



Bilateral vocal cord palsy after a posterior cervical laminoplasty

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Abstract

Purpose To report a patient with bilateral vocal cord palsy following cervical laminoplasty, who survived following a tracheotomy and intensive respiratory care.

Methods Acute respiratory distress is a fatal complication of cervical spinal surgery. The incidence of bilateral vocal cord palsy after posterior cervical decompression surgery is extremely rare. The authors report a 71-year-old woman who suffered from cervical myelopathy due to ossification of the posterior longitudinal ligament. Open-door laminoplasty from C2 to C6 and laminectomy of C1 were performed. Following surgery, extubation was successfully conducted. Acute-onset dysphagia and stridor had occurred 2 h following extubation. A postoperative fiber optic laryngoscope revealed bilateral vocal cord palsy. After a tracheotomy and intensive respiratory care, she had completely recovered 2 months after surgery.

Discussion One potential cause of this pathology was an intraoperative hyper-flexed neck position, which likely induced mechanical impingement of the larynx, resulting in swelling and edema of the vocal cords and recurrent laryngeal nerve paresis. Direct trauma of the vocal cords during intubation and extubation could have also induced vocal cord paralysis.

Conclusions We reported a case of bilateral vocal cord palsy associated with posterior cervical laminoplasty. Airway complications following posterior spinal surgery are rare, but they do occur; therefore, spine surgeons should be aware of them and take necessary precautions against intraoperative neck position, intubation technique, even positioning of the intratracheal tube.

Keywords Vocal cord palsy · Surgery · Respiratory distress · Cervical laminoplasty · Complication

Introduction

Acute respiratory failure due to postoperative hematoma at the anterior aspect of the cervical spine, edema of the larynx, and recurrent laryngeal nerve paralysis is well-known complication of anterior cervical discectomy and fusion [1–6]. Unilateral vocal cord palsy is generally caused by traumatic laryngeal nerve injury during an anterior surgical approach to the cervical spine [1–8]. The incidence of temporary unilateral vocal cord palsy following anterior spine surgery is 2–7%, whereas permanent palsy is 1–3.5% [9]. Clinically,

imperceptible injury to the recurrent laryngeal nerve occurs more frequently; an incidence of 24.2% was reported in one prospective study [10].

Bilateral vocal cord palsy is a life-threatening complication of thyroid or cervical spine surgery, endotracheal intubation, neck trauma, and neuromuscular disease [11–14]. The incidence of bilateral vocal cord palsy after anterior spinal surgery is rare; only three cases have been described in previous reports [2, 15, 16]. On the other hand, the incidence of bilateral vocal cord palsy following posterior cervical surgery is even more rare.

Here, we report a rare case of bilateral vocal cord palsy following posterior decompression surgery of the cervical spine, one of the most common procedures performed by spine surgeons.

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Case report

Presentation

A 71-year-old woman (height 5.0 feet, weight 136 lbs, BMI 26.5) complained of numbness in her upper and lower extremities, loss of manual dexterity, spastic gait, and bowel and bladder dysfunction.

No muscle weakness or sensory disturbance was observed; however, deep tendon reflexes were significantly increased. She had a medical history of hypertension. The Japanese Orthopedic Association (JOA) score for cervical myelopathy was 15 points (maximum 17 points). Radiographic findings did not show any apparent spinal instability at any level, but magnetic resonance imaging (MRI) revealed multilevel spinal stenosis from C1 to C6 without high intramedullary signal in the spinal cord (Fig. 1a). Spinal canal stenosis due to ossification of the posterior longitudinal ligament (OPLL) was seen in the CT image.

Surgical procedures and perioperative status

We were to schedule an elective surgery consisting of French, open-door laminoplasty from C2 to C6. While OPLL aggressively spread to dorsal aspect of the dens, further growth of the ossification which would cause recurrent symptoms was concerned. Therefore, C1 laminectomy was also planned in this case. As it was a routine posterior cervical surgery, her preoperative laryngeal condition was not assessed by an otolaryngologist. Anesthesia was induced with propofol, remifentanyl, and fentanyl,

followed by induction of muscle paralysis with rocuronium bromide. She was successfully intubated using an airway scope, and the spiral tube was fixed on the right corner of her mouth. (The inner diameter of the tube was 7.0 mm.) The amount of cuff air of the tube was a standard volume of 5 ml. Anesthesia was maintained with remifentanyl and a sevoflurane/air/N₂O mixture, with the occasional use of fentanyl and rocuronium.

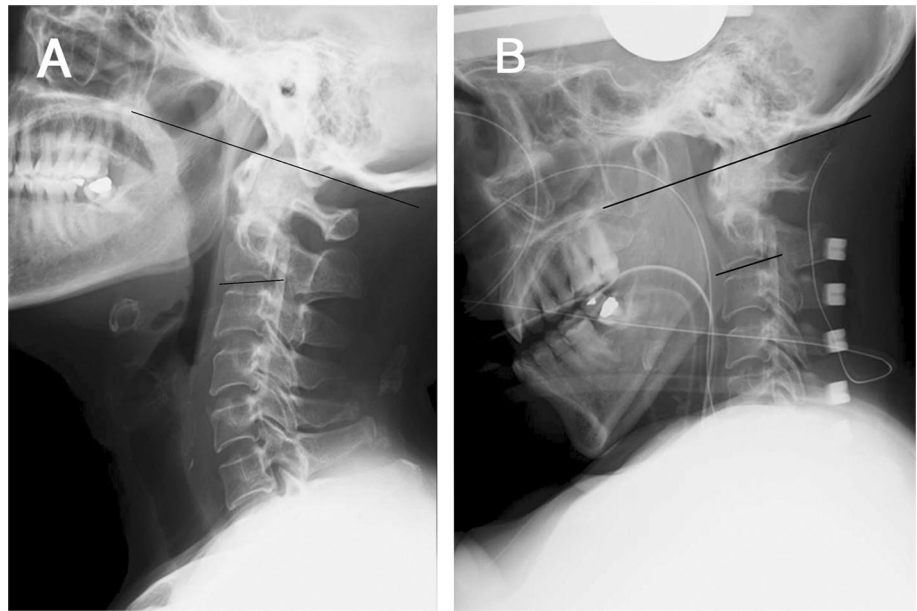
A three-pin head holder was applied to the patient's head, and she was placed in the prone position by medical staff. The surgeon moved her neck to the proper position, with mild flexion and pulling the chin. After settlement of the neck position, the airway pressure did not increase. Retrospectively, the intraoperative occipital-C2 (O-C2) angle was 10° greater than the preoperative O-C2 angle in a neutral position (Fig. 2a, b). In the end, the patient's intraoperative neck position was in hyper-flexion with retrusion of the mandible. The C1 laminectomy was accomplished for OPLL in the upper cervical spine. Following the laminectomy, a French-door laminoplasty (C2-6) was conducted, consisting of enlargement of the spinal canal by a median opening of the spinous process using artificial bone blocks, without any intraoperative complications (Fig. 1b). Continuous intravenous fentanyl was administered for postoperative pain control.

The total anesthesia and operation time were 277 and 186 min, respectively. The amount of estimated intraoperative blood loss was 180 ml. The patient had an uneventful extubation following confirmation of full recovery of respiration and consciousness, positive cuff leak test, and voluntary movement following our instruction.

Fig. 1 **a** Preoperative MRI (sagittal plane) showing multilevel spinal stenosis from C1 to C6 due to ossification of the posterior longitudinal ligament, and **b** postoperative MRI showing effective decompression of the spinal cord



Fig. 2 Cervical lateral radiographs showing the O-C2 angle (black lines) **a** before (standing position) and **b** during surgery (prone position). The O-C2 angle is 19° at a preoperative standing position (**a**) and -8° intraoperatively (**b**)



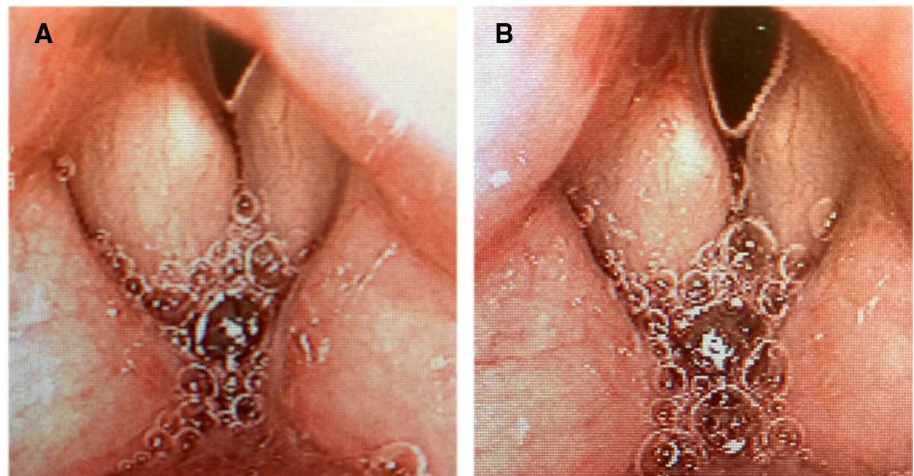
Postoperative course

After the surgery, the patient was transferred to the general ward. She began to complain of acute dysphagia and stridor 2 h after extubation, and her oxygen saturation of the peripheral artery (SpO_2) gradually decreased. A rigid fiber optic laryngoscope found no deviation of the tongue and pharynx, but remarkable swelling and absent movement of the bilateral vocal cords, which were resting in the center of the larynx (Fig. 3a, b). According to Bernoulli's principle, it represented paradoxical vocal cord movement, which closed at inspiration and opened at expiration. As an emergent life-saving measure, re-intubation was performed immediately after the diagnosis of vocal cord palsy. In addition, hydrocortisone was administered to reduce the edema of the vocal cords.

The patient was artificially ventilated in the intensive care unit (ICU) for several days to recover vocal cord function. There was no apparent motor dysfunction and no deterioration of other neurological symptoms in her extremities. In addition, there was no evidence of surgical neurological complications, such as intraoperative iatrogenic spinal cord injury, dural injury, or leakage of cerebrospinal fluid. No evidence of an acute disorder of the central nervous system, such as intracranial hemorrhage or infarction, was observed. Shy-Drager syndrome was also ruled out by a neurologist.

Using a fiber optic laryngoscope, recovery of vocal cord function was not observed 10 days after surgery. A tracheostomy was performed 21 days postoperatively, and a nasogastric feeding tube was inserted to reduce the risk of aspiration pneumonia. Artificial ventilator support was removed. A tracheal cannula was changed to a speech cannula 28 days

Fig. 3 Laryngoscopic findings 2 h after extubation during the **a** inspiration and **b** expiration phase. Vocal cord swelling and paradoxical vocal cord movement (closed at inspiration and opened at expiration) are observed



postoperatively, and then, transoral intake commenced. It took 6 weeks following surgery for vocal cord movement to begin to improve (Fig. 4a, b). Two months after surgery, vocal cord function was completely reinstated (Fig. 5a, b). Early intervention with a speech therapist for rehabilitation helped restore swallowing and respiratory ability. The intratracheal tube was removed 2 months after the surgery, and the patient was discharged from the hospital 3 months postoperatively.

Discussion

Acute respiratory distress is one of the fatal complications in cervical spinal surgery. While some reports describe acute respiratory failure caused by postoperative edema of the larynx or anterior cervical hematoma [1, 3, 4], other authors have reported vocal cord palsy due to recurrent laryngeal nerve palsy after anterior cervical surgery [2, 5, 7, 15, 16]. In contrast, vocal cord palsy following posterior cervical surgery is extremely rare; there are only a few previous reports

in the literature. The mechanisms of this rare complication are still unclear. The larynx and vocal cords are innervated by the recurrent laryngeal nerve, a branch of the vagal nerve, downstream from the pharyngeal nerve; thus, vocal cord palsy could occur through bilateral recurrent laryngeal nerve paresis [17].

Gokaslan et al. [18] retrospectively analyzed 1345 patients who received a cervical spinal surgery and found 19 cases (1.4%) with recurrent laryngeal nerve palsy. In their report, most cases of vocal cord palsy occurred following anterior cervical surgery; however, one case occurred after posterior cervical spinal surgery, though patient details were not clearly described [18]. Bekelis et al. reported a rare case of severe neurogenic dysphagia and bilateral vocal cord paresis following a posterior C1–C3 fusion surgery. The authors concluded that the bilateral vocal cord palsy in their patient might be related to a transient dysfunction of the vagal nerves due to trauma of the trunk between the C1 and jugular foramen by the tip of bicortically inserted lateral mass C1 screws [17]. In contrast, in our patient, no instrumentation was inserted

Fig. 4 Laryngoscope findings 6 weeks after the surgery during the **a** inspiration and **b** expiration phase. The patient recovers from paradoxical vocal cord movement, while swelling of the vocal cord is still observed

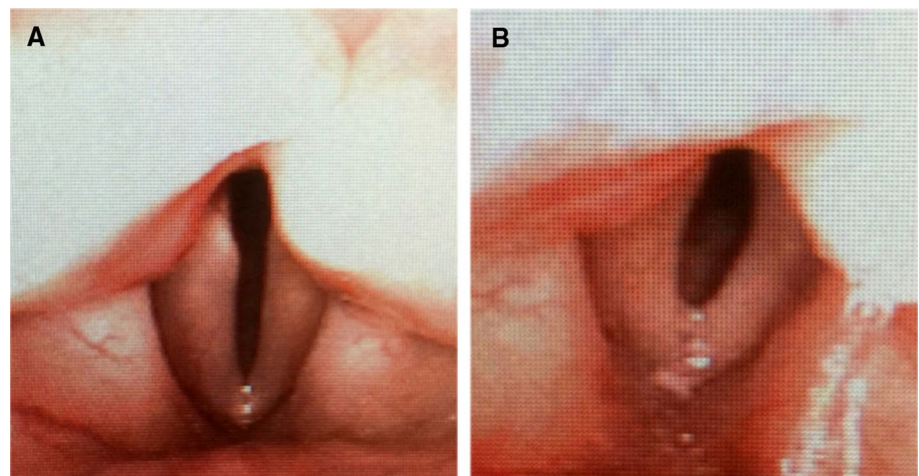
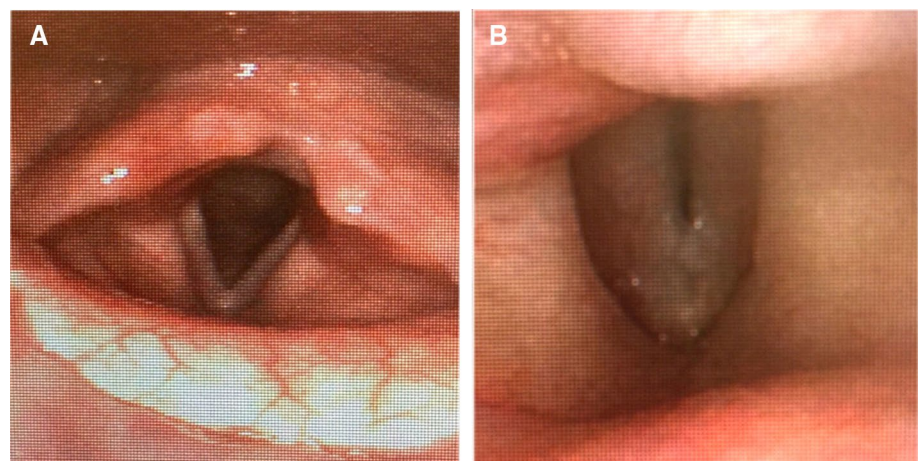


Fig. 5 Laryngoscopic findings 2 months after the surgery at the **a** inspiration and **b** expiration phase. Normal vocal cord movement is regained



in the C1 lateral mass, and there was no surgical invasion of the jugular foramen. Therefore, different mechanisms should be considered when determining potential causes of vocal cord palsy in the present case.

We hypothesized that one of the potential causes of our patient's pathology was an excessively flexed neck position intraoperatively. Park et al. [19] reported a case of Tapia's syndrome, vocal cord palsy accompanied by hypoglossal nerve palsy, following posterior cervical foraminotomy and discectomy. They concluded that the potential cause of this pathology was an excessively flexed neck position during surgery. When the neck is flexed excessively, the ramus of the mandible moves closer to the cervical spine. In this situation, the laryngeal nerve may be entrapped in the offending endotracheal tube, the ramus of the mandible, and the transverse process of the cervical vertebra [19]. Silva et al. [20] also reported a similar case of a patient who suffered from Tapia's syndrome following a posterior cervical foraminotomy by a similar mechanism. In our present case, according to an intraoperative lateral radiograph, the endotracheal tube steeply curved due to the excessively flexed neck position (Fig. 2b), which might indicate mechanical entrapment of the laryngeal space.

On the other hand, there are some reports that describe the relevancy of the neck posture to airway space. Ota et al. [21] demonstrated the relationship between the diameter of the airway space and the postoperative O-C2 angle in posterior cervical fusion surgery. They have shown that a 10° decrease in the O-C2 angle causes a 37% reduction in oropharyngeal airway space. Thus, the postoperative O-C2 angle should not be less than 10° compared to the neutral preoperative position. In our present case, the O-C2 angle of the intraoperative neck posture was -8°, 27° less than her neutral standing position (Fig. 2a, b). Therefore, this decreased airway space may have induced mechanical impingement by the intratracheal tube, resulting in swelling and edema of the larynx. However, the standard and ideal neck position during posterior cervical surgery is still unclear [22]. Kim et al. [23] strongly recommended that the patient's cervical spine be maintained in a neutral or slightly extended position. This suggestion is compatible with the prevention of airway impingement in the case of posterior cervical surgery in the prone position. In our present case, a lateral radiograph of the neck should have been taken prior to the surgery following stabilization of the head to the operating table in order to determine whether the neck posture was acceptable. This is an easy way to verify the intraoperative cervical alignment and one of the most essential measures in order to prevent airway complications. Most medical facilities use a chin-tucked position for posterior cervical spinal surgery, and there are only a few reports of vocal cord palsy following these surgeries. Therefore, many factors may contribute to this pathology.

The other potential mechanism leading to bilateral vocal cord palsy is direct trauma to the vocal cords during intubation, extubation, or intraoperative compression by the tube or air-cuff of the tube. Direct stimulation of the vocal cord by the intratracheal tube and cuff during intubation and extubation procedures may be a factor in inducing vocal cord edema. A few reports proposed a similar mechanism induced by direct surgical trauma, nerve division or ligature, pressure- or stretch-induced neuropraxia, and postoperative edema [8, 24]. Direct nerve injury by an unrecognized and overinflated, "high-riding" air-cuff is also reported as a cause of postoperative swelling of the larynx [11]. A gentle technique of intubation and extubation may be an essential measure to prevent traumatic edema of the vocal cords [12]. Furthermore, there may be slight migration of the intratracheal tube from its original position associated with preoperative posture change from a supine to prone position. Thus, the migrated intratracheal tube or air-cuff may impinge on tissue around the vocal cords. An intraoperative radiographic scan can verify the position of the tracheal tube and allow for strict control of the tube position.

Airway complications following spinal surgery are rare, but can occur even in cases of posterior cervical surgery, and should be a known complication among spine surgeons.

We reported a case of bilateral vocal cord palsy associated with posterior cervical laminoplasty. This complication is extremely rare, but can occur. Particular attention should be paid to the intraoperative neck position in order to avoid hyper-flexion of the neck and retrusion of the mandible. A gentle intubation technique and appropriate positioning of the intratracheal tube are also essential.

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Compliance with ethical standards

Conflict of interest The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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