mind that avoiding traumatic intubation, adequate positioning of endotracheal tube, eliminating the use of endotracheal tubes with uneven inflated cuffs, preventing tube migration and monitoring and adjusting cuff pressure regularly during general anesthesia, especially if nitrous oxide is used.

**REFERENCES**