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Difficult Extubation After Thoracoscopic Surgery in a Patient With Hypothyroidism: A Case Report

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Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Patient: Female, 62-year-old
Final Diagnosis: Laryngeal edema • hypothyroidism
Symptoms: Dyspnea • edema • hypoxemia • muscle weakness • stridor
Clinical Procedure: Endotracheal Intubation
Specialty: Anesthesiology

Objective: Rare disease
Background: Hypothyroidism is a common endocrine disorder with a prevalence of 0.4% to 1.7%, affecting predominantly women and older adults. Common anesthetic and surgical complications in patients with hypothyroidism include delayed emergence, myocardial and pulmonary infarction, respiratory arrest, hemorrhage, and fluid or electrolyte imbalance. Severe hypothyroidism can rarely trigger myxedema. This case report describes the case of a patient with hypothyroidism who underwent difficult extubation after thoracoscopic lung surgery.
Case Report: A 62-year-old woman with hypothyroidism developed shortness of breath, stridor, hypoxemia, and decreased muscle strength in the extremities following the removal of a double-lumen endotracheal tube after thoracoscopic resection of the right lung S6 segment. Reintubation revealed marked periglottic edema. Upon admission to the intensive care unit, she presented with lethargy, cold extremities, and edema of the eyelids and dorsum of the feet. A detailed medical history revealed irregular use of levothyroxine sodium and self-discontinuation of the medication for 3 days prior to surgery. Postoperative laryngeal edema was considered the direct cause of extubation difficulty, while inadequately controlled hypothyroidism was identified as a major contributing factor to tissue edema, impaired respiratory muscle function, and delayed postoperative recovery. Following respiratory support and supplementation with levothyroxine sodium, respiratory function and muscle strength gradually improved, and the patient was discharged on the sixth postoperative day.
Conclusions: This case suggests that in patients with hypothyroidism experiencing difficulty with extubation during the peri-operative period, a comprehensive assessment of common causes, such as airway edema and residual neuromuscular blockade, is necessary, and the adverse effects of hypothyroidism on airway edema and recovery from anesthesia should be given due attention.

Keywords: Hypothyroidism • Laryngeal Edema

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Introduction

Hypothyroidism is a common endocrine disorder with a prevalence of 0.4% to 1.7% in the general population and is more prevalent among women and older adults [1]. Common complications of anesthesia and surgery in patients with hypothyroidism include delayed emergence, myocardial and pulmonary infarction, respiratory arrest, bleeding, and fluid and electrolyte imbalances [2]. Under the stress of surgery, patients with severe hypothyroidism may develop the rare condition of myxedema [3]. This case report describes a patient with hypothyroidism who experienced difficulty in extubation following thoroscopic lung surgery.

Case Report

The patient was a 62-year-old woman weighing 60 kg who was scheduled to undergo right lung S6 resection via video-assisted thoracic surgery under general anesthesia. She had a history of hypothyroidism for over 1 year and had been taking levothyroxine sodium tablets (75 µg/day) orally. She had undergone surgical treatment for breast nodules more than 10 years prior. Otherwise, her medical history was unremarkable. Preoperative blood tests, urinalysis, coagulation studies, liver and kidney function tests, electrocardiogram, and echocardiogram revealed no significant abnormalities. Thyroid function tests performed 4 days before surgery showed free triiodothyronine level of 4.40 pmol/L, free thyroxine level of 17.50 pmol/L, and serum thyroid-stimulating hormone level of 2.48 uIU/mL.

The patient fasted for 8 hours as per standard protocol. Upon admission to the operating room, routine tests were performed. The anesthesia induction protocol consisted of 0.5 mg pentobarbital, 200 mg hydrocortisone, 30 mg etomidate, 6 mg remimazolam tosylate, 25 µg sufentanil, and 8 mg vecuronium bromide. Under video laryngoscope guidance, a 35-Fr left-sided video-assisted double-lumen endotracheal tube was inserted to a depth of approximately 27 cm. Respiratory parameters were adjusted until the airway pressure reached 20 cm H₂O. The surgical procedure proceeded smoothly, with no significant fluctuations in vital signs during surgery. The surgery lasted 95 minutes, with 80 minutes of 1-lung ventilation; a total of 1000 mL of fluids was administered. Upon completion of the surgery, the double-lumen tube was retracted to the main trachea, and the patient was transferred to the postanesthesia care unit (PACU).

Upon admission to the PACU, neostigmine and atropine were administered for antagonism. Within 5 minutes, the patient regained consciousness and spontaneous breathing and was able to cooperate with commanded movements. After thorough suctioning, the double-lumen tube was removed. Subsequently,

the patient developed tachypnea and stridor. Oxygen saturation dropped to 80%, and extensive cyanosis appeared on the anterior chest and cold extremities. Oxygen therapy was immediately switched to a mask hyperbaric oxygenation. The team suspected upper airway obstruction; therefore, dexamethasone was administered intravenously, and oxygen saturation gradually returned to 100%. However, after switching to nasal cannula oxygenation, oxygen saturation could not be maintained; therefore, endotracheal intubation was performed. Under video laryngoscopy, severe periglottic edema was observed, making visualization of the glottic cleft difficult. A 6.5# endotracheal tube was barely passed through the glottis. Following intubation, 60 mg of hydrocortisone was administered. The patient was alert but apathetic, stridor had resolved, and the patient was able to move limbs on command, although muscle strength was poor, with a grade 3 muscle strength in all 4 limbs. Breathing was shallow and rapid, and the patient reported shortness of breath. Multiple attempts at extubation failed, and the patient was transferred to the intensive care unit (ICU) while still intubated.

Upon admission to the ICU, arterial blood gas analysis was performed, showing an oxygen partial pressure of 80.2 mm Hg and potassium level of 3.8 mmol/L. Chest color Doppler ultrasound revealed no significant pleural effusion. The patient was placed on mechanical ventilation via endotracheal intubation. The endotracheal tube leak test was positive. The patient was alert but apathetic, with cold extremities and edema of the eyelids and dorsum of the feet. The patient tolerated mechanical ventilation without sedation. Upon further inquiry into the patient's medical history with the family, it was reported that the patient's daily intake of levothyroxine was extremely irregular, and the patient had discontinued it on her own for 3 days prior to surgery. After administering 75 µg of Letis via a nasogastric tube, the patient's respiration and muscle strength improved compared with before. Thyroid function was reevaluated 3 days postoperatively, showing free triiodothyronine level of 3.05 pmol/L, free thyroxine level of 18.60 pmol/L, and serum thyroid-stimulating hormone level of 4.58 uIU/mL. On the third postoperative day, the endotracheal tube was removed, and the patient was transferred back to the ward. She was discharged 6 days postoperatively (see **Figure 1**).

Discussion

The existing literature reports that the incidence of laryngeal edema following endotracheal tube extubation ranges from approximately 1.5% to 26.3% [4]. Common causes include mechanical irritation from intubation, difficult intubation, repeated intubation, and excessive fluid infusion [4-6]. In the present case, timely thyroid hormone testing and laryngeal biopsy were not performed, resulting in a lack of evidence to

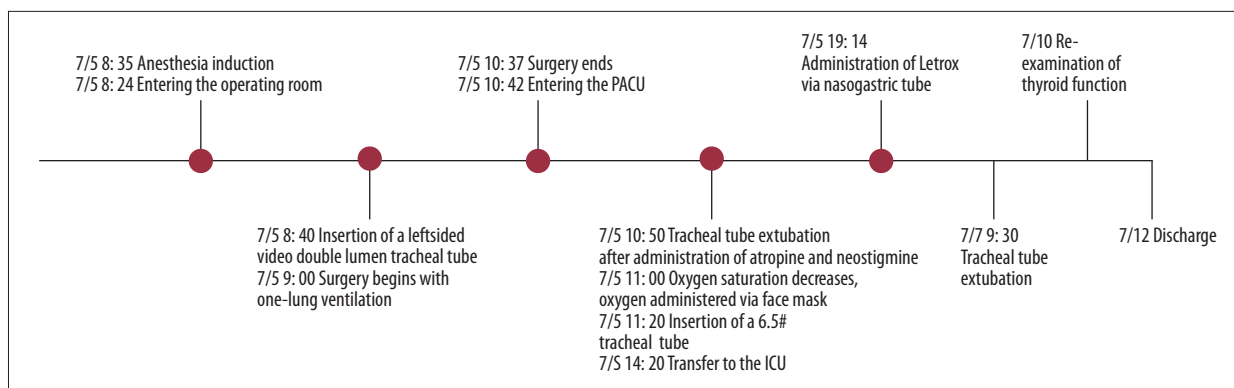


Figure 1. Key events in the case. Note: The patient took levothyroxine continuously from July 5, after the postoperative fasting period ended until July 12, when she was discharged.

directly distinguish among the aforementioned causes; therefore, the direct cause remains unclear. Regarding mechanical stimulation from intubation, the procedure was smooth, without difficulty or repeated attempts, and the duration of 1-lung ventilation was brief. Additionally, the patient exhibited psychiatric symptoms and signs of generalized edema, which do not support mechanical stimulation as the primary cause of laryngeal edema; however, since double-lumen bronchial tubes are thicker than single-lumen endotracheal tubes, intubation and extubation procedures inevitably cause some stimulation, so mechanical factors cannot be completely ruled out. Regarding fluid-related factors, in this case, only 1000 mL of fluid was infused intraoperatively; this relatively small volume is insufficient to explain the laryngeal and systemic edema on its own. However, since there are no complete records of perioperative fluid shifts or infusion rates, the involvement of volume-related factors cannot be entirely ruled out. Regarding factors related to hypothyroidism, the patient had a confirmed history of hypothyroidism, poor medication adherence, and had discontinued medication on her own for 3 days prior to surgery. Following the failed extubation attempt, she exhibited systemic symptoms such as lethargy, cold extremities, decreased muscle strength, and edema of the eyelids and dorsum of the feet, suggesting that hypothyroidism increased susceptibility to tissue edema and respiratory dysfunction [7]. Following experimental thyroid hormone replacement therapy, thyroid function normalized and muscle strength improved by the third postoperative day, further supporting the significant role of hypothyroidism in laryngeal edema and difficult extubation. Myxedema associated with hypothyroidism commonly occurs in the eyelids, periorbital region, face, and the dorsum of the hands and feet, and may also involve the tongue and pharynx [8-10]. In the present case, laryngeal edema was located around the glottis and was accompanied by systemic edema; it responded well to thyroid hormone replacement therapy, consistent with the characteristics of hypothyroidism-related laryngeal edema [8,9]. Hypothyroidism may also increase the risk of postoperative respiratory dysfunction through mechanisms such as reduced

sensitivity of the respiratory center to hypoxia and hypercapnia, decreased respiratory muscle function, and slowed metabolism of anesthetic agents [1,2,7]. Common precipitating factors, such as surgical stress, sedative medications, infection, and hypothermia [3,7], may trigger severe thyroid hormone depletion, particularly when combined with the patient's self-initiated discontinuation of thyroid hormone therapy prior to surgery. However, as no timely, direct evidence of thyroid hormone levels was obtained and no laryngeal biopsy was performed, this diagnosis can only be considered highly suspected; intubation-related stimulation cannot be ruled out. Based on the above analysis, we believe that hypothyroidism was a major cause of the laryngeal edema and difficulty in extubation in this case, while factors such as mechanical stimulation from endotracheal intubation may have played a contributing or exacerbating role on top of this underlying condition.

Timely and accurate diagnosis, along with immediate treatment, is key to managing thyroid crisis. Treatment recommendations call for intravenous thyroid hormone replacement [7,11]; however, as our institution did not have intravenous formulations available, the medication was administered via a nasogastric tube. Recent case reports indicate a favorable prognosis with oral or enteral administration [8,11], and this approach yielded definitive therapeutic results in our case. Respiratory and circulatory support are equally important; however, the role of glucocorticoids remains unclear [11].

This case suggests that even patients with hypothyroidism who have normal thyroid function prior to surgery may experience severe thyroid hormone depletion under surgical stress due to irregular treatment. The American Thyroid Association guidelines recommend that patients with clinical hypothyroidism undergo elective surgery only after thyroid hormone therapy has restored normal thyroid function [12]; although medication may be withheld once on the day of surgery due to fasting, intravenous supplementation should be administered if oral administration is not possible within 5 days

postoperatively [2,12]; during induction, consideration should be given to these patients' increased sensitivity to sedatives and opioids, as well as delayed gastric emptying [2], and dosages should be reduced as appropriate.

Our report also has some limitations: thyroid hormone levels were not measured urgently, no laryngeal biopsy was performed, and no objective neuromuscular function monitoring was conducted; therefore, factors such as residual neuromuscular blockers and mechanical injury cannot be completely ruled out. Levothyroxine sodium was administered at the standard dose, and whether a higher dose is necessary due to poor medication adherence and impaired absorption caused by intestinal edema remains to be investigated.

Conclusions

This case suggests that in patients with a history of irregular long-term thyroid hormone therapy or in those who have recently discontinued treatment, hypothyroidism may contribute to impaired postoperative respiratory recovery through mechanisms such as increased tissue edema, reduced respiratory muscle function, and enhanced sensitivity to anesthetic agents. A thorough perioperative assessment of adherence to thyroid hormone therapy, followed by a multifactorial differential diagnosis and prompt supportive treatment upon the

occurrence of extubation difficulties, may help improve patient outcomes in the future.

Institution Where Work Was Done

Weihai Municipal Hospital, Weihai, Shandong, PR China.

Statement

We have not used AI assistance in the preparation of the manuscript.

Ethics and Informed Consent Statement

This case report obtained written informed consent from the patient, who agreed to the use of their clinical data (including medical history, diagnostic and therapeutic processes, laboratory and imaging results, and figures) for publication in a medical case report. Any personally identifiable information that could reveal the patient's identity has been appropriately redacted in this article.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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