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Case Report

An Unusual Case of Symptomatic Hypercalcemia from Graves' Disease in a Young Filipino Female

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ABSTRACT:

Hypercalcemia in hyperthyroidism is usually asymptomatic, and related to a concurrent primary hyperparathyroidism. In this report, we describe a case of symptomatic hypercalcemia secondary to Graves' disease alone. A 24-year-old Filipino female presented to the emergency department with generalized weakness, vomiting and abdominal pain. No other symptoms were noted. She was otherwise previously healthy. Family history was unremarkable. During physical exam, she was noted to have a non-tender palpable thyroid gland without bruit. Her ECG showed sinus tachycardia. The complete blood count and electrolytes were normal however, ionized calcium was high at 1.6mmol/L (NV 1-1.3). Renal function was normal. Hydration with saline and Furosemide 20mg once daily was started though calcium levels remained elevated. Other causes of hypercalcemia were excluded as PTH was appropriate suppressed (8.8ng/L; NV 14-72), vitamin D was also suppressed (15.29nmol/L; NV >30). CT scan of chest and abdomen and bone scan did not point to any underlying malignancy nor metabolic bone disease. Medication history was also unremarkable. She was hyperthyroid with a suppressed thyroid stimulating hormone level of 0.004pmol/L (NV 0.55-4.78), free T3 of >20pmol/L (NV 2.3-4.2), free T4 of 8.4pmol/L (NV 0.89-1.76). Thyroid receptor antibody levels were raised at 41.07kU/L (NV <1) supporting the diagnosis of Graves' disease. She was started on propylthiouracil 50mg four times daily, along with propranolol 40mg three times daily. She was subsequently seen after two weeks with normal repeat calcium level and thyroid function test. This report aims to highlight that thyroid disease should always be considered as a cause of hypercalcemia. A concomitant primary hyperparathyroidism should also be evaluated. The definitive treatment for the hypercalcemia is correction of thyroid function.

Keywords: *thyrotoxicosis, primary hyperparathyroidism, graves' disease.*

Introduction

Hypercalcemia is most frequently caused by primary hyperparathyroidism or malignancy. Thyrotoxicosis is an uncommon endocrine etiology and typically results in only mild, asymptomatic elevations of serum calcium [1,2]. In patients with hyperthyroidism, hypercalcemia is often attributed to coexisting primary hyperparathyroidism [3]. Symptomatic hypercalcemia arising solely from thyrotoxicosis without underlying parathyroid disease is extremely rare. We describe such a case and emphasize the need to consider thyroid dysfunction when evaluating unexplained hypercalcemia.

Presentation

A 24-year-old Filipino female presented to the emergency department with generalized weakness, vomiting, and abdominal pain of two days' duration. She denied polyuria, constipation, neuropsychiatric changes, or bone pain. She had no significant past medical history, was not on medications, and her family history was unremarkable.

On examination, she was tachycardic at 112 beats/min, blood pressure 118/74 mmHg, afebrile, BMI 22.5 kg/m². A diffusely enlarged, non-tender thyroid gland was palpable without bruit. No ophthalmopathy or dermopathy was observed. Cardiovascular and abdominal examinations were unremarkable.

Laboratory findings showed the following: Ionized calcium: 1.60 mmol/L (reference 1.00-1.30); creatinine: 64 μmol/L (44-97); parathyroid hormone (PTH): 8.8 ng/L (14-72), suppressed; 25-hydroxyvitamin D: 15.3 nmol/L (>30), low; TSH: 0.004 pmol/L (0.55-4.78), suppressed; Free T3: >20.0 pmol/L (2.3-4.2), elevated; Free T4: 8.4 pmol/L0.89-1.76), elevated; thyrotropin receptor antibody (TRAb): 41.07 kU/L (<1.0), elevated.

Electrolytes, complete blood count, and renal function were normal. CT chest/abdomen and bone scan excluded malignancy and metabolic bone disease.

She was hydrated with intravenous saline and started on furosemide 20 mg daily, but calcium remained elevated. Propylthiouracil 50 mg four times daily and propranolol 40 mg three times daily were initiated. Her calcium normalized (1.21 mmol/L). On her two week follow up, laboratory showed normocalcemia and improvement in thyroid function tests.

Over the next 6 months, she remained euthyroid on antithyroid therapy, after which she underwent radioactive iodine (RAI) ablation. On long-term follow-up (18 months), she developed hypothyroidism, requiring levothyroxine replacement. Importantly, her calcium levels remained within normal range throughout follow-up, with no recurrence of hypercalcemia.

Discussion

This case illustrates a rare presentation of symptomatic hypercalcemia occurring in the setting of Graves' disease without concomitant primary hyperparathyroidism (PHPT). PHPT was effectively ruled out by suppressed parathyroid hormone (PTH) levels, low vitamin D, normal renal function, and the absence of skeletal or malignant pathology on imaging. The patient's calcium levels normalized with antithyroid therapy and remained stable following definitive radioactive iodine ablation, confirming thyrotoxicosis as the sole etiology.

Although hypercalcemia has been reported in thyrotoxicosis, it is typically mild and asymptomatic. A systematic review found that up to 20% of patients with hyperthyroidism may show elevated calcium, but symptomatic cases are uncommon [1]. In a retrospective cohort, hyperthyroidism was recognized as a cause of hypercalcemia in emergency settings, though most cases were confounded by PHPT or malignancy [3]. Previous reports of thyrotoxicosis with symptomatic hypercalcemia showed improvement after antithyroid therapy, with suppressed PTH and normalization of calcium once euthyroidism was achieved, thereby excluding PHPT [4,5]. The proposed mechanism involves the direct effect of excess thyroid hormones on bone metabolism—accelerating bone turnover and favoring osteoclastic resorption—which explains the calcium mobilization seen in uncontrolled hyperthyroidism.

How Our Case Differs

While prior reports have described similar biochemical findings, this case adds several unique aspects:

1. Severity of Hypercalcemia: Our patient presented with an ionized calcium of 1.60 mmol/L, well above the upper reference limit. In most earlier reports, hypercalcemia was mild or borderline.

2. Patient Age and Demographics: The patient was a 24-year-old Filipino woman. Previous cases largely involved middle-aged patients, and very few have been reported in Southeast Asian populations, making this

a valuable regional contribution.

3. Long-term Follow-up After Definitive Therapy: Unlike earlier reports limited to short-term outcomes with antithyroid medication, our patient underwent radioactive iodine ablation, developed hypothyroidism requiring levothyroxine, and maintained normocalcemia for 18 months. This strongly supports the sufficiency of correcting thyrotoxicosis to prevent recurrence of hypercalcemia.

Taken together, this case reinforces the evidence that thyrotoxicosis alone, without PHPT, can produce significant symptomatic hypercalcemia and demonstrates that definitive treatment of Graves' disease can achieve long-term biochemical stability.

Conclusion

We present a rare case of symptomatic hypercalcemia arising solely from Graves' disease, without coexisting primary hyperparathyroidism. This case stands out for the marked severity of hypercalcemia, the patient's young age, and the documented long-term normalization of calcium levels following definitive radioactive iodine therapy.

The key lessons from this case are threefold. First, thyroid disease should be considered in the differential diagnosis of hypercalcemia even when PHPT has been excluded. Second, thorough biochemical evaluation is crucial to avoid unnecessary parathyroid investigations. Third, effective treatment of thyrotoxicosis - whether with antithyroid medication or definitive therapy - remains central to management and can result in sustained normocalcemia.

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