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AN UNUSUAL CASE OF SYMPTOMATIC HYPERCALCEMIA FROM GRAVES' DISEASE IN A YOUNG FILIPINO FEMALE

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INTRODUCTION
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.

CASE
Her ECG showed sinus tachycardia. The complete blood count and electrolytes were normal however, ionized calcium was high at 1.6 mmol/L (NV 1-1.3). Renal function was normal. Hydration with saline and furosemide 20 mg once daily was started though calcium levels remained elevated. Other causes of hypercalcemia were excluded as PTH was appropriately suppressed (8.8 ng/L; NV 14-72), vitamin D was also low (15.29 nmol/LNV: >30). CT scan of chest and abdomen together with 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 0.004 pmol/L (NV: 0.55-4.78), elevated free T3 of >20 pmol/L (NV: 2.3-4.2), and elevated free T4 of 8.4 pmol/L (NV: 0.89-1.76). TSH receptor antibody levels were raised at 41.07 (NV: <1 kU/L) supporting the diagnosis of Graves'disease. She was started on propylthiouracil 50 mg four times daily, along with propranolol 40 mg three times daily. She was subsequently seen after two weeks with normal repeat calcium level and thyroid function tests.

CONCLUSION
This report highlights thyroid disease as a cause of hypercalcemia. The definitive treatment for the hypercalcemia is correction of thyroid function.

KEY WORDS
hypercalcemia, hyperthyroidism, philippines

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A CASE OF PARATHYROID CRISIS SECONDARY TO BENIGN PARATHYROID ADENOMA

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INTRODUCTION
Hypercalcaemic parathyroid crisis is a rare and potentially fatal complication of primary hyperparathyroidism (PHPT) in which patients develop severe symptoms and signs of hypercalcemia.

CASE
67-year-old lady presented to Accident and Emergency with increasing generalized lethargy, vomiting and severe epigastric discomfort for 1 week. She had significant history of osteoporosis with T12 compression fracture and multinodular goiter. At admission, she looked lethargic and clinically dehydrated. There was no palpable neck lump, goiter or cervical lymphadenopathy. Her corrected calcium on admission was markedly raised at 4.1 mmol/l. Other biochemistries revealed significantly elevated intact PTH 189 [1.3-7.6 pmol/l] and serum alkaline phosphatase 618 [32-103 IU/l]. Her urea, creatinine, thyroid functions were normal. She was started on aggressive intravenous hydration with 0.9% sodium chloride followed by subcutaneous calcitonin and iv pamidronate. Technetium-99 sestamibi scan showed hyperfunctioning parathyroid tissue at the region of lower lobe of left thyroid. There was a prompt decrease in her calcium and parathyroid hormone level immediately after removal of a 5 cm left inferior parathyroid tumor. Histology revealed benign parathyroid adenoma.

CONCLUSION
Parathyroid crisis is a syndrome characterized by a serum calcium level usually greater than 3.5 mmol/l resulting from marked elevation of PTH with multiple organ dysfunction and profound dehydration. 3% of PHPT patients present with parathyroid crisis in which 88% are caused by parathyroid adenoma. It is important to institute multimodality treatment to lower serum calcium before early parathyroidectomy.

KEY WORDS
hypercalcaemia parathyroid crisis parathyroid adenoma