Hammersmith Abstracts

Friday 5th December 2025

20th Hammersmith Multidisciplinary Endocrine Symposium 2025

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20th Multidisciplinary Endocrine Symposium, Friday 5th December
Wolfson Conference Centre, Hammersmith Hospital

08.30am Registration & Coffee

08.55am Welcome and Introduction
Prof Fausto Palazzo, Prof Waljit Dhillo and Prof Karim Meeran

Session 1 Chair: Miss Aimee DiMarco & Mrs Debbie Papadopoulou

09.00am What you must know about thyroid surgery in 2025: What is new?
Mr David Scott-Coombes (Cardiff, Wales)

09.30am Avoiding surgery for benign symptomatic thyroid nodules
Professor Neil Tolley (Imperial)

10.00am Hyperthyroidism: When is surgery appropriate?
Professor Andrea Gillis, Assistant Professor, University of Alabama, Birmingham USA

10.30am A Single-Centre Review of Referral Patterns Post-RAI Outcomes in Graves (**E016**)

10.45am Parathyroid Storm: Beyond Typical Presentation (**E007**).

11.00am Coffee Break

Session 2: Chair: Dr Preeshila Behary and Dr James Ahlquist

11.30am Making a watertight diagnosis of primary hyperparathyroidism.
Dr Jeremy Cox (Imperial)

12.00noon International Guest Lecture: Primary Hyperparathyroidism: the forgotten disease
Prof Herbert Chen (University of Alabama, Birmingham USA)

1.00pm Lunch & Poster session

Session 3: Chair: Prof Tricia Tan

02.00pm Improving bone health in patients with renal disease: when and what operation?
Dr Sophie Dream: (University of Alabama, Birmingham USA)

02.30pm Adrenal incidentaloma - an American perspective
Dr Brenessa Lindeman: (University of Alabama, Birmingham USA)

03.00pm The New Approach to Mild Autonomous Cortisol Secretion (MACS)
Dr Florian Wernig (Imperial)

03.30pm Hungry bones and Gargantuan glands: A case of Pseudohypoparathyroidism IB complicated by Tertiary hyperparathyroidism and Pathological fractures (**E044**)

03.45pm Primary Bilateral Adrenal Lymphoma Revealed by Adrenal Insufficiency: From Constitutional Symptoms to Crisis (**E011**)

04.00pm A Diagnostic Puzzle: Hypercalcaemia and High prolactin in a Young Woman (**E015**)

04.15pm Certificates and close

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E002	Associations between Pre-existing Mental Health Diagnoses and Post-Bariatric Surgery Outcomes
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E006	Determining the optimal timing for parathyroidectomy to preserve bone mineral density in patients with MEN1.
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E008	Complex Graves' Disease: Navigating Multilayered Complexity When All Standard Treatment Pathways Are Constrained
E009	Comparative Analysis of Ultrasound, SPECT-MIBI, and co registered CT Concordance with Surgical Outcomes in Parathyroid Adenomas
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E013	Recurrent Hypoglycaemia post Gastric-Sleeve: A case of pancreatic insulinoma managed with EUS guided radiofrequency ablation
E014	Biochemically quiescent vasopressin insufficiency secondary to suprasellar metastasis unmasked by nausea and vomiting in the context of progressive intracranial disease
E015 OC05	A Diagnostic Puzzle: Hypercalcaemia and Hyperprolactinaemia in a Young Woman
E016 OC01	A Single-Centre Review of Referral Patterns and Post-RAI Outcomes in Graves' Disease

E017	Back pain secondary to osteoporotic vertebral fractures as the presenting feature of Cushing’s disease in a 17-year-old
E018	Hyperlipidaemia, think again.
E019	When Antibodies Collide: An Endocrine and Ophthalmologist's Challenge of TRAB Positive Thyroid Eye Disease in a Hypothyroid Patient
E020	Unmasking the Pituitary: GnRH Agonist Triggered Apoplexy Revealing a Silent Macroadenoma: A Multidisciplinary Success Story
E021	A Convergence of Cardiology and Endocrinology: Understanding Carney`s Complex
E022	Navigating Starvation Ketosis in Expectant Mothers with Gestational Diabetes Mellitus
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E024	Adrenal Infarction and Haemorrhage in Pregnancy: The Crucial Role of Early MRI and Multidisciplinary Management
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E027	Adalimumab-Induced Hypophosphataemia: An Unknown Adverse Effect
E028	Management of Hyponatraemia in Metastatic SCLC
E029	Complex management of Alemtuzumab-induced Grave’s Disease complicated by thyroid eye disease with concurrent unplanned pregnancies
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E034	The Intersection of Genetics and Endocrinology: Idiopathic Infantile Hypercalcemia from CYP24A1 deficiency and a Decade-long Missed Diagnosis
E035	Testosterone Replacement Therapy and Raised Intracranial Pressure: A Complex Interplay in a Patient with Congenital Hydrocephalus and Obesity
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E037	A complex case of stature: transitional endocrinology
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E045	Pulmonary Cryptococcosis Masking an ACTH-Secreting Pulmonary Carcinoid: A Diagnostic Challenge

E001

The Dilemma in Diagnosis and Management of Insulinoma.

HH Zaw[1], G Farah[2], N Murphy[2], J Stedman[3], T Hta[2.]

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2. Diabetes and Endocrinology Department, Royal Berkshire NHS Foundation Trust, Reading.
3. Radiology Department, Royal Berkshire NHS Foundation Trust, Reading.

Abstract

Background
We presented a case of an elderly gentleman experiencing disabling recurrent hypoglycaemia with insulinoma, posing diagnostic and management challenges

Case
A 91-year-old individual with background of moderately severe aortic stenosis and epilepsy presented with recurrent episodes of confusion and seizures, initially attributed to epilepsy. Notably, capillary glucose was 1.8 mmol/L on admission and iatrogenic hypoglycaemia secondary to diltiazem and phenytoin were considered; however, hypoglycaemia persisted despite medication adjustment.

Supervised 72-hour fasting confirmed endogenous hyperinsulinemia (insulin 68pmmol/l C-peptide 800pmmol/l) in severe hypoglycaemia (2.1 mmol/L) alongside negative sulphonyl urea screen. Initial conventional CT and whole-body MRI scans did not localize a pancreatic or non-pancreatic nodule.

After regional MDT discussion, diazoxide was initiated, achieving euglycemia but resulting in significant fluid retention leading to exacerbation of heart failure. Therefore, the diazoxide dose was lowered to 50 mg BD and short-acting somatostatin analogue, octreotide, was introduced. However, this was discontinued due to intolerance (abdominal discomfort and diarrhoea). Hence, prednisolone 5mg twice daily was added resulting in euglycemia.

Subsequent Ga-68 DOTATATE PET/CT identified an 8-mm avid nodule at the head of pancreas. Considering the patient's significant comorbidities and personal preference, resection was deemed unfavourable. As an alternative approach, EUS RFA[1] was discussed in regional MDT, however as the nodule was ill-defined radiologically this appeared to have reduced likelihood of success and so conservative medical therapy was continued as achieving euglycemia. PRRT is also concluded as a treatment option which could bring the similar outcome.

Discussion
In endogenous hyperinsulinism, when conventional imaging fails to localise the culprit lesion, functional PET/CT plays a pivotal role in detection and tumour characterization. It highlights the crucial importance of MDT discussion in achieving an optimal clinical outcome while minimising potential iatrogenic burden.

1. EUS-RFA: Endoscopic US-guided radiofrequency ablation
2. PRRT: Peptide receptor radionuclide therapy

E002

Associations between Pre-existing Mental Health Diagnoses and Post-Bariatric Surgery Outcomes.

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1. Imperial College Healthcare NHS Trust, London.
2. Barts Health NHS Trust, London.
3. Imperial College London, London.
4. Brunel University, London.
5. West London Mental Health NHS Trust.

Abstract

Background
There is a high prevalence of mental health comorbidity in people with obesity, but most of the published literature does not report psychological outcomes post-bariatric surgery. We conducted a retrospective cohort study of patients managed in our specialist weight management service to investigate metabolic and psychological outcomes following bariatric surgery.

Methods
Data were collected from the electronic health records of consecutive patients who had bariatric surgery in our service between 2021 and 2024 with up to 2 years follow-up. Data were analysed using linear regression (for continuous data) or Poisson regression (for categorical data), with models adjusted for age, sex, body mass index (BMI) and type of bariatric surgery (sleeve gastrectomy, one anastomosis gastric bypass and Roux-en-Y gastric bypass).

Results
482 adult patients were included in the analysis (mean age 44.6±11.5 years, mean BMI 45.2±8.2 kg/m^2). 217 (45%) of the cohort had ≥1 pre-existing mental health (MH) diagnosis. Patients with ≥1 mental health diagnosis (MH group) had similar baseline characteristics to the non-MH group, apart from the gender distribution (female: MH group 88% vs non-MH group 76%, p=0.0009). %Total weight loss, cessation of glucose-lowering and anti-hypertensive medication, as well as the risk of weight regain were similar between the two groups. However, having ≥1 pre-existing MH diagnoses was associated with significantly increased risks of post-bariatric surgery suicidal ideation (RR 9.7 95%CI 1.5-61.0, p=0.016) and deliberate self-harm/attempted suicide (RR 7 95%CI 1.1-45.5, p=0.042).

Conclusion
Weight loss and metabolic outcomes are similar in people with and without pre-bariatric surgery MH diagnoses. There is a striking association between pre-bariatric surgery MH diagnoses and adverse psychological complications post-bariatric surgery. Specialist management services should include dedicated psychological follow-up as part of the multi-disciplinary management of patients following bariatric surgery.

E003

Tyrosine kinase inhibitors associated thyroid dysfunction in middle aged female with chronic myeloid leukemia - a case report.

A Nyein Thu, N Bhatia, A Hitesh Rana, M Babar.

Worcestershire Acute Hospitals NHS Trust.

Abstract

Introduction

First-generation tyrosine kinase inhibitors (TKIs), including imatinib, sunitinib, are known to cause thyroid dysfunction, including subclinical hypothyroidism, overt hypothyroidism, hyperthyroidism, and augmentation of autoimmune thyroiditis.

According to the previous studies, subclinical hypothyroidism is the most often reported thyroid dysfunction, particularly associated with imatinib and second-generation TKIs such as nilotinib and dasatinib.

TKIs have transformed CML from a once-fatal disease into a manageable condition, with most patients now enjoying near-normal life expectancy.

As TKIs became more widely used, various endocrine side effects were seen, among them, thyroid dysfunction is one of the less common but important adverse effects to monitor and follow.
This case report aims to explore the importance of thyroid function monitoring in TKI-treated CML patients.

Case presentation

A 43-year-old lady of Asian origin presented with increasing tiredness, abdominal distention, and urinary retention secondary to uterine prolapse. US abdomen and CT TAP admission revealed free fluid in the abdomen and new peritoneal deposits. The drainage tube was inserted to drain the ascites, which were hemorrhagic, and a peritoneal biopsy was taken, which supported peritoneal infiltration by CML.

She is a known case of TKI-treated CML. She was initially diagnosed with BCR-ABL gene-positive CML in 2009 and started on Imatinib. She achieved a major molecular response on imatinib, and it was later stopped in 2019. She received 10 years of TKIs in total, including a few months of dasatinib.

On admission, thyroid function test showed subclinical hypothyroidism with a markedly elevated TSH level of 22.0mIU/L and an FT4 level of 8.9mIU/L. TSH receptor antibodies were negative. There was no palpable goiter on examination. Low-dose levothyroxine was started immediately.

Investigations

TSH	Free T4	TSH receptor antibodies
22.0 mIU/L	8.9 mIU/L	<0.1 IU/L

CT Abdomen and pelvis	Gross ascites. Marked splenomegaly, mild hepatomegaly. Multiple ill-defined lower abdominal and pelvic peritoneal deposits likely ovarian masses/deposits.
Peritoneal biopsy	Acute and chronic inflammation and neoplastic lymphoid cells with a conspicuous number of eosinophils in keeping with infiltration by CML. No blasts identified.

Discussion
A subclinical hypothyroidism pattern was noted in the records in earlier months of 2023. However, she was not started on treatment, and thyroid function was not checked regularly.
Although TKI was stopped in 2019, she had received imatinib for a total duration of 10 years. Her current subclinical hypothyroidism was suspected to have started during imatinib treatment before 2019.

Potential mechanisms have been found to explain thyroid dysfunction, such as direct toxic effects of TKIs on follicular cells, triggering destructive thyroiditis, accelerated thyroid hormone clearance, regression of thyroid capillaries caused by inhibition of vascular endothelial growth factor (VEGF), and reduced iodine uptake.

Conclusion
Studies reported that the longer the duration of imatinib treatment is associated higher the risk of persistent thyroid dysfunction.

If left untreated, there is a chance of progression to overt hypothyroidism, especially if TSH is markedly elevated above 10mIU/L. Although the data are well-known to clinicians, the monitoring of thyroid function in TKI-treated patients is still a neglected area.

We recommend monitoring TFT every 6 months, and lifelong in all patients started on Tyrosine kinase inhibitors.

This case highlights Imatinib as a potential cause of thyroid dysfunction, even in patients with no preexisting thyroid dysfunction. It underscores the importance of baseline and periodic monitoring of thyroid dysfunction patients receiving long-term TKI.

References

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2. Sardarova N, Patel T, Abugoukh TM, Kim D, Yousuf S, Hammoude M. Impact of Tyrosine Kinase Inhibitors on Thyroid Function in Chronic Myeloid Leukemia: A Systematic Review. Cureus. 2025 Jun 1; 17(6):e85196.

E004

Hyponatraemia in the Absence of an Obvious Cause: Lessons from Idiopathic SIAD.

TT Soe, Z Pann Ei, S Zac-Varghese.

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Abstract

Background
Hyponatraemia is a common and potentially life-threatening electrolyte disorder, frequently indicating an underlying pathology. Sodium is critical for maintaining fluid balance, neuronal function, and muscle activity, and its deficiency can result in serious complications, including seizures and coma. Identifying the underlying cause is critical, though some cases require extensive investigation and multidisciplinary management[1].

Case Summary
A 60-year-old lady presented with recurrent symptomatic hyponatraemia following gallstone-induced necrotising pancreatitis, requiring a prolonged intensive care unit admission in 2023. In September 2024, the patient presented to the hospital with euvolemia on clinical assessment, sodium 122 mmol/L, cortisol 589 nmol/L, serum osmolality 258 mmol/Kg, urine sodium 116 mmol/L and urine osmolality 618 mmol/kg, consistent with SIAD, with normal thyroid function, calcium, serum electrophoresis, renal and liver function and no identifiable triggering factors such as medications. Multiple admissions occurred thereafter with sodium <120 mmol/L, confusion, and a fall resulting in a fracture. Each episode responded transiently to fluid restriction, demeclocycline, or tolvaptan, but relapsed when therapy ceased after discharge. The patient underwent extensive evaluation, including CT thorax–abdomen–pelvis, gut hormone profile, standard PET-CT, Gallium-68 Dotatate PET-CT and MRI brain, all of which were unremarkable. Following hospital discharge in December 2024, a trial of demeclocycline (150mg tds) caused side effects, including abdominal pain, elevated liver enzymes, and impaired renal function, although sodium remained stable. Consequently, the medication was discontinued and later restarted at a lower dose (150mg bd) while awaiting tolvaptan approval. Endocrinology input from a tertiary centre was sought, with ongoing liaison with nuclear medicine, the individual funding request team for tolvaptan approval, and the surgical team for elective cholecystectomy. Tolvaptan 7.5 mg alternate days was started in April 2025 and successfully normalised sodium (137–142 mmol/L) and maintained stability, allowing safe completion of cholecystectomy. Cognitive and emotional symptoms resolved, and biochemical parameters remained stable.

Conclusion
Despite extensive investigation and a comprehensive multidisciplinary assessment, no secondary cause was identified, confirming idiopathic SIAD[2]. Tolvaptan proved an effective and well-tolerated long-term therapy, maintaining normonatraemia and improving quality of life. Continued endocrine follow-up and biochemical monitoring are required, and this case highlighted the importance of coordinated care in complex hyponatraemia.

References
1. Verbalis JG, et al. Diagnosis, evaluation, and treatment of hyponatremia: expert panel recommendations. Am J Med. 2013;126(10 Suppl 1): S1–S42.
2. Hannon MJ, Thompson CJ. The syndrome of inappropriate antidiuretic hormone: prevalence, causes, and management. Eur J Endocrinol. 2010;162 Suppl 1: S1–S8.

E005

Should surgery be considered earlier in the management of lithium induced hyperparathyroidism?

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Imperial College Healthcare NHS Trust.

Background

Lithium is an effective mood stabilising drug and remains the treatment of choice for bipolar affective disorder and other psychiatric conditions. Lithium induced hyperparathyroidism is a well-recognised complication of chronic lithium therapy, however there is no definitive consensus on its management. Although dose reduction or discontinuation of lithium may be considered, this is associated with a significant risk of a psychotic relapse and patient harm. Herein we present a case where parathyroid surgery could offer a safe and effective treatment modality for lithium induced hyperparathyroidism.

Clinical case

A 63-year-old female was initially referred to the Endocrinology clinic with constipation, polyuria and polydipsia. She had a longstanding history of bipolar disease and was stable on her antipsychotic medication including lithium and aripriprazole for around 25 years. Her biochemistry demonstrated mildly elevated calcium (2.69mmol/L) and PTH (5.3pmol/L) levels in keeping with lithium induced hyperparathyroidism. She also failed to concentrate her urine osmolality following water deprivation (210 mOsmol/kg, RR >300) and exogenous desmopressin (190 mOsmol/kg) administration, thus confirming lithium induced arginine vasopressin (AVP) resistance. She had a short course of low dose desmopressin without any improvement in polyuria and polydipsia. The possibility of discontinuing lithium to improve symptoms of AVP resistance was also discussed with the patient and her psychiatrist; however, this was considered risky. Given that she had no other complications of hyperparathyroidism at the time, she was managed conservatively.

During a period of observation, she remained polyuric and her calcium (peak 2.85mmol/L) and PTH (peak 12.4 pmol/L) levels began to rise. Furthermore, her renal function deteriorated (estimated GFR 34ml/min/1.73m^2) and an ultrasound revealed nephrocalcinoses. Her bone densitometry also demonstrated osteopenia of her left wrist and hips. She was subsequently commenced on cinacalcet and considered for parathyroid surgery. Following discussion at the MDT and given the high prevalence of lithium induced multi-gland parathyroid disease, a three-and-a-half gland parathyroidectomy with an intra-operative PTH level was recommended.

Questions for discussion

- What are the concerns of parathyroid surgery in this patient?
- When and what type of surgery should be considered?

E006

Determining the optimal timing for parathyroidectomy to preserve bone mineral density in patients with MEN1

B Patel, J Todd, F Wernig.

Imperial College Healthcare NHS Trust.

Background

Multiple endocrine neoplasia type 1 (MEN1) is associated with an increased prevalence of osteopenia and osteoporosis. The early onset reduction in bone mineral density (BMD) is multifactorial but predominantly driven by hyperparathyroidism (HPT). Parathyroid surgery is an effective treatment modality for restoring parathyroid hormone (PTH) and calcium levels, and improving BMD. However, given the high risk of recurrence of HPT in MEN1, determining the optimal timing for surgery remains debatable. Herein we present two young patients with MEN1 being considered for parathyroidectomy to preserve BMD.

Case 1

A 17-year-old female with a family history of MEN1, presented with secondary amenorrhoea and was found to have elevated prolactin (5094mU/L, RR 100-550), and gastrin (72pmol/L, RR<40) levels, and a small 2mm pancreatic lesion. She also had asymptomatic HPT (calcium 2.83mmol/L, RR 2.2-2.6; PTH 19.8pmol/L, RR 1.6-7.2) with normal renal function and osteopenia on bone densitometry. Her case was reviewed by the MDT and given her young age, risk factors for reduced BMD (hyperprolactinaemia, hypergastrinaemia, HPT) and the need to attain peak bone mass, parathyroid surgery was recommended.

Case 2

A 23-year-old female was diagnosed with MEN1 at the age of 11 years following cascade screening from her father. She previously underwent a partial pancreatectomy for a pancreatic neuroendocrine tumour and a surgical wedge resection for a lung carcinoid tumour. Following surgery her gastrin (124pmol/L, RR<40) levels improved but remained elevated, consistent with omeprazole therapy use. She also had a macroprolactinoma which was previously treated with cabergoline; however, due to significant mental health concerns it was discontinued. A subsequent trial of hormonal treatment also failed due to the development of intolerable side effects. Currently, she is not receiving any treatment for her macroprolactinoma and her prolactin level (4069mU/L, RR100-550) remains high. She was also diagnosed with HPT at the age of 13 years and was initially managed conservatively. However, at the age of 17 years, she developed severe symptomatic hypercalcaemia (calcium 2.83, RR 2.2-2.6mmol/L; PTH 23.3, RR 1.6-7.2pmol/L) and was commenced on cinacalcet. In 2025, she developed osteoporosis and surveillance imaging identified multiple non-obstructive bilateral renal calculi. Additionally, she had persistently elevated calcium (2.89mmol/L) and PTH (20.9pmol/L) levels despite cinacalcet. The MDT therefore recommended parathyroidectomy in view of her osteoporosis, renal calculi and untreated hyperprolactinaemia.

Questions for discussion

- Is parathyroid surgery indicated or should it be postponed?
- How effective is hyperprolactinaemia treatment on BMD in MEN1?

E007

Parathyroid Storm: Beyond the Typical Presentation of Primary Hyperparathyroidism.

A Abbamin, Z Juman, N Lin, P Dimitriadis, A Patel, S Zac-Varghese.

East and North Herts Teaching NHS Trust

Abstract
A 62-year-old man was admitted with acute kidney injury secondary to rhabdomyolysis following a fall, loss of consciousness and a long lie. He was found to have a markedly elevated adjusted calcium level of 4.88 mmol/L, phosphate 0.55 mmol/L, magnesium 0.76 mmol/L and CK 26,684 U/L. His PTH was 159.3 pmol/L (nr 1.6-6.9).

He had a recent A&E attendance with a left elbow fracture (radial head) from standing height. He was known to have type 2 diabetes, hypertension, and COPD.

He was treated with intravenous fluids and pamidronate on admission. The vitamin D was replaced, and he was given cinacalcet.

He was investigated for causes of hypercalcaemia and had imaging of his neck, a CT CAP and myeloma screen. His USS neck revealed a U3 nodule and an FNAC demonstrated Thy 3a cytology.

Parathyroid surgery was expedited. Prior to surgery, day 9 of his admission, the adjusted calcium was 3.03 mmol/L.

He was consented for a parathyroidectomy, four gland exploration and hemithyroidectomy. A large left parathyroid adenoma and large left/isthmic thyroid nodule were noted. He underwent a left en block hemithyroidectomy/ parathyroidectomy and level 6 neck dissection. His intra-op PTH 59.4 dropped to 10.6 in 10 minutes.

Post-operatively, his calcium remained elevated, 3.09 mmol/l, with a low PTH, 1.5 pmol/L. He developed hypocalcaemia, 2.09 mmol/L, day 3 post op. He was treated with 1-alpha calcidol and Adcal D3. His calcium remained low despite this and dropped further to a nadir day 5, 1.96 mmol/L. This improved with increased 1-alpha calcidol and calcium supplementation.

The histology of the left thyroid and parathyroid specimen demonstrated a left 35 mm parathyroid adenoma and a pT1a, 1mm papillary microadenoma and 30 mm follicular adenoma.

His calcium remains normal post-operatively and he is recovering well.

A parathyroid storm, also known as a parathyroid crisis and in some reports as parathyroid intoxication, represents a rare, life-threating event with acute hypercalcaemic decompensation and signs and symptoms affecting multiple organs. This frequently occurs after a trigger, such as a fall and immobilisation, and presents with extremely high calcium and PTH levels. Hypocalcaemia was expected post-operatively, and the initial hypercalcaemia was possibly due to the biphasic calcium response following rhabdomyolysis. Careful monitoring and prompt treatment reduced the severity of the hypocalcaemia.

E008

Complex Graves' Disease: Navigating Multilayered Complexity When All Standard Treatment Pathways Are Constrained.

H Myat Mon[1], S Qureshi[2.]

1. Imperial College Healthcare NHS Trust
2. Chelsea and Westminster Hospital NHS Foundation Trust

Abstract
A 23-year-old woman was diagnosed with Graves' disease in 2023, presenting with persistent symptomatic thyrotoxicosis despite high-dose carbimazole (40 mg daily). She had palpitations, tremor, and progressive enlargement of her goitre with some swallowing difficulty. Thyroid function remained markedly deranged (free T3 >30 pmol/L, free T4 35 pmol/L, TSH <0.01 mU/L) with TSH receptor antibody 12.4 units/L.

She later disclosed poor adherence (taking only 2–3 doses per week) related to longstanding anxiety, depression, and a prior overdose of beta-blockers, resulting in significant fear of taking tablets.

Her management was further complicated by steadily rising liver enzymes, displaying a hepatocellular pattern with unremarkable abdominal ultrasound, raising concerns regarding the safety of continued antithyroid drug use.

On assessment, she had mild proptosis and she was vaping. She was encouraged to stop vaping. Prophylactic low-dose corticosteroid was planned to cover mild inactive thyroid eye disease after ophthalmology assessment to enable the radioiodine option. CT neck showed diffusely enlarged goitre without retrosternal extension or tracheal compression. However, thyroid uptake scan showed large goitre with extremely high tracer uptake (56%).

In this case, radioiodine therapy can potentially lead to inefficient radioiodine retention and high treatment failure risk and post-radioiodine thyrotoxicosis, as the radioactive tracer is both avidly taken up and quickly released due to extremely high uptake and rapid turnover. Therefore, the radioiodine option was deemed unfavourable after multidisciplinary discussion with the nuclear medicine team.
Subsequently, surgery was actively being considered with meticulous planning and multidisciplinary collaboration.

Highlights

- This case illustrates the complex situation where all three standard pathways—antithyroid drugs, radioiodine, and surgery—were constrained by psychosocial, hepatic, ophthalmic, and extremely high tracer uptake with large goitre on thyroid uptake scan.
- It is an uncommon scenario where radioiodine therapy deemed unfavourable in Graves' disease, due to potential high risk of treatment failure as the extremely high uptake and rapid turnover could result in poor radioiodine retention.
- Even surgical option carried unusually high perioperative risk of thyroid storm due to uncontrolled thyrotoxicosis, florid disease activity, poor compliance, and possible progression of liver function derangement on perioperative management with high-dose antithyroid medications, reinforcing the importance of early multidisciplinary collaboration.

E009

Comparative Analysis of Ultrasound, SPECT-MIBI, and co registered CT Concordance with Surgical Outcomes in Parathyroid Adenomas.

P SirDeshpandE[1], P MalloN[1], SNK Naido[1], A Clewlow[1], H Bola[1], R John[2], D Remedios[1], M Rahman[1]

1. London North West University Healthcare NHS Trust, London, United Kingdom.
2. Imperial college School of Medicine, London, United Kingdom.

Abstract

Introduction
Surgery is the definitive treatment for primary hyperparathyroidism. Accurate preoperative localisation of parathyroid adenomas is paramount for minimally invasive parathyroidectomy. Common imaging modalities include 99mTc-SPECT-MIBI, co-registered CT, and ultrasound (US), though adding CT increases radiation dose, cost, and time. This retrospective study evaluated concordance among SPECT, CT, and US in lesion detection and surgical outcomes.

Methods
Patients with suspected primary hyperparathyroidism who underwent imaging and successful surgery between September 2023 and August 2025 were included. Out of 348 records screened, 47 met inclusion criteria, as most others were either waitlisted for surgery or managed medically.

Results
Out of 47 patients, 77% were female (n=36), aged 28-85 years (median 52, IQR 20). Ten were <40 yrs, 10 aged 41-50 yrs, 21 aged 51-65yrs, and 6 were >65 yrs. FECa-based genetic testing (n=8) showed two positives. Mean serum calcium was 2.92 mmol/L (median 2.82, IQR 0.23). Forty-six patients underwent all three imaging modalities; one had US only. US detected 31 lesions (4 inconclusive, 12 negative); 99mTc-SPECT-MIBI detected 30 (2 inconclusive, 15 negative); and co-registered SPECT/CT identified 29 (5 inconclusive, 13 negative). Twenty-two showed concordant SPECT/CT and US findings-20 of them (91%) matched surgical localisation, with 2 differing only by superior/inferior position. Seven lesions were diagnosed on CT-only, nine on US-only, and nine were undetected on imaging but underwent surgery due to severe hypercalcemia. Post-operative normocalcemia was achieved in all patients indicating excellent surgical outcomes.

Conclusion
This study demonstrated 91% accuracy for concordant preoperative parathyroid adenoma localisation, indicating that only two modalities are necessary-either 99mTc-SPECT-MIBI+ co-registered CT or 99mTc-SPECT-MIBI + US (when co-registered CT is unavailable). The choice depends on equipment and expertise. Simplifying from three to two modalities may reduce patient burden, streamline workflow, and lower costs without compromising diagnostic accuracy.

E010

Immune Checkpoint Inhibitor-Related Hypophysitis and Pituitary Dysfunction: A Systematic Review of Clinical Presentation, Diagnosis, and Management.

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Abstract

Objective
To review the clinical presentation, diagnosis, and management of immune checkpoint inhibitor (ICPi)-associated hypophysitis and pituitary dysfunction.

Method
A systematic review of studies published from 2005 to 2025 was conducted to evaluate pituitary immune-related adverse events (irAEs) linked to CTLA-4 inhibitors, PD-1/PD-L1 inhibitors, and combination regimens. Eighty-four eligible studies comprising 7,259 patients were included.

Extracted data included ICPi type, demographics, cancer type, treatment duration, imaging findings, type of pituitary dysfunction, clinical presentation, and management.

Results
The weighted average proportion of male patients was 68.3%, with a pooled mean age of 63.9 years. Common symptoms included fatigue, headache, hyponatraemia, nausea, anorexia, and neuropsychiatric changes.

While MRI is a key diagnostic tool, it may not always detect subtle or early-stage pituitary involvement. In the CTLA-4 group, patients received ipilimumab for 2-12 cycles (mean: 3.3) before hypophysitis onset. In the PD-1/PD-L1 group, median time to onset was 28 weeks (range: 10-46 weeks).

Hypophysitis induced by CTLA-4 inhibitors-particularly ipilimumab and CTLA-4-based combination therapies-is more commonly associated with hypopituitarism than that caused by other ICPi classes.

In contrast, isolated ACTH deficiency, more frequently observed with PD-1 and PD-L1 inhibitors, typically presents as secondary adrenal insufficiency, often without distinct abnormalities on early MRI imaging. Reported MRI abnormalities included radiological evidence of hypophysitis, pituitary stalk abnormalities, pituitary enlargement, micro-adenoma, pituitary atrophy, and empty sella. Some patients showed no radiological abnormalities.

The most common biochemical abnormalities in the combination group were hypopituitarism and secondary adrenal insufficiency. High-dose glucocorticoid initiation, careful tapering, and tailored long-term hormone replacement remained the mainstays of management.

Conclusion
Our systematic review delineates the distinct clinical, radiological, and therapeutic profiles of immune checkpoint inhibitor (ICPi)-associated hypophysitis across CTLA-4 inhibitor monotherapy, PD-1/PD-L1 inhibitor monotherapy, and combination ICPi regimens.CTLA-4 inhibitors, particularly ipilimumab, are linked to earlier onset and multifaceted hypopituitarism with more frequent MRI abnormalities, while PD-1/PD-L1 inhibitors typically cause delayed, isolated ACTH deficiency with variable imaging findings.

Combination therapy presents the widest clinical variability and greatest risk of hypophysitis. high-dose glucocorticoid therapy remains the primary intervention, with subsequent tapering to physiological maintenance doses complemented by individualised hormone replacement therapy.

Notably, MRI sensitivity is limited for early or subtle pituitary involvement, necessitating a multidisciplinary diagnostic approach integrating clinical assessment and endocrine evaluation. Vigilant monitoring and early, multidisciplinary diagnosis are essential to reduce morbidity and optimise outcomes in patients receiving immune checkpoint therapy.

E011

Primary Bilateral Adrenal Lymphoma Revealed by Adrenal Insufficiency: From Constitutional Symptoms to Crisis.

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Abstract

Introduction
Primary adrenal lymphoma (PAL) is a rare extranodal lymphoma that often presents with bilateral adrenal masses and adrenal insufficiency. We describe a case of a 50-year-old man with constitutional symptoms, initially presenting with splenomegaly and inflammatory mesenteric changes, without clear evidence of malignancy. On readmission with worsening symptoms, bilateral adrenal masses were identified on CT imaging, and adrenal insufficiency was biochemically confirmed. Adrenal biopsy revealed diffuse large B-cell lymphoma (DLBCL). The patient was promptly started on corticosteroid replacement therapy and underwent systemic chemotherapy with R-CHOP. This case underscores the importance of recognising adrenal insufficiency in patients with adrenal masses, as early intervention with corticosteroids and chemotherapy can significantly improve outcomes.

Case
A 50-year-old man with no significant past medical history presented with a six-month history of progressive fatigue, night sweats, unintentional weight loss of 10 kg, and intermittent left upper quadrant pain. Initial investigations revealed leukocytosis, normocytic anaemia, and an elevated lactate dehydrogenase (LDH) level. Abdominal ultrasound showed splenomegaly and mesenteric inflammatory changes, but no definitive malignancy. The patient was extensively investigated for pyrexia of unknown origin (PUO), including CT TAP; however, no clear cause was identified. He improved clinically and was discharged with outpatient follow-up.

The patient was readmitted three months later with worsening fatigue, dizziness, and postural hypotension. Repeat imaging with contrast-enhanced CT demonstrated bilateral adrenal masses measuring 6.5 cm and 5.8 cm, respectively, with heterogeneous enhancement. Biochemical evaluation revealed hyponatraemia, hyperkalaemia, and a low morning cortisol level, suggesting adrenal insufficiency. An ACTH stimulation test confirmed an inadequate cortisol response. Given the high suspicion for malignancy, an ultrasound-guided adrenal biopsy was performed, which demonstrated features of diffuse large B-cell lymphoma (DLBCL) on histopathology and immunohistochemistry.

The patient was immediately initiated on corticosteroid replacement therapy with hydrocortisone and fludrocortisone, leading to clinical stabilization. An oncology referral was made, and systemic chemotherapy with R-CHOP (rituximab, cyclophosphamide, doxorubicin, vincristine, and prednisone) was commenced. He demonstrated clinical and biochemical improvement over subsequent cycles of chemotherapy, with complete resolution of adrenal mass size on follow-up imaging. However, adrenal insufficiency persisted.

Conclusion
Primary adrenal lymphoma is an uncommon but aggressive malignancy that can present with bilateral adrenal masses and adrenal insufficiency. This case highlights the importance of early recognition and treatment of adrenal insufficiency, as it can be life-threatening if undiagnosed. Clinicians should maintain a high index of suspicion for PAL in patients presenting with constitutional symptoms and adrenal masses. Prompt initiation of corticosteroid therapy alongside chemotherapy is crucial for improving patient outcomes.

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E012

Plasmapheresis and multidisciplinary collaboration in managing severe thyrotoxicosis complicated by antithyroid drug hepatotoxicity.

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Abstract

Introduction
The conventional management of severe thyrotoxicosis, using a combination of antithyroid drugs, iodine, beta-blockers and corticosteroid, is effective in most patients. However, therapeutic options become limited when antithyroid medications are contraindicated. In such cases, plasmapheresis offers a safe and effective alternative, enabling stabilization before definitive interventions like thyroidectomy. Here, we describe an adult patient with severe thyrotoxicosis who was successfully managed with plasmapheresis as a bridging therapy to thyroidectomy in the setting of drug-related toxicity.

Case Presentation
An 18-year-old male with Graves' disease, initially managed with carbimazole and later with propylthiouracil (PTU) because of neutropenia, was transferred to Leeds Teaching Hospitals Trust from Manchester in June 2024, with jaundice and deranged liver function tests (bilirubin 206 µmol/L, ALT 3,500 U/L, INR 2.1). PTU-induced hepatotoxicity was suspected. Clinical examination showed exophthalmos, fine tremor, and a non-tender goitre, with no features of thyroid storm. Initial thyroid function tests (TFTs) showed TSH <0.05 mIU/L (Ref rage 0.2-4.3), Free T4 100 pmol/L (Ref. range 10.0-20.0), and Free T3 >30 pmol/L (Ref. range 3.50-6.80). Liver biopsy showed features of necrosis suggestive of drug related liver injury or "burnt out" autoimmune hepatitis. Given the contraindication to antithyroid drugs, a multidisciplinary team (Including Endocrinologists, Endocrine Surgeons, Hepatologist, Haematologist and Intensivist) initiated serial plasmapheresis, propranolol, cholestyramine followed by Lugol's iodine and corticosteroids (for? autoimmune hepatitis). Eventually, the patient underwent successful thyroidectomy.

Management and Outcome
The patient underwent **10 plasma exchange sessions** between 6–23 July 2024, resulting in a progressive decline in thyroid hormone levels. When biochemical control was achieved (Free T4 11.3 pmol/L), a total thyroidectomy was performed on 30 July 2024. Postoperatively, calcium and parathyroid function were normal, and levothyroxine 100 µg daily was commenced. The patient was discharged on 6 August 2024 on tapering prednisolone for probable autoimmune hepatitis. Subsequent follow-up demonstrated normalized thyroid and liver function.

Date	Free T4(pmol/L)	Intervention	ALT	Bili	IN R
04/07	100	Pre-plasmapheresis	2422	226	2.1
06/07	43	After 1st session	1501	227	1.7
15/07	71	After 5th session	124	162	1.4
22/07	19.9	After 9th session	115	112	1.6
25/07	11.3	After 10th session	126	96	1.7
31/07	11.9	Post-thyroidectomy	166	95	1.6

Conclusion
This case highlights plasmapheresis as a safe and effective bridge therapy in severe thyrotoxicosis when antithyroid drugs are contraindicated, facilitating biochemical stabilization before definitive surgical treatment. It also highlights the importance of MDT in the management of complex cases.

E013

Recurrent Hypoglycaemia post Gastric-Sleeve: A case of pancreatic insulinoma managed with EUS guided radiofrequency ablation.

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Abstract

Background
Post-prandial hypoglycaemia is a recognised complication of certain forms of bariatric surgery. The prevalence is estimated to be between 25% and 75% based on continuous glucose monitoring studies (Hazlehurst et al., 2024).Typically, presentation with post bariatric hypoglycaemia first occurs >1 year after the surgery and usually occur 1 to 3 hours after eating (Salehi et al., 2018). Symptomatic hypoglycaemia, very early in the post-operative period (<6 to 12 months), in the fasting state or more than 4 hours after calorie intake is not typical and should instead raise concerns for the other causes of hypoglycaemia (Salehi et al., 2018).

Case Presentation
A 43-year-old male presented with recurrent hypoglycaemic episodes 6 months following gastric sleeve surgery. Symptoms-light headedness, lethargy, sweating and confusion were first noted during Ramadan fasting and resolved with frequent meals. Episodes predominantly occurred during fasting periods exceeding 4 hours post meal.

These symptoms have not occurred prior to the weight-reduction surgery, he did not have type 2 diabetes and was taking no regular medications.

During a clinic visit, he exhibited confusion suggestive of neuroglycopenia, with a capillary glucose reading of 2mmol/L following a 4 hour fast. Symptoms resolved with glucose administration fulfilling Whipple's triad. Formal laboratory evaluation at the clinic visit revealed plasma glucose of 1.4mmol/L, inappropriately elevated insulin of 39.1mu/L and C peptide of 1768pmol/L with negative sulphonylurea screen-consistent with endogenous hyperinsulinemia.

Ga 68 DOTATATE PET/CT demonstrated intense uptake in the uncinate process of the pancreas and MRI confirmed the 20mm lesion typical of neuroendocrine tumour.

Initial management with diazoxide and octreotide provided partial symptom control. Three options for definite treatment were considered: surgical enucleation, a Whipple's procedure or radiofrequency ablation (RFA). Anxious about recovery time (the patient was a head teacher), he requested RFA, understanding the potential issues of lack of histopathology and possible incomplete cytoreduction. Although initial RF ablation provided temporarily relief, symptoms recurred 6 weeks later with biochemical evidence of persistent inappropriate insulin secretion. A second RFA was performed 5 months later which was complicated by mild pancreatitis. Follow up imaging showed post ablative changes and remains symptom free a year later. He continues to be monitored in the endocrinology clinic.

Conclusion
This case highlights the importance of considering insulinoma as a differential in patients with post bariatric hypoglycaemia. It demonstrates the potential role of EUS guided RFA as a minimally invasive alternative to surgery in carefully selected patients.

E014

Biochemically quiescent vasopressin insufficiency secondary to suprasellar metastasis unmasked by nausea and vomiting in the context of progressive intracranial disease.

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Introduction
Metastatic infiltration of the hypothalamic-pituitary axis is uncommon, and is often associated with hypopituitarism.(1) Cranial vasopressin insufficiency can evade detection owing to intact thirst-driven compensatory polydipsia maintaining normal serum osmolarity(2); this can be disrupted by intercurrent illness. We present a case of suprasellar metastasis in which the diagnosis of vasopressin insufficiency was made following dyselectrolytaemia in the context of symptomatic nausea secondary to progressive intracranial disease.

Case summary
A 69 year old woman diagnosed with metastatic right upper lobe adenocarcinoma in May 2024, initially presented with symptoms of fatigue, dry mouth, and low appetite. As part of her cancer staging, CT head was performed, identifying an irregular mass centred over the hypothalamus. A solitary enhancing 7 x 12 x 11 mm suprasellar metastasis (hypothalamic, chiasmatic, and possible anterior pituitary stalk involvement) was shown on MRI. Endocrinology input was sought; initial investigations revealed anterior pituitary deficiencies (ACTH <5 ng/L, cortisol 83 nmol/L, TSH 0.19 mIU/L, FT4 12.5 pmol/L, LH <0.1 IU/L, FSH 0.2 IU/L and IGF-1 8.5 nmol/L). Her short Synacthen test showed a rise in cortisol from 46 nmol/L to a peak of 190 nmol/L; hydrocortisone was commenced with the later addition of levothyroxine 50 mcg. She received 4 cycles of pemetrexed, carboplatin, pembrolizumab followed by pemetrexed/pembrolizumab maintenance, achieving partial response. She concurrently reported persistent dry mouth, thought to be oral mucositis related to antimetabolite therapy, which was partially mitigated by artificial saliva sprays; she also reported ongoing fatigue.

Disease progression was observed in April 2025 with new leptomeningeal disease and the suprasellar lesion doubling in size; stereotactic radiosurgery (18 Gy in #1) was offered. This was followed by craniospinal radiotherapy (25 Gy in 10#) in September 2025; shortly thereafter she was acutely admitted with odynophagia precluding fluid intake, on a background of worsening nausea and vomiting. Palliative care advised switching from hydrocortisone to dexamethasone and starting a continuous subcutaneous infusion (cyclizine, hyoscine, morphine) for nausea.

A new hypernatraemia was observed, peaking at 163 mmol/L despite liberal intravenous fluids. On focused enquiry, the patient volunteered a previously unreported history of >6 L/day water intake (drinking to thirst) for several months, and dilute polyuria (>7 L/day urine output when charted).

The cause of vasopressin insufficiency was likely progressive infiltration of her suprasellar metastasis, though recent cranial radiotherapy may have also contributed.

A water deprivation test was undertaken, during which her serum osmolality rose from 296 to 305 mOsm/kg. There was impaired urine concentration during her 8-hour fast (UOsm 93 → 93 mOsm/kg); post-DDAVP, her urine osmolality rose from 166 to 502 mOsm/kg. Cranial vasopressin insufficiency was diagnosed, and she was discharged on 50 mcg BD of oral desmopressin, and a dexamethasone weaning regime titrated to nausea control. Given progressive weight loss, fatigue, and declining performance status, she was not considered fit for further systemic therapy.

Learning points

- While the prevalence of cranial vasopressin insufficiency in patients with all-cause hypopituitarism is low (5.1% in a single-centre series),(3) it is frequently observed in metastatic disease affecting the hypothalamic-pituitary axis,(1) and should be specifically screened for with focused history and further investigations.
- Dysnatraemia precipitated by intercurrent illness (e.g. nausea and vomiting) may reflect previously compensated central vasopressin insufficiency.
- Early multidisciplinary involvement, including palliative care, for symptom control can prevent metabolic decompensation in patients with hypopituitarism.

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E015

A Diagnostic Puzzle: Hypercalcaemia and Hyperprolactinaemia in a Young Woman.

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Abstract

Background
Managing patients with multiple endocrine abnormalities presents significant diagnostic and therapeutic challenges. When two distinct derangements co-exist, such as hypercalcaemia and hyperprolactinaemia, the differential diagnosis becomes complex raising several possibilities and clinicians should consider syndromic associations such as Multiple Endocrine Neoplasia type 1 (MEN1). Familial hypocalciuric hypercalcemia (FHH), a cause for hypercalcaemia, is an autosomal dominant condition caused by inactivating mutations in the calcium-sensing receptor (CaSR), is often misdiagnosed as Primary Hyperparathyroidism (PHPT), leading to unnecessary parathyroid surgery.

Case
A 37-year-old woman was admitted with acute shortness of breath and diagnosed with recurrent pulmonary embolism. During routine admission blood tests, she was incidentally found to have marked hypercalcaemia, with an adjusted calcium of 3.20 mmol/L and non-suppressed parathyroid hormone (PTH). Interpretation was challenging given her history of familial hypercalcaemia and previous parathyroidectomy of 4.5 glands in the United States for ectopic parathyroid tissue. She was referred to the endocrine clinic, where she reported persistent symptoms of thirst, polyuria (>5 L/day), generalised body aches, and long-standing menstrual irregularities, experiencing 3-5 periods per year since age 18.

Investigations

- **Adjusted calcium:** 3.12 mmol/L (persistent)
- **PTH:** 6.1 pmol/L
- **24-hour urine volume:** >5 L
- **Urinary calcium excretion:** Low
- **Calcium: creatinine clearance ratio:** 0.00496 (FHH typically <0.01; PHPT >0.02)
- **Prolactin:** 1713 mIU/L
- **Vitamin D:** Low
- **Renal ultrasound:** Normal
- **DEXA scan:** Negative for osteoporosis
- **PET-CT:** No parathyroid tissue detected
- **MRI pituitary:** initial report - normal appearance

Genetic testing confirmed a CaSR mutation, consistent with FHH, and MEN1 genetic testing was negative. Despite the usual benign course of FHH, this patient had symptomatic hypercalcaemia and on trial of Cinacalcet. Marked hyperprolactinaemia prompted a cannulated prolactin test, which confirmed true hyperprolactinaemia (1508 → 1509 → 1482 mIU/L). A subsequent radiology multi-disciplinary team (MDT) review was requested, to which a pituitary nodule was identified, and the diagnosis of microprolactinoma was made one year after initial review. This highlights the importance of MDT in making a diagnosis and the difficulty of interpreting imaging and pathology results in the context of multiple endocrine disorders.

Discussion
This case illustrates the diagnostic complexity when multiple endocrine abnormalities co-exist. The initial presentation of hypercalcaemia with non-suppressed PTH after previous parathyroidectomy raised suspicion for:

- Recurrent Primary Hyperparathyroidism (PHPT)
- MEN1 syndrome, given concurrent hyperprolactinaemia
- Familial Hypocalciuric Hypercalcaemia (FHH)

The low calcium: creatinine clearance ratio (0.00496) strongly favoured FHH, later confirmed by CaSR mutation testing in this case. This underscores the importance of biochemical profiling and genetic testing before considering repeat surgery, as parathyroidectomy is ineffective in FHH.

The co-existence of true hyperprolactinaemia and pituitary nodule initially suggested MEN1, but negative MEN1 genetic testing and isolated CaSR mutation excluded the syndromic disease. This highlights the need for systematic evaluation and multidisciplinary review to evaluate the imaging results.

Finally, although FHH is typically benign, this patient had symptomatic hypercalcaemia requiring pharmacological intervention, Cinacalcet, an uncommon scenario that challenges the assumption that FHH is often clinically silent.

Summary
- Co-existence of hypercalcaemia and hyperprolactinaemia should prompt consideration of MEN1, but alternative or multiple diagnoses such as FHH and pituitary pathology should be explored
- Comprehensive biochemical, genetic and imaging evaluation can aid diagnosis and avoid unnecessary surgery
- Normal imaging report does not rule out pituitary disease; MDT review and dynamic testing may be required

E016

A Single-Centre Review of Referral Patterns and Post-RAI Outcomes in Graves' Disease.

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Abstract

Introduction
Radioactive iodine (RAI), a definitive treatment option for Graves' disease (GD), carries a risk of de novo or worsening Graves' orbitopathy (GO). Early identification of high-risk patients and implementation of preventative strategies are essential to minimise complications. Pre-RAI risk assessment including ophthalmology referrals can vary between centres, highlighting the need for ongoing local evaluation.

Objective

1. Review pre-RAI risk assessment practice and ophthalmology referral patterns including use of prophylactic corticosteroids among patients with Graves' disease
2. Assess post-RAI outcomes including incidence of Graves' orbitopathy

Methods
A retrospective cohort review was conducted of all patients with GD who received RAI therapy at our centre between June 2019 and June 2024. Data collected included demographics, smoking status, treatment regimen, thyroid function and antibody titres, ophthalmic assessments, use of prophylactic corticosteroids, RAI dose along with de novo or progression of GO and remission rates.

Results
A total of 162 patients received RAI for GD between June 2019 and June 2024, with an average dose of 379.85 MBq (SD ± 74.86). The mean time from diagnosis to RAI was 3.2 years. Thirty patients (18.5%) were active smokers at diagnosis. Thirty-two patients (19.8%) were referred for ophthalmology review pre-RAI: 30 due to symptoms or clinical evidence of GO and/or smoking, one for family history of GO, and one for previous history of GO with high thyroid-stimulating immunoglobulin (TSI) levels. Steroid prophylaxis was administered in 35 patients (21.6%) using the local six-week tapering regimen starting on the day of RAI (prednisolone 0.5mg/kg/day for a week followed by tapering 5 mg/week).

Six out of thirty-two referred patients had post-RAI ophthalmology follow up. This included one with post-RAI de novo GO and five with stable eye disease. Two additional patients were referred post-RAI for suspected GO; one was subsequently found to have no GO, while the other had mild active disease. Pre-RAI TSI levels varied widely (<0.01–>40 IU/L) with no correlation with post-RAI GO disease activity.

At six months post-treatment, 108 patients (66.7%) were hypothyroid, 26 (16%) were euthyroid, and 28 (17.3%) remained thyrotoxic. At the time of data collection, 4/28 thyrotoxic patients received further RAI therapy, while the remaining were under active medical follow up. Pre-RAI ophthalmic risk assessments and prophylactic steroid use amongst endocrinologists remained variable.

Conclusion
Pre-RAI ophthalmic risk assessment remains variable, including amongst smokers and those without overt ocular symptoms. Our review demonstrates a lack of correlation between TSI and post-RAI GO incidence and disease activity. Our review highlights the need for standardised local screening protocols and multidisciplinary coordination between endocrinology and ophthalmology to optimise GD management pre- and post-RAI to ensure early detection and prevention of GO.

E017

Back pain secondary to osteoporotic vertebral fractures as the presenting feature of Cushing's disease in a 17-year-old.

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Abstract

Introduction
Exogenous steroids are a well-known cause of secondary osteoporosis, however excess endogenous cortisol production is a much rarer cause that may not always be considered if a patient with undiagnosed Cushing's syndrome first presents with the manifestations of bone degradation.

Case
We present the case of a 17-year-old young man with back pain who was found to have osteoporosis (lumbar spine Z-score -4.5) with multiple healed vertebral end plate fractures, treated with IV Zolendronate elsewhere. During a later unrelated A+E attendance, he was incidentally noted to have a cushingoid appearance and referred to our centre.

His 24-hour urinary free cortisol was raised and serum ACTH was not suppressed (32ng/L with a paired serum cortisol of 752 nmol/L at 9am). Midnight serum cortisol was raised (913 and 805 nmol/L) and a 48-hour low dose dexamethasone suppression test showed failure to suppress (0-hour cortisol 764 nmol/L, 48-hour cortisol 597 nmol/L).

MRI Pituitary identified a 2-3mm non-enhancing focus within the adenohypophysis. However, inferior petrosal sinus sampling (IPSS) found no significant central to peripheral gradient of ACTH, mandating a search for an ectopic source using both functional and cross-sectional imaging. Foci of dotatate avidity were seen in the pancreas (6mm) and small bowel (7mm) but without a cross-sectional correlate. There was no evidence for an intrathoracic source of ACTH. This was discussed by the MDT and it was felt that, in a young patient, a pituitary source of ACTH remained most likely and the lack of a significant gradient on IPSS was due to other factors (no crossover flow seen during left-sided catheterisation and suboptimal catheter position on the right).
He underwent transphenoidal surgery and post-operative cortisol levels were low (paired random cortisol 16 nmol/L, ACTH 5ng/L), indicating remission of Cushing's disease. Histology confirmed a pituitary adenoma/ neuroendocrine tumour (TPIT-lineage corticotroph). He temporarily required treatment with hydrocortisone 10/10/5, which was subsequently weaned off, leaving colecalciferol as his only regular medication. His cushingoid features improved, as did his mood, and he went on to pursue higher education. Follow up MRI pituitary found that the previously seen 3mm lesion was no longer present, with no evidence of adenoma recurrence.

Discussion
This case demonstrates the importance of considering rare causes of secondary osteoporosis, including hypercortisolism, in patients who lack other risk factors. The Endocrine Society's 2008 guidelines recommend testing patients with unusual features for age, including osteoporosis and hypertension, for Cushing's syndrome[1] and some papers have begun to debate the best method for screening patients attending bone disorder clinics.[2] This case also highlights diagnostic challenges in ACTH-driven hypercortisolism, including potential technical difficulties surrounding IPSS, which must be taken into account when interpreting ACTH gradients. MDT involvement is crucial in these complex cases, as seen with this patient and the decision to proceed with pituitary surgery despite the lack of a significant central to peripheral ACTH gradient.

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E018

Hyperlipidaemia, think again.

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Abstract Case
A 28-year-old gentleman was referred by his general practitioner (GP) to the Endocrine team due to hyperlipidaemia (cholesterol 9.1mmol/L, triglycerides 2.8mmol/L) with queries if it was due to genetic issues. He had a background of hypothyroidism, pre-diabetes, vitamin D and folic acid deficiency. His father, aged 55, also had high lipid levels but otherwise no other significant family history. On examination, he had no xanthoma nor xanthelasma. Interestingly, 5 months later, prior to his Endocrine clinic appointment, it was found that his cholesterol levels improved to 3.8mmol/L, triglycerides 0.8mmol/L, without any treatment. Upon reviewing his blood results, he initially had severely underactive thyroid (thyroid stimulating hormone [TSH] 254.58mIU/L, Free T4<5.4pmol/L). Following treatment with levothyroxine, when his thyroid function improved (TSH 1.48mIU/L), his lipid levels improved as well.

Discussions
This case highlights the importance of considering secondary causes of hyperlipidaemia and addressing them, so that risks of cardiovascular diseases can be reduced. Hypothyroidism is well recognised to be closely associated with hyperlipidaemia. Thyroid hormone **(TH)** plays a central role in regulating cholesterol synthesis, metabolism, and clearance (1). Consequently, reduced TH levels are linked to elevated total cholesterol, low-density lipoprotein cholesterol, and triglycerides. Emerging evidence also suggests that high TSH levels alone may contribute to dyslipidaemia, although the underlying mechanisms remain unclear (2). Recent observational studies have reported that among patients with hypothyroidism, approximately 48% have hypercholesterolaemia and 32% have hypertriglyceridemia, underscoring the strong association between thyroid dysfunction and lipid abnormalities (3). Hence, a multidisciplinary team approach may be required once secondary causes of hyperlipidaemia are established when managing these patients.

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E019

When Antibodies Collide: An Endocrine and Ophthalmologist's Challenge of TRAB Positive Thyroid Eye Disease in a Hypothyroid Patient.

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Abstract

Background
Thyroid eye disease is typically associated with Graves' hyperthyroidism but can rarely occur in euthyroid or hypothyroid states. Mixed TSH receptor antibody (TRAb) activity stimulating and blocking may lead to atypical thyroid function and orbitopathy, posing diagnostic and therapeutic challenges.

Case Presentation
A 59-year-old woman presented with diplopia and bilateral restriction of elevation of eye to ophthalmology department. Orbital CT revealed inferior rectus involvement consistent with Thyroid eye disease and referred for endocrine input. Thyroid function showed hypothyroidism (TSH 10.8 mIU/L, FT4 11 pmol/L) with positive TPO (409 IU/mL) and TRAb (10.6 IU/L). Levothyroxine 25 µg daily, selenium supplementation, and smoking cessation were initiated. Despite achieving euthyroid status (TSH 1.71 mIU/L at 5 months), ocular restriction persisted. TRAb remained positive (8.8 IU/L), likely reflecting blocking antibody dominance. Levothyroxine was titrated to maintain TSH <2.5 mIU/L. Surgery was deferred pending disease stability.

Discussion
This case demonstrates thyroid eye disease manifesting in a hypothyroid patient who is TRAb positive, a rare but recognised overlap between Graves' disease and autoimmune thyroid disease.

Mixed antibody activity can cause fluctuating thyroid states. Management requires antibody profiling and multidisciplinary coordination. While thyroidectomy or block-and-replace therapy may be a treatment option, but evidence is limited regarding reducing antibody titres and decisions should be individualized.

These findings are consistent with reports by Bahn RS *et al.* (*Thyroid*, 2011; 21:593–646) and Weetman AP (*N Engl J Med*, 2000; 343:1236–1248), both noting that Graves' orbitopathy can present in euthyroid or hypothyroid states due to mixed antibody responses but rarely.

Conclusion
TRAb-positive hypothyroid thyroid eye disease is rare and demands tailored endocrine and ophthalmic care. Optimizing thyroid status and close MDT follow-up may stabilize disease without immediate surgery. Further research is needed on the role of immunomodulation and thyroidectomy in reducing TRAb activity.

Learning Points:

- Thyroid eye disease can occur in hypothyroid or euthyroid states due to mixed TRAb activity.
- Functional antibody profiling aids diagnosis and management.
- Multidisciplinary collaboration of endocrinology, ophthalmology, and radiology is essential for optimal outcomes.

E020

Unmasking the Pituitary: GnRH Agonist Triggered Apoplexy Revealing a Silent Macroadenoma: A Multidisciplinary Success Story.

S Shivaprasad, R Vanka, J Ambigapathy.

County Durham and Darlington NHS foundation Trust, United Kingdom.

Abstract

Background
Pituitary apoplexy is a rare but potentially life-threatening endocrine emergency caused by haemorrhage or infarction within a pituitary adenoma. While spontaneous cases are well documented, apoplexy precipitated by gonadotropin releasing hormone (GnRH) agonist therapy for prostate cancer is exceptionally uncommon. This report describes a case of GnRH agonist triggered apoplexy unmasking a previously silent macroadenoma, successfully managed through a multidisciplinary approach.

Case Presentation
A 76-year-old man with prostate adenocarcinoma received his first GnRH agonist injection as part of androgen deprivation therapy. Within days, he developed sudden severe headache, nausea, vomiting, and diplopia. MRI revealed a haemorrhagic pituitary macroadenoma (20 × 17 × 17 mm) compressing the optic chiasm. Laboratory evaluation showed secondary adrenal insufficiency (morning cortisol 37 nmol/L, ACTH <3 pmol/L) and mild hyperprolactinaemia, with other pituitary axes preserved. The case was reviewed at the Pituitary MDT. Given stable vision and absence of severe mass effect, conservative management was chosen. Hydrocortisone replacement was initiated. Over subsequent months, symptoms resolved, MRI showed progressive tumour shrinkage, and adrenal function recovered, allowing steroid withdrawal. The patient remained clinically stable with normal pituitary function and controlled prostate cancer.

Discussion
GnRH agonist–induced pituitary apoplexy is rare but clinically significant. The temporal association suggests that rapid gonadotroph stimulation may precipitate intratumoral ischemia or haemorrhage in susceptible adenomas. Early recognition, prompt steroid therapy, and coordinated MDT input enabled successful non-surgical management and complete recovery.

Conclusion
Clinicians should maintain a high index of suspicion for pituitary apoplexy in patients presenting with acute headache or visual symptoms shortly after GnRH therapy. Conservative management can be effective in selected cases under close multidisciplinary supervision.

Learning Points:

- GnRH agonists for prostate cancer can rarely trigger pituitary apoplexy in undiagnosed macroadenomas.
- Acute headache, vomiting, or diplopia post-GnRH injection warrants urgent endocrine and imaging evaluation.
- Early corticosteroid replacement prevents adrenal crisis and supports recovery.
- MDT collaboration is essential for individualized management.
- Conservative treatment may achieve full endocrine and radiological remission in stable patients.

E021

A Convergence of Cardiology and Endocrinology: Understanding Carney`s Complex.

F Hussein, M Clarke, W Oo, M Dram, H Hussain, G Mlawa.

Barking, Havering and Redbridge University Hospitals NHS Trust.

Abstract

Introduction
Carney Complex is a rare multiple endocrine neoplasia syndrome involving skin pigmentation and multiple tumours affecting the adrenal, pituitary, and thyroid. It was described for the first time by J. Aidan Carney in 1985

A 52-year-old lady, who was referred for echocardiography following an episode of paroxysmal atrial fibrillation revealed a large mobile left atrial mass (Myxoma).

She underwent urgent surgical excision of the Atrial myxoma on the same day.

Cardiothoracic surgery was complicated by postoperative fast Atrial Fibrillation that was successfully chemically cardioverted with intravenous Amiodarone. She was discharged 7 days later.

She presented to the hospital 8 weeks later with cardiac sounding chest pain and breathlessness.

Findings
Chest clear, ECG sinus rhythm, troponin negative. Reviewed by the cardiology team and given an outpatient follow up appointment.

She was noted to have pigmented facial freckles, coarse facial features, and large hands during the review by the Endocrine team.

Medications included Cabergoline 250mcg weekly, hydrocortisone 15mg am, 5mg midday, and 5mg evenings, levothyroxine 125mcg once a day.

Her past medical history includes Acromegaly treated by transsphenoidal surgery in 1979, Goitre requiring partial thyroidectomy and hypothyroidism.

Her mother had myxoma.

The triad of acromegaly, facial freckles, and atrial myxoma made the diagnosis of Carney Complex most likely.

Discussion
Carney Complex is rare multiple endocrine neoplasia syndrome affecting adrenal, pituitary and thyroid.

It`s associated with other non-endocrine tumours such as cardiac, skin, mucosal, breast, myxomas, testicular tumours, melanotic schwannomas and abnormal pigmentation (spotty skin pigmentation/freckles).

The patient`s record shows that she had an echocardiogram in 2008 and 2010, and there was no mention of atrial abnormality.
Patients with Carney Complex should have annual review and annual blood test for IGF1, prolactin and thyroid function test.

They also need to have annual echocardiogram as well as a pituitary MRI, thyroid and testicular/ovarian ultrasound.

Annual colonoscopy is recommended if acromegaly is part of the Carney complex
This case report highlights the importance of an annual echocardiogram during endocrine clinic review and the importance of MDT approach in managing these patients as other speciality input would be needed for best management and outcome.

E022

Navigating Starvation Ketosis in Expectant Mothers with Gestational Diabetes Mellitus.

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Barking, Havering and Redbridge University Hospitals NHS Trust.

Abstract

Introduction
Starvation ketosis occurs when available glucose stores are inadequate to meet metabolic demands, resulting in a compensatory breakdown of free fatty acids into ketone bodies. In pregnancy, this metabolic complication is uncommon but potentially life-threatening, and a history of gestational diabetes (GDM) can markedly accelerate its development. Prompt recognition and treatment of this condition is vital to prevent adverse maternal and foetal outcomes.

Case report
We present a case of a 30-year-old female that was 29 weeks pregnant with history of GDM managed by diet alone. She presented with a 6 day history of persistent vomiting and abdominal pain, on a background of previous appendectomy four years ago. Upon investigation, blood ketones were elevated without accompanying acidosis. The urinalysis revealed 4+ ketones, and blood ketones 1.6 mmol/L despite normo-glycaemia.

Initial management comprised of IV thiamine followed by IV dextrose and further imaging in view of her surgical history. MRI results displayed a small bowel obstruction secondary to postoperative adhesions. Despite conservative management, vomiting persisted and decision to operate was initiated, managed with sliding scale prior and during surgery. Adhesiolysis was successfully performed, freeing the obstructed small bowel. Post-operatively, the patient had resumed oral intake, with ketone levels decreasing to 0.1 mmol/L.

Discussion
This case highlights the importance of considering starvation ketosis in pregnant women with GDM, presenting with persistent vomiting, even in the absence of ketosis. Management in such cases can be multifactorial, involving both medical and surgical care. In such cases, administration of intravenous thiamine prior to dextrose is essential to prevent lactic acidosis and Wernicke's encephalopathy. Prompt recognition and management of starvation ketosis is vital to ensuring positive maternal and foetal outcomes, preventing metabolic decompensation and impaired foetal development. The lack of local trust policy in managing starvation ketosis in pregnancy highlights the requirement of increased awareness into this under-recognised but potentially serious complication.

Conclusion
This case underscores the need for prompt recognition and early multidisciplinary involvement in cases of starvation ketosis, to enable optimal outcomes for maternal and foetal health.

E023

Cultural Challenges in DSD Management.

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Prime Health Hub-DEW.

Abstract

Case Study
A 17-year-old male patient presented with gynecomastia, decreased libido, absence of nocturnal penile tumescence, and sparse secondary sexual characteristics. Physical examination revealed developed breast tissue, a smaller right testicle, and an absent left testicle. The flaccid penile length measured 6 cm, with a girth of 5.5 cm.

Lab Investigations, including liver function tests (LFTs), renal function tests (RFTs), and Serum electrolytes, were within normal limits. The hormonal profile results showed the followingly: LH 6.52 mIU/mL (1.7-8.6 mIU/mL), FSH: 9.31 mIU/mL (1.5-12.4 mIU/mL), Prolactin: 25.40 ng/ml (4.0-15.2ng/mL), Testosterone: 1.28 ng/ml (1.75-7.81 ng/mL), Free Testosterone: 7.45 pg/ml (1.75-7.81pg/mL), Estradiol: 34.08 pg/ml (15 to 31.5 pg/mL), Progesterone: 0.17 ng/ml (14-2.06 ng/mL), SHBG: 27.81 nmol/L (18.3-54.1 nmol/L),17-OHP: 1.42 ng/ml (1-2 ng/ml), DHT: 468.33 pg/ml (300-850 pg/mL).

Scrotal ultrasound revealed bilateral atrophic testes, with the right testis volume measuring 3.4 ml, and a non-descended left testis accompanied by minimal hydrocele. Pelvic ultrasound showed non-visualisation of the prostate and seminal vesicles, as well as structures resembling a rudimentary uterus. An MRI confirmed the presence of a rudimentary uterus with a narrow vaginal canal and the absence of the prostate and seminal vesicles. A genetic karyotyping analysis has been performed, but the results are pending.

Management
The radiological investigations revealed the presence of male external genitalia along with Mullerian duct derivatives, and confirmed the absence of both the prostate and seminal vesicles. When it was time to discuss the diagnostic findings with the patient, his father insisted on being present, and the patient agreed. During the conversation, the patient experienced significant emotional turmoil, and both father and son found the results difficult to process.

The decision on how to proceed further depends on the patient's sexual orientation. The patient was encouraged to attend sessions with a clinical psychologist to help him better understand his sexual orientation. However, his father was reluctant to participate in these sessions and preferred to pursue a more traditional masculine path. Despite this, the patient was strongly encouraged to attend sessions with a clinical psychologist. When discussed separately about his sexual orientation after the psychologist sessions, he agreed with his father and chose to pursue a male gender identity.

Based on this decision, Testosterone replacement therapy was commenced, and a surgical consultation was arranged for the removal of breast tissue. A referral to a uro-gynecologist was also made for consideration of the removal of rudimentary female organs.

Discussion
This case shows the complexities of managing Disorders of sexual development (DSD) in Pakistani society, strongly influenced by traditional beliefs, which significantly impact social norms and perceptions of male dominance in a male-preferred society. A key issue involves a young person who has identified as male for 17 years within this cultural framework. This

raises an important question: how is it possible that, for 17 years, no one noticed his absence of testes and, more strangely, the development of breast tissue? This delay in recognising absent testes and developed breast tissue may reflect social discomfort in discussing sexual health and physical development, particularly during adolescence.

This scenario raises an additional question: is this person able to independently and without coercive influence, determine their future identity as male, female, or in terms of their sexual orientation? Or are their choices primarily shaped by family and cultural norms? Given the existence of scientifically acknowledged gender identities, we should also consider whether societal influences restrict this individual to a single option.

E024

Adrenal Infarction and Haemorrhage in Pregnancy: The Crucial Role of Early MRI and Multidisciplinary Management.

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Abstract
Haemorrhagic infraction of the adrenal gland during pregnancy is rare, but is probably under-reported and important to recognise because of the potential risk of hypoadrenalism, previous reports of vascular events within the adrenal gland in the literature describe presentations with acute ipsilateral abdominal or flank pain, nausea, and vomiting. Similar symptoms have been reported in other hypercoagulable conditions, and it is likely that the hypercoagulable state of pregnancy and the physiological changes in the adrenal glands during gestation are responsible for this observation. The adrenal gland has a rich arterial supply and a small venous return so venous thrombosis can give rise to back-pressure and haemorrhagic infarction. Given the overlap with other causes of abdominal pain in pregnancy, the diagnosis can be challenging and relies heavily on clinical suspicion and imaging. Magnetic resonance imaging (MRI) is the preferred modality during pregnancy, as it provides a clear assessment of adrenal pathology without exposing the foetus to ionizing radiation.

This report describes two cases of adrenal haemorrhage and infarction in pregnant patients. A 23-year-old female at 36 weeks of gestation presented with sudden-onset severe abdominal pain and left renal angle tenderness. MRI revealed left adrenal gland enlargement with surrounding fluid, consistent with acute adrenal haemorrhage. She underwent an emergency caesarean section, and the neonate was admitted for close monitoring. Follow-up confirmed intact adrenal function with normal Short Synacthen Test (SST) results, and a repeat MRI was planned in two months to monitor resolution of the haemorrhage.

In the second case, a 30-year-old female at 33 weeks gestation presented with severe right upper quadrant pain, elevated amylase levels, and neutrophilia, initially raising concerns for pancreatitis or appendicitis. MRI revealed findings consistent with right adrenal infarction, characterized by increased signal on T2-weighted imaging and marked diffusion restriction. The patient was started on low-molecular-weight heparin, and she delivered uneventfully at term. Follow-up MRI at one year showed atrophic changes in the right adrenal gland, but adrenal function remained normal. Thrombophilia screening was negative, and anticoagulation therapy was subsequently discontinued.

These cases underscore the importance of early diagnosis through clinical suspicion and MRI in managing adrenal haemorrhage and infarction during pregnancy. Both our patients were managed conservatively and did not have adrenal insufficiency. The interpretation of cortisol levels can be difficult in the 3rd trimester of pregnancy because raised SHBG will over-estimate cortisol assays, so clinical judgement is key. In cases of adrenal infarction, anticoagulation may be considered once haemorrhage is excluded. Early recognition and appropriate management of haemorrhagic adrenal infarction with consideration of adrenal function and appropriate imaging are crucial for optimizing both maternal and foetal outcomes in this rare but probably under-recognised condition.

E025

Multiple Endocrine Neoplasia Type 1 Presenting as Recurrent Overt Gastrointestinal Bleeding and Ulceration: A Diagnostic Challenge.

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The Hillingdon Hospitals NHS Foundation Trust.

Abstract
Multiple endocrine neoplasia type 1 (MEN1) is a multisystem endocrine disorder marked by tumors of the parathyroids, pancreatic islet cells, and pituitary gland. Although most cases are inherited through an Autosomal dominant pattern, a proportion arises through sporadic mutation. As the clinical presentation is highly variable, it often leads to diagnostic delay in patients with de novo mutation.

We report a 48-year-old man with no family history and a past medical history of renal calculi with intermittent hematuria. Over several months, he had multiple hospitalisations due to progressive epigastric pain and melena. Initial imaging suggested a localised small bowel perforation which was treated conservatively. Endoscopic evaluation revealed severe oesophagitis and duodenitis with biopsies suggesting peptic duodenitis. His symptoms were initially attributed to peptic ulcer disease and later to possible Inflammatory bowel disease (IBD), and he was managed with proton pump inhibitors (PPI) and steroids. Despite this, he continued to re-present similarly and also developed an episode of upper-limb thrombophlebitis requiring anticoagulation. Biochemical investigations demonstrated persistent hypercalcemia, elevated parathyroid hormone, and significantly raised fasting gastrin levels whilst on high dose PPI, raising suspicion for MEN1 syndrome. Functional imaging identified a parathyroid adenoma and multiple pancreatic and duodenal neuroendocrine tumours. Genetic testing confirmed a pathogenic MEN1 mutation. He was referred for parathyroidectomy followed by total pancreatectomy with duodenectomy, while first-degree relatives were offered genetic screening.

This case highlights the diagnostic complexity of MEN1 in the absence of a family history, where gastrointestinal ulcerations with bleeding, fleeting thrombophlebitis and hypercalcemia may serve as early clinical clues.

E026

Graves thyrotoxicosis with agranulocytosis secondary to anti thyroid medication: Difficult Medical Management leading to prolonged hospital admission and urgent thyroidectomy.

M Khan, K Norman, P Vankayalapati, J Cox, N Tolley, T Vakilgillani, M Siddiqui, M Yee.

Imperial College Healthcare NHS Trust.

Abstract

Background
Agranulocytosis is a rare complication of anti-thyroid medication with reported incidence of 0.3-0.6% with carbimazole[1,2] and 0.2-0.81% with PTU[3]. Mild neutropenia in thyrotoxicosis is reported in 10% of cases, but seldom significant[4]. This case highlights the challenges of managing uncontrolled thyrotoxicosis in the context of agranulocytosis and neutropenic sepsis. This case highlights the challenges of achieving the euthyroid state needed to proceed for necessary urgent surgical management.

Case Presentation
A 31-year-old white female previously diagnosed with Graves' disease presented with fever and tachycardia. She was non-compliant with anti-thyroid medication since diagnosis. She restarted propylthiouracil prior to admission. She was found to have absolute neutropenia and relapsed Graves' thyrotoxicosis with blood test showing thyrotoxicosis with bloods showing WBC of 0.8 x10 9 /l, neutrophils of 0.0 x10 9 /l, TSH <0.01 mU/L, fT4 50 pmol/L, fT3 >30 pmol/L on admission. She was initially managed with propranolol and cholestyramine. Due to ongoing fever and tachycardia, she required careful titration of beta blockers (Propranolol) up to 240mg daily, and management of neutropenic sepsis as per hematology and infectious disease input with filgrastim (G-CSF) and broad-spectrum antibiotics. Cholestyramine is not a standard treatment but has been used as an adjunctive treatment, although it had limited effect in her case. Steroids were not considered due to concern of further immunosuppression.

She remained inpatient due to ongoing fever, neutropenia, and difficulty in achieving biochemical euthyroid status. Decision of inpatient urgent thyroidectomy was made. In preparation for curative surgery, she was started on potassium iodide 65mg three times a day to block thyroid hormone synthesis via Wolf-Chaikoff effect to achieve an euthyroid state. She subsequently underwent a total thyroidectomy. Postoperatively, required propranol 20mg three times a day for two weeks. She developed no complications, with stable calcium (2.17 mol/L) and mild suppression of PTH (0.5mmol/L). She reported no hypocalcaemia symptoms or voice changes. She was started on lifelong levothyroxine.

Conclusion
This case illustrates the complexities of managing severe thyrotoxicosis with agranulocytosis and severe sepsis. A multidisciplinary strategy involving endocrinology, infectious disease, and surgery teams enabled safe optimization for definitive surgical management.

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3. Dose-dependent incidence of agranulocytosis in patients treated with methimazole and propylthiouracilJaeduk Yoshimura Noh 1 , Kosuke Inoue 2 , Nami Suzuki 1 , Ai Yoshihara 1 , Miho Fukushita 1 , Masako Matsumoto 1 , Hideyuki Imai 1 , Shigenori Hiruma 1 ,

Masahiro Ichikawa 1 , Masakazu Koshibu 1 , Akiko Sankoda 1 , Rei Hirose 1 , Natsuko Watanabe 1 , Kiminori Sugino 3 , Koichi Ito 3.

4. Neutropenia in patients with hyperthyroidism: Systematic review and meta-analysis
Lorenzo Scappaticcio 1, Maria Ida Maiorino 1 2, Antonietta Maio 2, Katherine Esposito 2, Giuseppe Bellastella

E027

Adalimumab-Induced Hypophosphataemia: An Unknown Adverse Effect.

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Imperial College Healthcare NHS Trust.

Abstract
Hypophosphataemia is a common electrolyte derangement in clinical practice, associated with a wide range of causes including drug-induced hypophosphataemia. Adalimumab, a tumour necrosis factor-alpha (TNF-α) inhibitor widely used in autoimmune diseases, has not been commonly linked to hypophosphataemia. Here, we describe a case of recurrent, treatment-resistant hypophosphataemia temporally associated with the initiation of adalimumab therapy, which subsequently resolved upon its discontinuation.

A 59-year-old woman of Bangladeshi origin was admitted in May 2025 with incidental severe hypophosphataemia (0.26 mmol/L) and hypomagnesaemia on routine outpatient bloods. She had multiple similar admissions since 2023 with varied symptoms including muscle spasms and myalgia. She denied any acute or chronic gastrointestinal losses, alcohol intake, or use of over-the-counter antacids.

Initial investigations on admission revealed low phosphate (0.26 mmol/L), low magnesium (0.46 mmol/L), normal calcium (2.41 mmol/L), normal parathyroid hormone (4.0 pmol/L), and low 25-hydroxy vitamin D (30 nmol/L).

Her past medical history included rheumatoid arthritis, type 2 diabetes, chronic kidney disease 3b (eGFR 30–40 mL/min), fatty liver disease with cirrhosis, hypertension and peptic ulcer disease. Additional history included a benign breast lesion (intraductal papilloma) and a necrotising granulomatous inflammatory pulmonary nodule resected via video-assisted thoracoscopic surgery (VATS). Her regular medications included Adalimumab, Hydroxychloroquine, Insulin, Metformin, Amlodipine and Omeprazole. She had previously been treated with methotrexate for rheumatoid arthritis, which was discontinued in 2023 due to abnormal liver function tests, after which she was switched to adalimumab.

On admission, she was treated with intravenous phosphate and magnesium, and a vitamin D loading regimen was initiated. Omeprazole was switched to Famotidine due to concerns that it might have been contributing to her electrolyte abnormalities. She was discharged on high-dose Vitamin D replacement.

Follow up at 1 month revealed ongoing hypophosphataemia. Her key biochemical findings and further investigation results were as follows:

	Normal range	**Jul-24**	**Feb-25**	**Early May-25**	**Late May-25**
Phosphate (mmol/L)	*0.8-1.5*	0.23 ↓	0.26 ↓	<0.2 ↓	0.35 ↓
Calcium (mmol/L)	*2.2-2.6*	2.87 ↑	2.63	2.41	2.67 ↑
Magnesium (mmol/L)	*0.7-1*	0.57 ↓	0.41 ↓	0.46 ↓	0.50↓
PTH (pmol/L)	*2.2-14*	0.8 ↓	—	4	1.2
Vitamin D (nmol/L)	*> 50*	58.8	45.2 ↓	30 ↓	—
eGFR (ml/min)	*> 90*	—	38	30–33	32
FGF-23 (RU/ml)	*<100*			112 ↑	

1,25 Vitamin D (pmol/L)	*55-139*			144 ↑	
24-hour urine phosphate (mmol/day)	*13- 42*			<2.13↓	
24-hour urine calcium (mmol/day)	*2.5- 7.5*			5.27 ↔	
Urinary protein (g/L)	-			13 ↔	

- **PET CT 2024:** pulmonary nodule and breast lesion (both were later identified as benign on biopsy)
- **DEXA 2024:** normal bone density

Though the FGF-23 level was mildly elevated, her 24-hour urinary phosphate excretion was low, with normal acid-base balance, normal urinary protein and only marginally raised 1,25 Vitamin D, making diagnoses of tumour-induced osteomalacia and renal tubular dysfunction less likely. Notably, the onset of hypophosphataemia in 2023 coincided with the initiation of Adalimumab for the management of her rheumatoid arthritis.

Given the patient's rheumatoid arthritis was stable, a decision was made with Rheumatology to temporarily withhold Adalimumab. This resulted in a normalisation of her biochemistry and symptoms, which have remained stable for four months following discontinuation of Adalimumab. These findings support the hypothesis of Adalimumab-induced hypophosphataemia. She is currently under close follow-up by the Rheumatology team, who are exploring alternative treatment options for her rheumatoid arthritis.

Although the underlying pathophysiological mechanism remains unclear, a potential association between tumour necrosis factor-alpha (TNF-α) inhibition and downstream effects on renal phosphate handling or systemic metabolism cannot be excluded. Furthermore, the presence of cirrhosis may have exacerbated hypophosphataemia in this patient, as chronic liver disease can impair gluconeogenesis and ATP turnover, thereby disrupting nutrient storage and promoting an intracellular phosphate shift [1,2].

This case describes a rare case of recurrent severe hypophosphataemia in a patient treated with Adalimumab. With the rising use of biologic therapies in multiple specialities, clinicians should remain alert to the potential of electrolyte disturbances as a possible adverse effect.

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E028

Management of Hyponatraemia in Metastatic SCLC.

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East and North Herts Teaching NHS Trust.

Background
Hyponatremia occurs frequently in small cell lung cancer due to inappropriate antidiuretic hormone secretion i.e. SIAD, a paraneoplastic syndrome with an incidence of 11-15%. It is associated with high morbidity and mortality and often delays appropriate treatment. In SCLC patients, SIAD is diagnosed predominantly in advanced stages and may induce a significant reduction in plasma sodium levels leading to poor outcome.

Case Presentation
A 59-year-old female presented with a 3-month history of headache, nausea, vomiting and epigastric pain and an admission Na of 114 mmol/L.

A hyponatremia screen was done and SIAD was diagnosed. A lesion was identified on her chest x-ray. CT thorax showed a left hilar mass with mediastinal invasion and lymphadenopathy consistent with primary lung carcinoma. Bronchoscopy confirmed small cell lung cancer with thoracic nodal disease and liver metastases.

She was fluid restricted and given a trial of demeclocycline which was ineffective. Tolvaptan 7.5mg improved her Na to 124 mmol/L and she was discharged.

She was readmitted by her oncologist prior to chemotherapy with a Na of 116 mmol/L and persistent vomiting and lethargy. The ward teams caring for her presumed the hyponatraemia was due to SIAD. The tolvaptan was increased to 15 mg and the fluid restriction was increased. This led to a drop in Na to 113 mmol/L.

She was admitted to ITU and given 2.7% hypertonic saline. Her Na improved to 127 mmol/L and was maintained with multiple doses of hypertonic saline. Her Na however continued to fluctuate and it was very difficult to correct the hyponatraemia without treating the underlying cause. After an MDT discussion, it was decided to start chemotherapy in hospital with a slightly lower sodium with HDU support and hypertonic saline if required.

The team subsequently sought advice from Endocrinology at Imperial College London. A mixed diagnosis of SIAD secondary to SCLC and dehydration due to vomiting and excessive fluid restriction was made. The tolvaptan was decreased to 7.5mg and she was given 0.9% NaCl as she appeared clinically dry which was confirmed by ECHO and ultrasound assessment. Her Na remained stable >120 on the ward once given IV fluids and she was discharged with Oncology follow up to continue chemotherapy with routine checks of her Na levels.

Conclusion
Dehydration in people with background SIAD is often not recognized. The treatment is to correct the dehydration before resuming the SIAD treatment. Following chemotherapy, the SIAD resolved. She is maintaining normal sodium levels without fluid restriction or tolvaptan. This case highlights the difficulty in managing patients with SIAD secondary to SCLC and the balance between medical management of hyponatremia and treatment of underlying malignancy for correction of Na.

E029

Complex management of Alemtuzumab-induced Grave's Disease complicated by thyroid eye disease with concurrent unplanned pregnancies.

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Abstract

Introduction
Alemtuzumab, a monoclonal antibody therapy known for its use as disease-modifying treatment of relapsing-remitting multiple sclerosis (RRMS), has been linked to development of autoimmunity. One such consequence is seen in autoimmune dysfunction of the thyroid – Graves' disease. Here we present a case of a 32-year-old female who developed Graves' disease 2 years after receiving Alemtuzumab infusions for relapsing-remitting multiple sclerosis, and subsequent difficulties in management.

Case presentation
The patient received treatment with Alemtuzumab with her last dose in June 2017. She initially presented with a high TSH 14.73mIU/L in September 2019 and referred to endocrinology. When she was seen in January 2020, she was clinically and biochemically thyrotoxic with a suppressed TSH<0.01mIU/L, and the decision was to monitor her blood tests over a short period of time. She received no treatment, as free thyroid hormones remained in the normal range until June 2020, when overt thyrotoxicosis declared itself and she was commenced on Carbimazole therapy, and responded. By that time, she was also on Natalizumab therapy for the MS. By early 2021 she was pregnant, having been counselled on use of antithyroid drugs in pregnancy previously. TRAb levels were first checked in June 2021, at 20/40, and were raised at 27 u/L. Her pregnancy was complicated by intermittently suboptimal control and neonatal thyrotoxicosis in October 2021, but her son has been well since and reaching milestones appropriately. By May 2023, she reported changes in the appearance of her eyes, with proptosis, lid retraction and intermittent chemosis, while MRI imaging revealed active disease nut no optic neuropathy. Her thyroid function had fluctuated throughout that period of time and TRAb had remained elevated between 19u/L and 30u/L. As the thyroid eye disease became a therapeutic priority and she was not planning further pregnancy, she was switched to block and replace treatment, which helped stabilise her thyroid function, albeit TRAb remained persistently elevated at 25.4 u/L. No orbital targeted therapy was deemed imminently necessary, other than single orbital steroid injection, and she was assessed in July 2024 to plan thyroidectomy. Her journey was complicated by an unplanned pregnancy shortly after that and was switched back to carbimazole monotherapy. Her thyroid function fluctuated throughout pregnancy, requiring up titration of medications, and lid retraction worsened. She delivered in May 2025 and in November 2025 free thyroid hormones were normal, with TRAb at 10.6u/L, while her thyroid eye disease remained unchanged. Repeat MRI imaging is planned, and she is due for a thyroidectomy soon.

Discussion
This case highlights the complexities of Graves disease and Graves eye disease in the following contexts:

1. Immunomodulatory agents such as Alemtuzumab are known to precipitate immune endocrinopathies. However, with this patient, there was a delay in initiating treatment, as when she was seen she was on Natalizumab, which has been reported to cause intermittent thyroiditis episodes, rather than autoimmune thyrotoxicosis, and this masked the diagnostic process.
2. Her journey highlights the importance of managing thyroid function in Graves, as uncontrolled hormone levels are linked to orbital symptoms and signs.

3. Immunomodulatory agents can often cause, not just severe disease, but also severely elevated TRAb levels, which are linked to thyroid orbitopathy incidence and severity, and her levels remained very elevated for many years, despite treatment for her Graves disease.
4. Relapses during antithyroid drug therapy and ongoing elevated TRAb levels are indications for definitive treatment, as they predict future relapse and relapse post-partum, in women planning pregnancy. In the context of pregnancy, neonatal thyrotoxicosis is a risk, and women need to be counselled on therapeutic options to facilitate pregnancy to risk stratify on an individual basis and acceptable risks to them specifically.
5. Pregnancy needs to be carefully planned, but those plans can be disrupted or interfered with due to waiting times for surgery, which are unavoidable.
6. Multi-disciplinary discussion and management of such patients is imperative, and in the case of our patient multiple professionals from endocrinology, ophthalmology, neurology, obstetrics, and obstetric medicine, endocrine surgery played a role in her complex journey.

E030

Between a Rock and a Hard Place: Surgical Decision-Making in High-Risk Amiodarone-Induced Thyrotoxicosis.

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Abstract

Case
A 68-year-old male, weighing 120kg, received amiodarone for atrial fibrillation from November 2022 until December 2023, after two unsuccessful direct current cardioversion attempts. He presented in August 2024 with symptomatic tachycardia and new, profound thyrotoxicosis (TSH <0.01mIU/L, fT3 21.7pmol/L, fT4 97.7pmol/L). He had no clinical signs of Graves' disease. He was initially treated with carbimazole 20 mg TDS and Lugol's iodine. Despite this, his symptoms worsened and thyroid function tests (TFTs) deteriorated (fT4 >100 pmol/L). TSH-receptor antibodies were undetectable. Despite observed inpatient compliance, his TFTs worsened. The patient's thyrotoxicosis proved refractory to a multimodal medical regimen of carbimazole, propylthiouracil, cholestyramine, and prednisolone (30mg daily). It was deemed that his surgical risk was too high for a thyroidectomy.

The patient went on to have a doppler ultrasound which demonstrated reduced vascularity, supporting a diagnosis of destructive AIT2. He started to improve after doubling his daily prednisolone dose to 60mg and became euthyroid following three months of treatment. At this point he remained on thionamides during prednisolone weaning. He commenced a GLP-1 agonist in the hopes of reducing surgical risk.

Eight months later he presented in fast AF, although his TFTs were normal. The local cardiology MDT felt ablative strategies were high risk given his BMI, yet his AF remains poorly controlled on calcium channel blockers. The only drug to which he has previously responded is amiodarone.

Management
Amiodarone-induced thyrotoxicosis (AIT) occurs in 6–10% of patients treated with amiodarone in the UK (1). Its highly lipophilic nature leads to extensive tissue accumulation, a large volume of distribution, and an extremely long and variable half-life. As a result, AIT can manifest unpredictably and long after drug cessation, creating significant management challenges. In the acute setting, the primary therapeutic goal is rapid control of thyrotoxicosis, as uncontrolled hyperthyroidism drives substantial cardiovascular instability, particularly in those with impaired ventricular function.

Current European Thyroid Association guidelines recommend emergency thyroidectomy as first-line therapy in life-threatening or drug-resistant AIT. When patients do not have significant premorbid cardiovascular disease, surgery should ideally be delayed until euthyroidism has been established (2). However, emerging observational evidence suggests that total thyroidectomy can be performed safely even in those with moderate to severe LV dysfunction when preceded by careful optimisation—including beta-blockade and glucocorticoids (3-5). Early restoration of euthyroidism is especially important, as persistent thyrotoxicosis itself is a major driver of cardiovascular decompensation and arrhythmia burden.

The alternative—prolonged high-dose glucocorticoid therapy, often required for weeks to months in AIT type 2—carries its own risks. Steroids may worsen hypertension, hyperglycaemia, fluid retention, and bone health, all of which can significantly increase morbidity in patients with underlying cardiac disease. In this case, delays in achieving euthyroidism contributed to repeated hospital admissions and ongoing clinical instability, highlighting the potential value of earlier surgical intervention despite cardiac comorbidities.

Amiodarone may be the only effective rhythm-control option for some individuals with complex arrhythmia profiles; however, re-exposure risks precipitating recurrent AIT, with its attendant cardiovascular consequences.

Overall, management requires multidisciplinary discussion between endocrinology, cardiology, endocrine surgery, and anaesthetics. The central priority is timely restoration of euthyroidism, balancing the risks of surgery in a physiologically unstable patient against the substantial harm of prolonged thyrotoxicosis, while integrating an approach to AF that minimises further decompensation.

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E031

Ivor Lewis Procedure – addressing the challenge of post-operative hypoglycaemia

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Introduction
The Ivor Lewis oesophagectomy is a two-stage surgical technique commonly utilised in the removal of malignancies in the distal oesophagus or gastroesophageal junction. The procedure involves mobilisation of the stomach and resection of the oesophagus to form an oesophagogastric anastomosis. This procedure carries the risk of metabolic complications, such as hypoglycaemia. We present a case of a patient with multiple hospital admissions due to symptomatic hypoglycaemia following an Ivor Lewis Oesophagectomy, with no previous diabetic history.

Case
A now 79-year-old gentleman initially presented in November 2021 with two-year history of progressive dysphagia and significant unintentional weight loss. Investigations confirmed a lower oesophageal Barrett's adenocarcinoma (T3N1M0) for which the patient underwent an Ivor Lewis oesophagectomy procedure in March 2022. Two months postoperatively, he began to develop recurrent episodes of symptomatic hypoglycaemia (CBG 1.8-3.0 mmol/L), characterised by shaking and light-headedness. Continuous capillary blood glucose monitoring was conducted with FreeStyle Libre, of which findings were consistent with reactive, postprandial hypoglycaemia secondary to late dumping syndrome. Episodes of hypoglycaemia were closely linked with high carbohydrate-meals, with avoidance of high-sugar maintaining more stable blood glucose levels. The patient was counselled on the importance of dietary modification, including small, frequent, low–glycaemic index meals, which has since led to a marked improvement in his symptoms of late dumping syndrome.

Discussion
The mechanism for the hypoglycaemia in this non-diabetic patient can be attributed to late dumping syndrome, typically occurring 1-3 hours after meals, with an exaggerated insulin response to rapidly absorbed carbohydrates and resultant reactive hypoglycaemia. Recognition of this syndrome is critical to ensuring efficient management and prevention of adverse outcomes. The management of this complication is mainly dietary – changing meal frequency and quantity to minimise this under-recognised complication of Ivor Lewis procedures.

Conclusion
Early identification of reactive hypoglycaemia and late dumping syndrome following an Ivor Lewis Oesophagectomy can allow for early and efficient management of symptoms, significantly improving post operative quality of life.

E032

Looking beyond the Common: A case of Severe Cushing's Syndrome Revealing Metastatic Adrenocortical Carcinoma.

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Abstract

Background
Cushing's syndrome results from prolonged exposure to excessive glucocorticoid activity. It is a multisystem disorder and increases mortality approximately threefold compared with the age- and sex-matched general population. Adrenocortical carcinoma (ACC) is a rare but aggressive cause of Cushing's syndrome, and all patients with suspected or confirmed ACC should be managed by a multidisciplinary expert team.

Case Presentation
A 68-year-old man, usually fit and in good general health, with a background of hypertension and hypercholesterolaemia, presented to a district general hospital with rapidly progressive breathlessness, bilateral pedal oedema, and abdominal distension over three weeks, initially thought to represent heart failure. He also reported upper back pain, two episodes of chest infection over three months, and profound fatigue. His breathlessness was considered disproportionate to the preserved cardiac function on echocardiography performed six weeks earlier, and the combination of persistent hypokalaemia and hypertension despite ramipril and spironolactone prompted further investigation. A CT pulmonary angiogram revealed a pulmonary embolism with pleural infarction and right heart strain, while contrast-enhanced CT of the abdomen and pelvis demonstrated a 9.2 cm left suprarenal mass with internal necrosis, intra-abdominal lymphadenopathy, and vertebral compression fractures. The local endocrine team was consulted for detailed assessment and endocrine work-up. Distinctive Cushingoid features were identified, including truncal obesity, thin skin with bruising over the chest wall, purple abdominal striae, and proximal myopathy, together with associated findings of venous thromboembolism, recurrent infections, and osteopenia. Initial adrenal investigations showed markedly elevated cortisol (1,417 nmol/L) unsuppressed by overnight dexamethasone 1 mg (1,321 nmol/L), with normal plasma metadrenalines. Given the severity of hypercortisolism, treatment was commenced immediately under expert tertiary guidance using a metyrapone–dexamethasone block-and-replace regimen, without waiting for further confirmatory testing. The clinical course was complicated by severe hospital-acquired pneumonia with respiratory failure requiring intensive care, followed by temporary improvement. Subsequent endocrine studies demonstrated suppressed ACTH (<5 ng/L), elevated DHEA (50 µmol/L) and markedly raised cortisol and adrenal androgen metabolites on random urinary steroid profiling, consistent with a strong possibility of adrenocortical carcinoma complicated with adrenal Cushing's syndrome. FDG-PET-CT revealed widespread metastases, and the tertiary adrenal MDT deemed the tumour inoperable, advising oncology review for chemotherapy and palliative care input. Advanced care planning was initiated, but the patient deteriorated and passed away before receiving input from the oncology team. Histology from a supraclavicular lymph node confirmed a metastatic poorly differentiated malignant tumour consistent with a deposit from ACC.

Learning Points
- Persistent hypokalaemia and resistant hypertension should prompt consideration of an underlying endocrine cause, and clinicians must maintain a high index of suspicion to avoid missing features of Cushing's syndrome.
- Endogenous Cushing's syndrome carries an approximately threefold higher mortality risk, primarily due to cardiovascular, thromboembolic, and infectious complications, with the greatest risk observed in those with adrenocortical carcinoma or uncontrolled cortisol excess.

- Early diagnosis, rapid biochemical control, and proactive risk management of hypercortisolism are crucial to improve outcomes.
- All patients with suspected adrenocortical carcinoma should be referred early for expert multidisciplinary discussion to guide investigation and management. Prognosis remains very poor in metastatic disease.

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E033

Complex Interplay: Graves' Thyrotoxicosis, Hypercalcaemia and Deranged LFTs.

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Introduction

Thyrotoxicosis is defined as a clinical condition caused by excess circulating thyroid hormones (T3 and T4). These hormones are responsible for increasing sympathetic activity thorough increasing basal metabolic rate and upregulating beta-adrenergic receptor. Therefore, an overexpression of circulating thyroid hormones can lead to a hypermetabolic state, presenting with signs and symptoms across multiple organ systems.

Graves' thyrotoxicosis can occasionally present with concomitant hypercalcaemia and liver function derangement which remit following resolution of thyrotoxicosis. We present a five-patient case series illustrating this biochemical triad from presentation to post-treatment resolution.

Cases

1. A 49-year-old female with a past medical history of type 2 diabetes mellitus (Metformin 500 mg BD), hypertension (Amlodipine 5 mg OD), hyperlipidaemia (Atorvastatin 20 mg ON), and Grave's disease (First diagnosed at 37, relapsed at 41). She presented to the emergency department with tremors and palpitations.

   Initial bloods showed thyrotoxicosis with associated hypercalcaemia and hepatic dysfunction:

Test	Result
TSH	0.1
Free T4	100
Adjusted Ca	2.73
Vitamin D	27
ALP	107
ALT	126
Bilirubin	22

   Management in hospital included carbimazole 30 mg OD, propranolol 40 mg TDS and a 5-day course of prednisolone 30 mg OD.
   Follow-up bloods 4 weeks post treatment showed the resolution of hypercalcaemia and LFTs with T4:

Test	Result
Free T4	24.8
Adjusted Ca	2.54
ALP	120
ALT	35
Bilirubin	12

2. An 80-year-old female with a past medical history of goitre and frequent falls. She presented to the emergency department with shortness of breath, peripheral oedema and palpitations. Initial assessment noted a goitre with an irregularly irregular pulse and bi-basal crepitations, with ECG noting fast atrial fibrillation and an echocardiogram noting LVEF 45%.

Initial bloods showed thyrotoxicosis, with associated hypercalcaemia and deranged LFTs:

Test	Result
TSH	< 0.01
Free T4	31.8
Adjusted Ca	2.62
ALP	142
ALT	28
Bilirubin	11

She was treated with furosemide 40 mg OD, carbimazole 10 mg OD and prednisolone 30 mg OD. As she remained in atrial fibrillation, she was commenced on rivaroxaban 20 mg OD. Following discharge her adjusted calcium and LFTs normalized with her TFTs:

Test	Result
Free T4	12.1
Adjusted Ca	2.51
ALP	136
ALT	23
Bilirubin	7

3. An 83-year-old female with a past medical history of asthma (Fostair, Salbutamol inhalers) and hypertension (Lisinopril 20 mg OD). She presented to the emergency department with palpitations, diaphoresis, coarse tremor and delirium.

   Initial bloods showed thyrotoxicosis with associated hypercalcaemia and hepatic dysfunction:

Test	Result
TSH	< 0.01
Free T4	> 100
Adjusted Ca	2.68
Vitamin D	26
ALP	124
ALT	49
Bilirubin	11

   Thyroid ultrasound noted a heterogenous thyroid with hyperaemia in keeping with Graves' disease.
   She was treated for an impending thyroid storm with associated atrial fibrillation and congestive heart failure with hydrocortisone, carbimazole, colestyramine, amoxicillin, clarithromycin, diltiazem, furosemide and apixaban.

   Follow-up bloods a few months post-discharge noted the resolution of her adjusted calcium and LFTs along with her thyroid function tests:

Test	Result
Free T4	21
Adjusted Ca	2.52
ALP	80
ALT	20
Bilirubin	8

4. A 52-year-old female with a past medical history of a mitral valve and tricuspid valve replacement (Warfarin 4.5 mg OM), atrial fibrillation (Amiodarone), hypertension (Doxazosin 8 mg OD, Candesartan 8 mg OD), type 2 diabetes (Metformin 500 mg OD). She presented to the emergency department with atrial fibrillation with atrial fibrillation with rapid ventricular rate.

   Initial bloods revealed a deranged ALP with thyrotoxicosis with normal adjusted calcium:

Test	Result
TSH	< 0.01
Free T4	40.1
Adjusted Ca	2.37
ALP	292 (Baseline 180)

   Workup with a thyroid ultrasound revealed features in line with amiodarone-induced thyrotoxicosis, type 2 and a multinodular thyroid (U2).
   She was treated with carbimazole 30 mg BD.
   Liver function tests resolved following the resolution of said hyperthyroid state post-discharge:

Test	Result
TSH	1.09
Free T4	17.3
Adjusted Ca	2.28
ALP	189

5. A 76-year-old male with a past medical history of ASMA-positive autoimmune hepatitis, hypertension (Amlodipine 5 mg OM, Ramipril, Indapamide), type 2 diabetes (Metformin), hyperlipidaemia (Simvastatin). He presented to the emergency department with chest pain, revealed following workup to be an NSTEMI with associated congestive cardiac failure with pulmonary oedema and thyrotoxicosis.

   Initial bloods revealed thyrotoxicosis with hypercalcaemia with normal vitamin D and PTH:

Test	Result
Free T4	52.2
Adjusted Ca	2.63
Vitamin D	61
PTH	4.8

   His NSTEMI was treated medically with aspirin and clopidogrel, with his thyrotoxicosis with hydrocortisone and PTU 100 mg BD.
   His calcium normalized post-discharge with his thyroid function:

Test	Result
Free T4	21
Adjusted Ca	2.57

Discussion/Conclusion

Hypercalcaemia and deranged LFTs can occur concomitantly with thyrotoxicosis. Some of the postulated mechanisms for abnormal liver function tests include direct toxicity to the liver due to excessive hormones, drug-induced (anti-thyroid medications), autoimmune liver injury, and high-output heart failure leading to congestive hepatopathy. Hypercalcaemia in thyrotoxicosis is explained by increased bone resorption through osteoclast activity activation. It is important to check baseline TFTS and liver function tests in this cohort of patients and regularly monitor them while the patients are on antithyroid therapy.

E034

The Intersection of Genetics and Endocrinology: Idiopathic Infantile Hypercalcemia from CYP24A1 deficiency and a Decade-long Missed Diagnosis.

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Abstract

Introduction
Hypercalcemia is commonly caused by primary hyperparathyroidism, malignancy, or medications. Idiopathic infantile hypercalcemia (IIH) is a rare autosomal recessive condition due to an inactivating mutation or deletion in the *CYP24A1*gene on chromosome 20, which results in loss of function of the enzyme vitamin D 24-hydroxylase, leading to impaired inactivation of active vitamin D metabolites. Although classically presenting in infancy, adult cases are increasingly recognized.

Case Presentation
We report a 72-year-old male with an 11-year of recurrent hospital admission due to persistent hypercalcemia, nephrolithiasis, and haematuria. He was referred by his GP to the endocrinology clinic for further assessment. Routine investigations repeatedly showed elevated calcium 2.89 mmol/L with suppressed/normal PTH. Ultrasound, technetium-99m sestamibi scanning, CT CAP, and PET imaging were all normal. He was referred for genetic testing which eventually confirmed a *CYP24A1* mutation which reduces the activity of the vitamin D 24-hydroxylase enzyme, an enzyme responsible for vitamin D breakdown. The diagnosis of (IIH) was made. He was commenced on fluconazole 50 mg daily and cinacalcet 30 mg twice daily, resulting in normalisation of calcium levels to 2.50 mmol/L. Three first-degree relatives tested positive for the same mutation. Since treatment initiation, he has remained normocalcemic without further renal colic episodes.

Discussion
CYP24A1 mutations lead to reduced vitamin D breakdown, resulting in elevated levels of active vitamin D and persistent hypercalcemia with suppressed PTH. Although typically diagnosed in childhood, adult presentations may occur after years of unexplained hypercalcemia. Previously, a diagnosis of (IIH) was made only after exclusion of more common causes of hypercalcemia. Standard acute management follows conventional hypercalcemia treatment. Long-term therapy includes placing patients on low-calcium and vitamin D–free diets. In addition, antifungals such as fluconazole—which inhibit 1α-hydroxylase and 25-hydroxylase involved in vitamin D activation—can be used. Patients with persistent hypercalciuria and recurrent nephrolithiasis may be treated with thiazide diuretics, which reduce urinary calcium excretion.

Conclusion
Early recognition of (IIH) prevents complications including nephrolithiasis, nephrocalcinosis, and renal impairment. Genetic screening is recommended for relatives due to autosomal recessive inheritance.

This case highlights that *CYP24A1*-related IIH can present in adulthood after prolonged unexplained hypercalcemia. Genetic testing should be considered in patients with PTH-independent hypercalcemia and recurrent renal stones. Targeted treatment with fluconazole and cinacalcet can successfully normalise calcium levels and prevent complications.

E035

Testosterone Replacement Therapy and Raised Intracranial Pressure: A Complex Interplay in a Patient with Congenital Hydrocephalus and Obesity.

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Abstract

Background
Testosterone therapy has been infrequently associated with raised intracranial pressure (ICP), although the association remains rare and not fully understood. Proposed mechanisms include androgen-mediated alterations in cerebrospinal fluid (CSF) production or absorption. This relationship is particularly unclear in patients with pre-existing structural brain abnormalities such as congenital hydrocephalus and cerebral palsy.

Case Presentation
A 47-year-old man with congenital cerebral palsy and obesity class III presented with reduced frequency of erections, fatigue and difficulty concentrating. He was diagnosed with hypogonadotrophic hypogonadism (testosterone 10.6 nmol/L [RR 10–30], SHBG 21.3 nmol/L [RR 15–55], free testosterone 0.246 nmol/L). He was initially commenced on testosterone gel for one year and subsequently switched to testosterone undecanoate (Nebido) injections due to persistent symptoms of hypoandrogenism.

To achieve stable symptom control, the Nebido dosing interval was gradually shortened, resulting in improvement in hypogonadal symptoms over two years. After transfer of care to our Trust, an MRI pituitary was arranged to evaluate for structural causes of central hypogonadism beyond metabolic contributors. The patient additionally reported a two-year history of worsening headaches and memory difficulties.

MRI demonstrated no pituitary adenoma but revealed marked enlargement of the third and lateral ventricles secondary to aqueductal stenosis, without features of acute CSF obstruction. Within two months of his MRI pituitary been performed, the patient presented acutely following a holiday, with severe headache, vomiting, diplopia and abducens nerve palsy. CT venogram and repeat MRI confirmed stable triventricular hydrocephalus with porencephalic dilatation of the right lateral ventricle, and old perinatal venous haemorrhage.

He was referred to neurosurgery and underwent endoscopic third ventriculostomy, which significantly improved his headaches. The endocrine plan is to gradually wean testosterone therapy and support weight reduction in an effort to assess for recovery of endogenous gonadal function.

Discussion
While testosterone therapy is not known to cause acute hydrocephalus or aqueductal obstruction, androgen-related increases in intracranial pressure may unmask or exacerbate symptoms in patients with pre-existing CSF flow abnormalities. In this case, chronic aqueductal stenosis was already present, and testosterone replacement therapy may have contributed to decompensation of previously compensated hydrocephalus.

This case highlights the rare but important interplay between testosterone therapy, raised ICP and hypogonadotrophic hypogonadism in an obese patient with congenital hydrocephalus. It underscores the importance of clinical vigilance when prescribing testosterone in individuals with underlying neurological abnormalities or risk factors such as obesity. A multidisciplinary and individualised approach is essential to ensure safe hormonal optimisation while preserving neurological stability.

E036

Not all calcitonin is cancer: a case of mistaken identity.

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Absract

Introduction
Diagnosis of medullary thyroid carcinoma (MTC) can be challenging, as fine-needle aspiration (FNA) cytology has variable sensitivity.[1] When MTC is suspected, NICE guidelines recommend serum calcitonin measurement to support diagnosis.[2] Some international guidelines suggest measuring calcitonin in FNA washout fluid;[3] however this is not currently practiced in the United Kingdom (UK). The increasing use of glucagon-like peptide-1 (GLP-1) receptor agonists adds complexity, as preclinical studies suggest these agents may induce C-cell hyperplasia and be associated with MTC.[4]

This report describes a patient with Hashimoto's thyroiditis and GLP-1 receptor agonist use, initially suspected to have MTC due to markedly elevated thyroid FNA calcitonin level.

Case presentation
A 32-year-old woman with Hashimoto's thyroiditis presented in Cyprus with a recent neck lump. Her medications included levothyroxine 150mcg daily, and semaglutide (Wegovy). She had no family history of thyroid malignancy. Ultrasound in Cyprus revealed a 1cm thyroid nodule; FNA demonstrated elevated calcitonin levels (>2000pg/ml), raising concern for MTC. She was referred to our centre for evaluation and possible thyroidectomy.

Repeat ultrasound demonstrated a small heterogeneously hypoechoic thyroid gland, compatible with autoimmune thyroiditis, with no discrete nodules or areas suspicious for malignancy; a repeat FNA was therefore not indicated. Laboratory investigations showed normal thyroid function, calcium and parathyroid hormone levels, with undetectable serum carcinoembryonic antigen (CEA) and calcitonin levels. Multidisciplinary team review concluded there was no concern for malignancy. The patient remains well and is expected to be discharged following review.

Discussion
The role of FNA calcitonin measurement in diagnosing MTC is uncertain, and not endorsed by UK guidelines. Results require cautious interpretation, especially in Hashimoto's, where C-cell hyperplasia may spuriously elevate results.[5] Pre-clinical studies suggest GLP-1 receptor agonists may have a similar effect.[4] A proposed FNA calcitonin threshold of 4085.5pg/ml provides a high sensitivity and specificity for MTC detection,[6] but requires validation.

Current guidelines support continued GLP-1 receptor agonist use in the absence of a personal or family history of MTC or multiple endocrine neoplasia type 2 (MEN2).[7] If further investigation is indicated, ultrasonography, cytology and serum calcitonin measurement should be prioritised over FNA calcitonin.

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E037

A complex case of stature: transitional endocrinology.

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Abstract
An 18-year-old male underwent surgery for a slipped upper femoral epiphysis; significant scoliosis was also noted. 6 months later, he was referred to an endocrine colleague with a history of increasing stature. He started out being the shortest in his class at 6-13 years, when he started gaining height, similar to his peers by 15 years, and then rapidly becoming the tallest. In addition, he started noticing a change in facial features and an increased size of his feet (14.5 UK size). He did not need to shave. Also reported increased headaches with 'brain fog', increased introverted behaviour, and not being able to cope with schoolwork. He had a large black spot in his vision, bigger on the right compared to the left, around the age of 13-14 years and had seen the opticians, but this had not been commented on.

Father was 168cm, mother 165cm, sister 155cm and brother 180cm. Mother was investigated for pituitary disease 10 years earlier, but did not return for treatment. The mother's paternal grandfather was apparently 192cm and died at the age of 71 years, attributed to lung disease.
At initial endocrine assessment, he was 200.7cm, had a large head, deep voice, macroglossia, dorsolumbar scoliosis, and large hands/feet with specially made shoes for different heel height. Testes size was 15mls. His biochemical tests showed Growth Hormone(GH) >100mcg/L, IGF-1 1.65xULN(upper limit of normal), prolactin normal, secondary adrenal insufficiency, hypothyroidism and hypogonadism. We consider that his IGF-1 at diagnosis may not completely reflect the burden of GH excess at the time due to hypothyroidism, hypogonadism and hypoadrenalism. Pituitary MRI revealed a lobulated sellar/suprasellar lesion (3.7x3.6x2.5cm) with extension into the right cavernous sinus. The optic chiasm was displaced superiorly within the suprasellar cistern. His bone age was 14 years at the chronological age of 18 years. He was started on thyroxine and hydrocortisone and monthly octreotide injections, and 3 months later had transsphenoidal surgery. Histology confirmed a sparsely granulated somatotroph adenoma, Ki67 proliferative index of 1-2%. Post-operative GH was 16.1mcg/L, IGF-1 1.24xULN. Two months after surgery and 6 months after diagnosis, he was 205 cm.

Points to consider for medical therapy options were:

1. Young age
2. Large tumour remnant
3. Open epiphysis
4. AIP mutation positive state (this was predicted due to his clinical picture and family history, even before genetic results arrived), which suggests poor responsiveness to first-generation somatostatin analogues
5. The need to build undetectable testosterone levels up to the adult range
6. Already very tall stature with skeletal complications (hip, spine)

He was started on pegvisomant and a gradually increasing dose of testosterone. MDT recommended proton beam radiotherapy. He reported persistent sharp eye pains following radiotherapy, which was diagnosed as radiotherapy-associated blepharitis.

His genetic testing identified a novel stop mutation in the *AIP* gene. Her mother and sister were also positive. Ten years earlier, the mother had a thyroidectomy due to multinodular goitre, elevated IGF-1, OGTT GH nadir of 0.5mcg/l and an 'abnormal MRI'. The current contrast-enhanced pituitary MRI did not find any abnormality, while her hormonal status for IGF-1 was

borderline. She shows wide hands, mild facial features consistent with acromegaly. The 21-year-old sister had amenorrhoea, elevated prolactin and a 4mm lesion on pituitary MRI.

One year after diagnosis and 6 months after start of pegvisomant and testosterone, his height is 207cm and his IGF-1, which decreased after surgery to 1.24xULN, is now 1.62xULN (with apparent good compliance with pegvisomant), probably due to the testosterone-induced pubertal changes. We are wondering which IGF-1 ULN should be used for his assessment: matching his chronological age, or rather matching his bone age, with additionally considering the normalising testosterone levels.

This case illustrates the complexity of management of a patient with genetically determined GH excess, large tumour, delayed puberty and emphasises the importance of MDT-based management of pituitary disease involving paediatric/adult endocrinology, neurosurgery, radiotherapy, physiotherapy, orthopaedics, ophthalmology, and genetics.

E038

A challenging case of Pituitary apoplexy with anticoagulation dilemma.

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Abstract

Introduction
Pituitary apoplexy (PA) is defined as acute hemorrhagic or ischemic infarction of the pituitary gland, most commonly within a pre-existing adenoma. Incidence is approximately 0.17 episodes per 100,000 person-years globally, with a UK prevalence of around 6 per 100,000, and precipitating factors including anticoagulation, surgery, hypertension, and others such as COVID-19 or GnRH agonists. Acute secondary adrenal insufficiency is the most immediate life-threatening consequence, occurring in 50-70% of cases. Partial recovery of pituitary function varies by axis after apoplexy (e.g., 17-50% for specific hormones, often necessitating lifelong replacement for deficient axes). Management is controversial when therapeutic anticoagulation is indicated, as re-bleeding risk must be balanced against venous thromboembolism (VTE). We present a case illustrating successful conservative management with early, safe re-introduction of anticoagulation guided by pituitary MDT.

Case
A 73-year-old male generally fit and well who underwent elective shoulder replacement and post op was commenced on Edoxaban 60mg OD for new onset Atrial Fibrillation. After 3 weeks, presented to ED with sudden onset headache and vomiting. Initial CT head report showed no evidence of hemorrhage or infarct and space occupying lesion. However, 2 days after he represented with profound lethargy, fluctuating levels of consciousness, acute hyponatremia of 119 mmol/l and low blood pressure of 105/60mmHg. Repeat CT head showed slight prominence in cavernous sinus with seller extension.

He was immediately commenced on treatment for suspected adrenal crisis with IV hydrocortisone 100 mg 6-hourly initially then 50 mg 8 hourly after 48 hours with fluid rehydration. Edoxaban was withheld, and mechanical VTE prophylaxis commenced. There were no visual concerns and visual fields by bedside were normal to confrontation. Blood test confirmed anterior hypopituitarism. CT venogram was done as opposed to MRI, due to initial concerns of shoulder metal prosthesis which excluded cavernous sinus thrombosis. MRI pituitary with contrast performed after checking MRI compatibility of prosthesis, demonstrated a 20 × 21 × 16 mm seller mass with internal T1-hyperintensity and minor suprasellar extension with no optic chiasm compression.
Case and images were shared with the neurosurgery team for immediate advice to continue with management of hypopituitarism.

He was closely monitored with hourly urine output due to the risk of unmasking of diabetes insipidus by steroids if present. Levothyroxine commenced 48 hours after establishing steroid replacement.

Pituitary profile:

9 am cortisol	46 nmol/L	170 – 700 nmol/L
TSH	1.6 mIU/L	0.4 - 3.6 mIU/L
free T4	6.3 pmol/L	11 – 22 pmol/L
Total prolactin	109 mU/L	<360 mU/L
LH	1.6 IU/L	1.5 - 9 IU/L
FSH	2.4 IU/L	1.5 - 12 IU/L
Testosterone	<0.42 nmol/L	10 – 35 nmol/L (male)
IGF-1	pending	

The challenge

Clinical improvement was noticed with all symptoms resolved within 36 hours. Formal visual fields were reported as normal. However, on day 4 of admission he developed a new oxygen requirement. CTPA reported no large pulmonary embolism but raised suspicion of segmental pulmonary embolism and raised left hemidiaphragm.

Discussion with neurosurgical team and specialist pituitary MDT advised that he could restart anticoagulation within 7-14 days due to risk of thromboembolism if needed for Pulmonary embolism assuming he remains well. Monitor for any new headaches, visual and visual field or cranial nerve issues. To continue with Hormonal replacement as needed, to repeat MRI pituitary in 3 months' time with visual field and return to pituitary MDT and local endocrine clinic follow up with dynamic tests (Short synacthen test).

He was commenced on prophylactic anticoagulation with Apixaban 2.5mg BD, switched to oral hydrocortisone 10 mg-5 mg-5 mg and levothyroxine titrated to 75 µg daily prior to discharge.
He remained well with no neurological concerns. The sodium was 138 mmol/L.
Sick day rules with emergency hydrocortisone injection with steroid identifier were provided prior to discharge. He was discharged on day 12.

Conclusion

This case exemplifies two key learning points:

1. Rapid recognition and management of adrenal crisis. Pituitary apoplexy was suspected based on clinical presentation, acute hyponatremia, and initial CT findings. He was not known to have pituitary macroadenoma.

2. Anticoagulation dilemma in high risk of venous thromboembolism in confirmed Pituitary apoplexy. Small case series indicate an increased (but unquantified) risk of re-hemorrhage with therapeutic anticoagulation. A pragmatic approach to balance risk of further bleeding versus thromboembolism was advised bridging with prophylactic dose from day 7 – 14 days while monitoring for new neurological or visual compromise with follow up MRI pituitary and visual field testing

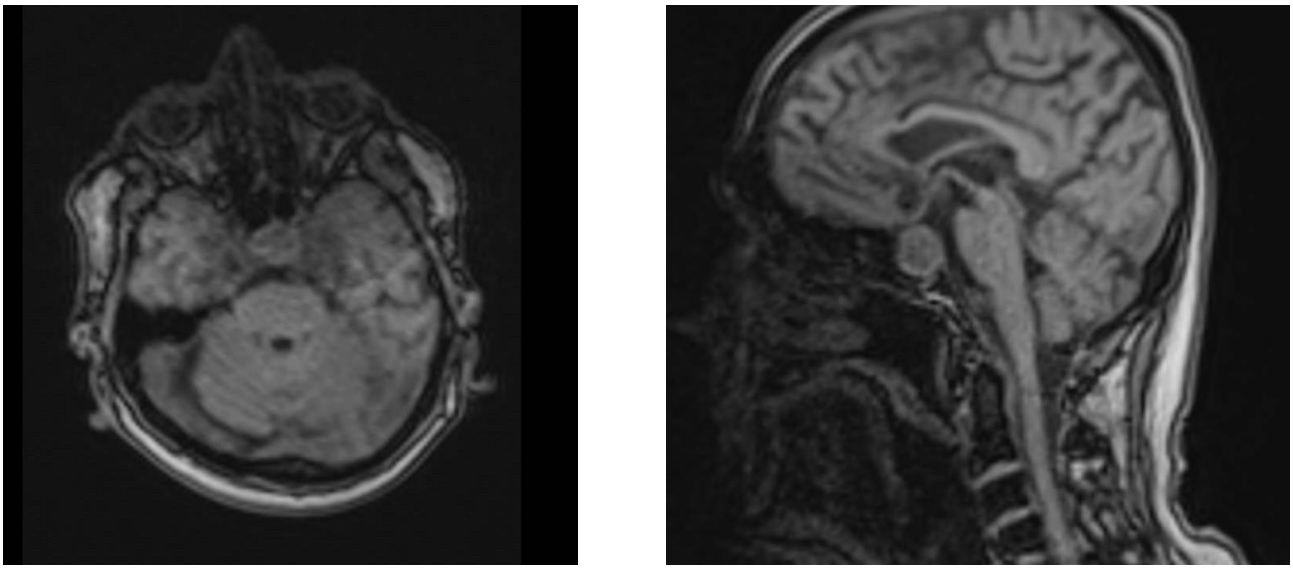

Figure. Horizontal (left) and sagittal (right). MRI with contrast showing 20 × 21 × 16 mm sellar mass with subacute blood products. Minor suprasellar extension abuts but does not compress the optic chiasm.

Learning Points

- Pituitary apoplexy can present subacutely with headache progressing to adrenal crisis within 24–72 hours.
- Empiric IV hydrocortisone (100 mg 6-hourly) is lifesaving and must not await cortisol results.
- Conservative management is appropriate when visual acuity/fields and consciousness are preserved.
- A multidisciplinary management approach is required including advice regarding reestablishing anticoagulation in high-VTE-risk patients.

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E039

A Tale of Three Crisis.

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Abstract

Introduction
Adrenal insufficiency, resulting in low circulating glucocorticoids from the inadequate production from the adrenal glands, can be classified as either primary, secondary or iatrogenic, from exogenous corticosteroid use. Acute presentations of adrenal insufficiency, or adrenal crisis, can be life threatening, necessitating urgent treatment with emergency doses of corticosteroids. Here we present three cases to highlight the differing ways in which adrenal insufficiency may present.

Case One
A 20-year-old student presented to the emergency department with a five-day history of postural dizziness, vomiting and 6kg weight loss over the last two years. He had no past medical history and was on no regular medications. Initial assessment found; heart rate 115bpm, blood pressure 102/66mmhg, with no postural blood pressure change.

Admission blood tests showed; sodium 123mmol/L, potassium 4.3mmol/L, creatinine 71umol/L, eGFR >90 ml/min/1.73m^2, glucose 4.1mmol/L, serum osmolality 262mOsm/kg, urine osmolality 1085mOsm/kg, urine sodium 200mmol/L, urine potassium 44.2mmol/L. Chest x-ray was normal.

He was initially treated for syndrome of inappropriate anti-diuretic hormone (SIADH) secretion, which was supported by a rise in serum sodium to 130mmol/L following a 1 litre fluid restriction. However, subsequent test results (Table 1) demonstrated a low-normal 9am cortisol and elevated ACTH, confirming a diagnosis of primary adrenal insufficiency.

Table 1.

Case 1: Laboratory Investigation Values		
	Result	**Reference Range**
Serum Cortisol	208 nmol/L	160-550 nmol/L
Serum ACTH	2114 ng/L	
Serum Aldosterone	<60 pmol/L	90-270 pmol/L
Serum Renin	29.6 nmol/L/h	0.5-3.5 nmol/L/h
Short Synacthen Test Result		
Time (min)	Serum Cortisol Level (nmol/L)	Reference Range (nmol/L)
0	197	160-550
30	219	160-550
60	204	160-550

Case Two
A 44-year-old gentleman with severe psoriasis, refractory to multiple treatments, was referred by his Dermatologist following a serum cortisol measurement <0.28nmol/L. For 20 years, he had used topical Dermovate cream (clobetasol 0.05%) on his scalp, face and body. Repeat early-morning serum cortisol (off topical steroids) confirmed a measurement of <0.28nmol/L. Remaining blood tests showed a normal serum sodium (144mmol/L). Clinical assessment demonstrated blood pressure 161/95 and abdominal striae. He was advised to wean off topical steroids. A follow-up

short synacthen test, off steroids, demonstrated a low baseline cortisol, and elevated ACTH, measurement suggestive of a degree of adrenal insufficiency.

Table 2.			
Case 2: Short Synacthen Test Result – Off topical steroid			
Time (min)	Serum Cortisol Level (nmol/L)	Reference Range (nmol/L)	ACTH (ng/L)
0	93	160-550	217
30	257	160-550	
60	248	160-550	

Case Three

A 73-year-old gentleman was referred by his GP after one week of nausea, vomiting, and diarrhoea, and a serum sodium 106mmol/L. Medical history was notable for a large pituitary adenoma with two previous debulking surgeries. He since suffered from secondary hypoadrenalism, and post-operative SIADH. He was on maintenance prednisolone that had recently been weaned from 2mg daily to 1.5mg daily. A pre-admission SST demonstrated an insufficient response. He subsequently required an intensive care admission for monitoring and management.

Table 3.			
Case 3: Laboratory Investigation Values			
	Result	**Reference Range**	
TSH	0.23	0.3-4.2 milliunit/L	
Free T4	18.8	9.0-23.0 pmol/L	
Sodium	103	135-145mmol/L	
Potassium	4.4	3.5-5.0mmol/L	
Serum osmolality	216 mOsm/kg	mOsm/kg	
Urine osmolality	373 mOsm/kg	mOsm/kg	
Urinary sodium	65 mmol/L	mmol/L	
Pre-Admission Short Synacthen Test Result			
Time (min)	Serum Cortisol Level (nmol/L)	Reference Range (nmol/L)	ACTH (ng/L)
0	<28	160-550	6.7
30	34	160-550	
60	46	160-550	

Discussion

Acute presentations of adrenal insufficiency can be life threatening, requiring prompt diagnosis and management. Our cases demonstrate how these presentations can differ but also pitfalls in their identification due to the potential unreliability of SST cutoffs.

E040

Diagnostic and Management Challenges of Pembrolizumab-Induced Thyroid Dysfunction.

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Abstract

Introduction
Immune checkpoint inhibitors (ICIs) have altered the therapeutic landscape in oncology. These monoclonal antibodies unleash a potent T-cell-mediated anti-tumour response. However, ICIs can block protective signals on normal cells, producing endocrine immune-related adverse events (irAEs), with the thyroid being particularly susceptible.

Anti-PD-1/PD-L1 agents such as pembrolizumab frequently cause destructive thyroiditis due to PD-L1 expression on the thyroid follicular cells. The initial immune insult causes destruction of thyroid follicles and unregulated release of preformed thyroid hormones. This causes a transient thyrotoxic phase, which is often asymptomatic, often followed by permanent hypothyroidism when the hormone stores are depleted [1,2].

Less commonly, ICIs trigger true Graves' disease, characterised by persistent hyperthyroidism and occasionally ophthalmopathy [2,3]. Distinguishing these entities is essential because management differs: antithyroid drugs are indicated for Graves' but not for the self-limiting destructive thyroiditis.

Case
A 33-year-old female with metastatic gestational choriocarcinoma presented to the hospital with a three-week history of nausea, vomiting, and fevers.

She was initiated on pembrolizumab immunotherapy 3 weeks before admission and finishing a weaning regimen of dexamethasone for brain metastases.

On admission, she was tachycardic and tremulous. Initial investigations revealed normal white cell count and CRP, but profound thyrotoxicosis: TSH <0.01mIU/L, fT3 >30.7pmol/L (above assay's upper limit), and fT4 44.5pmol/L. There was no personal or family history of thyroid disease. β-hCG, which can confound thyroid function tests, was low and remained undetectable throughout the toxic phase.

She was initially commenced on 20mg carbimazole and propranolol, and her symptoms improved and fT3/fT4 were both down-trending. She did not have a goitre or thyroid eye disease, but her TSH-receptor-antibody (TRAb) was slightly elevated (0.558units/L; Normal <0.4). She was discharged on carbimazole and planned for urgent out-patient review within 2-4 weeks. However, she rapidly developed hypothyroidism weeks later, confirming the diagnosis of destructive thyroiditis. The carbimazole was stopped and levothyroxine was initiated

	Day 1	Day 8	Day 24	Day 28	Day 36	Day 56	Day 77*	Day 98	Day 118
TSH mIU/L (0.3-4.2)	1.60	1.08	<0.01	<0.01	<0.01	<0.01	39.68	28.36	2.96
fT3 pmol/L (2.4-6)		5.6	>30.7	26.9	8.1	4.4		4	
fT4 pmol/L (9-23)	13.2	12.5	44.5	37.7	34.7	12.6	6.6	10.2	16.2
Morning Cortisol nmol/L	267		243	668	369	187	233		248
Tumour hCGIU/L (<2)	8	8	<2	3	<2	<2	3	<2	<2

Day 1- Baseline bloods
20mg carbimazole commenced from D25 onwards
*Commenced on levothyroxine Day 77

Discussion

This case illustrates the diagnostic challenges in ICI-induced thyroid dysfunction. Destructive thyroiditis is the most common presentation, so there should be a high index of suspicion [2,3]. European Society of Endocrinology guidelines support a watch-and-wait approach, recommending initial symptomatic management with beta-blockers and close biochemical monitoring [1,5]. Antithyroid therapy is reserved for persistent hyperthyroidism, which is more typical of Graves' [1,5].

A common mistake is misinterpreting mild, transient TRAb positivity—which can occur from immune-mediated antigen release—as definitive Graves' disease [2,3]. While carbimazole is appropriate for convincing Graves' (e.g., high TRAb, goitre) [1], its empiric use in early thyroiditis may accelerate the progression to profound hypothyroidism [1,4,5]. If anti-thyroid medication is commenced, close biochemical monitoring is essential, although this is often compromised by logistical issues in arranging patient follow-up.
A technetium uptake scan could have differentiated Graves' from thyroiditis. However, its interpretability is often precluded by early carbimazole administration, which blocks tracer uptake and can create a false-negative result. Ultimately, this case demonstrates that the clinical course—rapid, transient thyrotoxicosis followed by hypothyroidism—is often diagnostic on its own.

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E041

More Than a Number: A Case of Diagnostic Uncertainty in Hypercalcaemia.

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Abstract

Introduction
Primary hyperparathyroidism (PHP) is the most common cause of hypercalcaemia (1), but familial hypocalciuric hypercalcemia (FHH) is a rare and important differential with a similar biochemical picture. Both conditions can have a significant impact on bone health, including osteoporosis and fractures (2). Making a watertight diagnosis is often challenging (3) but clinically important, as PHP often requires surgical treatment while FHH typically requires no treatment.

Background
A 77-year-old female with a background of osteoporosis and multiple fractures was referred to the Endocrinology team for investigation of hypercalcaemia. **She** underwent a hysterectomy at age 46 and was diagnosed with osteoporosis shortly thereafter. She was switched from alendronate **to** teriparatide, having developed osteonecrosis of the palate. She was subsequently managed with raloxifen and then commenced zolendronate in June 2025.

Case Report
Her blood tests are shown in the table below. Over the past year, her calcium and parathyroid hormone (PTH) levels have fluctuated between normal and elevated. A workup for other causes was negative, including myeloma screening and a renal tract ultrasound which showed no calculi. Two 24-hour urinary calcium collections were performed. The calculated urinary calcium creatinine ratios (UCCR) from both were highly suggestive of FHH (0.0039 and 0.0026, respectively), with the first collection obtained prior to the initiation of zoledronate. She was always Vitamin D replete (4). Despite these results, subsequent genetic analysis for FHH was negative, and the patient had no known family history of hypercalcaemia or fractures. A recent DEXA scan revealed her lumbar spine (L1-L4) bone mineral density worsened by 9.3% from baseline, while the T-score deteriorated from –2.4 to –3.1.

Date of test	*23/10/24*	*13/03/25*	*12/08/25*	*17/09/25*
Adj Ca2+ (mmol/L)	2.71	2.43	2.51	2.60
PTH (pmol/L) (1.6-6.9)	5.6	12.5	14.3	15.3
Phosphate (mmol/L)	-	1.15	1.15	0.97

Discussion
Given her worsening bone health and fracture history, a definitive diagnosis is crucial. Parathyroidectomy is the definitive treatment for PHP and would significantly improve her long-term bone health; however, it carries risks, so FHH must be confidently excluded first.

Significant diagnostic uncertainty remains; while her UCCR suggests FHH, this is also seen in 20% of PHP patients (3). After a specialist bone MDT discussion, the plan is to gather more diagnostic data. This includes anatomical (ultrasound) and functional (Sestamibi) imaging, as well as cascade screening (calcium and genetic testing) for her three children. Given FHH's autosomal dominant inheritance, negative family screening would help solidify the diagnosis before considering surgical intervention.

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E042

Bone Mineral Density Improvement in a Patient with Multiple Endocrine Neoplasia Type 1 (MEN1): A Case Report on the Impact of Intensive Lifestyle Intervention.

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Abstract

Background
Multiple Endocrine Neoplasia Type 1 (MEN1) is a rare, autosomal dominant disorder predisposing individuals to tumours of the parathyroid, pituitary, and pancreas. Primary hyperparathyroidism (PHPT) can occur in up to 95% of patients, and is a significant driver of skeletal morbidity, osteoporosis and fractures [1]. Concurrently, pituitary prolactinomas can exacerbate bone loss through hypogonadism.

Case Presentation
We report a remarkable 27-year-old female with MEN1, diagnosed aged 12 (in 2011), with a complex history of insulinoma (sub-total pancreatectomy in 2012), macroprolactinoma, and symptomatic primary hyperparathyroidism (PHPT). Both her father and younger sister are also affected.

Her prolactinoma was initially treated with cabergoline and switched to the combined oral contraceptive pill (COCP) for dermatological benefits, before restarting cabergoline in 2020. This restart successfully reduced the size of the prolactinoma and normalised prolactin (see Table 1), leading to the resumption of normal menses (in 2022), allowing for successful cabergoline cessation last year.

She is a highly motivated patient, aware of the benefits of delaying parathyroid surgery. She engaged in significant exercise and dietary optimisation and subsequently decided to stop cinacalcet in 2021 once her calcium level reached the upper limit of normal range.

After finishing University, she further intensified her exercise regimen, becoming a professional yoga/pilates instructor. Notably, despite having stopped cinacalcet (and later cabergoline), she has remained normocalcaemic since 2021. Even more remarkably, a repeat DEXA scan in 2024 demonstrated a clinically significant increase in bone mineral density (BMD) from her baseline scan in 2020: +3.3% in the spine, +8.9% in the hips, and +2.0% in the forearm.

Discussion
This case highlights the role of non-pharmacological strategies in improving bone health in MEN1, presenting a clinical outcome that diverges from the expected disease trajectory [2].

As a professional yoga and pilates teacher, she engages in rigorous, daily weight-bearing and muscle-strengthening exercise. The mechanical loading from such activities is a well-established and potent stimulus for osteoblast activity and new bone formation [3, 4].

This positive outcome must be considered alongside initial pharmacological treatment: successful treatment of her prolactinoma with carbergoline restored her menses, which also aided the observed BMD improvement.

While this intensive lifestyle has deferred the need for intervention, it does not cure the underlying PHPT. Parathyroidectomy remains the definitive treatment, and the timing of surgery, particularly in relation to future pregnancy planning, remains a key point for future management [1,5].

Date	15/08/19	20/03/20	05/05/20	12/08/20	03/12/20	04/06/21	22/11/22	30/05/23	15/07/24	04/07/25
AdjustedCalcium2.69 mmol/L (2.20-2.60)	2.69	2.93	2.48 (ii)	2.60	2.70	2.51 (iii)	2.74	2.78	2.53	2.52
Vitamin D nmol/L (70-150)	-	59.3	66.6	-	-	100.2	96.3	115.9	88.6	84.4
Parathyroid Hormone Level pmol/L (1.6-7.2)	16.1	24.4	13.5	11.4	15.9	13.2	17.7	14.7	16.2	30.3
Prolactin/L (100-500)	1139	1414	1518	1238	694	349	313	280	415	582
Cabergoline Dose (mcg)	COCP (i)	NA	NA	250 O/W	500 O/W	500 2/W	500 2/W	500 3/W	Stopped	Stopped

(i) Taking COCP, not yet taking cabergoline; (ii) start taking 30mg of cinacalcet; (iii) stopped cinacalcet. Key: O/W = once weekly; 2/W = twice weekly; 3/W = three times a week

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E043

Hyperthyroidism: When is Definitive Management Appropriate? Lessons from a Complex Case of Graves' Disease.

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Abstract

Case Report

A 62-year-old male presented with fast atrial fibrillation (AF), decompensated heart failure, ascites, jaundice, and deranged liver/clotting functions. His background included Graves' disease (GD), presumed thyroid eye disease (TED), emphysema (ex-smoker), and previous excess alcohol intake. He reported both carbimazole and propylthiouracil discontinued due to agranulocytosis[1] 5 years prior in Hong Kong. He was non-compliant with medications, preferring traditional Chinese medicine. Care was complicated by language barriers (needing Mandarin translators) and unavailable previous medical records.

Initial thyroid function tests (TFTs) were: TSH <0.01 mU/L, fT4 24.1 pmol/L, fT3 4.8 pmol/L, TSH-receptor antibodies (TRAb) 22 IU/L. An echocardiogram showed poor biventricular dysfunction, biatrial dilation, and severe mitral regurgitation (MR) with torrential tricuspid regurgitation (TR). Specialist thyroid eye surgeons excluded active TED. A specialist cirrhosis MDT concluded his deranged LFTs were from hepatic congestion secondary to severe TR, not cirrhosis.
He had significant health anxiety and self-discharged from hospital. It transpired that he missed direct current cardioversion (DCCV) appointment 8 months previously. He stated he would refuse any thyroid surgery.

He was readmitted from cardiology clinic one month later with fast AF and heart failure; amiodarone was initiated. He remained profoundly thyrotoxic: TSH <0.01 mU/L, fT4 31.8 pmol/L, fT3 12.3 pmol/L, TRAb 22 IU/L.

Outcome

Cardiology advised DCCV would be unsuccessful now due to severe atrial dilatation and he was not a candidate for cardiac valve surgery. Untreated thyrotoxicosis was clearly driving his valvular disease and subsequent congestive hepatopathy[2]. Definitive GD treatment was imperative to halt further cardiac decline 2].

Amiodarone, a potentially life-saving cardiac intervention, significantly complicated definitive management by delaying radioiodine (RAI) treatment. The high iodine load from amiodarone impairs thyroid uptake scans and reduce RAI effectiveness [3]. A specialist Mitral MDT concluded that while his cardiac risk was high, it was not prohibitive to non-cardiac surgery, and rapidly correcting his thyrotoxicosis was the priority[4].

After lengthy multidisciplinary discussion with the patient and his daughter, he opted to wait for RAI over high-risk surgery. To facilitate this and counteract the amiodarone-induced iodine load, he will be given lithium as an adjunct, to increase thyroid iodine retention[5].

Discussion

This case presented a 'catch-22' situation, where the multisystem consequences of untreated Graves' disease meant that all definitive management with surgery now carries high-risk [2]. This was compounded by patient factors (language barriers, non-compliance, anxiety- itself can be exacerbated by thyrotoxicosis) and relocation, which led to several missed opportunities for intervention.

This case highlights the importance of involving specialist teams early. Earlier formal exclusion of TED by specialist surgeons might have clarified the pathway to RAI sooner.

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E044

Hungry bones and Gargantuan glands: A case of Pseudohypoparathyroidism IB complicated by Tertiary hyperparathyroidism and Pathological fractures.

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Abstract

Objective
Pseudohypoparathyroidism (PHP) is a rare genetic cause of hypocalcemia due to PTH resistance, caused by a mutation or altered methylation status of the imprinted GNAS gene. It is characterized by young onset hypocalcaemia and hyperphosphatemia despite high PTH levels. Tertiary hyperparathyroidism is an extremely rare complication, and we present the occurrence of this in a patient with PHP1b, highlighting the importance of a multimodal approach and a prudent surgical plan. A comprehensive literature review was also undertaken.

Case Presentation
A 35-years female patient had been diagnosed with hypocalcemia 6 years prior, with genetically confirmed pseudohypoparathyroidism type Ib. Medical treatment with alfacalcidol and calcium supplementation had initially corrected the hypocalcaemia. When she later developed hypercalcemia, this was discontinued. She had developed tertiary hyperparathyroidism with severe osteoporosis and was referred to the Imperial Department of Endocrine & Thyroid Surgery for consideration of surgical management. She had become unable to perform daily activities and was largely bed bound due to bone and muscle pain. At presentation, the serum calcium was 2.48 mmol/L with a serum parathyroid hormone level exceeding the measurable upper laboratory limit of 350 pmol/L and an ALP >300 IU/L. An US scan (performed primarily to exclude underlying thyroid lesions) revealed multi-gland parathyroid enlargement. On the day of parathyroid surgery, she fell while alighting from the taxi leading to a pathological left subtrochanteric femur fracture. A screening skeletal CT at this time revealed extensive intramedullary resorption, subperiosteal resorption and subchondral cyst formation affecting all bones along with multiple old, healed rib fractures and biconcave vertebral bodies. She was discussed in the Orthopedic MDT and underwent an open reduction and biopsy, gamma nailing and lateral proximal femoral non-contact bridging to manage the left femur fracture. This was followed by a brief period of pre-operative optimization for parathyroid surgery, which was scheduled following MDT discussion regarding the optimal surgical strategy. At surgery none of the 4 visualized glands presented preservable tissue and a total parathyroidectomy was performed leaving the thymus in situ. The post-operative course was complicated by severe hypocalcemia managed with re-introduction of oral alfacalcidol and calcium at high dose, combined with temporary intravenous calcium infusions. On POD-1, PTH levels were recorded at 1.2pmol/L. The patient was discharged on POD- 10 with an adjusted calcium level of 2.06 mmol/L. Histology confirmed parathyroids with weight ranging from 0.58g to 5.7g and pseudoadenomatous hyperplasia seen in all 4 glands. She is currently home undergoing physiotherapy for ambulation and rehabilitation.

Conclusion
There is a paucity of literature on the optimal medical management and surgical strategy for the management of tertiary hyperparathyroidism in PHP – we will present this. We can make the case for more proactive medical management and either a total or subtotal parathyroidectomy. Where the evidence base is weak the appropriately constituted MDT plays a pivotal role in the decision-making algorithm of such complex cases.

E045

Pulmonary Cryptococcosis Masking an ACTH-Secreting Pulmonary Carcinoid: A Diagnostic Challenge.

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Abstract

Background
Ectopic ACTH secretion causing Cushing's syndrome presents a major diagnostic challenge, particularly when the occult source is obscured by co-existing pathology. Pulmonary cryptococcosis is an uncommon but clinically important infectious mimic of malignancy in immunocompromised states, including hypercortisolism. Distinguishing infection-related pulmonary nodules from an ACTH secreting lesion is therefore essential, yet often delayed by overlapping radiological and clinical feature

Case report
A 47-year-old man with type 2 diabetes, hypertension and progressive Cushingoid features was found to have ACTH-dependent hypercortisolism (cortisol 700-800nmol/L, ACTH 632-156ng/L) with failure to suppress on overnight dexamethasone testing. Inferior petrosal sinus sampling confirmed ectopic ACTH secretion and cortisol-lowering treatment with metyrapone was commenced.

Ga^{68} DOTATATE PET/CT demonstrated a cluster of left lower lobe nodules with low-level uptake. Following neuroendocrine MDT review, FDG-PET/CT revealed 3-4 minimally cavitating FDG-avid nodules. Biopsy showed dense inflammatory change consistent with fungal infection, and positive serology supported pulmonary cryptococcosis. The patient was started on fluconazole but subsequently developed symptomatic hypocortisolism, attributed to a synergistic drug interaction, requiring block-and-replace therapy.

Over subsequent months, lung parenchymal changes regressed and cryptococcal antigen titres fell from 1:32 to negative. However, interval CT revealed new soft tissue foci in the anterior mediastinum, prompting repeat MDT review. Follow-up imaging identified a distinct basal left lower lobe nodule that had enlarged and developed new moderate DOTATATE uptake, in contrast to the regressing infection-related nodules.

CT-guided biopsy of this lesion confirmed a typical pulmonary carcinoid tumour (synaptophysin+, chromogranin+, CD56+), establishing the ACTH-secreting primary. Tumour resection is planned.

Discussion
This case illustrates the diagnostic complexity of ectopic Cushing's syndrome when complicated by opportunistic fungal infection. Pulmonary cryptococcosis can obscure or mimic the radiological appearance and FDG uptake of an underlying neuroendocrine tumour. A coordinated multidisciplinary approach involving neuroendocrine specialists, radiologists, cardiothoracic surgeons, infectious disease physicians and microbiologists, combined with serial imaging and targeted histopathology was crucial in successfully identifying the carcinoid tumour. Clinicians should maintain a high index of suspicion for dual pathology in ectopic Cushing's syndrome, as overlapping processes can significantly delay definitive diagnosis and treatment.

E046

Atezolizumab-Associated Isolated ACTH Deficiency With Concomitant Primary Hypothyroidism

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Immune checkpoint inhibitors (ICIs) are now integral to modern oncology treatments but could be associated with a spectrum of immune-mediated endocrine toxicities. Isolated ACTH deficiency is a recognised yet infrequent complication of PD-L1 inhibitors such as atezolizumab and may emerge following completion of treatment, highlighting the need for ongoing endocrine surveillance. Primary hypothyroidism is another ICI-related toxicity and may occur independently or alongside pituitary dysfunction.

A 67-year-old man with advanced squamous cell carcinoma of the lung received atezolizumab between July 2020 and July 2022. Four months after completing treatment, he presented with profound fatigue. Biochemical evaluation demonstrated secondary cortisol deficiency, with cortisol 59 nmol/L (normal 140–690) and ACTH 15 ng/L (normal 10–50). Thyroid tests revealed primary hypothyroidism: TSH 32.3 mU/L (normal 0.4–4.0) and free T4 6.5 pmol/L (normal 10–22). Anti-TPO and adrenal cortex antibodies were negative. Gonadal testing showed FSH 1.7 IU/L (normal 1.5–12), LH 1.5 IU/L (normal 1.8–9) and testosterone 7.8 nmol/L (normal 8–30), with normal prolactin and IGF-1. He failed his Short Synacthen Test with a low ACTH level, confirming secondary hypocortisolism.

Pituitary MRI showed no structural abnormality, and CT imaging confirmed normal adrenal glands. Hydrocortisone and levothyroxine were commenced, with marked symptomatic improvement. After hormone optimisation, FSH, LH and testosterone normalised. His clinical condition stabilised with ongoing endocrine follow-up. Repeated Short Synacthen Tests and thyroid function tests up to three years after the initial diagnosis showed no recovery of either axis.

This case illustrates that dual endocrine toxicity could be associated with PD-L1 inhibition, presenting as isolated ACTH deficiency consistent with immune-related hypophysitis together with concomitant primary hypothyroidism. These findings highlight the importance of long-term endocrine monitoring during and after ICI therapy to support timely diagnosis, appropriate hormone replacement and prevention of serious complications.

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