Case Report

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Bilateral vocal cord injury following anterior cervical discectomy: could a better preoperative exam have prevented it?

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ABSTRACT

We present a rare case of bilateral vocal cord injury (BVCI) following anterior cervical discectomy with fusion (ACD/F) in a 47 year old man. The patient experienced post-extubation stridor and whispering voice in the recovery room. Clinical assessment led to the diagnosis of BVCI. The patient was treated by tracheostomy and made a full recovery. What is unique about this case is that the patient had no reason for a preexisting unilateral vocal cord injury (UVCI) prior to this surgery. There have been only two similar cases in the English literature in which the patients had a preexisting unilateral vocal cord paralysis (UVCI). We recommend a more detailed preoperative airway exam to include a voice exam with specific voice fatigue questioning on all patients coming for ACD/F. Such detailed assessment may uncover hidden UVCI and allow a safer perioperative period.

INTRODUCTION

It is well known that UVCI could happen post ACD/F [1-7]. An incidence of 24.2% has been reported in one prospective study when including the clinically unapparent injury [5]. Only two cases of bilateral vocal cord injury (BVCI)

have been described in the English literature. One was after a whiplash injury which, by itself or in combination with the very extensive procedure, could explain the bilateral involvement. In the other case the patient has a history of cardiac surgery that could have been the cause of a silent UVCI [1,8]. We are presenting a case of BVCI with no indication of preoperative UVCI in order to alert practitioners to this possibility.

CASE REPORT

A 46 year old male with a history of smoking, hypertension and alcoholism with no significant surgical history or other medical problems scheduled for ACD for C 6-7 herniated discs. After establishing intravenous access and applying standard monitors, the patient was pre-medicated with midazolam 2 mg and robinol 0.2 mg. After a dose of 0.5 mg vecuronium, anesthetic induction was successfully achieved using fentanyl 250 mcg, thiopental/propofol, 140 mg/70mg and succinylcholine 100 mg. Atraumatic endotracheal intubation (ETI) was accomplished with size 8 tube inserted to 23 cm and secured at the lips uneventfully. The surgery was preformed to the right side using a microscope for the dissection followed by the graft placement under fluoroscopy. Anesthesia was maintained with desflorane/air/O2 mixture. As the patient was awakened at the end of the surgery, the endotracheal tube was removed and the patient was taken to the recovery room in a stable condition with no abnormal neurological signs and oxygen saturation (SpO2) of 99% on 3L/m nasal oxygen.

After thirty minutes, he developed wheezing and the SpO2 dropped to 96%. He received inhalation treatment of Albuterol (2.5mg). Hematoma was not noted at the operative site. Over the next 30 minutes the patient was noted to develop inspiratory stridor with sternal retraction and had a hoarse/whispering voice. 0.5 ml of racemic epinephrine (2.25%) was administered by inhalation as well as 100 mg of intravenous hydrocortisone. We entertained the possibility of vocal cord paralysis (VCP) and accordingly gave 1mg of midazolam. Chest and neck x-rays could not identify any pathology. The otolaryngologist fiberoptically visualized the upper airway and diagnosed BVCP.

Considering the risks of aspiration, the risks of trauma to the

neck if the airway had to be established urgently without proper preparation for neck protection, which may lead to paralysis from spinal cord injury, and the severity of the patient's symptoms; all these facts alone with the need for a permanent airway device for ventilation favored performing a tracheostomy.

Following the tracheostomy, the patient was discharged to his hospital room and had a stable postoperative course. Immediate postoperative evaluation could not identify any pathology for the BVCP. In a follow up visit six weeks later, it was found that the tracheostomy had been properly placed, the patient was able to eat and drink without difficulty, and he could also speak well. Laryngoscopy revealed that his vocal cords continued to be in a paramedian position; therefore, it was not considered safe to remove the tracheostomy tube at that time.

A voice analysis study (VA) indicated that his left vocal cord had some tone and some movement but his right vocal cord remained atonic. Six months later, the patient was noted to have a much stronger voice. A follow up visit for the removal of the tracheostomy tube was arranged with a local Otolaryngologist in his home town.

DISCUSSION

UVCI is a known neurological complication of ACD [2,3,4,6,7]. Some proposed mechanisms of this surgical complication includes direct surgical trauma, nerve division or ligature, pressure or stretch induced neuropraxia, and postoperative edema [2,9]. A UVCI occurs mostly on the right vocal cord due to the fact that the right approach is the preferred one to avoid injury to the thoracic duct, which resides on the left side [2,4,9]. While this would explain the recurrent laryngeal nerve injury on the right side, it would not explain the left side paralysis noted in our patient. Mark Kriskovich et al. reported a vocal cord paralysis rate of 6.4% in 250 consecutive patients undergoing ACD. That was then reduced to 1.69% in the next 650 patients [2]. This was done by deflating the endotracheal tube cuff after placement of the surgical retractors and then re-inflating the cuff to a pressure of 15 mm Hg.

When the paralyzed cord is near the midline (paramedian), the voice may appear near normal

although most of those patients may complain of voice fatigue [10]. This fact indicates that a patient may have normal voice but still have an underline injury. We hypothesize that our patient could have had an unrecognized pre-existing UVCI. Without a preoperative electromyographic study, it would be hard to determine if one of the paralyzed cords was an older injury. Another explanation for our BVCI is the endotracheal tube, which has been shown to lead to UVCP or BVCI [11,12]. Vocal cord paralysis (VCP) as a complication of ETI was reported to be 10-15% of all causes of VCP. Although it most commonly affects the left vocal cord, cases of BVCI have been described from ETI [11]. Also postoperative VCP has been reported even in patients who had a laryngeal mask airway during surgery [15-17]. It also could be manifested as late as 10 weeks after surgery [11]. Other causes that should be considered includes, but are not limited to, upper respiratory tract infection, thoracic tumor, stroke, diabetes neuropathy, and paradoxical vocal cord dysfunction [1,13,14].

Before a definitive diagnosis could be made, conservative treatment included: oxygen by mask, systemic steroids, racemic epinephrine, as well as sedation. After the diagnosis a permanent airway should be established if needed and that was our patient's treatment.

Given these occurrences, one should stay on guard for VCP postoperatively in their ACD patients. The addition of the technique of maintaining a specific cuff pressure and deflating followed by re-inflating it when the retractor is applied could be helpful in preventing VCP [2,3]. We cannot on the base of this one case report recommend a preoperative voice analysis study on every patient undergoing an ACD simply because this would not be cost effective; however, we do recommend a more detailed airway exam to include a voice exam with specific questioning about voice fatigue in order to identify patients at risk. Most importantly, we hope that this case presentation would alert the practitioners to the possibility of this rare complication after anterior cervical spinal surgeries.

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