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Transient Unilateral Recurrent Laryngeal Nerve Palsy Presenting as Post-extubation Stridor after Total Thyroidectomy for Non-toxic Multinodular Goiter: Anesthetic Implications and Airway Rescue

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A B S T R A C T

Total thyroidectomy is among the most frequently performed endocrine procedures worldwide, yet it carries a defined risk of recurrent laryngeal nerve (RLN) injury that can precipitate post-extubation stridor and emergent airway compromise. Transient unilateral RLN palsy is estimated to occur in 2 to 10 percent of cases for benign disease and remains an important anesthetic consideration even in carefully planned surgery. We report the case of a 53-year-old woman with controlled hypertension and a longstanding bilateral non-toxic multinodular goiter of 8.5 by 10 centimeters who underwent intraoperative conversion from subtotal to total thyroidectomy under general anesthesia with endotracheal intubation. Intraoperative course was stable on a balanced regimen of midazolam, fentanyl, propofol, atracurium, and isoflurane. Ten minutes after a smooth extubation, she developed inspiratory stridor, suprasternal retractions, and desaturation. Awake reintubation with preserved spontaneous ventilation was performed using low-dose sedation, intravenous lidocaine, and direct laryngoscopy, which simultaneously secured the airway and demonstrated paresis of the left vocal cord while the right cord was mobile. The patient was transferred to the intensive care unit, received systemic methylprednisolone, and was extubated successfully within 24 hours with complete recovery of bilateral vocal cord mobility on follow-up laryngoscopy. In conclusion, awake reintubation with maintained spontaneous breathing is a powerful maneuver that secures the airway and confirms the laryngeal diagnosis at a single procedure. Early systemic corticosteroid, vigilant monitoring, and otolaryngology liaison support rapid neurapraxia recovery, with multidisciplinary cooperation as the cornerstone of a favorable outcome.

1. Introduction

Thyroidectomy is one of the most frequently performed endocrine surgical procedures and is widely used to manage benign multinodular goiter, hyperfunctioning thyroid nodules, suspicious lesions identified by fine-needle aspiration cytology, and well-differentiated thyroid carcinoma.[1,2] The complexity of the operation is determined less by the resection itself than by the anatomical density of structures within the central compartment of the neck, where the trachea, common carotid arteries, internal jugular veins, parathyroid glands, the external branch of the superior laryngeal nerve, and the recurrent laryngeal nerve (RLN) are all closely apposed to the thyroid capsule. Consequently, the perioperative team must be prepared for a spectrum of complications including hemorrhage, hypocalcemia, airway edema, neck hematoma, and, most importantly, injury to the recurrent laryngeal nerve.[1,3,4]

RLN injury is the single most consequential nerve complication of thyroid surgery and the most relevant to the practicing anesthesiologist because of its capacity to

provoke post-extubation stridor and life-threatening airway compromise. The reported incidence of transient unilateral RLN injury after surgery for benign multinodular goiter ranges from 2 to 10 percent, while permanent injury occurs in approximately 0.3 to 2 percent of cases.[4-6] The reported figures vary widely between centers because detection is highly dependent on the timing and modality of postoperative laryngeal evaluation; routine immediate flexible laryngoscopy reveals motion impairment that subjective voice assessment will miss.[3,7,8] Anatomical variation is a powerful but underappreciated modifier of risk, as cadaveric series have shown two to five extralaryngeal RLN branches in up to 89 percent of nerves on the right and approximately three-quarters of those on the left, with non-recurrent variants also described.[8,9]

Post-extubation stridor after thyroidectomy may originate from several mechanisms operating singly or in combination. Mucosal edema from prolonged intubation, surgical manipulation, or fluid shifts can narrow the laryngeal inlet, while unilateral or bilateral vocal cord paralysis can produce mechanical glottic compromise.[10-13] Less common but equally urgent causes include expanding cervical hematoma, tracheomalacia after relief of long-standing compression, hypocalcemic laryngospasm in the setting of acute hypoparathyroidism, and aspiration of secretions or blood. The clinical task at the bedside is to distinguish between these etiologies rapidly because each has a different optimal intervention. Pharmacological measures such as nebulized epinephrine, systemic corticosteroids, and heliox-non-invasive ventilation are appropriate for edematous narrowing.[13,14] Mechanical glottic compromise from RLN injury, by contrast, demands airway control through reintubation, and direct laryngoscopic visualization is the most efficient diagnostic test.[11,12,15]

From the anesthesiologist's perspective, a thyroidectomy patient must be evaluated through four interlocking lenses: preoperative airway prediction, intraoperative airway maintenance, intraoperative protection of the recurrent laryngeal nerve (frequently through intraoperative neuromonitoring), and a deliberate plan for safe extubation and post-extubation surveillance.[1,2,8,16] When these four elements coalesce, even a difficult case proceeds smoothly. When one of them fails, the consequences become apparent within minutes of extubation, often in an environment with fewer resources than the operating room. Despite a robust descriptive literature, granular procedural reports that integrate the cognitive process of airway rescue with bedside diagnosis of nerve injury remain comparatively rare, and that gap motivates the present report.

The principal novelty of this report is the demonstration that awake reintubation with deliberately preserved spontaneous ventilation can serve simultaneously as a definitive airway rescue and as a real-time diagnostic procedure for unilateral RLN palsy after thyroidectomy. Two additional observations strengthen the contribution: transient unilateral palsy occurred after total thyroidectomy for a non-toxic multinodular goiter — a category historically associated with relatively low injury rates — emphasizing that even routine cases are not risk free; and complete bilateral cord mobility recovered within 24 hours under high-dose systemic corticosteroid therapy, consistent with neurapraxia rather than axonotmesis. The aim of this case report is therefore to describe the perioperative anesthetic course of a 53-year-old woman with post-thyroidectomy stridor due to transient unilateral RLN paresis, to articulate the diagnostic value of awake reintubation with preserved spontaneous ventilation as a transferable maneuver across resource settings, and to consolidate a practical anesthesia-led pathway for the recognition and rescue of this complication.

2. Case Presentation

2.1 Anamnesis and history

The full demographic and clinical profile of the patient is consolidated in Table 1 and is summarized in narrative form below. As detailed in Table 1, the patient was a 53-year-old woman with a body weight of 60 kg, height of 155 cm, and body mass index of 24 kg/m² who was referred to the surgical clinic for evaluation of a progressively enlarging bilateral anterior neck mass. The mass had been present for approximately two years, increasing gradually from a small palpable nodule to its referral dimensions of 8.5 by 10 centimeters as recorded in Table 1. The patient denied pain at the mass site, did not report rapid recent growth, and described intermittent mechanical discomfort when turning her head. There was no associated dysphagia, dyspnoea on exertion, weight loss, palpitations, tremor, heat intolerance, or change in voice. The mass had not been previously aspirated or biopsied at the initial referring facility.

Her past medical history was significant for essential hypertension, controlled with oral amlodipine 10 milligrams once daily, with no documented end-organ damage. She had received a course of antithyroid therapy for approximately two months prior to surgical referral, consisting of oral propylthiouracil 100 milligrams three times daily and oral propranolol 10 milligrams three to four times daily, combined with oral prednisone 10 to 20 milligrams once daily. Antithyroid medication had been initiated based on a clinical concern for thyroid hyperfunction during the initial workup at the referring facility; oral prednisone had been added empirically because of a suspected granulomatous component to the enlarging cervical mass and as adjunctive anti-inflammatory therapy. Thyroid function tests on referral demonstrated euthyroid status, and the patient was hemodynamically stable. She denied any prior surgical or anesthetic exposure, had no known drug allergies, did not smoke, did not drink alcohol, and had no family history of thyroid malignancy or anesthetic complications. The patient was assessed as American Society of Anesthesiologists physical status class II, as listed in Table 1.

2.2 Physical and airway examination

Focused airway and systemic findings, also enumerated in Table 1, were as follows. The airway was patent with spontaneous breathing. Mouth opening accommodated three finger-breadths between the upper and lower incisors, the Mallampati score was class II, and dentition was intact with no loose or prosthetic teeth. The neck was mobile in flexion and extension, and the thyromental distance measured 6 centimeters, consistent with the value tabulated in Table 1. The respiratory rate was between 20 and 22 breaths per minute with oxygen saturation of 97 percent on room air; chest auscultation revealed bilateral vesicular breath sounds without rhonchi or wheezing. A large bilateral cervical mass of approximately 8.5 by 10 centimeters was palpable, firm but not tender, with no audible bruit, normal overlying skin, and a smooth surface. The clinical appearance of the bilateral cervical mass with the surgical landmark drawing is shown in Figure 1, panel (a); the photograph has been cropped to exclude the face and any other identifying features in accordance with publication ethics requirements.

Table 1. Demographic, clinical, and airway characteristics of the patient at preoperative evaluation.

Variable	Finding
Age	53 years
Sex	Female
Body weight	60 kg
Height	155 cm
Body mass index	24 kg/m²
ASA classification	II
Comorbidities	Essential hypertension, controlled with amlodipine 10 mg once daily
Duration of neck mass	≈ 24 months, gradually enlarging
Size of neck mass	8.5 × 10 cm, bilateral, firm, non-tender, no retrosternal extension on imaging
Prior medication	PTU 100 mg three times daily; propranolol 10 mg three to four times daily; prednisone 10–20 mg daily — for two months
Airway: Mallampati	II
Mouth opening	3 finger-breadths
Thyromental distance	6 cm
Neck mobility	Full flexion and extension
Respiratory rate	20–22 breaths per minute
Resting SpO_2 on room air	97%
Blood pressure	158/95 mmHg
Heart rate	85 beats per minute, regular
Wayne Index	5 (euthyroid)*
Burch-Wartofsky score	5 (thyroid storm unlikely)†
Glasgow Coma Scale	15

*Notes: *Wayne Index ≤19 supports euthyroid status. †Burch-Wartofsky <25 indicates thyroid storm is unlikely.*

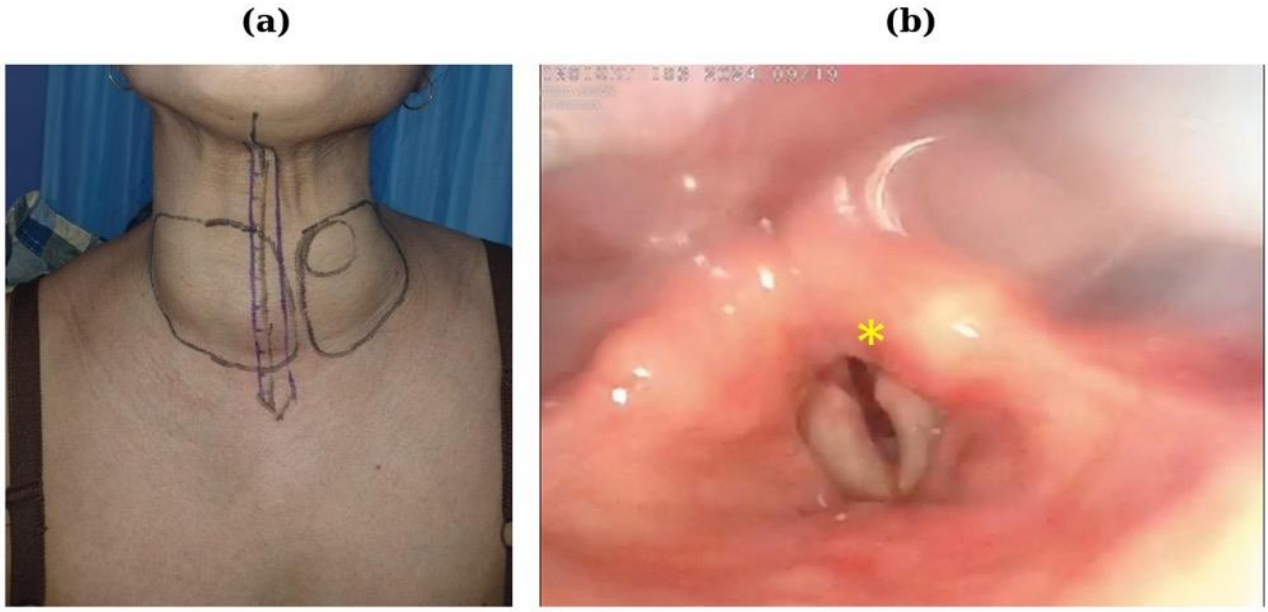

Figure 1. *Composite clinical and laryngoscopic findings of the index patient. (a) Preoperative clinical photograph of the bilateral anterior cervical mass with surgical landmark markings; the image is cropped to exclude the face and identifying features to preserve patient anonymity. (b) Direct laryngoscopic view at the time of awake reintubation showing the paretic left vocal cord (asterisk, yellow) held in a paramedian position while the right vocal cord remains mobile; bilateral cord mobility was confirmed on follow-up laryngoscopy at 24 hours.*

Cardiovascular examination, also tabulated in Table 1, demonstrated a blood pressure of 158 over 95 millimeters of mercury and a regular heart rate of 85 beats per minute. The heart sounds were normal with a single first and second heart sound and no murmurs, rubs, or gallops; capillary refill was less than two seconds. The Glasgow Coma Scale was 15 with no lateralising signs. The abdomen was soft and non-tender without organomegaly, urination and defecation were spontaneous and normal, and the extremities showed no peripheral edema, cyanosis, or clubbing. The Wayne Index for hyperthyroidism scored 5 (consistent with euthyroid status), and the Burch-Wartofsky score for thyroid storm risk was 5 (thyroid storm unlikely), with both scores entered into Table 1 alongside the airway parameters.

2.3 Laboratory and imaging findings

Preoperative laboratory and imaging findings are summarized in Table 2 with reference ranges and short interpretations for each parameter. As shown in Table 2, the complete blood count and coagulation profile were within reference limits (hemoglobin 12.60 g/dL, white blood cell count 7.23 × 10^3/µL, platelets 382 × 10^3/µL, prothrombin time 10.80 seconds with international normalised ratio of 1.04, and activated partial thromboplastin time 26.50 seconds). Renal and hepatic function were normal except for a borderline blood urea nitrogen of 24.8 mg/dL attributable to preoperative fasting, as flagged in Table 2. Serum potassium of 3.21 mEq/L, highlighted in red bold in Table 2, was the only frankly abnormal value; this mild hypokalaemia was consistent with the recent course of corticosteroid therapy. Free thyroxine of 1.14 ng/dL and thyroid-stimulating hormone of 0.38 µIU/mL fell within the reference ranges tabulated in Table 2, confirming biochemical euthyroidism preoperatively and an important determinant of safe induction.

Multiple radiological investigations, also entered into Table 2, established the local anatomy and degree of airway involvement. Chest radiography demonstrated cardiomegaly with otherwise clear lung fields. Cervical anteroposterior and lateral radiography demonstrated bilateral soft-tissue thickening from cervical vertebra C7 to thoracic vertebra T1 consistent with the goiter, with no radiographic evidence of airway compression or retrosternal extension, together with mild cervical spondylosis. Neck ultrasound documented a midline trachea and classified the right lobe as multinodular goiter (TIRADS 3) and the left lobe as containing a TIRADS 2 cyst, as listed in Table 2. Fine-needle aspiration cytology returned a nodular colloid goiter, Bethesda category III (atypia of undetermined significance), supporting a clinical decision toward

surgical resection for definitive histology and to relieve mass effect.

2.4 Perioperative anesthetic plan

Informed consent for general anesthesia and total thyroidectomy was obtained. The complete sequence of perioperative interventions, doses, and clinical responses is consolidated in Table 3. Standard fasting was observed (six hours for solids and two hours for clear fluids), and intravenous access was established with an 18-gauge peripheral cannula. Ringer's lactate solution was administered at 100 milliliters per hour during the fasting interval. One hour preoperatively the patient received intravenous metoclopramide 10 milligrams and ranitidine 50 milligrams to mitigate the risk of aspiration, as recorded in the Premedication row of Table 3. Emergency drugs for thyroid storm were prepared, and a difficult airway cart containing multiple endotracheal tube sizes, supraglottic airway devices, a video laryngoscope, and a flexible bronchoscope was placed at the head of the bed. A nasogastric tube and core temperature probe were positioned after induction. Continuous capnography, electrocardiography, non-invasive blood pressure monitoring, and pulse oximetry were employed throughout the case and were continued in the recovery area.

Table 2. Preoperative laboratory and imaging findings with reference ranges and interpretation.

Parameter	Value	Reference	Interpretation
Free T4 (ng/dL)	1.14	0.93–1.70	Normal
TSH (µIU/mL)	0.38	0.27–4.20	Normal (low-normal)
Sodium (mEq/L)‡	137	135–145	Normal
Potassium (mEq/L)‡	**3.21**	3.5–5.1	**Mild hypokalemia§**
Chloride (mEq/L)‡	100	98–107	Normal
Hemoglobin (g/dL)	12.60	12.0–16.0	Normal
WBC (×10³/µL)	7.23	4.0–11.0	Normal
Hematocrit (%)	39.90	36–46	Normal
Platelet (×10³/µL)	382	150–450	Normal
PT (seconds)	10.80	Control 11.7	Normal (INR 1.04)
aPTT (seconds)	26.50	Control 26.1	Normal
AST (U/L)	23	<37	Normal
ALT (U/L)	13	<41	Normal
Albumin (g/dL)	4.25	3.5–5.2	Normal
BUN (mg/dL)	24.8	**8–24**	Upper limit‖
Creatinine (mg/dL)	0.68	0.6–1.1	Normal
Chest X-ray	Cardiomegaly	—	Lungs clear
Cervical XR	Soft-tissue C7–T1 bilateral	—	No tracheal obstruction; no retrosternal extension
Neck ultrasound	MNG right TIRADS 3; left cyst TIRADS 2	—	Bilateral disease; trachea midline
FNAB cytology	Nodular colloid goiter	—	Bethesda III¶

Notes: ‡mEq/L is equivalent to mmol/L for monovalent ions. §Probably steroid-related and dietary in origin. ‖Mild prerenal contribution from preoperative fasting. ¶Bethesda III: atypia of undetermined significance, favoring surgical management.

Because the patient had been receiving oral prednisone preoperatively, the perioperative team considered the possibility of relative adrenal suppression and elected to give intravenous hydrocortisone 100 milligrams at induction as a conservative stress-dose strategy, as documented in the Stress-dose steroid row of Table 3.[1,2] Induction of general anesthesia was performed with intravenous midazolam 2 milligrams, fentanyl 150 micrograms, propofol 100 milligrams, and atracurium 30 milligrams (Induction row, Table 3). Direct laryngoscopy was straightforward, with a Cormack-Lehane grade II view, and oral endotracheal intubation was achieved on the first attempt with a cuffed 7.0 millimeter internal diameter tube. The tube cuff was inflated to a leak-free seal, and the tube was secured at 21 centimeters at the lip. Anesthesia was maintained with isoflurane in an oxygen-air mixture; muscle relaxation was titrated to surgical requirement (Maintenance row, Table 3). Intraoperative analgesia comprised intravenous ketorolac 30 milligrams during induction and intravenous morphine 5 milligrams prior to skin closure (Intraoperative analgesia row, Table 3). Vital signs remained within acceptable hemodynamic limits throughout the procedure, with the recorded ranges entered into Table 3: heart rate 65 to 82 beats per

minute, systolic blood pressure 115 to 136 millimeters of mercury, diastolic blood pressure 72 to 90 millimeters of mercury, and oxygen saturation of 98 percent or higher continuously.

2.5 Operative course

Surgery began under standard sterile precautions in the supine position with the neck slightly extended on a shoulder roll. The surgical team had initially planned a subtotal thyroidectomy, but on intraoperative assessment found bilateral nodularity that justified conversion to total thyroidectomy in order to minimise the risk of recurrence and avoid a future reoperation in a previously dissected field. Standard surgical technique included identification and ligation of the superior thyroid pedicles, capsular dissection along the lateral lobes with meticulous identification of the parathyroid glands, and identification of the recurrent laryngeal nerve in its course between the inferior thyroid artery and the tracheo-oesophageal groove on each side. Intraoperative blood loss was minimal and the operation lasted approximately 120 minutes. Two parathyroid glands were identified and preserved bilaterally; the surgical specimen was sent for histopathological confirmation.

2.6 Extubation and post-extubation course

At the end of the procedure, neuromuscular blockade was reversed and the patient was extubated when she demonstrated formal extubation criteria: regular spontaneous ventilation with an adequate tidal volume of more than 5 millilitres per kilogram, eye opening to command, sustained head lift greater than five seconds, intact gag reflex, and a satisfactory train-of-four ratio. Initial post-extubation breath sounds were clear without stridor or added sounds (Initial extubation row, Table 3), and the patient was transferred to the post-anesthesia care unit on supplemental oxygen by face mask at 4 liters per minute, with continuous electrocardiographic, blood pressure, capnographic, and pulse oximetry monitoring. Approximately ten minutes after arrival in the recovery area the patient developed acute inspiratory stridor with suprasternal retractions, an increased respiratory rate to 32 breaths per minute, and rapid desaturation from 99 percent to 87 percent on facemask oxygen, exactly as recorded in the Recovery room (T+10 min) row of Table 3. The differential diagnosis at the bedside included expanding cervical hematoma, laryngeal edema, unilateral or bilateral vocal cord paralysis, and laryngospasm. Manual examination of the surgical site did not reveal a tense hematoma, and an immediate decision was made to secure the airway by reintubation.

Awake reintubation was performed under topical local anesthesia, with intravenous lidocaine 60 milligrams as a bolus to attenuate airway reflex while preserving spontaneous ventilation, as detailed in the Awake reintubation row of Table 3. The total intravenous lidocaine dose was within the maximum recommended adult dose of 4.5 milligrams per kilogram and remained well below the systemic toxicity threshold; the patient was monitored continuously for early signs of local anesthetic systemic toxicity including circumoral paresthesia, tinnitus, and dysrhythmia, none of which were observed. Adjunctive sedation was kept deliberately light to permit ongoing laryngeal motion observation: intravenous midazolam 1.5 milligrams, fentanyl 100 micrograms, and propofol 40 milligrams were titrated to comfort without abolishing respiratory drive. Direct laryngoscopy at the moment of reintubation demonstrated paresis of the left vocal cord with the right cord moving normally; the dynamic view was preserved because the patient was spontaneously breathing. The trachea was intubated atraumatically with a cuffed 7.0 millimeter endotracheal tube. The laryngoscopic appearance of the cords at this moment is shown in Figure 1, panel (b), where the yellow asterisk marks the immobile left cord; the same figure also indicates that bilateral mobility had returned by the 24-hour follow-up examination.

The patient was then transferred to the intensive care unit (Intensive care unit row, Table 3) on synchronized intermittent mandatory ventilation with pressure support, sedated to comfort with low-dose propofol, and given intravenous methylprednisolone 31.25 milligrams as the initial dose, followed by a tapering regimen, in line with current evidence supporting systemic corticosteroid administration in airway edema and acute neural injury.[13] Sequential bedside laryngoscopy at 12 hours showed partial return of left cord motion, and by 24 hours both cords moved symmetrically, as summarized in the Response column of the same Table 3 row. A cuff leak test was satisfactory (audible expiratory leak of more than 110 milliliters at the end of expiration with the cuff deflated; Day 1 ICU row, Table 3), and the trachea was successfully extubated on intensive care unit day 1. Following extubation, the patient remained hemodynamically

stable and oxygenated, though her voice was initially mildly hoarse with progressive improvement over the following hours. The patient was discharged from the intensive care unit to the surgical ward on postoperative day 2 in stable condition, without recurrence of stridor (Day 2 row, Table 3).

Table 3. Treatment regimen, dosage, and clinical response during the perioperative and intensive care course.

Phase	Intervention	Response
Premedication	Metoclopramide 10 mg IV; ranitidine 50 mg IV (1 h preop)	Aspiration prophylaxis; well tolerated
Stress-dose steroid	Hydrocortisone 100 mg IV at induction (for preoperative prednisone exposure)	No adrenal crisis observed
Induction	Midazolam 2 mg + fentanyl 150 µg + propofol 100 mg + atracurium 30 mg IV	Cormack-Lehane II view; first-pass intubation
Maintenance	Isoflurane in O_2–air; titrated atracurium boluses	Stable hemodynamics; HR 65–82, BP 115–136/72–90, SpO_2 ≥ 98%
Intraoperative analgesia	Ketorolac 30 mg IV (induction); morphine 5 mg IV (closure)	Adequate analgesia at emergence
Initial extubation	Awake extubation in operating room; formal extubation criteria met	Smooth, no stridor on emergence
Recovery room (T+10 min)	**Inspiratory stridor; suprasternal retractions; SpO_2 87%**	**Airway emergency declared**
Awake reintubation	Lidocaine 60 mg IV + midazolam 1.5 mg + fentanyl 100 µg + propofol 40 mg IV (spontaneous ventilation preserved)*	Left vocal cord paresis observed at direct laryngoscopy; right cord mobile; trachea reintubated atraumatically
Intensive care unit	Methylprednisolone 31.25 mg IV (initial), then tapered regimen†; pressure-support ventilation; serial bedside laryngoscopy	Partial recovery at 12 h; full bilateral mobility at 24 h
Post-ICU analgesia	Ketorolac 30 mg IV three times daily; paracetamol 750 mg PO four times daily	Pain controlled; no NSAID-related adverse event
Day 1 ICU	Cuff leak test satisfactory (leak > 110 mL); extubation	Successful; persistent mild hoarseness only
Day 2	Ward transfer; voice progressively improving	Stable; no recurrence of stridor
Two-week follow-up	Outpatient assessment	Normal voice; no dysphagia or aspiration

*Notes: *Total intravenous lidocaine dose remained below the systemic toxicity threshold (< 4.5 mg/kg). †Methylprednisolone 31.25 mg ≈ dexamethasone 5.9 mg equivalence, consistent with multi-dose regimens shown to reduce post-extubation laryngeal edema in adults.*[13]

2.7 Final diagnosis and outcome

The final operative diagnosis was bilateral nodular colloid goiter with intraoperative conversion from subtotal to total thyroidectomy. The principal anesthetic complication was transient unilateral (left) recurrent laryngeal nerve palsy presenting as post-extubation stridor and respiratory distress, successfully managed with awake reintubation, brief intensive care unit ventilation, and systemic corticosteroid, with complete recovery of bilateral vocal cord mobility within 24 hours, as reflected in the response column of Table 3 and in panel (b) of Figure 1. The patient was discharged in stable condition with mild residual hoarseness that resolved progressively over the following two weeks at follow-up clinic visits. Histopathological examination of the surgical specimen confirmed colloid goiter without evidence of malignancy. Follow-up at three and six months is planned at the outpatient clinic to formally document long-term voice outcomes using the Voice Handicap Index instrument.

3. Discussion

3.1 Epidemiology and risk stratification of recurrent laryngeal nerve injury

Recurrent laryngeal nerve injury remains the most clinically significant nerve complication of thyroid surgery, even at high-volume centers with experienced surgeons.[3,6,8] The risk of transient unilateral injury reported in large case series and meta-analyses ranges between 2 and 10 percent for benign disease, with permanent injury accounting for 0.3 to 2 percent.[4-6] Zakaria and colleagues documented temporary injury in 2.9 percent and permanent injury in 0.33 percent of

benign cases, with the rate climbing to 21.7 percent for recurrent goiter where adhesions and altered anatomy increase nerve vulnerability.[4] Total thyroidectomy is statistically associated with slightly higher transient injury rates compared with subtotal procedures, but the difference narrows with surgical experience, intraoperative neuromonitoring, and protocolized dissection.[5,6] The case described here fits firmly within the expected envelope: a 53-year-old woman with longstanding non-toxic multinodular goiter, whose preoperative parameters tabulated in Table 1 and Table 2 reflect a low-risk patient, underwent total thyroidectomy and developed transient unilateral palsy with full functional recovery within 24 hours.

Risk stratification based on the underlying pathology is more important than the surgical title alone. Hyperthyroid goiter increases vascularity and inflammation around the recurrent laryngeal nerve, while malignancy can entrap the nerve in invasive disease.[3-5] Bilateral disease and intraoperative conversion of plan from subtotal to total resection, as occurred here, can raise risk by extending the dissection field and the duration of nerve exposure. Discussion of these probabilities during preoperative consent is essential, and the consensus statement of the International Neural Monitoring Study Group has proposed a structured informed-consent framework that should be adopted as a standard of practice.[17]

3.2 Anatomic and embryologic determinants of vulnerability

Embryologically the recurrent laryngeal nerve arises from the sixth pharyngeal arch and accompanies the developing aortic arch into the thorax, retaining a recurrent looping course as the cardiac structures descend. The right nerve typically loops under the subclavian artery and ascends in the tracheo-oesophageal groove, while the left nerve descends to loop under the aortic arch before ascending. Anatomic variations are common: cadaveric data show two to five extralaryngeal branches in up to 89 percent of right nerves and approximately three-quarters of left nerves, and non-recurrent right nerves are described in approximately 0.5 to 1 percent of patients, often associated with an aberrant right subclavian artery.[8,9] These variations explain why anatomic dissection alone may not protect every patient and underpin the rationale for intraoperative neuromonitoring.

Mattsson and colleagues describe in detail the pathophysiology of intraoperative nerve injury: most acute injuries are due to traction, compression, or thermal injury rather than transection, producing axonal conduction block (neurapraxia or partial axonotmesis).[18] Neurapraxia preserves axonal continuity and typically resolves within days to weeks, whereas axonotmesis with Wallerian degeneration requires regeneration measured in months. The rapid resolution observed in this case, within 24 hours of injury and depicted in panel (b) of Figure 1, is consistent with neurapraxia rather than axonotmesis, and the prognosis is generally excellent. Clinicians should be cautious about claiming permanent injury before at least six months of expectant observation have elapsed and electrodiagnostic studies have been performed where available.[3,18]

3.3 Preoperative airway and endocrine assessment in thyroidectomy

Anesthetic care of the thyroidectomy patient begins with two parallel evaluations: confirmation of euthyroid status and prediction of airway difficulty.[1,2] Biochemical euthyroidism is necessary to mitigate the risk of perioperative thyroid storm, and clinical scores such as the Wayne Index and the Burch-Wartofsky score remain practical bedside tools — the patient was scored at 5 on both, as recorded in Table 1, predicting low risk.[1] Airway prediction in goiter patients should incorporate Mallampati score, mouth opening, thyromental distance, neck mobility, and importantly, dynamic respiratory symptoms and imaging findings of retrosternal extension, tracheal deviation, or compression.[2,19] When difficulty is anticipated, plans should include awake fiberoptic intubation or video laryngoscopy and the immediate availability of a difficult airway cart. Our patient had a reassuring airway examination, a chest film and cervical radiograph free of compression, and a midline trachea on ultrasound (Table 2); these findings together justified a standard induction. Nevertheless, the bilateral 8.5 by 10 centimeter mass illustrated in Figure 1, panel (a) justified caution and the presence of a difficult airway cart at the head of the bed.

Routine preoperative fiberoptic laryngoscopy is increasingly recommended as a standard of care before any surgery in which the recurrent laryngeal nerve is at risk, to establish baseline cord mobility and to detect occult preoperative dysfunction.[1,8] In our institution this

examination is not yet routine for benign multinodular goiter; however, our experience with this case is part of an ongoing departmental quality-improvement effort to introduce routine preoperative fiberoptic examination for all thyroidectomy patients. The corticosteroid exposure that preceded surgery is also clinically relevant: chronic preoperative corticosteroid may attenuate the hypothalamic-pituitary-adrenal axis response to surgical stress, and a conservative stress-dose strategy such as intravenous hydrocortisone 100 milligrams at induction, listed in Table 3, is reasonable when in doubt.[1] In our patient this dose was administered without adverse effect and without overt evidence of relative adrenal insufficiency.

3.4 Intraoperative neural monitoring and surgical technique

Intraoperative neural monitoring (IONM) is now considered the standard of care in many centers for thyroid and parathyroid surgery, with international standards laid out by Randolph and colleagues and extended to the external branch of the superior laryngeal nerve by Barczyński and colleagues.[8,20] Continuous vagal monitoring and structured loss-of-signal algorithms allow early detection of impending injury, and current consensus emphasises both the safety and ethical advantages of disclosing monitoring to the patient.[17,21,22] Despite this, randomised controlled trials have not shown that IONM reduces permanent injury rates compared with experienced visual identification alone; rather, the strongest signals are reduction in operative time, more accurate prognostic information for the patient, and identification of nerves at risk in re-operative or complex anatomy.[16,21] In this case, although IONM is institutionally available, intraoperative monitoring was not employed; the intraoperative course tabulated in Table 3 was nevertheless smooth and the surgical team was experienced with bilateral nerve identification, factors known to mitigate but not eliminate risk. The episode of transient unilateral palsy in the recovery period — captured in Figure 1, panel (b) — has prompted a local protocol review to consider IONM use in all bilateral thyroid resections going forward.

3.5 Mechanism of stridor in the post-thyroidectomy patient

Stridor immediately after extubation in a thyroidectomy patient is not a unitary diagnosis but a final common pathway. The principal differential diagnoses are post-extubation laryngeal edema, unilateral or bilateral vocal cord paralysis, expanding cervical hematoma, tracheomalacia, and laryngospasm, with rarer etiologies including hypocalcemic laryngospasm from acute hypoparathyroidism, dehiscence of the airway, or aspiration of blood or secretions.[1,10,11,12,13,15] Each has different bedside features and management implications. Bilateral palsy is the most feared because the cords assume a paramedian position and the glottic aperture collapses on inspiration, producing immediate hypoxia; unilateral palsy can also generate stridor if mucosal edema accompanies the cord weakness, as in our case (Figure 1, panel b). Mucosal edema responds to nebulized epinephrine, head-up positioning, systemic corticosteroid, and adjuvant heliox.[13,14] Hematoma demands immediate evacuation. Vocal cord paralysis with critical glottic compromise demands a controlled airway.

3.6 Diagnostic value of awake reintubation with spontaneous ventilation

A central learning point of this case is the diagnostic value of awake reintubation performed with preserved spontaneous ventilation. The conventional approach to emergent reintubation is rapid sequence induction with apnoea, which secures the airway quickly but abolishes the opportunity for dynamic laryngeal observation. By using topical and intravenous lidocaine to blunt reflex, light sedation with midazolam and fentanyl, and a small dose of propofol that did not abolish respiratory drive — the full regimen of which is enumerated in the Awake reintubation row of Table 3 — we maintained the patient's intrinsic ventilatory effort while bringing the larynx into clear view at direct laryngoscopy.[11,12,15] The instant the cords came into view, the left cord was seen to be immobile in a paramedian position with the right cord moving normally, as illustrated in Figure 1, panel (b) — diagnosing unilateral RLN palsy at the same moment that the airway was being secured. This approach has three transferable advantages: it provides physiologic redundancy through preserved spontaneous breathing if intubation fails; it generates immediate diagnostic information that guides ICU management; and it spares the patient an additional invasive procedure such as a separate flexible laryngoscopy at a later moment.

Awake or lightly sedated reintubation requires a cooperative patient, an experienced operator, and

rapidly available rescue options including a video laryngoscope and surgical airway access. The indications for this technique are a cooperative patient who is alert and not severely agitated; oxygen saturation maintained above 90 percent on facemask oxygen; a predicted Cormack-Lehane grade I or II view; and immediate availability of an experienced anesthesia and surgical team. Contraindications include profound hypoxia that does not permit even short delays, severe agitation that prevents safe positioning, anticipated severe Cormack-Lehane grade III or IV anatomy, profound hemodynamic instability, and inability to recognise local anesthetic systemic toxicity at the bedside. Special attention must be paid to local anesthetic systemic toxicity: the maximum recommended dose of intravenous lidocaine in adults is 4.5 milligrams per kilogram, and the team must be vigilant for circumoral paresthesia, tinnitus, dysrhythmia, and altered mental status that signal toxicity. In our case, oxygen saturation rebounded with face mask oxygen and the patient remained alert, providing a workable window to execute the maneuver safely.

3.7 Pharmacologic adjuncts in the management of post-extubation stridor

Pharmacotherapy plays a supporting but not primary role. Pluijms and colleagues describe a multimodal pharmacologic strategy for post-extubation laryngeal edema centered on nebulized epinephrine, multi-dose dexamethasone or methylprednisolone administered 12 to 24 hours before high-risk extubation, and head-up positioning.[13] In our patient the stridor was not anticipated, so prophylactic steroid had not been administered; however, the patient received early postoperative methylprednisolone in the intensive care unit when the diagnosis of laryngeal edema with cord paresis was established, as listed in the Intensive care unit row of Table 3. The mechanism of systemic corticosteroid in this setting is principally a reduction in local edema and inflammatory mediators rather than a direct acceleration of axonal regeneration; the airway benefit is well documented, while the neural-recovery benefit remains more controversial and supported by lower-grade evidence.[13,18] Punj and colleagues have shown that adjuvant heliox delivered through bilevel positive airway pressure can rescue patients with post-extubation stridor refractory to standard medical therapy.[14] In our case heliox was not required because the airway was already secured by reintubation, but it remains a useful tool when the airway is patent and reintubation is contemplated as a last resort.

The cuff leak test deserves separate comment. As shown by Kuriyama and colleagues in a systematic review and meta-analysis, the cuff leak test has high specificity (~0.88) but only moderate sensitivity (~0.66) for predicting post-extubation airway events.[10] A positive cuff leak test (small or absent leak) in a high-risk patient justifies postponement of extubation and corticosteroid administration; a negative test does not guarantee that stridor will not occur and should not engender false confidence. The cuff leak test was originally validated for prolonged intensive care unit intubation, and its predictive performance after short operative intubations is less well established. In our case, the test was applied prior to elective extubation in the ICU at 24 hours and supported safe removal of the tube, as documented in the Day 1 ICU row of Table 3. We did not perform a cuff leak test at initial operating room extubation, a decision that reflects the absence of recognised risk factors apparent at that time but which we now recognise as a learning point for future practice.

3.8 Bedside flexible laryngoscopy as a postoperative tool

Bedside flexible fiberoptic laryngoscopy is a low-cost, low-resource maneuver that should be available in any center performing thyroid surgery and that provides high-yield diagnostic information in the postoperative period.[11,12] In our patient, serial bedside laryngoscopy in the intensive care unit documented the partial recovery of left cord motion at 12 hours and full bilateral mobility at 24 hours, with the recovered appearance captured in the right portion of Figure 1, panel (b), supporting the diagnosis of neurapraxia and informing the decision to extubate. Centers without access to a flexible scope can substitute direct laryngoscopy with a video or standard blade if the patient is intubated or readily intubatable; in non-intubated patients, indirect mirror laryngoscopy by an otolaryngology colleague is an acceptable alternative. The principle is that serial direct observation of vocal cord motion is the single most informative bedside investigation in this clinical context, and reliance on subjective voice assessment alone misses motion impairment in a substantial proportion of patients.[3,7]

3.9 Prognosis and follow-up of unilateral vocal cord palsy

Most acute unilateral RLN palsies after thyroid surgery recover spontaneously, especially when the mechanism is neurapraxia from traction or thermal injury rather than transection.[3,18] Recovery is reported within days to weeks for neurapraxia and weeks to months for axonotmesis. Lynch and Parameswaran describe a graduated treatment algorithm: voice therapy first-line for mild dysphonia, injection laryngoplasty with hyaluronic acid or autologous fat for persistent symptoms beyond approximately 6 weeks, and medialization thyroplasty for confirmed palsy at 6 to 12 months.[3] Wen and Wang, comparing 51 patients with post-thyroidectomy unilateral vocal fold paralysis, showed that medialization thyroplasty produced the largest improvement in voice-related quality of life, followed by hyaluronic acid injection, autologous fat injection, and voice therapy alone.[7] In our patient, the rapid 24-hour recovery of full mobility — also shown in Figure 1, panel (b) — argued against the need for any of these interventions, and follow-up speech assessment two weeks postoperatively demonstrated normal voice quality without dysphagia or aspiration. Formal voice outcomes at three and six months will be captured using the Voice Handicap Index as part of an ongoing departmental follow-up protocol.

3.10 Comparison with similar published cases

Table 4 compares our case with four similar published reports of post-thyroidectomy or thyroid-related airway compromise. As demonstrated in Table 4, the most commonly reported scenarios are delayed presentations of bilateral palsy requiring tracheostomy, whereas our patient illustrates the comparatively favorable outcome of unilateral neurapraxia with rapid functional recovery. In particular, the Sanapala et al. report (row 1 of Table 4) describes successful airway rescue by reintubation in benign goiter, conceptually closest to our case, while the Tan et al. report (row 4 of Table 4) describes anticipatory awake fiberoptic intubation for a massive compressive goiter. The early intervention with awake reintubation and preserved spontaneous ventilation distinguishes the present report (final row of Table 4) from cases that proceeded directly to tracheostomy or relied on rapid sequence induction without dynamic laryngeal observation.

Table 4. Comparison of the present case with similar published cases of post-thyroidectomy or thyroid-related airway compromise.

Reference	Pathology / Procedure	Airway event	Rescue strategy	Outcome
Sanapala et al., 2015 [15]	Total thyroidectomy, benign goiter	Bilateral RLN paresis with post-extubation stridor	Reintubation, ICU corticosteroid	Recovery, tracheostomy avoided
Omura & Kurahashi, 2022 [12]	Post-COVID-19 prolonged intubation (not thyroid)	Delayed bilateral cord paralysis day 4	Awake tracheostomy	Long-term tracheostomy
Belitova et al., 2023 [11]	Total thyroidectomy	Delayed respiratory distress	Stepwise reintubation	Recovery
Tan & Zhang, 2023 [19]	Massive goiter (preop airway compression)	Anticipated difficult airway pre-induction	Awake fiberoptic nasotracheal intubation	Successful intubation, uneventful surgery
Present case, 2026	**Total thyroidectomy for non-toxic MNG***	**Acute post-extubation stridor at 10 min**	**Awake reintubation with preserved spontaneous ventilation; bedside diagnostic laryngoscopy**	**Full bilateral cord mobility at 24 h; uneventful discharge**

*Notes: RLN, recurrent laryngeal nerve; ICU, intensive care unit; *MNG, multinodular goiter.*

3.11 Learning points and a practical rescue algorithm

Several practical lessons can be extracted from this case for trainees in anesthesia and surgery. First, even benign multinodular goiter carries a real risk of RLN injury, and routine extubation does not eliminate the need for vigilant observation in the recovery area. Second, the initial five to fifteen minutes after

extubation are the highest-risk window for post-thyroidectomy airway compromise — exemplified by the T+10-minute event recorded in Table 3 — and continuous bedside monitoring with capnography, pulse oximetry, and the ability to summon airway support within minutes should be standard. Third, the differential diagnosis of post-extubation stridor must be considered systematically rather than reflexively attributing the event to edema alone. Fourth, an awake or lightly sedated reintubation with preserved spontaneous ventilation is a versatile and underused maneuver that provides both control and information, as demonstrated in this case by direct visualization of the paretic cord captured in Figure 1, panel (b). Fifth, early systemic corticosteroid administration and intensive care unit observation give a transient neurapraxia the chance to recover under controlled conditions, and most unilateral palsies will resolve within hours to days if the mechanism is neurapraxia.[3,18] The case suggests a five-step rescue algorithm that may be transferred across centers: (1) recognise post-extubation stridor immediately and call for airway support; (2) rule out cervical hematoma by surface inspection; (3) optimise oxygenation with facemask oxygen and head-up positioning while preparing for reintubation; (4) perform awake reintubation with topical and intravenous lidocaine, light sedation, and preserved spontaneous ventilation, observing the cords during laryngoscopy; (5) transfer to intensive care for systemic corticosteroid, serial laryngoscopy, and structured extubation when the cuff leak test is satisfactory and cord function has recovered.

3.12 Limitations and broader applicability

Several limitations of this report deserve acknowledgement. As a single case it does not establish a generalizable management algorithm, and the favorable outcome may be unrepresentative of patients with axonotmesis or those with bilateral involvement. We did not employ intraoperative neuromonitoring during the procedure, and although the surgical team identified both nerves visually, the absence of IONM precludes definitive comment on the exact intraoperative moment of nerve compromise. Cuff leak testing was not performed at the initial operating-room extubation. We did not formally measure voice-related quality of life with a validated instrument at the time of discharge, although follow-up at three and six months using the Voice Handicap Index is planned. Nevertheless, the case captures common elements of an anesthesia-led airway rescue and is structured to be transferable across centers that share comparable resources. The case was managed in accordance with the principles of the Declaration of Helsinki and the institutional policy on case publication.

4. Conclusion

Transient unilateral recurrent laryngeal nerve palsy is a recognised complication of total thyroidectomy that can present as acute post-extubation stridor and respiratory distress. The key transferable lessons are that post-thyroidectomy stridor demands a structured differential diagnosis with immediate airway control, that awake reintubation with preserved spontaneous ventilation is a powerful diagnostic-and-rescue maneuver, and that early systemic corticosteroid administration combined with intensive care observation allows transient neurapraxia to recover under controlled conditions. Multidisciplinary cooperation between anesthesia, surgery, and intensive care, combined with bedside flexible or direct laryngoscopy and a willingness to act on a five-step rescue algorithm, is the cornerstone of a favorable outcome.

5. References

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