sphenoidal surgery (TSS), and histopathology confirmed a pituitary adenoma. However, she still had persistent Cushing disease post-operatively with non-suppressed serum cortisol, poor glycemic control with HbA1c of 11-13% and mild hypokalemia. A repeat pituitary MRI was scheduled, and a repeat TSS is likely warranted.

CONCLUSION
Although hypokalemia is not a determining feature of CD, it can be a significant presentation. Hence, a high index of clinical suspicion of the possible etiologies in evaluating hypokalemia is essential.

EP_A077

A RARE CASE OF THIOAMIDE-INDUCED PANCYTOPENIA

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INTRODUCTION
Thioamides play a central role in the management of hyperthyroid disorder due to their efficacy and relatively lower risk of adverse events. While serious adverse effects are relatively uncommon, the more frequently reported are agranulocytosis, hepatotoxicity and vasculitis. Notably, propylthiouracil has been associated with a higher incidence and severity of agranulocytosis and hepatic dysfunction compared to carbimazole. We report a case of a patient with toxic multinodular goitre who developed pancytopenia shortly after initiation of various thioamide agents.

CASE
A 72-year-old female with toxic multinodular goitre developed recurrent neutropenic sepsis following exposure to multiple thioamides. She was initially treated with carbimazole but was complicated with neutropenic sepsis after 2 weeks of treatment; hence, she was switched to cholestyramine and prednisolone. Due to a lack of clinical response, propylthiouracil was introduced, resulting in initial improvement but with subsequent pancytopenia. Iodine therapy was then attempted but failed to produce clinical benefit. A low dose of methimazole was initiated as a final medical option, which eventually precipitated a third episode of neutropenic sepsis. In all three episodes, she was treated with appropriate antibiotics and received granulocyte-colony stimulating factor (G-CSF) support, leading to hematologic recovery. Extensive work-up excluded other potential causes of pancytopenia. Eventually, despite persistently elevated thyroid hormone levels and being at a high risk of intra-operative thyroid crisis, she underwent a successful semi-emergency total thyroidectomy following a multi-disciplinary team discussion.

CONCLUSION
This case highlights a rare and potentially life-threatening complication associated with thioamides, distinct from more commonly observed isolated agranulocytosis, emphasising the need for heightened vigilance when prescribing these medications.

EP_A078

PROLONGED HYPOTHYROIDISM AS A RARE COMPLICATION AFTER ANTITHYROID TREATMENT FOR A PATIENT PRESENTING WITH THYROID STORM

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INTRODUCTION/BACKGROUND
Hypothyroidism rarely occurs following anti-thyroid therapy (ATT). We present a case of prolonged hypothyroidism following ATT for thyroid storm.

CASE
A 50-year-old female presented to the emergency department with a 3-week history of failure symptoms, 10 kg weight loss and diarrhoea. She was in respiratory distress, hypotensive with a high fever and had atrial fibrillation in rapid ventricular response (170 beats/min) with congestive heart failure. She had no goitre or ophthalmopathy. She was diagnosed with thyroid storm (Burch-Wartofsky Score 90) with free T4 79.9 pmol/L and TSH <0.005IU/L. Despite prompt initiation of carbimazole, IV hydrocortisone, Lugol's iodine, non-invasive ventilation, IV amiodarone and electrical cardioversion, she suffered cardiorespiratory arrest. She was revived after cardiorespiratory resuscitation, intubation and triple inotropic support. Her 21-day ICU stay was eventful with multiorgan failure (ischaemic hepatitis, cardiogenic shock, oliguric kidney injury) complicated by nosocomial infection, critical illness myopathy and bedsore. She spent three months in the hospital, including one month of inpatient rehabilitation. Thyroid-wise, she responded to ATT with fT4 dropping to 36 pmol/L on day 3 of admission. All ATT was discontinued on day 11 when fT4 was reduced to 3.64 pmol/L and TSH <0.005 IU/L. On Day 28, her fT4 remained suppressed, reaching a nadir of 1.36 pmol/L (TSH 0.084IU/L, fT31.60 pmol/L [normal