

A Nonsurgical Trauma Causing Bilateral Adductor Palsy of the Vocal Folds following Total Thyroidectomy

Santosh Kumar Swain, Debasmita Dubey¹

Department of Otorhinolaryngology and Head and Neck Surgery, Manipal TATA Medical College, Jamshedpur-831017, Jharkhand, Manipal Academy of Higher Education, Manipal, ¹Medical Research Laboratory, IMS and SUM Hospital, Siksha "O" Anusandhan University, Bhubaneswar, Odisha, India

ORCID:

Santosh Kumar Swain: <https://orcid.org/0000-0001-7933-4414>

Debasmita Dubey: <https://orcid.org/0000-0002-2406-4145>

Abstract

Vocal fold palsy is an uncommon cause of hoarseness of voice following thyroidectomy. The vocal fold palsy can be unilateral or bilateral, with presentation varying accordingly. There are often surgeons blamed for postthyroidectomy vocal fold palsy. However, there are certain subtle factors associated with vocal fold palsy that should be known to clinicians. Bilateral adductor palsy following thyroidectomy is a morbid clinical situation where the patient presents with aphonia and aspiration. Here, we present a case of postop-total thyroidectomy with adductor palsy of bilateral vocal folds. A nonsurgical reason for the vocal fold palsy was suggested by the clinical findings and the patient's recovery. The knowledge of this case management is very important for surgeons to avoid such morbidity among patients.

Keywords: Bilateral adductor palsy, recurrent laryngeal nerve, total thyroidectomy, vocal fold

INTRODUCTION

In the head and neck area, thyroid surgery is a common surgical treatment. The common complications that occur in thyroid surgery include bleeding hypoparathyroidism and recurrent laryngeal nerve (RLN) injury.^[1] Thyroidectomy is the most common surgical procedure that puts the risk of injury to the RLN and vocal fold palsy. Even though it does not happen often, RLN injuries can have a negative impact on a person's quality of life.^[2] RLN injury is a major concern in thyroid surgery. A lesion of the RLN, whether by section, stretching, or injury by electrocoagulation is considered the main cause of postthyroidectomy dysphonia.^[3] A rare side effect of thyroid removal is bilateral RLN paralysis, which occurs in 0.4% of cases.^[4] A known side effect of thyroidectomy surgery is an injury to the RLN. In most cases, the injury of the RLN is surgical. However, despite meticulous surgery and preservation of the nerve, RLN paralysis can still occur. The RLN may sustain a unilateral or bilateral, permanent, or temporary injury. Near-total thyroidectomy is associated with an increased risk of transient RLN paralysis.^[5] Paralysis of the bilateral RLN is an important risk factor for dysphonia, aspiration, and airway compromise.

CASE REPORT

A 28-year-old female attended the outpatient department for swelling in the front of the neck for 2 years. She was diagnosed with Graves' disease and resistant to the treatment, so referred by an endocrinologist for total thyroidectomy. She had a history of palpitation, excessive sweating, and tremulousness. She had protrusions in both eyes. Ultrasonography of the neck revealed swelling of 7 cm × 4 cm × 3 cm in both the lobes of the thyroid. With antithyroid medication, serum calcium levels and thyroid function tests were within normal ranges. She was undergone total thyroidectomy under general anesthesia. The patient had a short and thick neck. Throughout the entire procedure, the patient was positioned in the customary extended position. The

Address for correspondence: Dr. Santosh Kumar Swain,

Professor, Department of Otorhinolaryngology and Head and Neck Surgery, Manipal TATA Medical College, Jamshedpur - 831 017, Jharkhand, Manipal Academy of Higher Education, Manipal, India.

E-mail: santoshvoltage@yahoo.co.in

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full course of the RLN was identified and preserved during the intraoperative phase. There was no use of electrocautery near the RLN. After locating the RLN, all main blood vessels were only then tied off. The patient was extubated smoothly. The immediate postoperative period was uneventful. On the 1st postoperative day, the patient developed a breathy voice. At the time of eating, she was not experiencing any breathing issues, stridor, or coughing. With the use of calcium gluconate, oral calcium, and Vitamin D, hypocalcemia was actively treated. The patient was also started on a replacement dose of thyroxine. A fiberoptic nasopharyngolaryngoscopy (NPL scopy) was performed to examine the throat and revealed that the patient had vocal fold adductor palsy on both sides [Figure 1]. The patient had no signs of hypocalcemia and was released from the hospital with a normal calcium level. The same results of the NPL scopy for aphonia were obtained at the time of discharge. After voice therapy, the patient was called for a follow-up appointment. There were minor voice improvements noted, and a follow-up NPL scopy performed after 4 weeks revealed a slight flickering movement of the vocal folds. At this time, there was no indication of hypocalcemia. Calcium, Vitamin D, and magnesium levels in postoperative blood tests were all within acceptable ranges. At 12 weeks, the voice had much improved, and an NPL scopy at that time revealed that the vocal folds were meeting normally in the middle on both sides when producing voice.

DISCUSSION

One of the important complications of thyroid surgery is an injury to the RLN.^[6] Bilateral RLN injury during thyroid surgery may cause vocal folds in the paramedian position and result in stridor where the patient requires tracheostomy and further treatment depends on the recovery of the nerves.^[7] Although rare, RLN palsy can be occurred due to cuff over-expansion by endotracheal tube.^[8] Under the superior rim of the cricoid cartilage, it was discovered that the RLN splits into an anterior and a posterior branch.^[8]

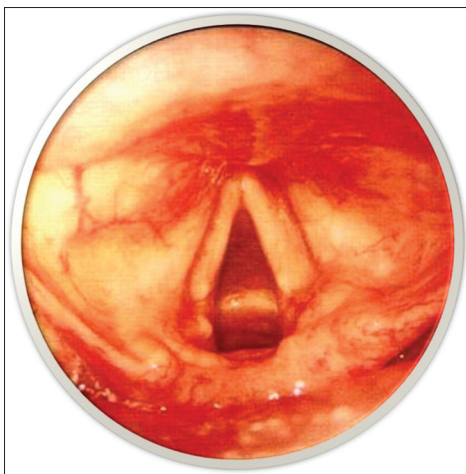


Figure 1: Fiberoptic nasopharyngolaryngoscopy showing bilateral adductor palsy

The lateral cricoarytenoid and thyroarytenoid muscles are supplied by the anterior branch of the RLN, which travels medially through the thyroid cartilage.^[9] By diffusing gases across the semipermeable membranes of the cuff, the cuff pressure may rise during operation. This could be a significant contributing factor in lengthy operations. The anterior branch of the RLN may experience pressure neuropraxia due to the stretched neck position used during thyroid surgery.^[8] The adductor muscles of the vocal folds receive neural feed from the RLN's anterior branch. An abducted vocal fold will occur if this nerve is damaged. Clinically, the patient would show signs of aspiration and a raspy voice, but the airway would not be compromised.^[10] There may be no evidence of aspiration or breathing difficulties. If there is no chronic damage from ischemia, the vocal folds will eventually regain their normal function.^[10] To prevent nerve damage during thyroid surgery, adequate knowledge of the relationships of RLN at the level of the thyroid gland is necessary. Typically, the RLN enters the larynx through the inferior fibers of the cricopharyngeal section of the inferior constrictor muscle.^[7] During tracing the RLN superiorly, the first branch (posterior branch) that originates, innervates the posterior cricoarytenoid muscle. Then, the anterior branch of the RLN to come off supply the interarytenoid muscle.^[11] The lateral cricoarytenoid and thyroarytenoid muscles are innervated by the terminal branches of the RLN. The inferior thyroid artery cross-point, the nerve segment related to the thyroid gland capsule, the nerve segment related to the gland ligament, and the point of entry into the larynx are the common sites of RLN injury (below or between the fibers of cricopharyngeal part of the inferior constrictor muscle). Transection, clamping, stretching, electrothermal injury, ligature entrapment, or ischemia are some of the possible causes of RLN injury.^[12] However, the actual causes of nerve injury are not well understood, particularly in situations where the integrity of the nerve is confirmed during surgery.^[13] The bilateral symmetrical vocal fold paralysis that was observed in our patient cannot be explained by the aforementioned mechanisms. RLN palsy is a rare complication of endotracheal intubation that occurs when a cuffed intubation tube is used even though the nerve was not surgically manipulated and no signs of intubation trauma were discovered postoperatively.^[9] Traditional neck positioning during neck surgery involves neck hyperextension. This can strain and paralyze the vagus nerve, which is anchored by the RLN in the mediastinum, during intubation or surgery.^[9] This hyperextended position of the neck can also cause migration of the cuff of the endotracheal tube proximally to just below the vocal folds which aggravate the pressure injury.^[14] In the case of differential diagnosis of intubation-induced vocal fold paralysis, the possibility of arytenoid dislocation should be kept in mind.^[9] However, laryngoscopic examination and palpation of the arytenoids revealed no signs of arytenoid dislocation in this case. Rare but well-known nonsurgical causes of vocal fold dysfunctions include hypocalcemia, low vitamin D levels, and adverse effects of chemotherapy drugs based on cisplatin and vinca alkaloids.^[12] A common side effect of complete thyroidectomy

is hypocalcemia.^[15] In the present case, postoperative blood tests for calcium, Vitamin D, and magnesium all came back within normal ranges.

CONCLUSION

Bilateral adductor palsy of the vocal fold is a rare complication of total thyroidectomy. However, the nonsurgical etiology for bilateral adductor paralysis of the vocal fold is extremely rare in thyroid surgery. Bilateral adductor palsy due to endotracheal intubation often recovers completely. Along with the meticulous surgical procedure, the monitoring of the cuff pressure and avoidance of prolonged hyperextension must be ensured to avoid probably nerve injury. Understanding this minimizes the morbidity caused by vocal fold adductor palsy on both sides. As long as there are no persistent ischemia-related injuries, voice function normally returns.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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