Case Report
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A Case of Unilateral Vocal Cord Palsy after Endotracheal Intubation: Could it be due to Arytenoid Subluxation?

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Abstract

Endotracheal intubation can result in complications in 1% of patients, of which hoarseness of voice is a common complaint. One of the rare causes is arytenoid dislocation. A 24 year old patient diagnosed with ruptured ectopic pregnancy was posted for emergency laparotomy. She underwent emergency laparotomy under general anaesthesia. Post operatively, patient developed hoarseness of voice. Further evaluation with fibreoptic bronchoscopy revealed immobile arytenoid cartilage, which improved with conservative management.

Keywords: Arytenoid Subluxation, Laryngeal Electromyography, Recurrent Laryngeal Nerve Palsy, Vocal Cord Palsy

Introduction

Endotracheal intubation can have many complications among which sore throat and hoarseness are the most common symptoms[1]. Indeed, prolonged or even permanent hoarseness occurs in approximately 1% of patients. Vocal cord paralysis not only causes patient's dissatisfaction, but it also becomes a risk factor for aspiration pneumonia, which in turn increases postoperative morbidity and mortality[1].

The causes of unilateral vocal cord paralysis are mainly divided into joint disorders and nerve damage[1]. It is essential to distinguish between these two entities as the management of each varies.

Case Report

A 24-year-old female patient with ruptured ectopic pregnancy was posted for emergency laparotomy. The patient was gravida 3, para 2 and living 2, on examination she was pale with heart rate of 120 beats/min, blood pressure 80/42mmHg, saturation 97% on room air and airway was Mallampati grade 1. The investigations revealed a haemoglobin of 7.4g%. The patient was nil oral for 4 hours. Written informed consent was taken. She was shifted to the operation theatre and monitors like electrocardiogram, noninvasive blood pressure and pulse oximeter were connected and basal parameters recorded. Two intravenous 16G cannulae were inserted and 500ml Ringer lactate (RL) fluid was connected. The patient was premedicated with Inj metoclopramide 10mg and inj ranitidine 50mg intravenously. She was preoxygenated for 5 min with 100% oxygen, and inj glycopyrrolate 0.2mg, inj midazolam 1mg, inj fentanyl 100µg were administered intravenously. Rapid sequence induction was done with ketamine 60mg and muscle relaxation achieved with inj succinylcholine 100mg. After 60seconds, direct laryngoscopy was done by our postgraduate. Since the postgraduate could not intubate after one attempt the anaesthesiologist on duty took over and the trachea was intubated with a 7.5mm cuffed endotracheal tube in the first attempt. The cuff was inflated with 5ml of air. After confirming bilateral equal air entry, the tube was fixed at 20cm and connected to the ventilator with tidal volume

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of 400ml, respiratory rate of 12/min and a Positive End Expiratory Pressure of 5cm H_20. Anaesthesia was maintained with O_2 50%, N_2O 50%, isoflurane 0.6% and inj vecuronium 0.1mg/kg.

After the vessel clamping, hemostasis was secured and the patient was stable. Ringers lactate 1000ml, 500ml hetastarch and 2 pints packed cells were transfused throughout the procedure which lasted for 60mins. Intraoperative blood loss was 1.2 litres and urine output was 500 ml towards the end of surgery. After skin closure, patient was adequately reversed and extubated. She was monitored for 24 hours postoperatively. After 6 hours in the postoperative room the patient complained of sore throat and hoarseness of voice which was clinically evident. Postoperative vitals were stable. Suspecting oedema of the vocal cords, she was given inj dexamethasone 8mg IV. After 24 hours fibreoptic bronchoscopy revealed unilateral left complete paralysis of the vocal cords with no oedema, overhanging and immobile arytenoid cartilage on the same side as palsy. She was given inj dexamethasone 8mg IV twice daily for 7days, tab nervigen P (Jenburkt pharmaceuticals Ltd, Mumbai) once a day for 30days and was advised voice rest. After one week the patient showed some improvement in phonation and hence was advised discharge with review every 15days. Further follow up showed that she had complete improvement in her voice after 3months.

Discussion

Vocal cord palsy is classified based on the site of the lesion (supranuclear, bulbar, peripheral nerve, myoneural junctions, or laryngeal muscles) or the nature of the disorder (inflammatory, neoplastic, traumatic, postsurgical, systemic or idiopathic)[2]. The most common causes of vocal cord palsy are joint disorders and nerve injuries. Age, diabetes mellitus, hypertension, and prolonged intubation would all potentially increase the risk of vocal cord paralysis[1].

Traumatic intubation can occur in emergency situations that require rapidity in the access of the airway, in difficult exposition of glottis or when carried through by professionals inexperienced[3]. In a prospective observational study, it was observed that 63% of acute injuries of the larynx are due to orotracheal intubation and that they had been reversible in 30days[4]. The true anatomical analysis of the cause was done on a cadaver by Ellis and Pallister in 1975. They proposed that it was the anterior branch of the recurrent laryngeal nerve which was compressed between the cuff of the endotracheal tube and the lamina of the thyroid cartilage[5]. John and coworkers noted that the suspected point of vulnerability was located 6-10mm below the posterior end of the vocal cord in their work[6].

Although vocal cord function contributes to the ability to breathe, swallow, and phonate effectively, up to 40% of individuals with vocal cord palsy may be asymptomatic[7]. Conversely, mild persistent symptoms of hoarseness can be the initial signs of serious disease secondary to invasion or compression of the RLN[7].

In our case, intubation was first attempted by a less experienced anaesthesiologist and then was taken over by the staff on duty, who intubated the trachea in one attempt. Hence there could be chances of trauma to the vocal cord during intubation. We had inflated 5ml of air in the cuff but we did not monitor the cuff pressure throughout the surgery, hence RLN injury due to compression could not be ruled out although the surgery lasted for just 60mins. Postoperatively bronchoscopy revealed immobility of the left vocal cord hence unilateral vocal cord palsy was confirmed.

Arytenoid cartilage dislocation is a rare complication with an incidence of 0.023% to 0.097%[8]. Vocal cord subluxation is joint dislocation with some remaining contact of the joint surface. Laryngoscopy is the first step in diagnosis. Clues that can raise the suspicion of an arytenoid cartilage dislocation when evaluating unilateral VF mobility include arytenoid cartilage oedema, difference in vocal fold level, and absence of a 'jostle sign', which is a brief lateral movement of the arytenoid cartilage on the immobile side during glottic closure caused by contact from the mobile arytenoid[2]. In our case although there was no oedema of the arytenoid, we observed an overhanging left arytenoid with no mobility during phonation which pointed towards arytenoid subluxation/dislocation although not the definitive cause of vocal cord palsy. Pregnancy related increase in ligament laxity may cause joint instability[9], but there are no studies done that assess the effect of hormones on the arytenoid joint.

Symptoms common to both RLN palsy and arytenoid subluxation/dislocation include hoarseness, aphonia, and dysphagia[10]. Currently used assessments include laryngoscopy, Computed Tomography (CT), and Laryngeal Electromyography (LEMG), although none are ideal[10]. Long standing recurrent laryngeal nerve palsy can lead to structural changes in the arytenoid cartilages predisposing them

to dislocation. Other causes include use of laryngeal mask airway, insertion of transesophageal echocardiography probe or physical force on the tube[8]. Alexander et al., reported the use of helical CT in patients with arytenoid dislocation[11]. If the CT is equivocal, LEMG is useful to confirm diagnosis[2]. LEMG shows almost normal action potential in the case of arytenoid dislocation and aberrant action potential in those with RLN palsy[12]. Additionally, asymmetry in the joint space with specific obliteration or widening is the HRCT characteristic of arytenoid cartilage dislocation[13].

Spontaneous recovery of vocal cord function may occur due to spontaneous axonal regrowth[14]. Dana and associates recommended that a 12month waiting period is essential before surgical vocal cord medialization is required[14]. Closed reduction of the arytenoid cartilages may be required for dislocation[12].

Further follow up of our patient revealed complete improvement in voice after 3months. Due to financial restrictions we were not able to conduct a Computed Tomography (CT) or Laryngeal Electromyography (LEMG) (which was not available in our institution) and hence we could not conclude whether, unilateral vocal cord palsy in our case was due to RLN involvement or arytenoid subluxation.

Conclusion

Unilateral vocal cord palsy can occur during intubation due to RLN injury or arytenoid subluxation, and can be reversible. A CT and LEMG are both essential along with the vocal cord visualization by bronchoscopy for accurate diagnosis.

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