



Hungry bone syndrome after thyrotoxicosis factitia complicated with thyroid storm: a case report

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Background: Although the hungry bone syndrome (HBS) is a well-established thyroidectomy hypocalcemic complication in Graves' disease, other etiologies of hypocalcemia after thyrotoxicosis treatment are rare. We aimed to report the first case of persistent hypocalcemia after thyrotoxicosis factitia treatment.

Case Description: A 56-year-old woman presented a thyroid storm at the Emergency Department of a referral hospital in Bangkok, Thailand. Thyrotoxicosis factitia was diagnosed due to a low thyroglobulin (Tg) level together with a history of using levothyroxine as an over-the-counter weight loss pill. Systemic glucocorticoid, together with comprehensive end-organ supportive therapy, was administered to achieve euthyroidism. During admission, the serum calcium level gradually decreased to as low as 6.25 mg/dL, whereas serum intact parathyroid hormone and the alkaline phosphatase (ALP) level considerably increased. Intravenous calcium gluconate, oral calcium carbonate, and vitamin D were administered aggressively to treat severe hypocalcemia. Oral administration of calcium carbonate, ergocalciferol, and alphacalcidol was successfully reduced and discontinued two months after the patient was discharged from the hospital.

Conclusions: The abrupt discontinuation of exogenous levothyroxine in the thyrotoxicosis factitia led to an acceleration of the skeletal calcium uptake and resulted in hypocalcemia. Therefore, the close monitoring of the calcium and bone mineral metabolism in patients diagnosed with thyrotoxicosis factitia receiving treatment is recommended.

Keywords: Thyrotoxicosis factitia; hungry bone syndrome (HBS); persistent hypocalcemia; case report

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Introduction

The hungry bone syndrome (HBS) is reported as a well-established thyroidectomy complication of in Graves' disease, especially in the case of severe thyrotoxicosis. This phenomenon is caused by a rapid increase in the skeletal uptake of blood calcium leading to persistent symptomatic hypocalcemia (1,2). The severity of hypocalcemia would depend on the severity of the thyrotoxicosis, previous low bone mass density (BMD), and the degree of the elevated

preoperative serum alkaline phosphatase (ALP) (3). Although the incidence is still unclear, there have been many observational trials and case reports (3-5). Apart from post-thyroidectomy hypoparathyroidism and reversal of the high bone turnover in Graves' disease, other etiologies of hypocalcemia after thyrotoxicosis treatment have been rarely reported. To our knowledge, there have been no case reports of hypocalcemia originating from the HBS after thyrotoxicosis factitia treatment.

Therefore, we aimed to report the first case of thyrotoxicosis

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factitia presenting a thyroid storm complicated by profound hypocalcemia after discontinuing the offending drug. Hence, we present this case in accordance with the CARE reporting checklist (available at <https://aot.amegroups.com/article/view/10.21037/aot-21-18/rc>).

Case presentation

Clinical history

A 56-year-old married woman with a history of intermittent treatment of bipolar disorder and body dysmorphism was admitted to our hospital with thyrotoxicosis factitia in 2017. She had been previously healthy. Her condition was diagnosed by a history of taking diet pills (identified as levothyroxine tablets) and signs of thyrotoxicosis. A laboratory investigation showed incomplete suppression of thyroid-stimulating hormone (TSH), very high value of free thyroxine (FT4) compared to free triiodothyronine (FT3), and a markedly decreased level of serum thyroglobulin (Tg). She was treated with cholestyramine and dexamethasone and was discharged in a full recovery condition. Then, the patient became lost in the follow-up shortly after being discharged.

Two years later, in 2019, the patient presented herself at the Emergency Department with alteration of consciousness and shortness of breath with signs of thyrotoxicosis, which was similar to the first episode. We were informed that the patient had again taken large indeterminate doses of levothyroxine tablets as diet pills for a four-month duration and did not do the follow-up with her psychiatrist.

Physical examination

On admission, the patient was 155 centimeters (cm) tall and weighed 43 kilograms (kg), with a body mass index (BMI) of 17.92 kg/m². She lost 6 kg in four months (changes in bodyweight of 49 to 43 kg and BMI of 20.42 to 17.92 kg/m²). The physical examination at the Emergency Department revealed an irregular heart rate of 169 beats per minute, rapid and shallow breathing patterns at a rate of 28–32 per minute, and fine crepitation in both lower lung areas. The thyroid gland was only around 10 grams, almost non-palpable, and without thyroid bruit. She had tremors of both hands and sweating palms.

Laboratory and radiographic investigations

The laboratory data were as follows: TSH <0.005 μ IU/mL

(0.27–4.20 μ IU/mL), FT4 >7.77 ng/dL (0.93–1.70 ng/dL), FT3 24.57 pg/mL (2.0–4.40 pg/mL), serum Tg 1.42 ng/mL (3.50–77 ng/mL), antithyroglobulin antibody 0.77 IU/mL (<4.11 IU/mL), and TSH receptor antibody <0.8 IU/L (<1.75 IU/L). The albumin corrected calcium, ALP, and serum creatinine values were in a normal range (*Tables 1,2*). The chest radiography showed cardiomegaly with pulmonary congestion. Acute respiratory failure was diagnosed, and the patient was immediately intubated, assisted with mechanical ventilation, and transferred from the Emergency Department to the intensive care unit (ICU). The Burch and Wartofsky score (BWS) was 80, which was highly suggestive of a thyroid storm. The bedside thyroid color-flow Doppler sonography revealed a normal size thyroid gland with decreased blood flow. The echocardiography showed cardiomyopathy with reduced left ventricular ejection fraction (LVEF) to 42%, despite normal coronary angiography (CAG) afterward. As a result of the clinical history and investigation, again thyrotoxicosis factitia was diagnosed, but this episode was additionally complicated with a thyroid storm.

Management of thyroid crisis

The offending drug (levothyroxine) was immediately stopped, and intravenous dexamethasone 4 mg was initiated every 12 hours with the aim to decrease the peripheral conversion of thyroxine. Propranolol was not prescribed according to her active low-output heart failure on top of the thyrotoxic dilated cardiomyopathy. Seventy-two hours after admission to the ICU, the patient developed flank pulmonary edema with cardiogenic shock. The cardiologist initiated dobutamine as an inotropic agent to rescue the hemodynamics. The cardiorenal syndrome subsequently occurred; thus, emergent continuous renal replacement therapy was also initiated on the seventh day of admission. After intensive organ support therapy and treatment of the thyroid crisis, the patient's FT3 and FT4 levels dramatically declined to a near-normal range (*Table 2*), and the patient gained self-consciousness, so we discontinued dexamethasone on the fourth day of admission. The patient also resumed her normal renal function and discontinued renal replacement therapy within one week.

Clinical progression and HBS

Two weeks after admission, the patient complained of a tingling sensation and muscle cramps. Her serum calcium values gradually decreased to as low as 6.25 mg/dL

Table 1 Laboratory data on admission

Laboratory parameters	Results
Hematology	
White blood cell (cell/mm ³)	11,360
Hemoglobin (g/dL)	10.2
Platelet ($\times 10^3/\text{mm}^3$)	217
Blood chemistry	
Glucose (mg/dL)	79
Creatinine (0.5–1 mg/dL)	0.67
Sodium (135–145 mEq/dL)	137.9
Potassium (3.5–4.5 mEq/dL)	3.82
Carbon dioxide (21–31 mEq/dL)	19
Calcium/albumin corrected calcium (8.8–10.6 mg/dL)	9.8/10
Phosphate (2.5–4.5 mg/dL)	2.8
Albumin (3.5–4.5 g/dL)	3.74
Magnesium (1.8–2.6 mg/dL)	2.0
ALP (30–120 IU/L)	119
25(OH) vitamin D (≥ 30 ng/dL)	8.5
Thyroid autoantibody	
Anti-thyroglobulin (<4.11 IU/mL)	0.77
TSH receptor antibody (<1.75 IU/L)	<0.8
Tg (3.50–77 ng/mL)	1.42

Units and normal ranges are provided in parentheses. ALP, alkaline phosphatase; 25(OH) vitamin D, 25-hydroxy vitamin D; TSH, thyroid-stimulating hormone; Tg, thyroglobulin.

Table 2 Thyroid function test during admission

Laboratory value	Day 0	Day 1	Day 3
TSH (0.27–4.2 $\mu\text{IU/mL}$)	<0.005	0.006	0.024
FT3 (2–4.4 pg/mL)	24.57	2.3	1.64
FT4 (0.93–1.7 ng/dL)	>7.77	7.16	2.51

Units and normal ranges are provided in parentheses. TSH, thyroid-stimulating hormone; FT3, free triiodothyronine; FT4, free thyroxine.

(8.8–10.6 mg/dL), the serum phosphate level was 2.5 mg/dL (2.5–4.5 mg/dL), serum magnesium level was 1.3 mg/dL (1.8–2.6 mg/dL), and ALP level increased to 139 IU/L (30–120 IU/L) (*Figure 1*). The intact parathyroid hormone level was increased to 78 pg/mL (15–68 pg/mL) (measured when the calcium value was 8.8 mg/dL). The plain radiograph, thoroughly reviewed by the radiologist, revealed a markedly advanced-than-age radiolucency of

both distal clavicles and non-union right distal clavicular fracture without appropriate callus, which was referred to as thyrotoxic osteodystrophy (*Figure 2*). The HBS was diagnosed and calcium gluconate was administered intravenously since early calcium descent. After 10 days of intravenous infusion of calcium gluconate (2,500 mg of calcium gluconate per day on average, equivalent to 225 mg of elemental calcium) together with oral administration

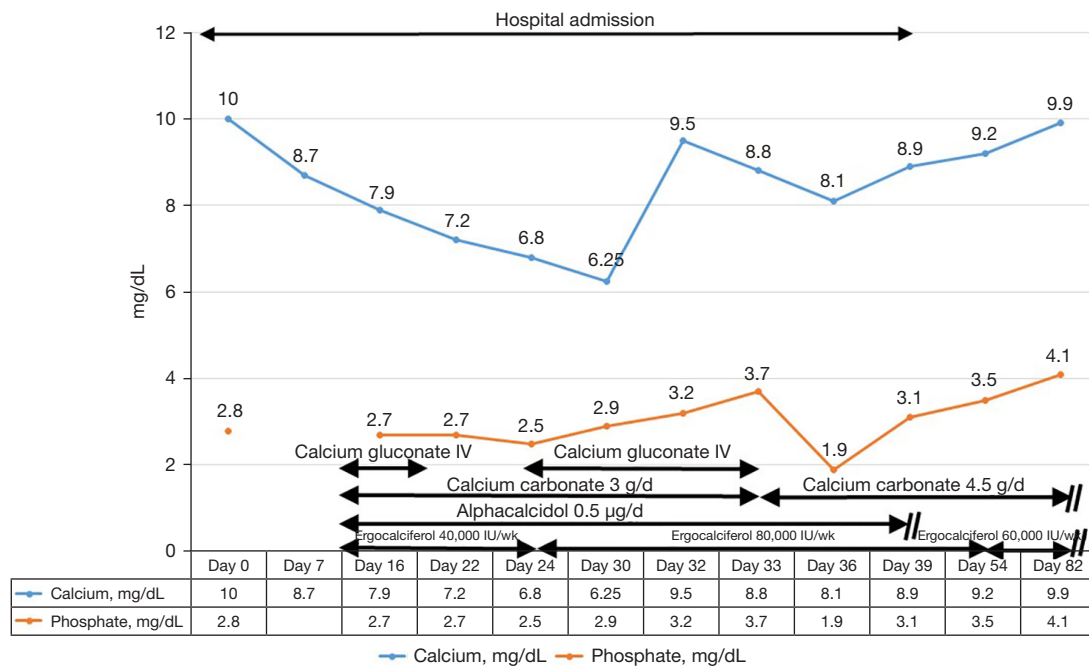


Figure 1 Trends of calcium level, phosphorous level, and management during the course of hospital stay.

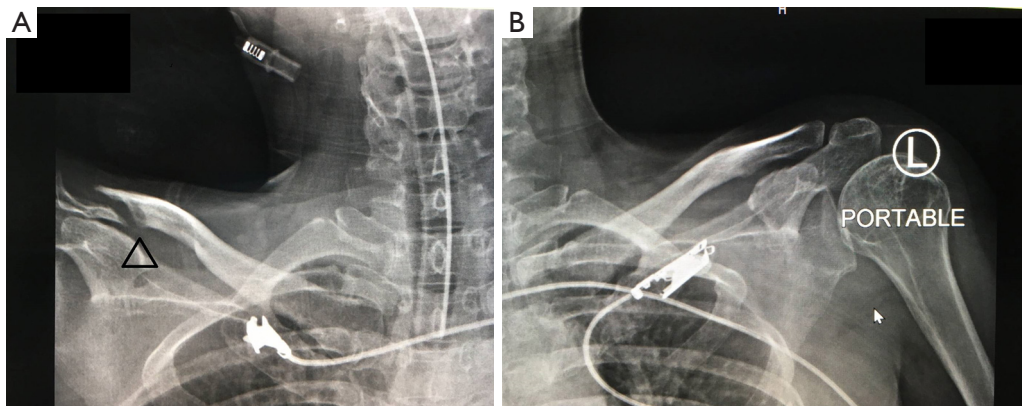


Figure 2 Plain radiograph of patient’s bilateral clavicular bones. (A) Right clavicular bone with non-union fracture (black open arrow). (B) Left clavicular bone.

of precipitated calcium carbonate 3 g/day (equivalent to 1,200 mg of elemental calcium per day), ergocalciferol 40,000 IU/week, and alphacalcidol 0.5 µg/day, the serum calcium level, paresthesia, and cramping eventually ameliorated (Figure 1). Throughout the hospital admission, the patient strictly needed oral calcium and vitamin D supplement. Tapering both agents had been tried but failed due to recurrent symptomatic hypocalcemia. Finally, two months after being discharged from hospital, the

patient gained her normal weight (49–50 kg) with clinical euthyroidism. The serum calcium, phosphate, and ALP returned to normal levels, so oral administration of calcium, ergocalciferol, and alphacalcidol was successfully reduced and eventually discontinued without any adverse events (Figure 1). All of the calcium values in this report were displayed as corrected calcium by Payne’s formula; in the case of low albuminemia (<4.0 g/dL), the corrected calcium (mg/dL) was calculated by total calcium (mg/dL)

+ 0.8 × [4.0 – albumin (g/dL)] (6).

After successful calcium and vitamin D discontinuation, the patient again shortly lost contact with our clinic and her psychiatrist; therefore, we could not obtain her informed consent. We nevertheless reassured for non-personally identifiable patient's specific information throughout the report. All procedures performed in this study were in accordance with the ethical standards of the institutional research committee and with the Helsinki Declaration (as revised in 2013).

Discussion

To the best of our knowledge, this was the first case of hypocalcemia resulting from HBS after thyrotoxicosis factitia treatment. The patient had been exposed to a large dose and long duration of levothyroxine enough to produce thyrotoxic changes in many organ systems, including bone and mineral metabolism. There were three obvious clinical clues. Firstly, the patient already had thyrotoxic osteodystrophy, which was the most important risk factor of developing HBS (3,7,8). Secondly, the patient had thyrotoxicosis factitia with thyroid crisis, rarely reported, but associated with the massive amount and, especially, longer duration of levothyroxine abuse (1,9,10). Lastly, the patient already had thyrotoxic dilated cardiomyopathy with acute low-output heart failure, which was strongly associated with untreated long-standing and severe thyrotoxicosis (11).

Thyrotoxic dilated cardiomyopathy is a rare condition accounting for less than 1% of initial thyrotoxicosis presentation (12) of which the hyperadrenergic state compensates in maintaining the cardiac output. This raised the concern of using beta-blockers in the management of the active low-output heart failure in thyrotoxic dilated cardiomyopathy, which could possibly halt this compensation and lead to abruptly reduced cardiac output and, consequently, hemodynamic instability (13).

Thyrotoxicosis is one of the established causes of increased bone turnover, both bone resorption and formation (14). In the state of thyrotoxicosis, excess T₃ binds to the thyroid hormone receptor alpha-1 subtype in the nucleus of both the osteoblasts and osteoclasts and activates new shortening remodeling cycles (14). The remodeling cycle even shows an imbalance between the resorption and formation that leads to a net loss of approximately 10% of bone per cycle (15). This would eventually result in a decrease in bone mass and increase

fragility fracture in thyrotoxicosis patients on both the trabecular and cortical bones, referred to as thyrotoxic osteodystrophy (3,14,16). The prospective study revealed subsequent significant improvement of BMD during 18±3 months of antithyroid treatment (17) and the fracture risk returned to normal more than one year afterward (16).

The calcium derangement in thyrotoxicosis, owing to the bone remodeling effect, was also a concerning issue. Treating with antithyroid medication, the thyroid hormone levels gradually decreased to a euthyroid state; thus, the high normal total serum level of calcium and urinary calcium excretion at diagnosis decreased without any symptoms, and the serum PTH level slightly increased during the follow-up (17). In contrast, the sudden decline of thyroid hormones would lead to rapid reversal of bone turnover and, consequently, a rapid skeletal uptake of calcium from the serum, thus causing hypocalcemia (3,7). This phenomenon was occasionally complicated with prolonged hypocalcemia with a simultaneous fall in the serum phosphorus and magnesium levels but with an appropriate increase in the PTH level, specifically called HBS (3,7,8). The clinical course of HBS would depend on the etiology of the excess bone remodeling state. In primary hyperparathyroidism, transient hypocalcemia occurs within 18 hours after parathyroidectomy and remains low up to several months (18). In thyrotoxic osteodystrophy, transient hypocalcemia is less severe and starts at 72- or 96-hour post-thyroidectomy and may last for up to 12 weeks (7,19).

Interestingly, this patient showed gradually progressive hypocalcemia, hypophosphatemia, and hypomagnesemia, defined as HBS, which started in the second week and peaked at the fourth week after the discontinuation of the offending drug. There were also case reports of HBS post thyrotoxicosis treatment (antithyroid medication, radioactive iodine therapy, and subtotal thyroidectomy) of which the onset beyond 96 hours, varied between 2 to 6 months (5,8,19) (Table 3). All of them concordantly had thyrotoxic osteodystrophy at the diagnosis, which was defined by osteopenia in either a plain radiograph or BMD. The presumably rather slow decline of the thyroid hormones after the treatment, compared to typical HBS post total thyroidectomy, may contribute to this phenomenon.

Additionally, a gradual decline of the tissue thyroid hormone might play a role in the prolonged onset of HBS in this patient. The pharmacokinetic study in a patient with massive levothyroxine ingestion illustrated that the serum T₄ half-life declined from seven days in euthyroid

Table 3 Reported cases of HBS post thyrotoxicosis treatment of which the onset prolongs beyond 96 hours, including ours

Age (y)/ ethnicity/sex	Thyrotoxicosis etiology	Duration of thyrotoxicosis (mo)	Definite thyrotoxicosis treatment	Onset of HBS since definite treatment (mo)	Serum Ca (mg/dL)	Serum PO ₄ (mg/dL)	Serum Mg (mg/dL)	Serum PTH (pg/mL)	Serum ALP (IU/L)	Treatment	
										Medication	Duration of Ca supplement (mo)
54/Chinese/ F (5)	Recurrent Graves' disease (partial thyroidectomy 4 years)	7	Radioactive iodine therapy	5	4.01 (8.42–10.42)	8.24 (2.48–4.49)	1.56 (1.53–2.28)	48 (35–90)	717 (30–120)	· Ca carbonate · Mg sulfate · Vitamin D2	22 (until the last follow-up)
61/American/ F (19)	NA	2	Radioactive iodine therapy	6	8.3 (NA)	NA	NA	105.2 (10–65)	239.95 (NA)	· Ca carbonate	3
25/Chinese/ F (8)	Graves' disease	NA	Subtotal thyroidectomy	2	7.5 (8.34–11.22)	5.42 (2.79–4.15)	NA	89.92 (15–65)	540 (0–150)	· Ca carbonate · Calcitriol	3
55/Thai/F [†]	Thyrotoxicosis factitia	4	Discontinue offending drug	1	6.25 (8.8–10.6)	2.5 (2.5–4.5)	1.3 (1.8–2.6)	78 (15–68)	139 (30–120)	· Ca carbonate · Vitamin D2 · Alphacalcidol	2

[†], our case. HBS, hungry bone syndrome; y, years old; mo, months; Ca, calcium; PO₄, phosphorous; Mg, magnesium; PTH, parathyroid hormone; ALP, alkaline phosphatase; F, female; NA, not available.

individuals to 5.7 days; conversely, the half-life of serum T3 was apparently prolonged from 25 hours to 5.3 days, which was explained by continuous conversion from a large dose of levothyroxine (20). The unmeasurable tissue T3, which was the better clinical predictor of thyrotoxicosis factitia, was supposed to have an even longer half-life than serum T3 (21,22). The timing of 3 to 4 weeks was also equivalent to five half-lives of the massive elimination of levothyroxine.

Other factors which may confound persistent hypocalcemia were the previous depletion of the vitamin D level, concomitant critical illness, and acute kidney injury. The vitamin D deficiency might play a role in mild hyperparathyroidism and hypocalcemia in this patient, not only the HBS itself. The critical illness and acute kidney injury would lead to malabsorption of intestinal calcium, impaired parathyroid hormone secretion and action, and impaired vitamin D synthesis (23,24). Nevertheless, these conditions could not entirely explain hypocalcemia with HBS in this patient.

Our obvious limitations were the lack of the comprehensive pre- and post-treatment BMD study and serum bone markers, i.e., osteocalcin and bone-specific ALP. This was, because to our knowledge, there was still a lack of normal cut-off levels for patients with acute kidney injury (25).

Conclusions

In conclusion, we reported the first case of thyrotoxicosis factitia with thyroid storm complicated by HBS after treatment. Therefore, it was recommended that close monitoring of calcium and bone mineral metabolism in patients under treatment for thyrotoxicosis factitia should be undertaken. When HBS occurs, prompt adequate supplementation of intravenous calcium, oral calcium, and vitamin D should be conducted until eucalcemia is achieved.

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Footnote

Reporting Checklist: The authors have completed the CARE reporting checklist. Available at <https://aot.amegroups.com/article/view/10.21037/aot-21-18/rc>

Peer Review File: Available at <https://aot.amegroups.com/article/view/10.21037/aot-21-18/prf>

Conflicts of Interest: Both authors have completed the ICMJE uniform disclosure form (available at <https://aot.amegroups.com/article/view/10.21037/aot-21-18/coif>). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. After successful calcium and vitamin D discontinuation, the patient again shortly lost contact with our clinic and her psychiatrist; therefore, we could not obtain her informed consent. We nevertheless reassured for non-personally identifiable patient's specific information throughout the report. All procedures performed in this study were in accordance with the ethical standards of the institutional research committee and with the Helsinki Declaration (as revised in 2013).

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