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An unusual case of hyperthyroidism with recurrent vomiting and hypercalcemia as the main manifestations

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Abstract

Complicated vomiting and hypercalcemia are clinically rare in patients with hyperthyroidism. We describe a case of a woman whose main symptoms were palpitations, sweating, and vomiting. She was diagnosed with Graves' disease by an analysis of thyroid function, thyroid-related antibodies, and color Doppler ultrasound. Biochemical tests showed that her serum calcium levels were greatly elevated. Her symptoms were relieved following the administration of antithyroid drugs, propranolol for heart rate control, fluid replacement, diuresis and calcium reduction, antiemesis, and liver protection. This case suggests that the thyroid function should be screened when hypercalcemia is seen in the clinic.

Keywords

Hyperthyroidism, hypercalcemia, Graves' disease, vomiting, liver damage, palpitation

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Introduction

Hyperthyroidism is an endocrine disease characterized by high metabolism and dysfunction of the nervous and cardiovascular systems. It is caused by the excessive secretion of thyroid hormone by the thyroid gland and its release into the blood. Hyperthyroidism can cause electrolyte

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imbalance, but hyperthyroidism with hypercalcemia is not common clinically. According to reports, the clinical incidence of hyperthyroidism with hypercalcemia is 10%–20%.[1] We present the clinical data analysis of a patient with hyperthyroidism combined with hypercalcemia.

Case presentation

Ethical approval was not required for publication of this case report. The patient provided her oral informed consent for treatment, but consent for publication was considered unnecessary as all identifying details had been removed. The study was conducted following CARE guidelines.[2]

A woman aged 65 years had palpitations with no obvious triggers, accompanied by heat intolerance, sweating, fatigue, and hunger for more than 1 month. She did not seek treatment for this. She presented at the emergency department of the Affiliated Hospital of Guangdong Medical University in June, 2021, after experiencing vomiting accompanied by tremors in both hands for the past 20 days. She was vomiting stomach contents 2–5 times a day around 10 minutes after eating, and the vomiting had been more severe for the 3 days prior to hospital attendance.

Thyroid function testing showed the following: free triiodothyronine (FT3), 46.650 pmol/L (normal range, 2.3–6.8 pmol/L), serum free thyroid hormone (FT4) >100 pmol/L (normal range, 10–23.5 pmol/L), and thyroid stimulating hormone (TSH) <0.005 mU/L (normal range, 0.34–4.0 mU/L). Biochemical test results showed serum calcium levels of 2.98 mmol/L, and serum potassium levels of 3.84 mmol/L, with myocardial markers and routine blood findings within normal ranges. During the past 20 days, she had lost around 4 kg in weight. She was already menopausal and denied a history of smoking, drinking, and vitamin and hormonal intake. She also denied a history of kidney disease and other similar diseases. Physical examination revealed a height of 156 cm, weight of 46 kg, and body mass index of 18.9 kg/m^2. Her facial expression was pained, but there was no exophthalmos, no bilateral thyroid enlargement, no palpable tremor, and no vascular murmur. Small tremors were observed in her hands. Breast examination showed no obvious abnormalities. Her heart rate was 137 beats/minute. Physical examination of the lungs, cardiovascular system, and abdomen was normal. The neurological examination was also negative.

After admission, her blood tests revealed TSH receptor antibody levels of 31.63 IU/L (normal range, 0–1.75 IU/L), anti-thyroid peroxidase levels of 156.0 IU/mL (normal range, 0–34 IU/mL), and anti-thyroglobulin levels of 26.740 IU/mL (normal range, 0–115 IU/mL). Biochemical results showed serum potassium levels of 3.92 mmol/L, serum calcium levels of 2.97 mmol/L, aspartate aminotransferase levels of 67.9 U/L, and albumin levels of 32.5 g/L. Osteoporosis test results were as follows: 25-hydroxyvitamin D, 57.37 ng/mL (normal range, 20.0–100.0 ng/mL), N-Mid osteocalcin, 95.530 ng/mL (normal range, 15–46 ng/mL), β-collagen degradation product, 4.16 ng/mL (normal range, 0.0–0.6 ng/mL), and total type I N-terminal extension peptide, 342.70 ng/mL (normal range, 0.0–37.1 ng/mL). Parathyroid hormone (PTH) levels at three measurements were 11.6, 7.9, and 8.2 pg/mL (normal range, 12–88 pg/mL). No obvious abnormalities in alkaline phosphatase, creatinine, β2 microglobulin, immunoglobulin, serum free light chains, serum protein electrophoresis, immunofixation electrophoresis, urine calcium, phosphorus, and Bence Jones protein, carbohydrate antigen CA125, carbohydrate antigen CA153, or carbohydrate antigen CA199 were observed. Thyroid ultrasound revealed the thyroid gland to

not be unusually large, the capsule to be smooth, the echo of the gland tissue to be rough, and the gland to have abnormally rich blood flow signals. Several spongy hypoechoic nodules were seen in the right lobe; the larger nodules were 0.8×0.4 cm (lower right pole), with an aspect ratio <1, smooth edges, a uniform internal echo, no focal hyperechoic or large comet tail, with a small amount of blood flow signal inside and around the nodules. The presence of hypoechoic nodules in the right lobe of the thyroid led us to consider goiter (2017 ACR total score: 3 points; TI-RADS category 3), while the presence of diffuse thyroid disease led us to consider hyperthyroidism. Dynamic electrocardiogram showed a sinus rhythm, with an average ventricular rate of 94 beats/minute (the fastest ventricular rate of 138 beats/minute, and the slowest ventricular rate of 80 beats/minute), multisource atrial premature beats, and moderately reduced heart rate variability. Echocardiography, brain computed tomography (CT), and CT scans of the upper and lower abdomen and chest showed no obvious abnormalities. Bone density analysis showed a T score of the lumbar spine of –2.6 SD and a T score of the left hip joint of –1.5 SD.

After admission, she was diagnosed with Graves' disease, liver damage, vomiting, and hypercalcemia. We administered methimazole 10 mg three times daily (Tid) to inhibit thyroid hormone synthesis, propranolol 10 mg Tid to control heart rate, fluid replacement, diuresis and calcium reduction, metoclopramide and ondansetron to relieve vomiting, nutritional support, and liver and stomach protection. Her blood calcium level gradually decreased from 2.99 mmol/L after fluid replacement and furosemide diuresis treatment. On the 7th day of admission, her blood calcium was 2.39 mmol/L and blood potassium was 3.41 mmol/L. However, she still vomited frequently. Considering that the onset of action of methimazole is slower than that of propylthiouracil, methimazole was replaced by propylthiouracil 100 mg Tid. On the 10th day of admission, the patient's vomiting and anorexia greatly improved. Oral and intravenous potassium supplementation led to fluctuations in her serum potassium levels during hospitalization from 3.16–3.92 mmol/L. Before discharge, her vomiting had improved markedly, and biochemical tests showed serum calcium levels of 2.41 mmol/L, and serum potassium levels of 3.16 mmol/L. Thyroid function testing revealed FT3, 6.500 pmol/L, FT4, 27.190 pmol/L, TSH <0.005 mU/L, and A-TSHR, 39.09 IU/L.

After discharge from hospital, the patient did not experience vomiting again. One month post-treatment, biochemical tests in the outpatient department revealed serum calcium levels of 2.29 mmol/L, and thyroid function tests showed FT3, 6.650 pmol/L, FT4, 9.830 pmol/L, TSH, 0.013 mU/L, and A-TSHR >40.00 IU/L. At the time of writing the patient is still undergoing follow-up.

Discussion

The cause of vomiting from hyperthyroidism is unclear, but it is thought to result from the following mechanisms: [3,4] 1) the synergistic effect of thyroid hormones and catecholamines having a strong effect on the hypothalamic vomiting center, 2) the chemoreceptor trigger area of vomiting being stimulated by excessive thyroid hormone, and 3) thyroid hormones increasing the sensitivity of adrenergic beta receptors to catecholamines, resulting in excitatory stimulation of the gastrointestinal tract.

Hypercalcemia is common in hyperparathyroidism, tumors, kidney diseases, and as a side effect of drugs, but it rarely results from hyperthyroidism; possible mechanisms for this are as follows:[3,5,6] 1) the excessive secretion of thyroid hormones

accelerating the bone metabolism rate and increasing the activity of osteoblasts and particularly osteoclasts, leading to bone loss, and 2) elevated blood interleukin-6 levels stimulating the activity of osteoclasts, leading to further bone loss.

Our patient had repeated vomiting and hypercalcemia, but her nervous system examination showed no positive signs. Vomiting was greatly relieved when serum calcium levels normalized after 3 days of treatment with methimazole and propylthiouracil to inhibit thyroid hormone synthesis, propranolol to control heart rate, and calcium-lowering therapy. Following this treatment, signs of anorexia improved. We therefore considered that the vomiting was associated with hyperthyroidism and hypercalcemia. Her hypercalcemia was not caused by drugs, she had no history of kidney disease, did not take vitamin A, D, or hormones, and was ruled out for secondary hyperparathyroidism. Her PTH levels were lower than reference values, but hypercalcemia caused by primary hyperparathyroidism was eliminated. No other obvious abnormalities, including multiple myeloma, were identified, so her hypercalcemia was considered to be caused by hyperthyroidism.

Conclusion

Despite being rare in clinical practice, hyperthyroidism in patients with hypercalcemia should nevertheless be considered a possibility.

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Author contributions

Xiao-Dan Wei analyzed the data and wrote the manuscript; Xiao-Ming Chen revised the paper; Jie-Ping Tan contributed to data collection.

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Declaration of conflicting interests

The authors have no relevant financial or non-financial interests to disclose.

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