CONCLUSION
This case highlights the management challenges in a case of Refractory Graves' disease. Adjunct to maximal medical therapy, plasmapheresis is a potential modality to achieve a euthyroid state prior to thyroidectomy.

PA-A-08

A CASE OF GRAVES' DISEASE WITH SEVERE HYPERCALCAEMIA

https://doi.org/10.15605/jafes.037.S2.14

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INTRODUCTION
Mild to moderate hypercalcemia is seen in up to 20 percent of thyrotoxic patients. However, thyroid hormone-mediated severe hypercalcemia is rare. We report a case of Graves' disease-induced symptomatic severe hypercalcemia.

CASE
A 25-year-old male presented with a three-week duration of abdominal pain, vomiting and constipation. He also complained of palpitations for one month and weight loss of 40 kilos within a year. Clinical examination revealed a temperature of 37.2°C, blood pressure of 120/72 mmHg and pulse rate of 140 beats per minute. He had fine tremors, exophthalmos and diffuse goiter with a thyroid bruit. Initial investigations showed significantly elevated free T4 of >64.35 pmol/L (9-19) with suppressed thyroid-stimulating hormone (TSH) of <0.008 μIU/mL (0.4-4.2). He had severe hypercalcemia with a serum calcium level of 3.97 mmol/L and a low serum intact PTH of 1.09 pmol/L (1.59-7.24). Serum phosphate, magnesium, creatinine and alkaline phosphatase were normal. TSH receptor antibody was raised at >40 IU/L (0-1.75) with normal anti-thyroid globulin and anti-thyroid peroxidase antibody. Neck ultrasound showed diffuse thyroiditis with increased vascularity in the thyroid gland. Tumour markers (CA19-9, CEA, AFP, PSA) were all normal. The skeletal survey revealed no lytic lesions. He was treated as a case of impending thyroid storm. Diagnosis of hyperthyroidism-induced hypercalcemia was made after excluding other common causes of hypercalcemia. He was started on propylthiouracil, propranolol, Lugol's iodine and steroids. An intravenous saline infusion was started for hypercalcaemia without calcitonin or bisphosphonate. His serum calcium levels progressively declined and hypercalcemia-related symptoms resolved. He was discharged on day 8 of hospitalization with a normal calcium level of 2.5 mmol/L and fT4 of 40 pmol/L.

CONCLUSION
Hyperthyroidism is a potential cause of severe hypercalcemia. The treatment of hyperthyroidism may cause normalization of serum calcium levels as observed in this case.

PA-A-09

COLLISION TUMOR OF THE THYROID: A CASE OF MEDULLARY AND PAPILLARY THYROID CARCINOMAS WITH NODAL METASTASES

https://doi.org/10.15605/jafes.037.S2.15

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INTRODUCTION
Papillary thyroid carcinoma (PTC) and medullary thyroid carcinoma (MTC) differ in their incidence, cell origin and histopathological features. Thyroid collision tumors have rarely been reported. We present a 69-year-old female who was recently diagnosed with a collision thyroid tumor consisting of PTC and MTC.

CASE
A 69-year-old female with underlying dyslipidemia presented with neck discomfort. On surveillance medical check-up, it was found that her carcinoembryonic antigen (CEA) was markedly elevated at 220.9 uG/L (<5). Computed tomography scan of thorax, abdomen and pelvis showed a suspicious left thyroid nodule and multiple enlarged lymph nodes at the left lower cervical and superior mediastinal. Subsequent fine needle aspiration (FNA) demonstrated medullary thyroid carcinoma. She underwent total thyroidectomy with modified radical neck dissection with sternotomy for removal of right paratracheal lymph nodes. Histopathology examination demonstrated medullary thyroid carcinoma measuring 35 mm with extensive perithyroidal soft tissue and lymph nodes infiltration. Seven out of twenty-two right lateral cervical lymph nodes, three out of four right paratracheal lymph nodes, and four out of thirteen left lateral lymph nodes were positive for metastases. Papillary microcarcinoma measuring 3 mm was also found at the right lobe. The calcitonin level was 2742 pg/ml postoperatively and decreased further to 1456 pg/ml six months later. CEA decreased to 45.4 uG/L. FDG-PET whole body scan reported no hypermetabolic lesions at the thyroid bed. Currently, she is on levothyroxine therapy (without TSH suppression) and on close follow-up.

CONCLUSION
The collision between two thyroid carcinomas is a rare entity. Management requires assessment of each carcinoma and tailored according to the extent of both tumors. In this case, the focus was on the predominant MTC.