OXFORD

Journal of Surgical Case Reports, 2024, **4**, rjae031
https://doi.org/10.1093/jscr/rjae031
Case Report

Case Report
Hungry bone syndrome following thyroid surgery

María De Armas-Conde*, Ángel Camarasa-Pérez, Rubén García-Martínez, Alejandro Hueso-Mor, Eugenia Caballero-Rodríguez, Julio César Jordán-Balanzá

General and Digestive Surgery Department, Hospital Universitario Nuestra Señora de la Candelaria, 38010 Santa Cruz de Tenerife, Canary Islands, Spain
*Corresponding author. Hospital Universitario Nuestra Señora de la Candelaria, Carretera General del Rosario, 145, 38010 Santa Cruz de Tenerife, Canary Islands, Spain. E-mail: mariadearmasconde@gmail.com

Abstract

The diagnosis of hypocalcemia-induced tetany following a total thyroidectomy is not common. However, there is a higher risk in patients with a history of gastric bypass surgery due to their malabsorption condition. This case describes postoperative hungry bone syndrome resulting from chronic malabsorption in a patient with a history of bariatric surgery. It is important to consider alternative treatment options if the initial management proves ineffective. Typically, this is a temporary condition, but it's crucial to prioritize prevention in high-risk patients by providing perioperative calcium and vitamin D supplementation.

Keywords: hypocalcemia; total thyroidectomy; thyrotoxicosis; hungry bone syndrome; bariatric surgery; indocyanine green fluorescence

Introduction

Hypocalcemia is the most common complication following total thyroidectomy, with an incidence of 27.4% for transient hypocalcemia and 12.1% for permanent hypocalcemia. The etiology can be attributed to intraoperative trauma, removal or devascularization of the parathyroid glands [1, 2].

Hungry bone syndrome (HBS) is an underdiagnosed etiology in patients with hypocalcemia, defined by calcium levels <8.4 mg/dL persisting for at least 4 days. It occurs more frequently during the postoperative period in patients undergoing thyroidectomy for thyrotoxicosis [3].

This may be exacerbated in patients with a history of bariatric surgery. Roux-en-Y gastric bypass (RYGB) achieves weight loss primarily through malabsorption, which can lead to vitamin and mineral deficiencies: calcium (15–48%) and vitamin D (30–60%) [3–5]. This combination of HBS and malabsorption introduces an elevated risk of hypocalcemia following a total thyroidectomy [4, 5].

Case report

A 53-year-old female with a medical history of morbid obesity, who had been treated with RYGB 7 years prior, was diagnosed with Graves disease. She was diagnosed with exophthalmos that was initially managed with methimazole and orbital decompression surgery but showed no clinical improvement. Subsequently, total thyroidectomy was performed.

During surgery, both superior and inferior parathyroid glands were identified and preserved. Their vascularization was confirmed using indocyanine green fluorescence (ICG) imaging, with a score of 2 for each gland. Bilateral recurrent laryngeal nerves were also identified, and intraoperative neuromonitoring was employed.

She had an uncomplicated postoperative course and was discharged on the first postoperative day with outpatient treatment: calcium carbonate (500 mg, 2 tablets/6 h), calcitriol (0.5 mcg/24 h) and levothyroxine (100 mcg/24 h).

Three days after surgery, she experienced paresthesias, muscle cramps and tetanic contractions in her extremities. Blood test revealed hypocalcemia, with a serum corrected calcium level of 5.8 mg/dl, while phosphorus and magnesium levels remained within normal limits.

She was initiated on high-dose intravenous calcium gluconate at a rate of 60 ml/day. However, due to persistently abnormal serum calcium levels and ongoing symptoms (paresthesia sans muscle cramps) after 2 days of intravenous treatment, an additional booster dose of 20 ml of calcium over 20 min was required (235 mg/h of calcium for 1 week). Furthermore, her serum parathyroid hormone level was found to be at 1.3 pc/mL (normal range: 15–125 pc/mL).

Subsequently and after 1 week of treatment, we initiated parathyroid hormone (PTH) 1–34 treatment (Teripatida) with an increasing serum calcium level to 8 mg/dL. Normocalcemia was sustained after the gradual discontinuation of intravenous calcium supplementation.

Received: December 3, 2023. **Accepted:** January 14, 2024

Finally, she was discharged one and a half months after surgery with no symptoms, normal PTH levels and outpatient treatment, which included teriparatide, calcium carbonate, calcitriol and a levothyroxine dosage.

Discussion

Day-case thyroid surgery has become the standard practice, as the incidence of certain complications such as hemorrhage or recurrent laryngeal nerve palsy has decreased. Nonetheless, hypocalcemia remains the most common complication, underscoring the importance of identifying patients with perioperative risk factors for PH. Fortunately, this condition is typically transient, as the parathyroid glands tend to recover their function [1].

Hypocalcemia typically manifests within the first 24–48 h following surgery. As part of our routine post-total thyroidectomy practice, we administer oral calcium and vitamin D supplements to prevent or alleviate hypocalcemia symptoms. Despite these, our patient experienced severe and symptomatic PH, necessitating the administration of high doses of intravenous calcium gluconate, calcium acetate and vitamin D. Additionally, it is advisable to address magnesium levels, as magnesium deficiency can lead to decreased PTH secretion. If intravenous prove ineffective, consideration may be given to PTH analog teriparatide, thiazide diuretics and pancreatic lipase [6–8].

Thyrotoxicosis can induce bone mineral loss by activating osteoclasts and osteoblasts through thyroid hormones, leading to increased bone resorption and bone formation. Following thyroidectomy, bone formation surpasses resorption, causing skeletal absorption of calcium from blood. It results in severe and persistent hypocalcemia, known as HBS [3].

HBS occurs in 12–13% of patients undergoing parathyroid and thyroid surgery, but in individuals with history of Graves's disease the incidence rises to 47%. The diagnosis is based on the presence of severe hypocalcemia (<8.5 mg/dL) resulting from extensive bone remineralization, concomitant with normal or low levels of phosphorus (<3 mg/dL) and elevated alkaline phosphatase. This acute hypocalcemia typically presents with carpopedal spasms (a positive Trousseau sign) and paresthesias, as observed in our patient. Magnesium plays a crucial role in the rate of calcium normalization, as its deficiency can suppress PTH secretion. Although magnesium levels in our case remained within the normal range (0.73–0.83 mmol/L), serum values may not accurately reflect the total body content of this intracellular ion [3, 9–11].

Bariatric surgery, for instance, is widely recognized for its role in inducing nutritional deficiencies. The primary site of dietary calcium absorption occurs in the duodenum, accounting for 80–100% of absorption. In procedures like RYGB, the duodenum is bypassed within the gastrointestinal tract, leading to reduced calcium absorption via a less efficient transport mechanism. Furthermore, vitamin D deficiency is a concern due to diminished intestinal absorption as well [2, 5, 6, 12].

Malfunctions of the parathyroids due to surgical stress during the operation or accidental excision can serve as an additional risk factor for PH. ICG fluorescence can be valuable for confirming the anatomical integrity of parathyroids and assessing their vascularization, thus aiding in the prediction of postoperative outcomes (92% sensitivity, 83% specificity). During the surgical procedure, we successfully identified all four parathyroid glands without congestion, and we assessed their vascularization using ICG, resulting it excellent for each gland [11–13].

In conclusion, the interplay of factors related to prior bariatric surgery, the chronic malabsorption of calcium and vitamin D, preexisting thyrotoxicosis, and post-surgery parathyroid gland dysfunction, culminated in a severe and prolonged hypocalcemic episode [9–11]. Therefore, it is crucial to emphasize the importance of preventive measures against PH in those high-risk patients. This can include administering calcium and vitamin D supplementation 4–6 weeks before surgery and initiating intravenous calcium gluconate after the operation [4, 7, 14].

Conflict of interest statement

None declared.

Funding

None declared.

References

1. Azadbakht M, Emadi-Jamali SM, Azadbakht S. Hypocalcemia following total and subtotal thyroidectomy and associated factors. *Ann Med Surg (Lond)* 2021;**66**:102417. https://doi.org/10.1016/j.amsu.2021.102417.
2. Shuchleib-Cung A, Garcia-Gordillo JA, Ferreira-Hermosillo A, Mercado M. Risk factors for hypocalcemia after total thyroidectomy. *Cir Cir* 2022;**90**:765–9. https://doi.org/10.24875/CIRU.21000579.
3. Kusuki K, Mizuno Y. Hungry bone syndrome after thyroidectomy for thyroid storm. *BMJ Case Rep* 2019;**12**:e231411. https://doi.org/10.1136/bcr-2019-231411.
4. Spartalis E, Thanassa A, Athanasiadis DI, *et al.* Post-thyroidectomy hypocalcemia in patients with history of bariatric operations: current evidence and management options. *In Vivo* 2019;**33**:1373–9. https://doi.org/10.21873/invivo.11614.
5. Cali B, Hasani A, Buffet C, Menegaux F, Chereau N. Is there a relationship between different types of prior bariatric surgery and post-thyroidectomy hypocalcemia? *Gland Surg* 2021;**10**:2088–94. https://doi.org/10.21037/gs-21-225.
6. Lorenz FJ, Tucker J, Milarachi EN, Hearn M, King TS, Goldenberg D. Hypocalcemia after thyroidectomy in patients with prior bariatric surgery: a propensity score matched analysis from a national administrative database. *Thyroid* 2022;**32**:1382–91. https://doi.org/10.1089/thy.2022.0312.
7. Morales-Conde S, Licardie E, Alarcón I, Balla A. Indocyanine green (ICG) fluorescence guide for the use and indications in general surgery: recommendations based on the descriptive review of the literature and the analysis of experience. *Cir Esp (Engl Ed)* 2022;**100**:534–54. https://doi.org/10.1016/j.ciresp.2021.11.018.
8. Pepe J, Colangelo L, Biamonte F, Sonato C, Danese VC, Cecchetti V, *et al.* Diagnosis and management of hypocalcemia. *Endocrine* 2020;**69**:485–95. https://doi.org/10.1007/s12020-020-02324-2.
9. Witteveen JE, van Thiel S, Romijn JA, Hamdy NAT. Hungry bone syndrome: still a challenge in the post-operative management of primary hyperparathyroidism: a systematic review of the literature. *Eur J Endocrinol* 2013;**168**:R45–53. https://doi.org/10.1530/EJE-12-0528.
10. Guo Z, Zhao L, Xie Y, Yan Y, Mo Z. Hungry bone syndrome secondary to subtotal thyroidectomy in a patient with thyrotoxicosis. *Am J Med Sci* 2021;**362**:314–20. https://doi.org/10.1016/j.amjms.2021.02.006.
11. Chandran M, Bilezikian JP, Salleh NM, Ying H, Lau J, Lee J, *et al.* Hungry bone syndrome following parathyroidectomy for

primary hyperparathyroidism in a developed country in the Asia Pacific. A cohort study. *Osteoporos Sarcopenia* 2022;**8**:11–6. https://doi.org/10.1016/j.afos.2022.03.004.

12. Vidal Fortuny J, Belfontali V, Sadowski SM, Karenovics W, Guigard S, Triponez F. Parathyroid gland angiography with indocyanine green fluorescence to predict parathyroid function after thyroid surgery. *Br J Surg* 2016;**103**:537–43. https://doi.org/10.1002/bjs.10101.
13. Kim DH, Kim SH, Jung J, Kim SW, Hwang SH. Indocyanine green fluorescence for parathyroid gland identification and function prediction: systematic review and meta-analysis. *Head Neck* 2022;**44**:783–91. https://doi.org/10.1002/hed.26950.
14. Verma H, Arun P, Sharan R, Manikantan K, Jain P. Risk of Hypocalcemia and the need to augment calcium supplementation after total thyroidectomy. *Indian J Surg Oncol* 2022;**13**:7–10. https://doi.org/10.1007/s13193-020-01098-3.