



Transient bilateral vocal fold paralysis after total thyroidectomy

Total tiroidektomi sonrası geçici iki taraflı vokal kord felci

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ABSTRACT

Vocal fold paralysis is a serious complication of thyroidectomy that is worrisome for health providers and potentially disastrous for the patient and family. A 56-year-old woman presented with bilateral vocal fold paralysis immediately after routine thyroidectomy and neck dissection for a large goiter with compressive symptoms. She was extubated the next day with full recovery of vocal fold motion. We discuss possible causes of vocal fold paralysis, including surgical, metabolic and anesthetic factors.

Keywords: Bilateral vocal cord paresis; complications; recurrent laryngeal nerve; thyroidectomy; total vocal cord paralysis; transient vocal fold paralysis.

ÖZ

Vokal kord felci, tiroidektominin ciddi bir komplikasyonu olmakla birlikte sağlık hizmeti sunucuları için endişe vericidir ve potansiyel olarak hasta ve ailesi için felaket olabilir. Elli altı yaşında bir kadın hasta sıkıştırıcı semptomları olan geniş bir guatr için rutin tiroidektomi ve boyun diseksiyonundan hemen sonra iki taraflı vokal kord felci ile başvurdu. Hasta vokal kord hareketinin tamamen iyileşmesi ile ertesi gün ekstübe edildi. Bu yazıda vokal kord felci nedenleri için cerrahi, metabolik ve anestetik faktörler dahil olmak üzere olası açıklamalar tartışıldı.

Anahtar Sözcükler: İki taraflı vokal kord parezi; komplikasyonlar; tekrarlayan larengeal sinir; tiroidektomi; toplam vokal kord felci; geçici vokal kıvrım felci.

Vocal fold paralysis is a major complication of thyroidectomy, with an incidence of 5.2% and 1.4% for temporary and permanent paralysis-- and involves the recurrent laryngeal nerve in 3.3% and 0.9%, respectively.^[1] It is also one of the most serious complications of tracheal intubation, resulting in severe vocal disability and increased risk of aspiration pneumonia.^[2] As it is not expected following routine thyroidectomy under general endotracheal anesthesia, its occurrence is worrisome for both surgeon and anesthesiologist, and potentially disastrous for patient and

family. We report the case of a 56-year-old woman presenting with bilateral vocal fold paralysis after an apparently - uneventful total thyroidectomy and central neck dissection.

CASE REPORT

A transcervical thyroidectomy and level-six neck dissection was uneventfully performed on a 56-year-old housewife for a 30-year large multinodular colloid goiter with compressive symptoms. The mass, which barely moved on deglutition, occupied her anterolateral neck with its inferior borders palpable below the clavicles.



Preoperative ultrasonography revealed solid lobulated masses measuring 7.7x3.4x2.4 cm and 7.8x4.1x3.2 cm on the right and left thyroid lobes, respectively, with substernal extension. Computed tomography scan confirmed extension into the thoracic inlet, with the right lobe measuring 8.5x3.2x3.7 cm and the left lobe measuring 9.9x4.6x4.1 cm with an upper segment cyst measuring 2.6x2.4x2.0 cm and a mid-segment complex nodule measuring 2.0x1.9x2.2 cm, and micro-calcifications in both lobes. A few non-enlarged lymph nodes (up to 0.6 cm) were seen in both submandibular regions and deep cervical chains.

Thyroid function tests, hemoglobin/hematocrit and chest radiographs were normal. Calcium was slightly low (2.12 mg/dL vs. 2.33-2.58 mg/dL) and albumin was slightly elevated (43 g/L vs. 35-38 g/L). She was taking once-daily simvastatin 40 mg, allopurinol 300 mg and amlodipine 5 mg, and had a family history of goiter in her mother and sibling, and a calcified solitary solid thyroid nodule in her daughter.

Intraoperatively, multiple branches of both recurrent laryngeal nerves were all identified and meticulously preserved. No electrocautery was used near the nerve, and potential bleeders were individually ligated. Shortly after extubation, severe stridor and oxygen desaturation from 100% to 88% at room air were noted. Direct laryngoscopy showed complete bilateral adductor paralysis of the vocal folds, with no perceptible inspiratory abduction. She was re-intubated and monitored overnight in the Post-Anesthesia Care Unit. Dexamethasone 4 mg intravenous was given six hours after the initial 8 mg dose that had been given at the start of surgery. Postoperative hemoglobin/hematocrit and albumin were normal, but calcium decreased further (1.86 mg/dL).

The next morning, a trial of extubation was successful and flexible laryngoscopy revealed fully mobile vocal folds. Fingertip paresthesia and a grade 1 Chvostek sign (with no carpopedal spasm) resolved with intravenous calcium gluconate. She was discharged on oral calcium supplements, and the rest of her postoperative course was uneventful. Final histopathologic diagnosis was "multinodular colloid goiter, bilateral lobes and isthmus; six (6) reactive

parathyroidal lymph nodes." She was well on levothyroxine supplementation alone at six months follow-up. Informed consent was obtained to report this case.

DISCUSSION

Unexpected total bilateral vocal fold paresis following an uneventful thyroidectomy can be devastating for providers (surgeons, anesthesiologists, nurses) and patients (and their families) alike. Regarded as the rarest among all complications post-thyroidectomy, the reported incidence is 0.7%, with it being permanent in 0.3%.^[3] The devastating effect is primarily due to the need for a tracheostomy and the loss of normal speech. Perplexed, we opted to wait for 24 hours and attempt another trial of extubation before converting to a tracheostomy, as we could think of no problems with our surgery. We searched the literature overnight for an explanation.

What could have caused bilateral vocal fold paralysis in our case? Branched recurrent laryngeal nerves may be a risk factor for transient and permanent vocal cord paresis after surgery.^[4,5] In a comparative analysis of 115 postoperative clinical outcomes between patients with and without branching RLN, the presence of branching RLNs had a relative risk of 13.25 for permanent RLN palsy and 7.36 for unilateral transient RLN palsy.^[4] In another study of 222 patients, vocal cord dysfunction (either paralysis or paresis) was twice as common in branched nerves than non-branching nerves with an odds ratio of 2.2.^[5] The hypothesized reason for branching being associated with vocal cord paresis is that more branches are prone to more manipulation/mobilization stress than a single-trunk.^[5] Branching also increases the risk for diathermy injury, despite efforts to visualize and preserve all branches of the RLN.^[4] However, we had dissected meticulously without manipulating nerve branches or employing cautery in their vicinity, individually ligating vascular structures.

Hypocalcemia (which leads to increased neuromuscular irritability and may present with circumoral numbness, paresthesias, muscular cramps, or when severe, seizures) has been noted to cause stridor in a patient due to laryngospasm.^[6] An early report implicated hypovitaminosis D from malabsorption

syndrome as the cause of hypocalcemia resulting in laryngospasm.^[6] Although laryngospasm secondary to hypocalcemia may conceivably give an impression of transient bilateral vocal fold paralysis, our patient did not have laryngospasm and her hypocalcemia was not severe (postoperative corrected calcium= 2.0 mg/dL).

Endotracheal intubation itself has been associated with transient bilateral vocal fold paralysis.^[7-10] Following several reported cases of post-intubation hoarseness, it was proposed that the endotracheal tube cuff can abut on the anterior branch of the recurrent laryngeal nerve, compress it against the posteromedial portion of the thyroid lamina and cause microcirculatory compromise.^[7] This pressure compression can cause ischemic neuronal degeneration and subsequent recurrent nerve paralysis and vocal fold immobility, affecting the cricoarytenoid muscles.^[8] Thru a series of anatomic dissections, the likely site of injury has been hypothesized at the junction of the vocal process of the arytenoid cartilage and the membranous true vocal cord approximately 6 to 10 mm below the level of the cord.^[9] Analysis of cuff pressures during anesthesia indicated that nitrous oxide diffuses into endotracheal tube cuffs causing a substantial increase in intracuff pressure.^[9]

Although not used in this case, Laryngeal Mask Anesthesia (LMA) has also been implicated in transient vocal fold paralysis, with the suspected etiology being compression of the anterior branch of the recurrent laryngeal nerve between the inflated LMA cuff in the midline of the hypopharynx and thyroid cartilage.^[10] Studies have shown a constant linear increase of mean cuff pressure secondary to diffusion of nitrous oxide across the cuff wall of the LMA^[11] potentially causing compression.

Analysis of the risk factors for transient vocal cord paralysis post-tracheal intubation showed it to be three times more common in patients aged 50 and above, two times more in patients intubated 3-6 hours, 15 times more in patients intubated six hours or more, and two times more in patients with diabetes mellitus or hypertension.^[2] Our patient was above 50, hypertensive (with good control) and had only been intubated three hours. Ensuring that the cuff of the endotracheal tube is distal to the cricoid cartilage and that the pressure in the cuff is kept to the minimum

required to prevent a gas leak may minimize this complication.^[12] Avoiding overextension of the neck during intubation, which can stretch and injure both vagus nerves may also prevent bilateral vocal fold paralysis.^[13]

While we cannot isolate the reason(s) for bilateral vocal fold paralysis in our patient, we have identified several possible factors. Indeed, although vocal fold paralysis after head and neck surgery is more likely to be considered a surgical complication, anatomic, metabolic and anesthetic factors-- including endotracheal intubation should not be ignored as a possible cause of this paralysis.^[14]

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