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Case Report

Post Thyroidectomy Bilateral Recurrent Laryngeal Nerve Palsy – Not Always Surgical!

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Abstract

Importance: The cause of Recurrent Laryngeal Nerve (RLN) palsy following thyroidectomy is usually attributed to the surgery. Rarely, however, the cause can be non-surgical and results in adductor palsy. The management of these patients is different and is prudent that surgeons be aware of this entity.

Observation: We encountered a patient who developed bilateral RLN palsy following total thyroidectomy. The clinical finding and recovery were suggestive of a non-surgical cause for the palsy. We reviewed the literature and also performed a cadaver dissection to better understand the anatomy of RLN and cause of the palsy.

Conclusion: The non-surgical cause of RLN palsy (adductor palsy) presents differently from that of the surgical cause. The presentation and treatment are also different. Tracheostomy is not required and recovery of the nerve is usually the norm.

Keywords: Recurrent laryngeal nerve; Total thyroidectomy; Palsy

Introduction

Recurrent Laryngeal Nerve (RLN) palsy occurring following thyroidectomy is a known complication. In most instances it is assumed that the cause of the nerve injury is surgical. However, in spite of meticulous surgery and preservation of the nerves, rarely the cause of RLN palsy could be unrelated to surgery. We encountered one such case where the patient developed bilateral adductor vocal cord palsy following total thyroidectomy. We felt the cause for the palsy is not related to the surgery. We present the events that occurred in our patient and discuss how the management of these patients should be different from those with a surgical cause of RLN palsy.

Case Report

A 32 year old female was referred to our department for the surgical management of her toxic multinodular goiter. The patient gave a history of a neck swelling since 1 year, associated with weight loss and palpitations for 6 months. Following the diagnosis of toxic multinodular goiter she had been started on antithyroid medication. Examination revealed a multinodular goiter involving both lobes measuring 6 x 5 x 4cm. Patient had no voice symptoms and an office laryngoscopy showed bilateral normal mobile vocal cords. Total thyroidectomy under general anesthesia was planned. Patient was induced with Propofol, intubated with a cuffed flexometallic endotracheal tube (size 7mm) and maintained with isoflurane, nitrous oxide and oxygen. During the intraoperative period the entire course of both RLNs was identified and the nerves preserved. No electrocautery was used in the vicinity of the nerves. All the major blood vessels were ligated and divided only after identifying the RLNs. No anatomical variation of the RLNs was noted. Duration of surgery was three hours and twenty minutes, and the patient was positioned in the traditionally described extended position throughout the surgery. General anesthesia was reversed with neostigmine and patient was extubated smoothly. The immediate post operative period was uneventful. Three hours post operatively, the patient was noted to have a breathy voice and developed cough on taking oral feeds. She had no breathing difficulty or stridor. Laryngoscopic evaluation revealed bilateral fixed vocal cords with a wide phonatory gap of

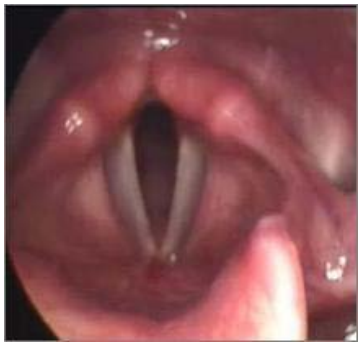

Figure 1: Laryngoscopy immediate post-op.

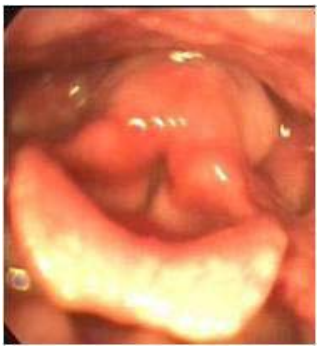

Figure 2: Laryngoscopy 6 weeks post-op.

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around 8 mm (Figure 1). There was no evidence of edema or signs of trauma. The patient was started on naso-gastric tube feeds and was referred to our speech therapist for vocal cord adduction exercises. The histopathological examination of the resected thyroid gland was suggestive of nodular colloid goiter. The patient was started on replacement dose of thyroxine and advised to review after two weeks. After fourteen days, office laryngoscopic examination revealed flickering movements of the vocal cords with a persistent phonatory gap. Though the voice appeared unchanged to us, the patient felt there was an improvement in her voice. She was tolerating soft food orally but had persistence of aspiration to liquid feeds and hence was advised to continue tube feeds. Six weeks post surgery the patient, visibly relieved, came to the clinic with a normal voice and was able to eat and drink normally. Repeat office endoscopy showed bilateral normal and mobile vocal cords with complete adduction (Figure 2). The naso-gastric tube was removed and the patient was advised to follow-up regularly.

Discussion

One of the main complications of thyroid surgery is injury to the RLNs. The surgical cause of the injury and the management of the complication have been described extensively. Patients with bilateral RLN palsy following thyroid surgery usually have their cords in the paramedian position and develop stridor. Tracheostomy is usually performed and the further management depends on the recovery of the nerves. It is assumed (and quiet rightly so) that the injury is surgical as the nerves are in the field of surgery. Rarely the nerves can be affected due to causes not related to the surgery. Yamashita et al in 1965 suggested that the bilateral RLN palsy they encountered was possibly related to the cuff overexpansion [1]. Cadaver studies have been undertaken to provide an explanation to this phenomenon. Ellis et al found that the RLN divides into an anterior and posterior branch below the superior rim of the cricoid cartilage. The anterior branch passes medial to the thyroid cartilage to supply the lateral cricoarytenoid and thyroarytenoid muscles. They suggest that a highly placed cuff can cause pressure neuropraxia of the RLN [2]. In the study by Cavo et al. they found that the anterior branch of the RLN could be compressed between the expanded cuff of the endotracheal tube and the overlying thyroid cartilage [3]. This would lead to paralysis of the muscles supplied by the anterior branch and lead to adductor palsy. A similar conclusion was drawn by the study done by Brandwein et al. [4]. A cadaver dissection done by us shows the findings described above (Figure 3). Another contributing factor could be an increase in cuff pressure during surgery due to diffusion of gases through the semipermeable membrane of the cuff. This may be a contributing factor especially for prolonged surgeries. Extended position of the neck, commonly used in head and neck surgeries, can cause the cuff to ride up towards the larynx and cause pressure neuropraxia of the anterior branch of the RLN [2].

The anterior branch of the RLN supplies the adductor muscles of the vocal cord. When injured, it will result in an abducted vocal cord.

Figure 3: (1) Main RLN, (2) Posterior branch RLN, (3) Anterior branch RLN, (4) Trachea, (5) Thyroid cartilage.

Clinically this would manifest with a breathy voice and aspiration, but should not compromise the airway. This was the picture seen in our patient and has been described earlier in patients undergoing surgery in regions other than the neck [5]. As the airway is patent, tracheostomy is not required in these patients. The aspiration symptoms are very distressing and tube feeds should be initiated to maintain the nutrition. Speech and swallowing therapy will be useful for these patients. Recovery of the function of the vocal cords will eventually occur provided there is no permanent damage due to ischemia. Knowledge of this phenomenon is useful as these patients can be counseled well and be avoided of the morbidity and stress of a tracheostomy.

Conclusion

The cause of the RLN palsy can rarely be non-surgical following thyroid surgery. Adductor palsy occurs due to compression of the anterior branch of the RLN. These patients should not have a compromised airway as the cords will be in abducted position. Tube feeds are usually required as aspiration is present initially. Surgical cause of RLN palsy will present differently and needs to be differentiated from adductor palsy as the management is different and less morbid.

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