Bilateral Vocal Cord Paralysis after Anterior Cervical Discectomy Following Cervical Spine Injury: A Case Report

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ABSTRACT

Bilateral vocal cord paralysis is a rare and preventable complication of anterior cervical discectomy and fusion. Herein, we report a fatal case of bilateral vocal cord paralysis after anterior cervical discectomy and fusion (ACD/F). A 65-year-old man with cervical spine trauma and anterior cord syndrome, following car overturn presented to our emergency department. The patient had C6-T10 prolapsed discs for which ACD/F was performed. In the recovery room he developed stridor and respiratory distress immediately after extubation, and was reintubated. Otolaryngological evaluation revealed bilateral vocal cord paralysis. He later required a tracheostomy but finally died in a rehabilitation center after an acute coronary event. Awake fibroptic intubation is recommended in patients at high risk for preoperative recurrent laryngeal nerve injury. Intraoperative tracheal tube cuff pressure monitoring and modification of surgical approach to neck are recommended to prevent bilateral nerve damage.

Keywords: Anterior cervical discectomy; Anterior cervical fusion; Cervical spine trauma; Vocal cord paralysis


Introduction

Anterior cervical discectomy with fusion (ACD/F) is now a common spinal surgical procedure all over the world. In earlier studies the unilateral recurrent laryngeal nerve palsy (RLNP) was reported to be the most frequent nerve complication during an anterior approach to the cervical spine [1-3]. The incidence of temporary unilateral vocal cord paralysis (UVCP) after this surgery is 2 to 7%. An incidence of 24.2% was reported in one prospective study of clinically inapparent injury [4]. The incidence of permanent unilateral vocal cord paralysis is 1 to 3.5% [5]. It has been pointed out that RLNP is infrequently reported [6]. Bilateral vocal cord paralysis after ACD/F is extremely rare and only three cases of bilateral vocal cord palsy (BVCP) have been described in the English literature [7-9]. Herein, we report a 65 year old man who sustained a cervical neck injury due to car overturn, and developed a bilateral vocal cord paralysis after ACD/F.

Case Report

A 65-year-old man was transferred to the emergency...
department of our center with cervical neck trauma following car overturn. Radiographies revealed cervical spine dislocation at C6-T1 and thus he was scheduled for urgent ACD/F. He had anterior cord syndrome with paraparesis and muscle power of 1/5 without any significant medical co-morbidity except for chronic hypertension. After establishing intravenous line, he was pretreated with midazolam 2 mg, and sufentanyl 10 μg. Anesthesia was induced with sodium thiopenthal 250 mg and propofol 100 mg, and after muscle paralysis with succinylcholine 100 mg, he was intubated with direct laryngoscopy and manual inline fixation using size 8 tube inserted to 23 cm. Anesthesia was maintained with Isoflurane/air/NO\textsubscript{2} mixture. Anterior approach at the right side of the neck was chosen and an oblique surgical incision was made to perform Discectomy at C6-7 and C7-T1 with anterior interbody cage insertion. A general surgeon was requested for help during the exploration of the lower neck. Finally anterior fixation at C6-10 level was accomplished with plaque under fluoroscopy.

The patient was taken to recovery room after operation and extubated while he was awake. He immediately developed acute respiratory distress with post-extubation inspiratory stridor. The problem was initially handled with jaw trust while cervical collar was in place. Intravenous hydrocortisone 200 mg was administered with possible diagnosis of post-extubation laryngeal edema. After 30 minutes of conservative management there was no improvement in stridor and patient was reintubated with sedation and topical anesthesia with the cervical collar still in its place. The patient was then transported to ICU. He was on continuous positive airway pressure (CPAP) overnight while receiving sedation with propofol and fentanyl infusion. His neurologic state was the same as before operation with paraparesis and muscle power of 1/5. The patient was extubated in the early morning after a positive cuff leak test. However the inspiratory stridor and respiratory distress continued after the extubation. Respiratory distress was not improved after managing the patient with jaw trust for about half an hour. An otolaryngologist was consulted who recommended reintubation and patient’s evaluation under direct laryngoscopy. The patient showed bilateral paramedian positioning of both vocal cords on direct laryngoscopy under general anesthesia. The patient was reintubated and transferred to ICU and trachiostomy was done two days later. On the following days, the patient developed progressive paraparesis and became paraplegic. He was managed conservatively with high dose methylprednisolone which was not effective. During ICU stay several attempts were made to block the trachiostomy which was not successful. He was then referred to a rehabilitation center but unfortunately died due to acute myocardial infarction after several days.

**Discussion**

UVCI is a known neurological complication of ACD [10,11]. However BVCP is an extremely rare complication of ACD with only three cases reported so far [7-9]. In the first case, the patient had a history of cardiac surgery that could have caused a silent UVCP [7]. The second was due to a whiplash injury alone or in combination with the very extensive procedure, which could explain the bilateral involvement [8]. The third patient, scheduled for ACD for C 6-7 herniated discs, developed BVCP with no indication of preoperative UVCP [9]. This is the second case report of BVCP following ACD/F after cervical neck trauma.

Some proposed mechanisms of this surgical complication includes direct surgical trauma, nerve division or ligature, pressure or stretch induced neuropraxia, and postoperative edema [12,13]. The UVCI occurs mostly on the right vocal cord due to the fact that the right approach is the preferred procedure to avoid injury to the thoracic duct, which resides on the left side [12,13]. Duration of surgery, multilevel exposure, and low-level exposure were also found to be associated with higher risk of UVCP [1]. Right RLN/P can be explained in our case considering right-sided approach, dislocation at the level of 6-T10, and prolonged surgery. The etiology of left vocal cord palsy in our case, however, remains elusive. Although rare, we assume that the injury to the left recurrent laryngeal nerve occurred after progressive spread of the retractor during exposure of the spine at the C7-T1 level. Considering the prolonged surgery and the problems associated with neck exploration as evidenced by the need to a general surgeon for better exploration, the left RLN may have been confused or excessively stretched along its course within the tracheoesophageal groove. Laryngeal nerve injury can be minimized if tissues are handled gently to avoid unduly vigorous retraction of the carotid sheath, larynx, and trachea. The surgeon should also ensure that the left-sided retractor teeth are placed into the muscles and not in the esophagus. Tension on the retractors should be released intermittently when they are not actually required during the procedure.

Another explanation for our BVCP is the endotracheal tube, which has been shown to lead to UVCP or BVCI [14,15]. Vocal cord paralysis (VCP), as a complication of endotracheal intubation (ETI), was reported to be 10-15% of all VCP causes. Although left vocal cord is most commonly affected, cases of BVCI have been described from ETI [14]. Apfelbaum et al. suggested
that injury is the result of nerve compression. They argue that the tube is tethered proximally at its point of fixation to the face and distally by its inflated cuff [1]. Insertion of the surgical retractor causes a marked lateral bowing of the tube, which they demonstrated radiographically in fresh cadavers. Presumably, any structures caught between the tube and the retractors are compressed. Deflating the cuff for a few seconds lessens its curvature and allows its shaft to move away from the tracheal wall. In this way, compression of any intervening tissue is alleviated. Another possibility is direct nerve compression by an unrecognized and overinflated, high-riding cuff [16,17]. We used digital palpation for adjusting intra-cuff pressure and deflating followed by its re-inflating during the retractor application could help prevent VCP [12].

Undiagnosed ULVCP before ACD/F is another possibility which may explain BVCP in our case. Manski et al. in the first reported case of BVCP after ACD/F, observed an undiagnosed UVCP which was due to previous cardiac surgery [7]. Although our patient did not exhibit any symptoms of vocal cord paralysis before surgery, the voice appeared almost normal when the paralyzed cord was near the midline (paramedian) [18]. We presume that our patient could have had an unrecognized preexisting UVCP which might have been due to neck trauma. Unilateral vocal cord paralysis after neck trauma has already been reported [19]. Bachar et al. reported the first case of BVCP after cervical neck trauma [9]. Our patient is the second reported case of BVCP following neck trauma. It would be rather impractical to submit every patient with a proposed anterior cervical disectomy to a thorough laryngoscopic examination, but would be quite possible to do so, at least on a limited basis for people with a strong history of trauma or previous thoracic intervention. From a more practical point of view, it would be advisable to recommend that these people be intubated using fibroptic laryngoscopy. If there is any evidence of malfunction, it would be appropriate to consider ipsilateral intervention.

We recommend awake fibroptic intubation in patients at high risk for preoperative recurrent laryngeal nerve injury. Intraoperative tracheal tube cuff pressure monitoring and modification of surgical approach to neck are recommended to prevent bilateral nerve damage.

Conflict of Interest: None declared.

References