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Vocal Fold Palsy Following Endotracheal Intubation – Diagnosis, Management, and Outcomes: Case Series

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Abstract

Background: Endotracheal intubation is a routine procedure for an elective operation under general anesthesia (GA) or an emergency case for airway protection. No matter being lifesaving or routinely performed, endotracheal intubation can cause vocal fold palsy (VFP) which may result in undesirable morbidities.

Goal: To spotlight the significance of the early diagnosis of VFP following endotracheal intubation, plan of management, and outcomes.

Method: We present consecutive 7 patients who developed VFP following endotracheal intubation for various kinds of surgeries. The VFP was identified on a flexible nasopharyngolaryngoscope (FNPLS) when they were referred to otolaryngology surgeons for hoarseness post-extubation. Data collections include the patient's demographic data, diagnosis, type of intubation (elective vs emergency), laterality of VFP, intervention, and duration of functional recovery. Objective and subjective voice assessments were performed to evaluate pre and post-treatment outcomes.

Results: Hoarseness and aspiration are the early symptoms of VFP in this review. Examination using FNPLS found left VFP in 5 patients, 1 patient with right VFP, and 1 patient with bilateral VFP. Two patients had undergone injection laryngoplasty (IL) with hyaluronic acid, three patients had self-recovery of function while the other two patients passed away due to acute illness before receiving any interventions. Duration of functional recovery ranges from 3-4 months in the intervention group while the non-intervention group ranges from 4-6 months. Malay Voice Handicap Index-10 (mVHI-10), Maximum Phonation Time (MPT), and Eating Assessment Tool (EAT 10) showed significant improvement at 1 month in the post-intervention group. The mVHI-10 showed a reduction from 33/40 and 29/40 to 11/40 and 0/40 respectively. MPT showed improvement from 6 seconds and 5 seconds to 16 seconds and 13 seconds respectively. EAT 10 showed a reduction from 8/40 and 5/40 to 0/40 for both patients in the intervention group.

Conclusion: VFP can occur following endotracheal intubation. Despite its rarity, it is crucial to identify early symptoms so that early referral can be done for definitive management. IL performed in our case series has shown significant voice improvement objectively and subjectively in the surgical intervention group.

Keywords: Endotracheal Intubation; Injection Laryngoplasty; Vocal Fold Palsy

Introduction

Endotracheal intubation may cause injury to the oral cavity, oropharynx,

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larynx, trachea, and oesophagus [1,2]. The injury in the larynx ranges from mild edema or mucosal ulcerations of the glottis, glottic web, granuloma, hematoma, arytenoid dislocation, or neurogenic injury which may lead to vocal fold palsy (VFP) [3,4,5]. VFP either unilateral or bilateral secondary to endotracheal intubation is a rare complication [2,3]. A study by Sehun Lim et al reported that among 100,941 cases of GA, only 43 patients (0.043%) developed VFP [3]. In this study, no detailed objective or subjective voice evaluation of VFP was performed and only flexible nasopharyngolaryngoscope (FNPLS) findings were described. Kikura et al reported that the incidence of VFP following endotracheal intubation was only 0.077% (A total of 24 patients) [6]. The 24 patients with VFP were all treated with mecobalamin (vitamin B12) and no active surgical intervention was performed on the patients [6]. Most of VFP following endotracheal intubation is unilateral vocal fold left side (70%) [3,4]. The possible mechanism of injury is direct pressure of the recurrent laryngeal nerve (RLN) by the endotracheal tube (ETT) cuff, cricoarytenoid joint dislocation, or subluxation or neurapraxia secondary to stretching during neck hyperextension [2,4,7]. The common symptoms of unilateral VFP are hoarseness, voice fatigue, or reduced voice intensity. Furthermore, VFP with decreased laryngeal sensation and poor cough reflex may cause pneumonia secondary to aspiration resulting in significant morbidity [1-4,8]. Here, we present a consecutive case series of 7 patients who developed VFP following endotracheal intubation in both elective and emergency cases and discuss its management and outcomes.

Case Report

All patients referred to otolaryngology surgeons for voice change post-extubation were examined using FNPLS. Data collections include the patient's demographic data, diagnosis of the disease, type of intubation (elective vs emergency), laterality of VFP, surgical intervention, and duration of functional recovery. Objective and subjective voice assessments were performed to evaluate pre and post-treatment outcomes. We present a summary of 7 consecutive case series of VFP (4 male, 3 female) following endotracheal intubation (Table 1). Two patients were under 50 years old and 5 patients were over 50 years of age. Five patients had co-morbidities such as diabetes mellitus, hypertension, ischemic heart disease, renal failure, chronic obstructive pulmonary disease (COPD), and systemic lupus erythematosus (SLE) while the other 2 patients were free from chronic diseases. All patients had a history of previous intubation. Four patients had been electively intubated for the surgical procedure under GA and the surgery lasted greater than 3 hours. Three patients were intubated during the acute life-saving event to secure the airway and were kept intubated minimally for three days. Six patients presented with unilateral VFP, five patients of them with left VFP, one patient with right VFP, and one patient who developed bilateral VFP. The objective and subjective voice assessments consist of Malay Voice Handicap Index-10 (mVHI-10) [9], Reflux Symptoms Index (RSI), Maximum Phonation Time (MPT) [10], and Eating Assessment Tool (EAT-10) were used to evaluate pre and post-treatment (Figure 2). All patients showed abnormal voice parameters at pre-intervention. In the intervention group, questionnaire evaluation showed significant improvement post-treatment. The mVHI-10 showed a reduction from 33/40 and 29/40 to 11/40 and 0/40 respectively. MPT showed improvement

Table 1: Summary of demographic data of the present case series together with the type of intubation, laterality, type of intervention, and the duration of Function recovery.

Gender/ Age	Diagnosis	Intubation (Emergency or Elective)	Laterality	Intervention	Duration of Function Recovery
Patient A Male / 48	Extraluminal Intestine Tumor	Elective	Left	Injection laryngoplasty	3 months
Patient B Female / 39	Right Parapharyngeal Schwannoma	Elective	Left	Injection laryngoplasty	4 months
Patient C Male / 65	Status Epilepticus secondary Uremic Encephalopathy	Emergency	Left	Voice Therapy	6 months
Patient D Male / 64	Sepsis secondary HAP	Emergency	Left	None	Patient passed away
Patient E Male / 67	Severe Sepsis with AECOPD secondary HAP	Emergency	Bilateral	None	Patient passed away
Patient F Female / 58	Splenic Marginal Zone Lymphoma Stage 4	Elective	Right	Voice Therapy	6 months
Patient G Female / 68	Strangulated Hernia	Elective	Left	Voice Therapy	4 months

*AECOAD – Acute Exacerbation Chronic Obstructive Pulmonary Disease
**HAP – Hospital Acquired Pneumonia

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Table 2: Objective and subjective voice parameters at baseline (before intervention), at 1 month and 3 months post-intervention.

Patient	Before intervention				1 month after intervention				3 months after intervention				Recovery
	m-VHI-10	RSI	EAT-10	MPT	m-VHI-10	RSI	EAT-10	MPT	m-VHI-10	RSI	EAT-10	MPT	
*Patient A	33/40	20/45	Aug-40	6 sec	20/40	13/45	Jun-40	12 sec	Nov-40	Feb-45	0/40	16 sec	3 months
*Patient B	29/40	17/45	May-40	5 sec	18/40	Oct-45	May-40	10 sec	0/40	Feb-45	0/40	13 sec	4 months
**Patient C	27/40	20/45	Aug-40	8 sec	21/40	18/45	Jun-40	8 sec	15/40	Dec-45	Jan-40	10 sec	6 months
**Patient F	25/40	22/45	Oct-40	6 sec	22/40	20/45	Oct-40	8 sec	17/40	Oct-45	Aug-40	9 sec	6 months
Patient G	27/40	22/45	Oct-40	6 sec	On tracheostomy	On tracheostomy	On tracheostomy	On tracheostomy	14/40	Nov-45	Aug-40	8 sec	4 months

*Injection laryngoplasty: Patient A and B

**Voice therapy: Patient C, F and G

Abbreviation: VHI – Voice Handicap Index; RSI – Reflux Symptom Index; MPT – Maximum Phonation Time; EAT-10 – Eating Assessment Tool

from 6 seconds and 5 seconds to sixteen seconds and thirteen seconds respectively. EAT 10 showed a decrease from 8/40 and 5/40 to 0/40 for both patients in the intervention group. In the intervention group, the subjective and objective voice parameters showed significant improvement at 1-month post-intervention compared to the baseline. Two patients (Patient A and Patient B) underwent injection laryngoplasty (IL) and achieved normal function of the vocal fold between 3 to 4 months with significant improvement of voice quality post-treatment. Three patients (Patient C, Patient F, and Patient G) who refused intervention, opted for voice therapy for voice rehabilitation. Two patients (Patient C and Patient F) had self-recovery after 6 months, however, their voice quality was still poor. One patient (Patient G) despite being on a tracheostomy tube initially, had fully regained the vocal fold function after 4 months and was decannulated. Unfortunately, 2 patients (Patient D and Patient E) passed away due to acute illness before any treatments were given.

Discussion

The incidence of VFP after endotracheal intubation is extremely low which is less than 0.1% [1,3,5,8]. Only 7 out of 23010 patients (or 0.03%) who underwent general surgical procedures with elective endotracheal intubation developed unilateral VFP, according to Jack Sariego et al [5]. VFP can either be due to neurogenic injury or mechanical injury [1,3]. Neurogenic injury is due to injury to the RLN or vagal nerve (CN X). Surgeries like thyroid surgery, carotid end-arterectomy, anterior cervical spine decompression, open-heart surgery, and thoracic surgery had been acknowledged to cause postoperative VFP due to neurogenic injury [2]. VFP also may be caused by direct neoplastic infiltration or distant metastases from thyroid, esophageal, laryngeal, or lung malignancy [1,2]. Mechanical damage of the vocal fold may additionally result from the direct insult of endotracheal intubation causing dislocation and subluxation of the cricothyroid or cricoarytenoid joints. The mechanism of neuropraxia during endotracheal intubation arises from the possibility that the posterior surface of the tube push the vocal fold or cricoid cartilage in an oblique manner, compressing the RLN [5,6,10]. Temporary VFP can also be brought on by glottis edema or inflammation following an intubation injury [4]. Before the RLN reaches the superior edge of the cricoid cartilage, it splits into anterior and posterior branches [4]. The inter-arytenoid and posterior cricoarytenoid muscles are innervated via the posterior branch. The lateral cricoarytenoid and thyroarytenoid muscles are innervated by the anterior branch as it goes medially to the thyroid cartilage lamina. This anatomical feature makes the anterior branch of the RLN vulnerable to compression between the lamina of the thyroid cartilage and the endotracheal tube if the cuff was placed at the level or slightly inferior to the vocal cords [2,5,11]. As a result, it inhibits glottic closure during phonation, which results in symptoms of aspiration and a breathy voice. The most frequent possible location of RLN compression, according to cadaveric dissection by Cova et al., is roughly 6 to 10mm inferior to the vocal cord [1,5]. Furthermore, an overinflated cuff near the glottis during intubation or improper deflation of the cuff prior to extubating might traumatized the anterior branch of the RLN [3,11]. The duration of the endotracheal intubation also plays a significant role in VFP. A longer duration of surgery has been associated with VFP due to the high incidence of laryngeal cartilage dislocation and laryngeal nerve ischemia [2,3]. There have been reports that surgeries lasting three to six hours result in a twofold rise in VFP [1,3,6,10]. The incidence of unilateral VFP in prolonged intubation lasting more than 12 hours has been reported as high as 41% [4,12]. The likelihood of VFP after endotracheal intubation can increase due to patient variables such as advanced age and the presence of other co-morbidities. Patients over the age of 50 are at a 3-fold higher risk, and those with diabetes or high blood pressure are at a 2-fold higher risk [3,11]. The left side accounts for almost 70% of reported cases of unilateral VFP related to

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endotracheal intubation [3,4]. Three explanations have been proposed to explain this incidence of left side VFP. Being right-handed medical professionals is the first theory. Since most medical professionals have a right-handed dominant grip, damage to the left vocal fold during endotracheal tube insertion is more frequent [4]. The preference for securing the endotracheal tube at the right side of the mouth angle, which increases pressure on the left vocal fold, is the second theory. The third explanation relates to the anatomical structure of the left RLN as it is much longer compared to the right RLN making it more vulnerable to insult along the course [4,11]. Following endotracheal intubation, it's important to recognize the symptoms of VFP. The first symptom that frequently indicates VFP is hoarseness [11]. Other symptoms may include vocal fatigue, weak or breathy voice, low or loss of voice projection, or decreased voice intensity [11]. There have also been reports of dysphagia, globus sensation, aspiration, and respiratory symptoms [1,5]. Reduced laryngeal sensation with the presence of the glottic gap increases the risk of aspiration which subsequently may lead to significant morbidity [4]. The duration of unilateral VFP can range from 6 to 9 months, however, the symptoms such as hoarseness may improve earlier from 2 to 6 months [3,5]. However with early surgical intervention such as injection laryngoplasty, the voice improvement may occur much earlier. FNPLS is routinely performed to analyze the structure and functionality of the vocal fold in the clinical setting [11]. Due to the low incidence reported, there are very few studies that advise specific management of VFP secondary to endotracheal intubation [4]. Consequently, the methods of treatment include voice therapy, IL, and type 1 thyroplasty [2,4]. IL has been proposed as a method for early surgical intervention in unilateral VFP [13,14]. It is safe and effective, and studies had proven early IL had shown better voice outcomes compared to one performed later [13]. According to Friedman et al., early medialization of the paralyzed vocal fold with IL results in a more favorable vocal fold position resulting in good voice outcome [13]. In the present case series, those who received IL showed significant improvement in both subjective and objective voice parameters compared to patients who received voice therapy.

Conclusion

Following endotracheal intubation, VFP, either unilaterally or bilaterally, is a rare consequence. Due to limited evidence from the literature, there are no specific guidelines on how to manage VFP that results from endotracheal intubation. IL is the treatment employed in our case series which depicted significant voice improvement objectively and subjectively.

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