CONCLUSION

Starvation ketoacidosis is an under-recognized cause of metabolic acidosis and may occur even in a diabetic patient who has been acutely unwell with poor oral intake. While the mainstay of therapy in a patient with starvation ketoacidosis is to provide an intravenous dextrose-containing fluid replacement, this has to be judiciously given in an anuric ESRD patient on fluid restriction. A careful balance between low-dose insulin infusion to maintain euglycemia and strict fluid management is crucial to stop gluconeogenesis and ketogenesis. The ultimate goal is to bring the patient out of starvation ketoacidosis while avoiding the deleterious effect of fluid overload in a patient who is already in ARDS.

PA-A-06

AN UNUSUAL SITE FOR THYROID CANCER: A CASE REPORT ON ECTOPIC PAPILLARY THYROID CARCINOMA

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INTRODUCTION

Ectopic thyroid tissue is rare, and the prevalence of ectopic thyroid cancer is even rarer. We report the case of a 37-year-old female with ectopic papillary thyroid carcinoma.

CASE

A 37-year-old female initially presented to the Ear, Nose and Throat (ENT) clinic with a midline upper anterior neck swelling that had gradually increased in size over several months. She did not complain of any compressive or infective symptoms. A computed tomography scan of the neck showed ectopic thyroid at the lingual area, thyroglossal cyst at the hyoid level, posterior to the thyroglossal cyst, and left supraclavicular locations. Fine needle aspiration for cytology of the left supraclavicular swelling was reported as papillary thyroid carcinoma. Subsequent thyroid scintigraphy further confirmed the presence of ectopic thyroid tissue or foci of metastasis. Pre-surgery blood investigation showed Free T4 of 13.4 pmol/L (11-22), TSH of 4.042 mIU/l, unstimulated thyroglobulin of >300 mcg/L (2—50 mcg/L), and negative anti-thyroglobulin. The patient underwent bilateral neck dissection, Sistrunk procedure, and ablation of the base of the neck. Histopathology showed ectopic thyroid tissue with papillary thyroid carcinoma from the Sistrunk specimen and bilateral lymph node metastases. Thereafter, she underwent radioiodine ablative therapy with 100 mCI of Iodine-131. Serial whole-body scans showed physiologic findings. Currently, she is on TSH suppression therapy and close monitoring for tumor recurrence.

CONCLUSION

This case is a reminder of the embryological journey of thyroid tissue, defects of which can lead to its ectopic location. In spite of its rarity, thyroid carcinoma can occur in ectopic thyroid tissue.

PA-A-07

MANAGEMENT CHALLENGES PRIOR TO SUCCESSFUL TOTAL THYROIDECTOMY IN A PATIENT WITH REFRACTORY GRAVES' DISEASE

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INTRODUCTION

Graves' disease is the most common cause of thyrotoxicosis. Restoration of euthyroidism is vital to prevent further complications including cardiac impairment. Refractory Graves' disease is uncommon and, thus, poses a challenge in preparing a patient for definitive therapy. We describe a case of refractory Graves' disease who successfully underwent definitive surgical therapy.

CASE

A 25-year-old female with a seven-month history of Graves' disease was referred for recurrent syncope due to multifocal atrial tachycardia. She had multiple previous admissions for severe thyrotoxicosis within the last five months where she was treated with thionamides and multiple five-to-seven-day courses of Lugol's iodine each time. On admission, thyroid functions tests showed free T4 (fT4) of 92.5 pmol/L (normal range: 11.5 - 22.7) and TSH of <0.01 mIU/L (normal range: 0.55-4.78). The thyroid ultrasound revealed diffuse enlargement of both thyroid lobes with increased vascularity. She was treated with carbimazole up to 80 mg/day, however, fT4 remained at a range of 77.9 – 90.1 pmol/L. Additional therapy with lithium carbonate (1200 mg/day), dexamethasone (8 mg/day) and cholestyramine resin (2 g twice a day) failed to normalize the fT4 level. Switching carbimazole to propylthiouracil (900 mg/day) also did not prove successful. Plasmapheresis was initiated which near-normalized her fT4 after 11 cycles. Tachyarrhythmias were controlled with carvedilol 25 mg twice a day, verapamil 80 mg thrice a day and ivabradine 7.5 mg twice a day. She underwent a successful semi-urgent total thyroidectomy and was eventually discharged after seven days post-operatively with levothyroxine replacement, calcitriol and calcium supplementation.

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

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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

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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.