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

Adult E-Poster

(>22000 U/L at 36th-hour post-op) and stage 3 acute kidney injury (serum creatinine 360 umol/L). He was diagnosed with rhabdomyolysis and was co-managed with the nephrology team, whereby aggressive fluid replacement with diuresis was initiated. He did not require kidney replacement therapy throughout his course of recovery. On day 10 post-op, the laboratory findings normalised and the patient was discharged home fully recovered.

CONCLUSION
Postoperative rhabdomyolysis is a severe complication of bariatric surgery, which is potentially life-threatening. Creatine kinase testing should be performed in high-risk patients after bariatric surgery for timely diagnosis and interventions.

EP_A081

NON-ISLET CELL TUMOR SECONDARY TO MALIGNANT PHYLLODES TUMOR OF BREAST

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INTRODUCTION/BACKGROUND
Non-islet cell tumour-induced hypoglycaemia (NICTH) is a rare but important cause of recurrent hypoglycaemia in patients with non-pancreatic tumours. Unlike insulinomas that cause hypoglycaemia through excess insulin secretion, NICTH is associated with large mesenchymal or epithelial tumours producing high-molecular-weight insulin-like growth factor 2 (IGF-2), leading to insulin-independent hypoglycaemia. We report a case of NICTH in a patient with a malignant phyllodes tumour of the breast.

CASE
A 50-year-old female was found unresponsive at home with a blood glucose level of 2.3 mmol/L. She regained consciousness following the administration of IV glucose. She had no history of diabetes or use of glucose-lowering agents. Examination revealed a large, firm 20 × 20 cm left breast mass. Hypoglycaemia work-up showed a random glucose level of 3.0 mmol/L, C-peptide of 35 pmol/L and insulin <2.78 pmol/L, suggesting hypoinsulinaemic hypoglycaemia. IGF-1 was within the normal range. She was treated with glucocorticoids while awaiting surgery. She underwent a left mastectomy, which revealed a 16 × 12.5 × 22.5 cm 7.6-kg malignant phyllodes tumour. Histopathology examination confirmed a malignant phyllodes tumour with high mitotic activity and a high risk of recurrence. An oncology referral was made for adjuvant therapy. At one-month follow-up, she remained asymptomatic with no hypoglycemia.

CONCLUSION
NICTH should be considered in patients with large tumours presenting with hypoglycemia. Corticosteroids may help manage hypoglycaemia before surgery, which remains the definitive treatment. A multi-disciplinary approach is essential for optimal care.

EP_A082

NOCTURNAL HYPOGLYCEMIA: THE TUMOR YOU DON'T SEE, BUT YOUR BLOOD SUGAR DOES

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INTRODUCTION/BACKGROUND
Non-islet cell tumour hypoglycemia (NICTH) is a rare but potentially life-threatening complication of malignancy, often driven by tumour overproduction of insulin-like growth factor 2 (IGF-2). Diagnosis can be challenging due to non-specific symptoms and limited access to specialised assays.

CASE
We report the case of an 87-year-old female with no known medical history who presented with reduced consciousness and was found to have symptomatic hypoglycemia with capillary glucose 2.1 mmol/L. She had experienced unexplained hypoglycemic episodes over the past 3 months. During hospitalisation, she showed a pattern of nocturnal hypoglycemia that temporarily resolved with continuous dextrose infusion, fulfilling Whipple's triad. The laboratory work-up revealed low serum insulin, low C-peptide, low insulin-like growth factor, negative serum sulfonylurea screen and normal random serum cortisol. Unfortunately, IGF-2 measurement was not available. A contrast-enhanced CT (CECT) of the thorax and abdomen exposed a large left lung mass with features suggestive of malignancy. The patient was initiated on glucocorticoid therapy, which led to partial improvement, although nocturnal hypoglycemic episodes persisted. Given her advanced age and overall condition, she declined surgical intervention and opted for conservative management.

CONCLUSION
This case underscores the importance of considering NICTH in elderly patients with recurrent, unexplained