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## CASE REPORTS

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### BILATERAL ADDUCTOR VOCAL CORD PALSY: COMPLICATION OF PROLONGED INTRAOPERATIVE HYPOTENSION AFTER ENDOTRACHEAL INTUBATION

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Endotracheal intubation for general anesthesia is usually a safe procedure. However, postoperative sore throat and mild hoarseness may occur due to laryngeal edema but bilateral vocal cord paralysis as a result of recurrent laryngeal nerve injury is a rare complication. We report a case of bilateral adductor vocal cord palsy following general anesthesia for abdominal surgery. Clinical presentation was hoarseness, aspiration pneumonia and hypoxemia requiring ventilatory support. Neuropraxia of recurrent laryngeal nerve due to prolong intra-operative hypotension, even with normal endotracheal tube cuff pressure was the likely mechanism.

**Keywords:** Endotracheal intubation, Adductor vocal cord palsy, Prolong intraoperative hypotension.

#### Introduction

Endotracheal intubation is essential part of anesthesia practice but rarely result in vocal cord paralysis which is a most serious complication resulting in vocal disability and aspiration<sup>1,2,3</sup>. Direct surgical trauma, quality of tracheal intubation and high cuff pressure are some common risk factors for this complication. In our current case, we report an incident of uncommon cause for vocal cords palsy.

#### Case History

A 50-year-old 60 kg male presented with abdominal pain in abdomen and hematuria and was diagnosed to have renal cell carcinoma with thrombus in inferior vena cava (IVC). He was posted for right radical nephrectomy with IVC thrombectomy. Patient had no comorbidities. His preoperative examination and laboratory investigations were insignificant. Airway assessment was normal with mallampati score grade-I.

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Balanced general anesthesia was given and trachea was intubated with 8.0 mm cuffed portex endotracheal tube in single attempt without any trauma. Cuff of the endotracheal tube was inflated with room air till no apparent leak. Endotracheal tube (ETT) cuff pressure was measured with aneroid manometer and it was less than 25 mmHg and was maintained throughout the procedure. Anesthesia proceeded with oxygen, air, isoflurane, and atracurium with IPPV. Unexpectedly there was massive blood loss as tumour was adherent to surrounding structures. Intra-operatively patient received 14 unit of packed cells volume (PCV), 6 unit of fresh frozen plasma (FFP), colloids and crystalloids even vasopressors were started to maintain blood pressure but most of time mean arterial pressure was less than 65 mmHg. Surgery lasted for 8 hours. After completion of surgery extubation was accomplished in the operating room with neostigmine and glycopyrrolate. Patient was shifted in stable hemodynamic condition.

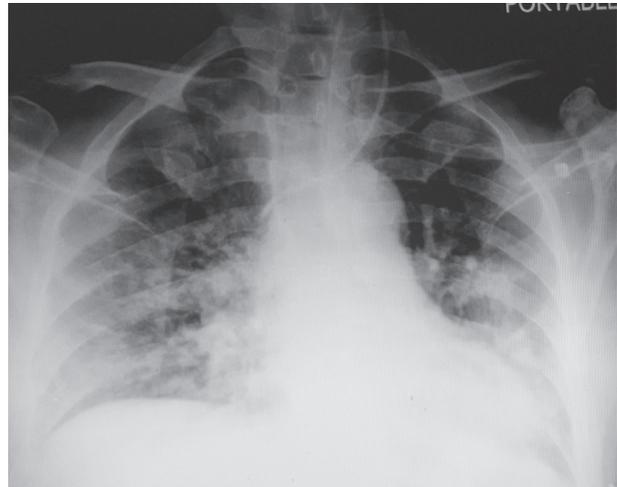
to ICU for observation.

On the next day patient had hoarseness, coughing on swallowing liquids, breathlessness and hypoxemia. For protection of airway and reversing severe breathlessness, the trachea was smoothly intubated and patient was put on mechanical ventilator. During direct laryngoscopy sluggish movement of vocal cords were noted. Chest x-ray was done and it was suggestive of bilateral aspiration pneumonia (figure 1). Patient was treated with antibiotics and supportive management. Patient was weaned off ventilator gradually and extubated under fiberoptic bronchoscopy (FOB) guidance. During extubation both the vocal cords were seen lying in intermediate position with a gap. Reduced movements of both vocal cords were also noted and on coughing vocal cords failed to approximate completely (figure 2). Adductor vocal cord palsy due to possible recurrent laryngeal nerve injury was suspected. The patient was started nasogastric feed and transferred to his room. After 10 days mild hoarseness and coughing on swallowing liquid still persisted. ENT opinion was sought and indirect laryngoscopy was done by ENT specialist which showed bilateral bowing with reduced movement of vocal cords more on right side than left. Beside speech therapy no active treatment was prescribed and after 25 days the nasogastric tube was

removed as patient could tolerate oral intake of liquid orally and by 40 days his voice recovered completely.

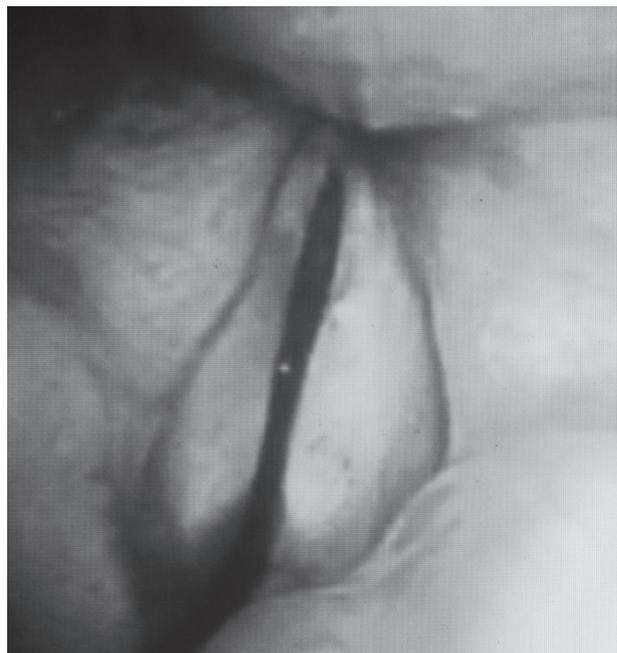
*Fig. 1*

*Chest X-Ray showing bilateral opacities*



*Fig. 2*

*Fiberoptic bronchoscopy view of larynx. Vocal cords failed to approximate completely during forced coughing*



## **Discussion**

Acute hoarseness and sore throat after endotracheal intubation is common and is usually due to mucosal injury. Prolong or permanent hoarseness occurs in approximately 1%<sup>4</sup> but postoperative vocal cord paralysis following endotracheal intubation,

leading to aspiration pneumonia and requiring mechanical ventilation is a rare occurrence.

Several risk factors for laryngeal injury have been identified including the size of the ETT, the cuff pressure and the quality and duration of tracheal intubation<sup>5,6</sup>. Kikura et al found that the risk of vocal cord paralysis was increased three folds in patients aged older than 50 years, 15-fold in patient intubated 6 h or more and two folds in patients with diabetes or hypertension<sup>7</sup>. In other cases, the cause of vocal cord paralysis still remains undetermined.

Minuck suggested that increasing endotracheal tube cuff pressure and asymmetrical cuff inflation might be the principle mechanism of recurrent laryngeal nerve palsy associated with endotracheal anesthesia<sup>8</sup>. According to Cavo John, the probable site of injury is the subglottic region where anterior branch of recurrent laryngeal nerve is vulnerable to compression between expanded cuff and overlying thyroid cartilage<sup>9</sup>.

General guidelines state that endotracheal tube cuff pressure should be not more than 25 mmHg (Possible maximal range of 15-30 mmHg) to maintain tracheal mucosa perfusion and thereby prevent mucosal ischemia, tracheal necrosis, rupture, stenosis, laryngeal nerve palsy and tracheoesophageal fistula<sup>10</sup>.

As far as our patient is concerned direct injury to recurrent laryngeal nerve is unlikely. Trachea was intubated without trauma in single attempt with

adequate size of ETT. Although surgery lasted for 8 hours but N<sub>2</sub>O was not used and cuff pressure was kept below 25 mmHg all the time; as such excessive cuff pressure could not be the reason for nerve injury. Our patient remained hypotensive (MAP less than 65 mmHg) for most of the time during the surgery and received vasopressors to maintain blood pressure. Prolonged intra-operative hypotension could have affected microcirculation of the larynx and led to the neuropraxia of the bilateral recurrent laryngeal nerve. Efrati et al suggested that when the patient is hemodynamically unstable and is being treated with vasoconstrictors the perfusion pressure in the tissue (tracheal mucosa) is significantly reduced. In this condition tissue hypoperfusion and hypoxia can happen even at lower cuff pressures (less than 25 mmHg)<sup>10</sup>.

## Conclusion

Severe and Prolonged intraoperative hypotension may adversely affect the microcirculation of larynx even with normal cuff pressure and can lead to neuropraxia of recurrent laryngeal nerve. We emphasize on keeping endotracheal tube cuff pressure in normal range and whenever patient is hypotensive and on vasopressors, ETT cuff pressure should be decreased as low as possible to maintain adequate tissue perfusion and prevent possible injury to recurrent laryngeal nerve and bilateral vocal cords palsy.

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