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Delayed Right Vocal Cord Palsy Following Normal Endotracheal Extubation: Report of a Rare Case

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Abstract: ***Vocal cord paralysis is a rare but severe complication after tracheal intubation. Delayed VCP after GA with endotracheal intubation usually involve left vocal cord in approximately 70% of the cases. It is usually due to a recurrent laryngeal nerve neuropraxia, and the course is benign with spontaneous recovery in most patients. It may occur due to delayed nerve swelling, traction on the recurrent laryngeal nerve during thyroid surgery retraction on RLN, thymectomy, cardiac surgeries like PDA ligation, BT shunt and aortic aneurysm and dissection repair or neck surgery. Also, compression from postoperative hematoma, or inflammation from endotracheal intubation. The symptoms of the VCP include hoarseness of the voice and breathing difficulties and slow silent aspiration, and pneumonia. we present a case of delayed right vocal cord palsy (VCP), 48 hrs after GA with tracheal intubation in a patient who has undergone exploratory laparotomy for intestinal obstruction. After ruling out the presence of a tumour or compressive infectious complications, direct injury during cardiac and thoracic surgery and cervical spine decompression surgery, and using the exclusion criteria, the most likely mechanism was direct vocal cord compression or trauma by the endotracheal tube as intubation was performed by the anaesthesia trainee resident, or RLN compression by the overinflated tube cuff as cuff pressure was not monitored.***

Keywords: Vocal cords paralysis, Intubation intratracheal, hoarseness of voice, Respiratory distress, general anaesthesia, Case presentation

1. Introduction

Delayed right vocal cord paralysis (VCP) is an uncommon complication of general anaesthesia (GA) with endotracheal intubation after surgery or prolonged intubation in ICU. VCP could be a stressful experience for patients following anaesthesia. It may be frequently caused due to vagal or recurrent laryngeal nerve inflammation or neuropraxia, a temporary blocking of the nerve conduction, rather than permanent nerve severing. In addition, vocal cords damage might result due to arytenoid dislocation, edema or inflammation of the glottis and vocal cords. Mostly the postoperative VCP occurs due to recurrent laryngeal nerve(RLN) damage following thyroid surgery, thymectomy, carotid endarterectomy for stenosis of the carotid artery, anterior cervical spine decompression, or even after open-heart surgery, and thoracic surgery.[1,2] However, in the absence of the iatrogenic RLN injury, the mechanical damage due to forceful endotracheal intubation using larger size of tube, or over extension and rotation of the head and neck, tube placement in improper position, and local ischemic nerve damage owing to the prolonged pressure of the over inflated cuff have been proposed precipitating factors.[3] We present a 47 years old male, who underwent an exploratory laparotomy under general anaesthesia with endotracheal intubation for 4 hours duration. Following uneventful tracheal extubation, patient had normal speech, however at 48 hours of the endotracheal extubation, he developed progressive hoarseness of voice due to right VCP.

A 47 years -old- male, presented with distension abdomen and vomiting for the last 4-5 days, and diagnosed with Subacute Intestinal obstruction, and planned for emergency exploratory laparotomy under general anaesthesia. He was alcoholic for 10 years and tobacco chewer for 5 years, and suffering from diabetes mellitus for 3 years, but not on any medication. Patient had no comorbidities like hypertension, bronchial asthma, coronary artery disease (CAD), epilepsy, tuberculosis and thyroid disorder. There were no symptoms of shortness of breath, hoarseness of voice or any history of voice change.

On examination, his heart rate was 104bpm, and blood pressure was 114/84mmHg and SPO2-99% on room air. A preoperative airway assessment revealed adequate mouth opening of 3 fingers with Mallampati score of grade-2, and normal dentition, adequate neck movements without any restriction and thyromental distance was more than 6cm, suggestive of easy intubation. Cardio-respiratory and neurological examination were unremarkable. His hematological and biochemical values were with in normal limits, except high blood sugar (RBS- 293 mg/dl) and total leucocyte count of 16960 cells/cumm. The blood sugar was maintained in a range of 80-180 mg/dl with act rapid insulin infusion (2 units/hr). The resting ECG showed normal sinus rhythm with no pathological findings, and chest X-Ray was unremarkable, and an antibiotic cover with Cefuroxime and metronidazole was used for underlying bacterial infection.Anaesthesia plan was to use a balanced GA along with epidural anaesthesia and analgesia.

In OR, all standard ASA monitors including non-invasive BP, EKG, SPO2, EtCo2, and temperature probe were attached and foley's catheter was inserted for the urine output. A 20 G epidural catheter was placed through an 18 G Tuohy's needle using L3-4 space and fixed at 11cm length, and morphine 3mg diluted in 5 ml saline was administered for perioperative analgesia. Induction of anaesthesia was performed using fentanyl (100 µg), Propofol (130 mg), midazolam (2mg), and Vecuronium (8 mg) was used as a muscle relaxant to facilitate the endotracheal intubation. He was easily intubated in first

attempt with 8mm.ID, cuffed ETT. The cuff was inflated with 6 ml of air and tube was fixed at 21 cm after confirming proper placement by chest auscultation and capnography. Anaesthesia was maintained with sevoflurane (1-2 MAC) in oxygen with air using FIO2 of 0.6, intermittent boluses of vecuronium (2mg/hr), and epidural morphine was also used for analgesia. Patient remained hemodynamically stable throughout the procedure. On abdominal opening, two intestinal bands were identified and same released and intestinal continuity was re-established. The surgery was completed smoothly. Tracheal extubation was achieved easily following reversal of neuromuscular block with neostigmine and glycopyrrolate (3.0 mg:0.6 mg). Surgery lasted approximately 4hrs and 30min which itself is a precipitating risk factor for vocal cord paralysis. Patient was speaking normally in the postoperative room. However, at 48 hours after the surgery, we received information that the patient had hoarseness of voice without dysphagia, and so ENT surgeon opinion was obtained, and on indirect laryngoscopy the right vocal cords were found to be fixed. [Figure 1, video-1] CECT Neck revealed mild dilatation of right sided vallecula and pyriform sinus with mild fullness and medial displacement of right aryepiglottic fold and right sided vocal cord, and likely right sided vocal cord palsy. He was put on nebulization and speech therapy and wait and see approach was followed and discharged from the hospital on 5th day of the operation with the advice of the speech therapy.On Regular followup, there was gradual improvement of the voice quality and hoarseness of voice. The indirect laryngoscopy revealed a gross improvement of the right sided vocal cord after 5th week.[video-2]

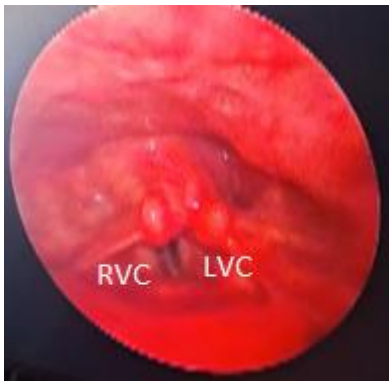
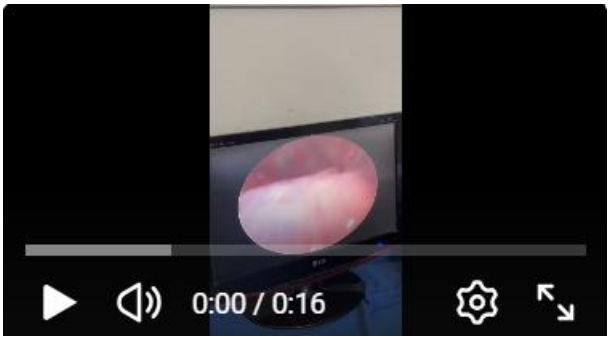


Figure 1: Laryngoscopic views of vocal cord paralysis (VCP). It shows a fixed right vocal cord suggestive of an unilateral (right) VCP. Above picture is of VCP patients who occurred at our hospital.

https://youtu.be/bJhpRdBh7yw

Video 1: The laryngoscopic view of the vocal cords revealed RVC palsy as RVC is fixed and normal mobility of the LVC after 48 hrs of the tracheal extubation.
RVC- right vocal cord, LVC- left vocal cord



https://youtube.com/shorts/IZqEwfor-0c

Video 2: The laryngoscopic view revealed a normal movements of the both vocal cords after 5 weeks of the surgery. The recovery occurred spontaneously and did not require any active therapy

2. Discussion

Delayed vocal cord palsy (VCP) following tracheal extubation is uncommon but a serious complication occurring in approximately 1 in 1500 (0.07%) to 0.1% of the intubated general surgical patients. [2] Higher incidence has been observed in cardiac surgical patients due to surgical proximity to the RLN, affecting 1 in 50–100 adult patients, and 18% to 56% after pediatric cardiac surgery. [4]

VCP can present within 36 hours to several days post-extubation, and the onset is further delayed in the ICU settings. Delayed edema or Inflammation can result in swelling around the nerve, induced by surgical manipulation or infection, can lead to functional impairment days after the initial procedure. The presented patient developed hoarseness of voice after 48 hours of the extubation. Lim et al have reported a vocal cord palsy incidence of 0.043% and 70% presented as unilateral.[5] Duration of the functional recovery ranges from 3-4 months with intervention and 4-6 months without intervention.[5]

The internal branch of the RLN is at risk of compression between the arytenoid cartilage and the thyroid cartilage and the inflated cuff of the endotracheal tube. It often presents with dysphonia, hoarseness of voice or respiratory stridor, or airway obstruction days or weeks after extubation.[6] Whereas, bilateral palsy of the recurrent nerve results in acute airway obstruction. [6,7]

The etiology of delayed VCP might be linked to direct trauma during endotracheal intubation, or transient recurrent laryngeal nerve neuropraxia due to compression of the nerve between the endotracheal tube(ETT) cuff and the thyroid cartilage, or neuropraxia secondary to the stretching during neck hyper extension, or even due to inflammation.[8,9,10] Even, neuropraxia may occur due to pushing of the vocal cords or cricoid cartilage in an oblique manner compressing the RLN by the posterior surface of the ETT. In addition, traumatic Intubation causing direct mechanical damage during tube insertion particularly by the inexperienced trainee anaesthetist, and even dislocation, subluxation or dislocation of the cricoarytenoid joint might play a major role in delayed VCP. Furthermore, over inflation of the cuff with a cuff pressure of more than capillary pressure i.e. 30 mmHg, inadvertently extubation with inflated cuff can directly

damage the vocal cords. Moreover, use of oversized ETT in oedematous airway in patients with CHF, eclampsia, respiratory infections, particularly viral infection should also be considered as another possible injury mechanism, and growth surrounding the upper airways and also post radiation therapy.[11,12,13] Some surgeries like thyroidectomy, carotid endarterectomy, anterior spine decompression, open heart surgery, and thoracic surgery can cause injury to RLN, or vagus nerve resulting in hoarseness of voice and voice fatigue and decreased voice intensity.[5] Surgeries of longer than 3-6 hours increases the VCP by two folds rise due to glottic edema and inflammation. Often, a prolonged intubation of longer than 12 hours might result in unilateral VCP in approximately 41% of VC palsies. Some authors have suggested that VCP secondary to upper airway tract infection presents late with in the median of 22 weeks with the symptoms of cough, dysphonia, vocal fatigue, odynophagia and vocal cord palsy.[14]

There are also some patient dependent factors involved in the VCP such as advanced age of more than 50 yrs increases the incidence by 3- fold, associated comorbidities (DM, hypertension) increased risk by 2-fold and 70% left sided VC is involved.[3] However, the presented patient had right sided VCP. The extensive rotation of the neck and over extension and flexion of the head during cervical spine or neurosurgery can precipitate VCP.

Some authors have proposed the precipitating factors sparingly involving in the left VCP; right- handed anaesthetists likely causing more stretch and pressure on the left sided VC and RLN, fixation of the tube on the right angle of the mouth causing pressure on left RLN and long tortuous course of the left recurrent laryngeal increases the chances of injury with cuff pressure.[15]

Prevention of delayed VCP includes selection of appropriate size of the ETT, inflation of the endotracheal tube cuff just enough to seal the airway and avoidance of over-inflation of the cuff limiting the intracuff pressure less than 30 mmHg, avidance of too high cuff position, avoid cord trauma during intubation, continuous cuff pressure monitoring particularly when nitrous oxide is used in anaesthesia, trainee anaesthetist should be supervised by experience consultant, central tube fixation can avoid pressure on the LRL nerve, use of anti-inflammatory agents like dexamethasone in long surgical procedures, use antibiotic cover to control the infection.[15,16]

Management of VCP depends on the cause, and severity of the symptoms and time of the symptoms onset. Management of right-sided vocal cord paralysis after surgery typically follows a "wait-and-see" approach for the first 6–12 months, as most of the time the nerve is only bruised and can recover spontaneously. During this period, the goal is to improve voice quality and prevent persistent silent aspiration pneumonitis. Hence, the immediate management starts with voice therapy as a primary non-surgical treatment and practiced with vocal exercises like "humming" or "pitch glides" to strengthen the cords. 70-80% of patients experience meaningful voice improvement with dedicated voice therapy. In addition, breath control learning for respiratory support to reduce the effort needed to speak. Furthermore, swallowing safety exercises are learnt to prevent the aspiration. [17]

In some instances, the cord palsy may improve without surgical treatment and therefore permanent surgery may be delayed for at least a year. The presented patient also had a significant improvement on the hoarseness of voice spontaneously within 6-8 weeks without any medical or surgical treatment.

In some refractory acute cases, a calcium-channel blocker (nimodipine) may be prescribed to encourage nerve recovery.[18] Selectively an Injection laryngoplasty as a "bulk injection" like hyaluronic acid or collagen, calcium hydroxyapatite, or autologous fat/fascia into the paralyzed cord has been recommended. This moves the paralyzed vocal cord closer to the moving well one, and thus significantly improves voice and swallowing within the first 3 months. [19, 20]

If permanent VCP persists for longer than one year, then permanent surgery may be considered as Medialization thyroplasty that relies on the use of an implant (silicone or Gore-Tex) in the larynx to reposition the vocal cord by pushing the paralyzed cord into a fixed position near the midline. It is the current gold standard treatment for unilateral vocal fold paralysis. It provides a permanent solution to glottic insufficiency caused by injury to the RLN.[21] Occasionally, a laryngeal reinnervation is attempted where a healthy nerve from another part of the neck is reattached to the damaged one. Though, it doesn't restore movement, but improves muscle tone and vocal quality over the next 6–9 months.[22]

The researchers are working on the emerging treatment involving with the linking the vocal cords to another source of electrical stimulus similar to pacemaker to restore opening and closing of the vocal cords that can't move.[23]

3. Conclusion

Delayed VCP after GA with endotracheal intubation usually involve left vocal cord in approximately 70% of the cases.[24] Delayed VCP can occur after uneventful tracheal extubation and normal voice recovery. In the presented patient the hoarseness occurred at 4th postoperative day. Initial management includes the speech therapy and anti-inflammatory agents in an ETT or ETT cuff pressure induced unilateral VCP.A "wait and see" approach is applicable for vocal cord palsy following general anesthesia, particularly when the injury is suspected to be due to temporary pressure from an endotracheal tube cuff (neuropraxia) or edema. Fortunately, vocal cord functions improves within a few days to weeks. The intubation-related VCP is temporary, with recovery typically occurring within4- 6 weeks.

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