sepsis. They received thionamides, glucocorticoid, Lugol's iodine, antiarrhythmic and one received cardioversion. All developed ischemic hepatitis with transaminases increased from the initial 2-7 x to 10-80 x upper limit of normal, and two had coagulopathy. Thionamide dose was reduced in two and withheld temporarily in one. Cholestyramine was added as an adjunct for all. All responded to therapy. Two were discharged with carbimazole and beta-blocker. Unfortunately, one succumbed despite initial improvement due to hospital-acquired infection.

CONCLUSION

Although beta blockers play an important role in the management of thyroid storm, caution should be exercised due to its potential life-threatening side effect especially in the presence of clinical or subclinical thyro-cardiac disease. Lugol's iodine and cholestyramine are useful adjuncts in the presence of severe liver dysfunction when choices of antithyroid drugs are limited.

EP_A152

A CHALLENGE IN MANAGING THYROID STORM WITH CONCURRENT PERFORATED GASTRIC ULCER PATIENT

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INTRODUCTION/BACKGROUND

Thyrotoxic crisis can be fatal if not treated promptly. Individuals with severe thyrotoxicosis who lack a functional gastrointestinal system present an uncommon but significant therapeutic challenge with a high fatality rate. We describe a case of Graves' disease in a thyroid storm with a concurrent perforated gastric ulcer.

CASE

A 36-year-old male presented to the emergency department with acute abdominal pain and vomiting. He also had heat intolerance and significant weight loss. He was borderline hypotensive, tachycardic with a regular pulse, and had generalized abdominal guarding. Chest radiograph showed air under the diaphragm. Thyroid function test (TFT) confirmed thyrotoxicosis with Thyroid Stimulating Hormone (TSH) <0.008 m IU/L and free T4 (FT4) 64.32 pmol/L. He was scheduled for an emergency laparotomy for a perforated gastric ulcer, thus contraindicated to taking anything by mouth. We gave him 200 mg of intravenous hydrocortisone. We did not administer an antithyroid drug because our centre did not have any intravenous or per-rectal antithyroid drug preparation. Post-operatively, his condition deteriorated, and he developed rapid atrial fibrillation which required inotropic support and synchronised cardioversion. After he was permitted to sip fluids for medication, he was started on oral propylthiouracil (PTU) and Lugol's iodine in addition to regular intravenous hydrocortisone. Following this, his general condition and thyrotoxic status improved. Upon discharge, he received oral carbimazole and propranolol.

In the 2-month follow-up, he was clinically euthyroid. His anti-thyroglobulin receptor antibody level was elevated, supporting the Graves' disease diagnosis. His TFT improved, with TSH <0.008 m IU/L and FT4 17.40 pmol/L. We further titrated down his oral carbimazole until the next appointment.

CONCLUSION

Managing a thyroid storm with concurrent perforated gastric ulcer is challenging due to limited antithyroid options other than oral medication and high mortality rates. The attending physician should collaborate with the surgical team to determine the optimal timing for oral antithyroid medication to manage the thyrotoxic crisis.

EP_A153

PITFALLS IN THE DIAGNOSIS OF AMIODARONE-INDUCED THYROTOXICOSIS: A CASE SERIES

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INTRODUCTION/BACKGROUND

One of the lesser known but unique mechanisms of action of amiodarone is its ability to inhibit the 5'deiodinase enzyme that converts T4 into the active T3. Inadvertently, this results in an elevated T4 and a lower T3 which suppresses pituitary TSH, giving the impression of a classic thyrotoxicosis. Only when this peripheral effect is overcome by the explosive release of T4 and T3 that they begin to manifest as amiodarone-induced thyrotoxicosis (AIT).

CASE
We reviewed the last 11 patients who were treated as presumed AIT in our institution who had an elevated fT4 >22 pmol/L and suppressed TSH <0.27 m IU/L at the time of diagnosis. All of them were treated with carbimazole whilst one was started with dexamethasone. However, when the heart rate, symptoms and signs of thyrotoxicosis as well as fT3 levels were analysed, 8 out of the 11 patients were deemed to have demonstrated harmless peripheral or physiological effects of amiodarone which did not require any active intervention. Only one out of the 8 patients had a heart rate above 70/minute at the time of diagnosis notwithstanding the fact they were on low doses of beta-blockers (2.5-5 mg of bisoprolol). Three out of the 8 patients had low fT3 whilst the remaining 5 had normal levels of fT3. Out of the 3 who had true AIT, two were treated as type 2 AIT and started on prednisolone whilst the third was treated as type 1 AIT and managed with carbimazole.

CONCLUSION
One of the pitfalls in managing AIT is the failure to recognise the peripheral effect of amiodarone which produces high T4 and suppressed TSH. Only by analysing the T3 level whilst assessing the heart rate and symptomatology will we be able to discern this phenomenon from that of AIT.

EP_A154

A UNIQUE ENCOUNTER OF PAPILLARY THYROID CANCER AND HODGKIN LYMPHOMA IN TANDEM

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INTRODUCTION/BACKGROUND
Papillary thyroid carcinoma (PTC) comprises the largest proportion of differentiated thyroid carcinoma cases. A notably uncommon scenario involves the simultaneous manifestation of PTC and Hodgkin's lymphoma as distinct primary malignancies.

CASE
A 35-year-old female with no history of radiation exposure presented with painless neck swelling for one year. Thyroid ultrasonography revealed 2 hypoechoic lesions over the left lobe measuring 1.8 x 1.6 cm and 0.7 x 0.9 cm (TIRADS 4) and the right lobe measuring 0.4 x 0.4 cm (TIRADS 1) with multiple prominent cervical lymph nodes. Her thyroid profile was normal. Cervical lymph node biopsy was performed and result suggestive of metastatic papillary thyroid carcinoma. She subsequently underwent total thyroidectomy with modified radical neck dissection. Histopathological examination confirmed multifocal (>5 foci) classical variant papillary thyroid carcinoma, with the largest nodule measuring 17 mm, demonstrating lymphovascular involvement and regional nodal metastasis. According to American Thyroid Association (ATA) guidelines, this case was stratified as high recurrence risk. An unexpected diagnosis of nodular sclerosis classical Hodgkin lymphoma was made during lymph node dissection. Bone marrow assessment ruled out lymphomatous involvement and her computed tomography neck, thorax, abdomen, and pelvis revealed extensive bilateral supraclavicular mediastinal and abdominal lymphadenopathy. Therefore, stage 3 Hodgkin lymphoma was diagnosed. After six cycles of escalated BEACOPP chemotherapy for Hodgkin lymphoma, her positron emission tomography (PET) scan revealed no active lymphoma and resolved thyroid bed issues. She is on TSH suppression with 100 mcg of Levothyroxine daily and plans for radio-ablation therapy. Thyroid function, calcium, and parathyroid hormone levels are all normal.

CONCLUSION
This case highlights the rarity of synchronous papillary thyroid carcinoma and Hodgkin's lymphoma. Thorough investigations are crucial to confirm both pathologies and prioritizing treatment becomes essential. Meta-analysis shows that delaying radio-ablative iodine treatment does not impact long-term overall survival in differentiated thyroid cancer. Therefore, lymphoma treatment takes precedence in this case.

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DELAYED-ONSET AMIODARONE-INDUCED THYROTOXICOSIS

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INTRODUCTION/BACKGROUND
Amiodarone is a commonly used antiarrhythmic drug for treatment of refractory tachyarrhythmias. However, its use can lead to development of amiodarone-induced thyrotoxicosis (AIT). AIT is classified into type 1, a form of iodine-induced hyperthyroidism, and type 2, which is a