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

3.1-6.8 pmol/L]). Her fT4 rebounded to 47.80 pmol/L (TSH<0.005 IU/L) after 6 weeks (or one month from the last dose of carbimazole). Carbimazole was reintroduced and continued up to her recent follow-up at 2 months post-discharge.

**CONCLUSION**

Hypothyroidism can occur with ATT for primary hyperthyroidism due to overdosage or increased individual sensitivity, but it is usually short-lived. Prolonged hypothyroidism shortly after presentation of thyroid storm is unusual. Possible explanations include the presence of TSH blocking or stimulating antibodies, sick euthyroid syndrome and the elusive "shock thyroid." A thyroid storm due to a thyrotoxic phase of thyroiditis is unlikely here due to the subsequent relapse of thyrotoxicosis. Careful clinical assessment and monitoring are essential to guide treatment direction.

## EP_A079

### UNMASKING MACRO-TSH: A CASE SERIES

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**INTRODUCTION/BACKGROUND**

Discrepancies between biochemical findings and clinical presentation—particularly isolated elevations in thyroid-stimulating hormone (TSH) with normal free thyroxine (FT4) and the absence of hypothyroid symptoms should prompt the consideration of assay interference. Macro-TSH is one of the important possible causes that should be considered. Failure to recognise macro-TSH can result in unnecessary investigations and inappropriate treatment. We describe two middle-aged male patients, both without a family history of thyroid disorders, who were referred for evaluation of discordant thyroid function tests.

**CASE**

***Case 1.*** A 52-year-old male with long-standing Type 2 diabetes and chronic kidney disease Stage 3a was referred for an abnormal thyroid function test (TFT). His TSH was 7.83 uIU/L (0.35-4.94), while free T4 (FT4) was within the normal limit at 16.59 pmol/L (9-19.05). Polyethylene glycol (PEG) precipitation was 0.67 uIU/mL, with a recovery rate of 93% and a confirmed diagnosis of macro-TSH.

***Case 2.*** A 29-year-old male had been treated for hypothyroidism with levothyroxine for 10 months following an initial TSH of 12.37 uIU/mL and free T4 of 13.27pmol/L. Despite adherence to treatment and titrating doses of thyroxine, his TSH persistently rose to 86.06 uIU/mL with free T4 of 11.64 pmol/L. He remained clinically euthyroid. PEG precipitation revealed pre-precipitation TSH of 76.46 uIU/mL with 84% recovery and post-precipitation TSH of 11.88 uIU/mL. These findings confirmed the presence of macro-TSH and led to the cessation of thyroxine treatment.

**CONCLUSION**

These cases underscore the importance of considering macro-TSH in patients with elevated TSH and normal FT4 who lack clinical symptoms of hypothyroidism. Failure to recognise this phenomenon may result in misdiagnosis and inappropriate treatment. PEG precipitation testing is a valuable tool in confirming macro-TSH and guiding appropriate clinical decision-making.

## EP_A080

### MUSCLE UNDER SIEGE: A CASE OF POST-BARIATRIC SURGERY RHABDOMYOLYSIS

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**INTRODUCTION/BACKGROUND**

Rhabdomyolysis after bariatric surgery is rare and under-recognised. It can lead to acute kidney impairment with an associated 25% risk of mortality. We report a patient with rhabdomyolysis after sleeve gastrectomy.

**CASE**

A 48-year-old male patient who has class III obesity (body mass index of 70 kg/m$^2$) was admitted for bariatric surgery. His medical history was significant for hypertension, gouty arthritis and moderate obstructive sleep apnea, with an American Society of Anesthesiologists (ASA) III physical status. He received 3 weeks of in-patient meal replacement therapy with a very low-calorie liquid diet and resistance exercise program before his operation. Intra-operatively, he was placed in a reverse Trendelenburg position. Initially, laparoscopic sleeve gastrectomy was planned, but a switch to open surgery was made due to technical difficulties. The total duration of surgery was 554 minutes. Post-operatively, the patient had a blister and grade II pressure injury at the left gluteus. He was oliguric (urine output less than 0.1 ml/kg/day) with elevated blood creatine kinase