

Case Report

Management of Post extubation stridor following Total thyroidectomy due to bilateral recurrent laryngeal nerve damage - a case report

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Abstract

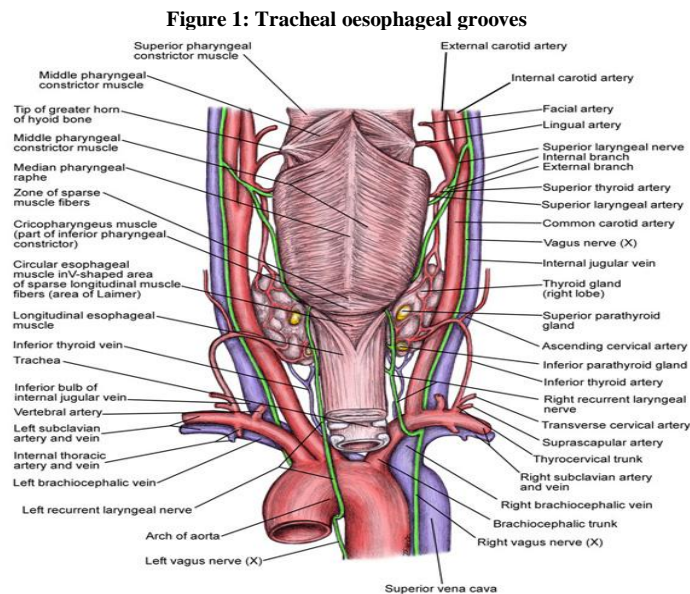
Recurrent laryngeal nerve (RLN) damage during total thyroidectomy is rare and estimated to occur in upto 14% of cases. Damage to RLN may be unilateral or bilateral. Bilateral RLN injury results in dysfunction of both vocal cords; which remain in midline during inspiration. After extubation, biphasic stridor, respiratory distress and aphonia occurs due to unopposed adduction of vocal cords and closure of glottic aperture necessitating immediate intervention and emergency intubation or tracheostomy. Deliberate intraoperative identification and preservation of the recurrent laryngeal nerve minimizes the risk of injury. Post operative visualization of vocal cord movements should also be performed as patients may be asymptomatic at first. Laryngeal electromyography EMG may be useful to distinguish vocal cord paralysis from injury to the cricoarytenoid joint secondary to intubation, and it may yield prognostic information. We report a case of post extubation stridor following total thyroidectomy probably due to accidental bilateral RLN damage and the management of the same after tracheal extubation.

Keywords: Total thyroidectomy, Post extubation stridor, Direct laryngoscopy(DLS), Recurrent laryngeal Nerve(RLN), Bronchoscopy, Tracheostomy

1. Introduction

Thyroid dysfunction can be treated medically or surgically. A thyroidectomy is performed as a definitive treatment for thyrotoxicosis and for malignancy. General anaesthesia (GA) with endotracheal intubation is the anaesthetic technique of choice for thyroidectomy, infrequently local anaesthetic techniques like cervical plexus block can be considered; in situations where GA is not indicated.¹

The patient is positioned supine with the head elevated 30 degree and neck extended by a roll behind the neck and shoulder (rose position)², During surgical excision haemostasis is crucial; the two anterior jugular veins must be avoided and ligation of the middle thyroid arteries is crucial. In addition the right and left recurrent laryngeal nerves, which lie along the tracheal oesophageal grooves, need to be identified. Figure 1.



The surgical identification and preservation of recurrent laryngeal nerve is essential to avoid injury. A study by Canbaz *et al*³ supports the practice of identification and exposure of recurrent laryngeal nerve and its branches during total thyroidectomy which decreases the rate of recurrent laryngeal nerve injury. Damage to recurrent laryngeal nerve may be unilateral or bilateral; however unilateral nerve injury is more common.¹

Unilateral recurrent laryngeal nerve damage causes ipsilateral vocal cord to remain in midline during inspiration, resulting in hoarseness. Bilateral recurrent nerve injury results in dysfunction of both vocal cords which remain in the midline during inspiration. After extubation, biphasic stridor, respiratory distress and aphonia occur due to unopposed adduction of the vocal cords and closure of the glottic aperture, unlike unilateral nerve injury, bilateral nerve injury necessitates immediate intervention with emergency intubation or tracheostomy^{1,4}.

Diagnosis of recurrent laryngeal nerve damage following surgery can be identified by visualizing vocal cords movement by either doing direct laryngoscopy/ fibreoptic bronchoscopy. Further management is based on the type and severity of the injury which can be managed by reintubation with small sized endotracheal tube/ tracheostomy.

In this case we report bilateral recurrent laryngeal nerve damage was the cause of upper airway obstruction with stridor following total thyroidectomy in a patient with toxic nodular goiter which was diagnosed on the table immediately after extubation by usage of direct laryngoscopy and fibreoptic bronchoscopy which was successfully managed by emergency tracheostomy.

2. Case Report

A 34 year old female weighing 38kg with swelling in front of the neck, FNAC of which was suggestive of hyper plastic nodular colloid goiter who was on antithyroid drug along with beta blocker since 4 months and also on lugol's iodine since 1 week before the patient was taken for total thyroidectomy and she was euthyroid at the time of surgery.

On examination of patient a swelling measuring 6×6 cm was present anteriorly in the neck with no retrosternal extension .Airway grading was Mallampatti grade II. Cardio respiratory assessment was normal with a Heart rate of 78 beats/min and a BP of 126/74 mm Hg. There was no history suggestive of airway compression symptoms; breath holding was about 25 secs and no pathology seen in her X-ray neck (AP and Lateral) and chest.

Investigations revealed haemoglobin of 11gm%, Fasting Blood Sugar (FBS) - 96mg%, Serum creatinine-0.8mg%, Total count-7000cells/cumm and differential count revealed Neutrophils-51%, Lymphocytes-39%, Eosinophils-7%, Monocytes-3%, Basophils - 0% with ESR - 40 mm/hr. ECG was within normal limits and recent thyroid profile done a week before suggestive of euthyroid state. An ultrasound of the neck done 4 days back showed bilateral diffuse enlarged hyper vascular thyroid gland. Indirect laryngoscopy done a day before surgery showed normal supraglottic anatomy and vocal cord movements.

Night before surgery 0.5mg Alprazolam was given orally .The antithyroid drugs (Carbimazole tablet 10mg QID) along with beta blocker (Propranolol 20mg BD) continued even on the day of surgery. Patient was shifted to OT and two intravenous lines were secured with 18G cannula over both upper limbs. Multiparameter monitor (ECG, NIBP, SPO₂, Temperature, RR and EtCO₂) connected. Airway management cart was kept ready along with the drugs like injectable beta blocker (esmolol).Pre medication included Inj. Glycopyrrrolate 0.2mg, Inj Fentanyl 80 mcg, Inj. Ondansetron 8mg and Dexamethazone 16mg IV. After preoxygenation for 3 mins. lidocaine hydrochloride 2% (without preservative) 1.5mg/kg IV was given and anaesthesia was induced with pentathol sodium 5mg/kg after confirming ventilation of the lungs, succinyl choline 2mg/kg was given IV ,following IPPV patient was intubated with 7.0mm ID cuffed portex endotracheal tube(ETT) effortlessly in first attempt , after confirming equal bilateral air entry ETT was fixed in place, anaesthesia was maintained with O₂:N₂:O supplemented with Isoflurane, Vecuronium bromide and paracetamol infusion was given intraoperatively. All the vital signs were maintained well during intra operative period which lasted for 3 and half hours. On completion of surgery lidocaine hydrochloride 2%(without preservative)1.5mg/kg IV was given and neuromuscular block was reversed with neostigmine and glycopyrrrolate in the dose of 2.5mg and 0.4mg respectively ,adequacy of neuromuscular reversal confirmed by TOF count(4 twitches) and extubation was done.

Immediately after extubation patient began breathing low Tidal Volume and with audible stridor. Patient desaturates to high 80's and low 90's. Positive pressure ventilation was applied with jaw lift, immediately blood was drawn and sent to evaluate the serum calcium level. Assisted ventilation was possible with movement of adequate tidal volume.O₂ saturation returned to 100%. Patient was then able to spontaneously breath adequate tidal volume with continuous audible stridor, serum calcium levels revealed normal level.

Anticipating recurrent laryngeal nerve injury, ENT surgeons were consulted and after discussion with operating surgeons and ENT consultants a decision was made to deepen the plane of anaesthesia with sevoflurane while maintaining spontaneous ventilation. Direct laryngoscopy was performed and both vocal cords were found to be in paramedian position leaving a glottic orifice of less than 2 to 3 mm, with no movement of left vocal cord and very minimal movement of right vocal cord and there was no laryngeal and cord edema noted, even then cords and the larynx were sprayed with racemic epinephrine but the stridor continued. A 6.5 sized CETT used for re intubation and fiber optic bronchoscope passed through the same and there was no significant pathology noted sub-glottically up to the carina.

Bilateral RLN damage was confirmed after discussing with the operating surgeons and ENT surgeon,the decision of temporary tracheostomy was made and proceeded with the same under GA after confirming complete recovery to spontaneous ventilation with tracheostomy the patient was monitored for about two hours in the post operative recovery room and then shifted to Surgical ICU for further management and care. On the 2nd day post operative day patient developed severe hypocalcemia (serum calcium levels= 7.8 mEq/dL) with perioral numbness and tingling abdominal pain, paraesthesia of extremities, carpopedal spasm, tetany but laryngospasm was not manifested as tracheostomy was already performed. The neuromuscular irritability secondary to severe hypoparathyroidism was confirmed by assessing for the Chvostek sign (facial contractions elicited by tapping the facial nerve in the periauricular area) and the Trousseau sign (Carpal spasm after inflation of a blood pressure cuff) and then severe hypoparathyroidism was treated with IV calcium gluconate, oral calcium and vitamin D supplementation.

3. Discussion

The most common post operative complication following total thyroidectomy include hypocalcaemia ,RLN damage and haematoma at the surgical site, especially operating over a large gland, malignant thyroid gland or a highly vascular gland. In our case we have faced two complications, firstly the biphasic stridor following extubation and secondly severe hypocalcaemia secondary to hypoparathyroidism due to inadvertent removal of parathyroid gland during total thyroidectomy.

There are studies showing planned tracheostomy done in anticipation of RLN injury during dissection(gross distortion of anatomical land marks) and tracheomalacia (due to large size of gland)⁵. On the contrary in our patient there were no pre operative risk factors like-large gland, significant tracheal deviation, retrosternal extension, difficult tracheal intubation and thyroid cancer to expect post extubation stridor but it was a total thyroidectomy which has caused bilateral accidental damage to RLN during dissection of gland bilaterally causing post extubation stridor. Hence we had to proceed with planned tracheostomy.

Injury to the RLN can occur by a number of mechanism like Ischemia(cautery use) contusion, entrapment, actual transection and anatomical variability and distortion will increase the risk of nerve injury⁶.The risk factors of RLN damage include no or incomplete dissection and exposure of RLN (visualization required along the distance between branching of inferior thyroid artery and entry of nerve into cricoids cartilage), thyroid cancer, total thyroidectomy(permanent nerve damage), re-do surgery(recurrence/cancer), sub-sternal goiter and ligature of the internal laryngeal artery^{7,8}

Therapeutic strategies for bilateral RLN palsy includes

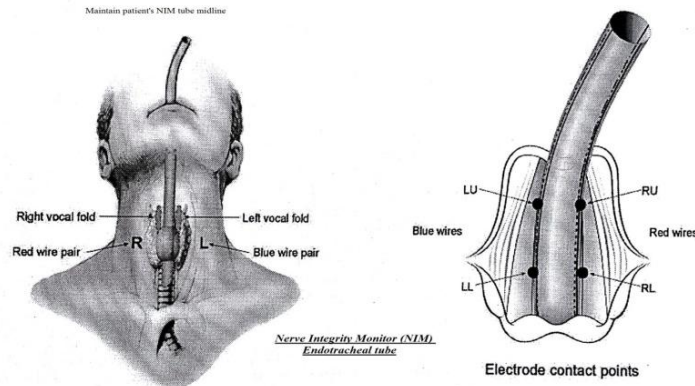
- ✓ Reintubation (if paralyzed in paramedian position)
- ✓ Tracheostomy
- ✓ Endoscopic posterior ventriculocordectomy
- ✓ Nerve decompression from ligatures or scar tissue

- ✓ Glottic widening procedures after 6-9 month^{9,10,11}

Prevention of RLN damage can be done by continuous RLN monitoring which may be useful in certain cases, but time consuming, requires spontaneous ventilation and incidence of false negatives also controlled trial have shown no statistical reduction in paralysis, paresis, or total injury rates to RLN^{12,13}.

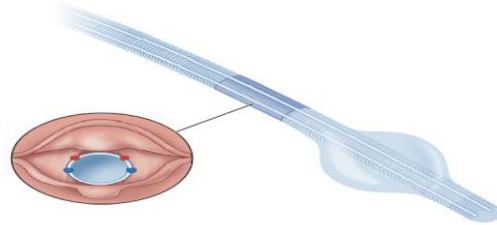
The Medtronic NIM electromyographic (EMG) endotracheal tube (Medtronic xomed) Figure 2. is constructed of a flexible silicone elastomer and has a distal inflatable cuff .

Figure 2: The Medtronic NIM electromyographic (EMG) endotracheal tube (Medtronic xomed)



The tube is fitted with stainless wire electrodes (2 pairs) that are embedded in the silicone of the main shaft of ETT and exposed only for a short distance, slightly superior to the cuff. The electrodes are designed to make contact with the patient vocal cords to facilitate EMG. Monitoring of RLN when connected to multichannel EMG monitoring device. If monitored correctly, the EMG monitor should show a consistent sound signal and a potential tracing¹⁶. The red wire pair of the NIM tube should contact the anterior and posterior portion of the right true vocal cord and the blue wire pair should contact the anterior and the posterior portion of the left true vocal cord. (Figure 3)

Figure 3: EMG monitoring device



The new guidelines in June 2013 includes a strong recommendation that surgeon performing the thyroid surgery should identify the recurrent the RLN or nerves^{15,16}.

In our case we did temporary tracheostomy for an accidental bilateral RLN damage following total thyroidectomy. Generally tracheostomy is performed on the basis of clinical judgement^{17,19}. Here we have confirmed the paramedian position of both vocal cords by DLS and fiberoptic bronchoscopy. Due to temporary tracheostomy, the possibility of keeping the patient reintubated with endotracheal tube and artificially ventilated for an unexpected number of days post operatively with heavy sedation/neuromuscular relaxants was avoided. Most importantly the temporary tracheostomy has helped us for further examination of the supraglottic area, glottic orifice and the position and movements of vocal cords as and when required in an attempt to judge the prognosis of RLN damage. Photograph showing the video laryngoscopic view of supraglottic area and vocal cord 2 weeks following the tracheostomy (fig 4.) which showed no movement of left vocal cord and slight flickering of right vocal cord during inspiration.

Figure 4: Laryngoscopic view of supraglottic area and vocal cord



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