Hammersmith Abstracts

Friday 2nd December 2022

17th Hammersmith Multidisciplinary Endocrine Symposium 2022

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Hammersmith Hospital 17th Multidisciplinary Endocrine Symposium

Programme Friday 2nd Dec 2022

Wolfson Conference Centre

Hammersmith Hospital, London

8.30am Registration & Coffee.

8.55am Welcome and Introduction **(FP/KM/WD)**

Session 1: Obesity

9.00am Bariatric new drugs and mechanisms.
John Wilding (Liverpool)

9.30am The new non surgical bariatric procedures.
Dev Bansi (Imperial)

9.50am Current & Future Role of Bariatric surgery.
Sanjay Purkayastha (Imperial)

10.20am Clinical case B001. Cushing's Syndrome due to ectopic ACTH production. from carcinoid of the lingula.
Ainesh Singh (Imperial).

10.35am B004. Audit of primary hyperparathyroidism.
Jaclyn Tan (Stevenage)

10.50am Coffee Break.

Session 2: COVID and Endocrine Disease

11.10am COVID in Thyroid & adrenal disease.
Sophie Clarke (UCH)

11.40am Endocrine Surgery & COVID.
Aimee Di Marco (Imperial)

12.10pm PET positive thyroid nodules: a management update.
David Scott Coombes (Cardiff)

12.40pm Case B007. A challenging adrenal incidentaloma.
M Mantega (Northwick)

12.55pm Case B005. Type 2 amiodarone-induced thyrotoxicosis.
Saif Rehman (Stevenage)

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1.10pm LUNCH and POSTER session.

Session 3: Afternoon session: Adrenal

2.00pm Adrenal growths: Finding the needle and making the haystack smaller
Prof Wiebke Arlt (Birmingham)

2.50pm Endocrine anaesthesia explained
Dr Parind Patel (Imperial)

3.15pm Tales from the T3 withdrawal Clinic
Karim Meeran (Imperial)

3.45pm Clinical Case B002: Medullary thyroid cancer presenting as ectopic Cushings

4.00pm B003: A tale of two multi-focal papillary thyroid cancers

4.15pm Feedback & Close

B001	Cushing's Syndrome due to ectopic ACTH production from carcinoid of the lingula
B002	Medullary thyroid cancer presenting as ectopic Cushing's
B003	A tale of two multi-focal papillary thyroid cancers
B004	Audit into pre-operative workup, surgery and outcomes for primary hyperparathyroidism
B005	A case of Type 2 Amiodarone-induced thyrotoxicosis
B006	Case report: Partial hypopituitarism & central diabetes insipidus in acute myeloid leukaemia monosomy 7 & chromosome 3 inversion
B007	A challenging adrenal incidentaloma
B008	Partial hypopituitarism with unknown aetiology followed by spontaneous recovery: a case report
B009	Analysing the hormonal characteristics of men self- administering hormonal therapy to facilitate their withdrawal from anabolic androgenic steroids

B010	Neutropenia in a Patient Taking Thionamides
B011	Transient hypophosphatemia secondary to iron infusion
B012	Highlighting Multidisciplinary Interdependence - Post Operative Hypoadrenalism following Unilateral Adrenalectomy
B013	A case of diabetes insipidus unmasked by initiation of steroids
B014	Severe Hyponatremia secondary to NSAIDs
B015	Abnormal LFT and Hypercalcaemia due to Thyrotoxicosis
B016	Interesting survivor of Di-George syndrome
B017	Thyroiditis presenting with abnormal LFTs, and weight loss
B018	Tachy Tea: The perils of a Kalamitous Cuppa
B019	A rare case of a functioning adrenal oncocytoma masquerading as adrenocortical carcinoma
B020	Covid-19 vaccination and new diagnosis adrenal insufficiency: cause or accelerated presentation?
B021	The Role of Fluconazole and Cinacalcet in the management of CYP24A1 mutation Induced Hypercalcaemia
B022	Peculiar numbers requiring clinical correlation.
B023	Are SGLT2 inhibitors over-prescribed in primary care without appropriate risk-stratification

Save the date for the 18th Hammersmith Multidisciplinary Endocrine Symposium Friday 8th December 2023

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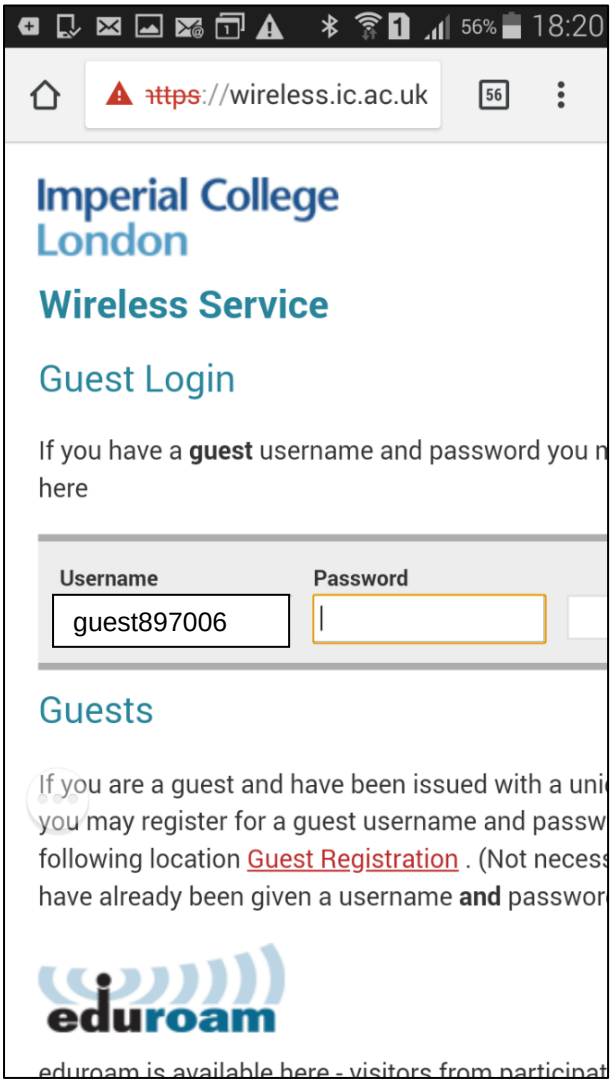

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B001

Cushing's Syndrome due to ectopic ACTH production from carcinoid of the lingula

A Singh[1], E Vainieri[1], R Agha-Jaffar[1], M Al-Sahaf[2], B Jones[1], F Wernig[1], J Cox[1] & S Robinson[1]
[1.]Department of Endocrinology, Imperial College Healthcare NHS Trust, London.
[2] Department of Cardiothoracic Surgery, Imperial College Healthcare NHS Trust, London.

Abstract: A 53-year-old woman with recent diagnoses of Type 2 Diabetes, hypertension, and dyslipidaemia, was referred by the lipid clinic with a 3-month history of facial swelling, hyperpigmentation, and progressive proximal myopathy. Clinical history and examination were highly suspicious of endogenous hypercortisolaemia. An initial overnight dexamethasone suppression test yielded an elevated morning cortisol (752 nmol/L, normal <50nmol/L). Two 24-hour urine collections showed markedly elevated urinary free cortisol values (1917nmol/day, 1511 nmol/day).

The low-dose dexamethasone suppression test demonstrated a failure of cortisol to suppress (basal cortisol 606 nmol/L, Time =48 hours 642 nmol/L), with a high ACTH (basal 48.5 ng/L, Time = 48 hours 49.5 ng/L) indicating ACTH-dependent Cushing's syndrome. Ketoconazole therapy was commenced to control hypercortisolaemia, alongside anti-hypertensive and glycaemic agents, producing reasonable benefits in blood pressure and glycaemic management. An MRI adrenal scan showed bulky adrenal glands with no discrete lesion. An MRI Pituitary showed increased size and heterogeneity of the signal at the left lateral aspect, with infundibulum deviation to the left; however, no discrete lesion or contrast-enhancing characteristics were identified.

The patient underwent inferior petrous sinus sampling which did not show a central-to-peripheral ACTH gradient, in keeping with an ectopic source of ACTH.

Upon review of a previous CTCA performed to look for unrelated coronary artery calcification, the presence of a small 6mm lesion was noted in the lingula of the lung. In light of the clinical, radiological, and IPSS findings, a Gallium-68 DOTATATE PET scan was performed, confirming the presence of an 8mm tracer-avid carcinoid nodule in the lingula believed to be the source of the ectopic ACTH production (not amenable to biopsy but resectable). The ketoconazole was stopped prior to surgery and the patient underwent a left VATS lingulectomy and lymphadenectomy for resection of the nodule, staged as T1a N0 PL0 R0, with histology findings consistent with Carcinoid.

Postoperatively, the hypercortisolaemia improved (morning cortisol of 45 nmol/L). She was provided with steroid cover and the prednisolone was weaned following surgery. She has remained stable with satisfactory blood pressure and glycaemic control: her symptomatology has additionally improved.

Ectopic ACTH secretion is a rare condition which accounts for approximately 10% of ACTH-dependent Cushing cases. This case illustrates the diagnostic approach in an individual with overt clinical and biochemical hypercortisolaemia and highlights the importance of an MDT approach for prompt and adequate intervention.

B002

Medullary thyroid cancer presenting as ectopic Cushing's

B Sharma, A Qureshi, A Di Marco, S Partridge, F Wernig. Imperial College Healthcare Trust, London.

A 68 year old male with a history of schizophrenia was admitted with cough and fever during the second UK covid surge. Two months earlier, he had been found to have hypertension and diabetes. On admission, he had hypernatremia, hypocalcemia, hypomagnesemia along with refractory hypokalemia and metabolic alkalosis. There were no obvious clinical features suggesting Cushing's syndrome. However, he failed to suppress cortisol during an overnight dexamethasone suppression test with plasma cortisol at 1045 nmol/l. 24-hour urinary free cortisol was 3536 nmol/l and midnight plasma cortisol was 856 nmol/l. Plasma ACTH was 275 ng/L with low dose dexamethasone suppression test not revealing fall in cortisol (remaining at 900 nmol/l).

Pituitary MRI was normal, CT chest, abdomen and pelvis showed bulky adrenal glands, multiple bronchovascular lung nodules and right lower lobe consolidation and a left supraclavicular soft tissue density of 2 cm which did not show significant DOTATATE uptake on a subsequent Gallium68 DOTATATE PET CT. A provisional diagnosis of ectopic ACTH secretion was made, and he was commenced on metyrapone which was gradually up titrated to 1.5 mg TDS to achieve a mean plasma cortisol of around 300 nmol/l. Eplerenone was added for better control of hypokalemia. He was eventually established on block and replace regime with high-dose metyrapone and 4mg prednisolone. He was started on rivaroxaban 10 mg for DVT prophylaxis as well as prophylactic co-trimoxazole.

A biopsy of the supraclavicular node was pursed. The histology was suggestive of a well differentiated neuroendocrine tumour with Ki-67 proliferation index of 2%. It stained negative for ACTH and positive for TTF-1, chromogranin and synaptophysin. Plasma calcitonin was raised at 28,000 pg/ml thus confirming a diagnosis of medullary thyroid cancer with ectopic ACTH secretion. A planned total thyroidectomy had to be abondoned due to very advanced disease and medical treatment with Cabozantinib was commenced. Then tumour tissue was found to harbour a somatic RET mutation. The patient had an excellent response to Cabozantinib, but due to side effects would only tolerate low-dose treatment which resulted in significant improvement, but not in normalisation of his calcitonin levels. Therefore, a change of treatment to a selective RET inhibitor is currently being considered.

This case highlights a conservative medical management approach in a patient with complex medical and psychosocial needs presenting with medullary thyroid cancer presenting and ectopic Cushing's syndrome with an aim to preserve and enhance his quality of life in a patient-centered approach. It also demonstrates recent advancement of medical therapy for medullary thyroid cancer.

Question: Should calcitonin levels routinely be sent for patients with suspected ectopic Cushing's syndrome?

B003

A tale of two multi-focal papillary thyroid cancers

Ni Tekkis, A Symons, A Kapoor, R Agha-Jaffar, J Cox, S Partridge, N Tolley, S Robinson.
Imperial College Healthcare Trust, London.

Case 1: A 31-year-old male presents to head and neck clinic with a painless lump in the right supraclavicular fossa. US imaging showed an 8mm partially cystic lymph node lesion at right level IV, in addition to a 5mm U5 lesion in the right lobe of the thyroid. Lymph node cytology showed follicular cells. The patient underwent a total thyroidectomy with selective right IIA, II, IVA and VB lymph node dissection. Histology demonstrated a focus of papillary thyroid carcinoma (PTC) measuring 3mm in the right lobe of the thyroid with no vascular invasion. 2/19 nodes resected contained metastatic disease. Multiple smaller foci were also found in the adjacent tissue, ranging from individual follicles to collections of follicles, with the largest focus measuring 1.3mm. All foci were confined to the thyroid and were fully excised. The patient underwent further radioiodine ablation and remains disease free at 6 months.

Case 2: A 44 year old female with a past medical history of hypothyroidism and a family history of thyroid cancer presents to thyroid clinic with a painless lump in the neck. US imaging showed an 8mm U4 THY4 lesion in the right lobe of the thyroid. She underwent a right hemithyroidectomy. Histology confirmed a 12mm PTC. She underwent a completion thyroidectomy, with histology demonstrating an additional focus of PTC in the left lobe measuring 1.25mm. She declined radioiodine ablation due to childcare commitments. A year later, a painless discrete nodule was identified on the left side of the neck, with 2 enlarged paratracheal nodes identified on ultrasound. FNA and I-131 SPECT-CT scanning identified these sites to be metastatic PTC. She underwent radioiodine ablation and now remains disease free at 6 months**.**

Conclusion: Multifocal Papillary thyroid cancer is defined as micro or macrocarcinoma in two or more thyroidal sites. Whilst a more aggressive form of PTC, there is uncertainty on management. Extrathyroidal mPTC disease represents intermediate response to treatment. Patients should have total thyroidectomy and be considered for radioiodine ablation.

B004

Audit into pre-operative workup, surgery and outcomes for primary hyperparathyroidism

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[4] Department of Diabetes and Endocrinology, East and North Herts NHS Trust.

Introduction: Prior to the introduction of a parathyroid MDT in 2015 at the East and North Herts NHS Trust, audit revealed a 25% failure rate (raised calcium) following parathyroid surgery. This was partly due to inadequate pre-operative workup.

Since 2015, audit has revealed marked improvement. Our parathyroid audit is carried out by final year medical students together with the medical endocrinology team. This is in contrast to many trusts where the audit is carried out by parathyroid surgeons.

Audit findings: Prior to 2015, around 17 parathyroidectomies were carried out annually. In 2015-2016, there was a marked drop to 9 parathyroidectomies per year. This is likely attributed to a more cautious approach by the parathyroid MDT. In 2017, the numbers increased back to 17 parathyroidectomies annually. This number did not decrease during the Covid pandemic.

This audit looked at 49 parathyroidectomies carried out between December 2018 and March 2021.

We found that pre-operative workup was incomplete. Renal ultrasound was not requested in 14 out of 49 patients (28.6 %). DEXA was not requested in 1 patient. Urine calcium (neither a ratio nor 24-hour collection) was not requested in 9 patients (18.4%). Vitamin D was not requested in 6 patients (12.2%).

Looking at the type of surgery, 31 patients had discordant imaging. However, out of these, 20 had targeted parathyroidectomy, not 4 gland exploration.

Post-operative calcium was raised in 4 patients, a failure rate of 8.2 %.

Conclusion: Whilst the operative outcomes have improved since 2015, we are keen to improve further. Since 2015, our parathyroid MDT has increased in frequency from quarterly to monthly. We have removed Sestamibi as an imaging modality from our pathway and we have replaced this with SPECT-CT. Finally, we are planning to introduce intra-operative PTH sampling for specific patients.

Our aims are to have 100 % of patients having a complete biochemical and radiological pre-surgery workup and appropriate surgery based on these findings.

B005

A case of Type 2 Amiodarone-induced thyrotoxicosis

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[2] Written in May 2022 as a medical student at University of Cambridge School of Clinical Medicine

Case history: This 74-year-old woman presented to the emergency department with a 1-2 week history of tremors, palpitations and anxiety as well as a 2 month history of progressive shortness of breath on exertion and 1-2 weeks of leg swelling. On examination, she was noted to have a tremor, a regular heart rate of 100 bpm, and bilateral oedema to the mid-shins. There was no palpable goitre or proptosis. She has a complex medical background including diabetes, pulmonary HTN, OSA and previous CABG. Her medications included amiodarone 100mg for sinus tachycardia with supraventricular ectopy, and propranolol 10mg. Blood test results included NT-proBNP 517pg/mL, TSH <0.02mU/L and FT4 of 93 pmol/L. Given her history of pulmonary hypertension, she was considered to have suspected heart failure complicated by amiodarone-induced thyrotoxicosis (AIT). She was initially treated with IV furosemide 80mg, carbimazole 30mg OD, prednisolone 40mg, and propranolol 10mg. An ultrasound thyroid scan showed multiple <1cm nodules compatible with benign U2 thyroid nodules, which could be suggestive of type 1 AIT, however an iodine uptake scan revealed reduced uptake. Given this, the absence of TSI antibodies, and following endocrinology review, the aetiology was deemed to be type 2 AIT and the carbimazole was subsequently stopped. Review one month later revealed a normal thyroid examination with no palpable goitre or asymmetrical movement on swallowing. Blood test results included normal TFTs (TSH 0.55mU/L, FT4 18.1 pmol/L) and therefore her prednisolone was tapered down, with regular TFT monitoring for any exacerbations that could require higher steroid dosing. Review in clinic three months later revealed normal TFTs.

Discussion: Type 2 AIT reflects a thyrotoxic process caused by direct amiodarone toxicity to thyroid follicular cells, releasing preformed T3/T4. It classically occurs in patients without underlying thyroid disease and toxicity may take years to develop from initiation of amiodarone therapy[1]. Whilst type 1 and 2 AIT are better differentiated via colour flow doppler studies[2], the thyroid ultrasound and iodine uptake scans in this patient revealed no features suggestive of an autonomous nodular goitre or Graves' disease, indicating type 2 aetiology. Type 2 AIT is treated with corticosteroids such as prednisolone due to their anti-inflammatory and membrane-stabilising effects[3].

References:

1. Tsang W, Houlden RL. Amiodarone-induced thyrotoxicosis: a review. Can J Cardiol [Internet]. 2009 Jul;25(7):421–4. Available from: https://pubmed.ncbi.nlm.nih.gov/19584973
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3. Bartalena L, Brogioni S, Grasso L, Bogazzi F, Burelli A, Martino E. Treatment of amiodarone-induced thyrotoxicosis, a difficult challenge: results of a prospective study. J Clin Endocrinol Metab. 1996 Aug;81(8):2930–3.

B006

Case report: Partial hypopituitarism & central diabetes insipidus in acute myeloid leukaemia monosomy 7 & chromosome 3 inversion

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[3] St Bartholomew's Hospital, Radiology Department, Barts Health NHS Trust, London.

We present a case of partial hypopituitarism in acute myeloid leukaemia (AML) with chromosome 3 or 7 abnormalities.

A 56 year old male initially presenting in Pakistan with fevers on a background of intermittent increased urinary frequency and difficulty voiding. He was found to be pancytopenic and referred locally to haematology and subsequently underwent a bone marrow showing 13% blasts. On arrival in the UK, he was treated for pyrexia of unknown origin and found to be profoundly hypernatremic (Na 163) despite intravenous fluids with intermittent polyuria.

Initial endocrine investigations showed partial hypopituitarism and hypernatraemia secondary to diabetes insipidus - T4 5.6 pmol/L, TSH 0.82 mU/L, cortisol 80nmol/L, FSH 5 unit/L, LH 4.1 unit/L, prolactin 531 mU/L, testosterone < 0.5 nmol/L, GH 0.89 mcg/L, urine osmolality 285 mmol/kg and serum osmolality 324 mmol/kg. He was commenced on desmopressin, hydrocortisone, and levothyroxine. On the MRI pituitary scan with contrast the posterior bright spot could not be visualised.

A blood film showed circulating blasts and a bone marrow biopsy confirmed AML. The AML FISH panel showed monosomy 7 and paracentric inversion of long arm chromosome 3. He was commenced on his first cycle of chemotherapy. Post chemotherapy bone marrow biopsy showed evidence of refractory disease.

The association with AML monosomy 7 or chromosome 3 abnormalities and hypopituitarism has been reported in the literature. Our patient with both abnormalities presented with partial hypopituitarism and central diabetes insipidus likely secondary to AML infiltration.

B007

A challenging adrenal incidentaloma

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[2]University College London Hospital, London, UK.

Case History: 69 years old gentleman was referred to endocrinology for investigation of a benign appearing left incidental adrenal adenoma (1cm) after being investigated for abdominal pain. He had a past medical history of an abdominal aortic aneurysm (AAA), type 2 diabetes mellitus and hypertension.

Investigations: Initial endocrine investigations revealed normal 24h urinary free cortisol levels (twice), metanephrines and ARR. His overnight dexamethasone suppression test was abnormal (370 nmol/l – NR <50). The vascular surgeons were keen to undertake endovascular repair of his AAA. There were no stigmata of Cushing's syndrome and together with unconvincing biochemical results, surgery was undertaken. His low dose dexamethasone suppression test failed to suppress his cortisol (135 nmol/l) with a baseline ACTH of 52.3 ng/l (NR 1.6 – 63.3). Moreover, his midnight cortisol was elevated (107 nmol/l). A pituitary MRI scan demonstrated a 4mm microadenoma. His repeated urinary free cortisol levels were abnormal (128 and 187 nmol/24h – NR 1-124). The patient declined pituitary surgery, and was commenced on Metyrapone titrating up to 1.5g BD. 11 months later, he agreed to have surgery. IPSS then confirmed Cushing's Disease: left petrosal ACTH sampling showed a peak concentration of 1,203 ng/l and the right petrosal one was 1,165 ng/l (NR 1.6 – 63.3). The ratio favoured left sided hypersecretion. A repeat pituitary MRI scan, 24 months after initial scan, showed that the lesion had increased in size and had proteinaceous content. He is scheduled for explorative surgery.

Conclusion: We present a diagnostically challenging case of Cushing's Disease, referred initially as adrenal incidentaloma. He had no clinical features of hypercortisolaemia and with unconvincing biochemistry, he underwent vascular surgery that was deemed necessary. Subsequent investigations confirmed pituitary source rather than ectopic or adrenal. Pituitary surgery has now been scheduled.

Learning Points

1. Adrenal incidentalomas should be referred to endocrine MDT for further workup for hypersecretion or malignant features. Initially his biochemistry was normal, then it showed overproduction of cortisol, without any clinical feature.

2. Aorto-bi-iliac quadruple fenestrated EVAR (percutaneously) was performed, as it was deemed necessary, without any complications, whilst still investigated for hypercortisolaemia. Subsequently, investigations confirmed pituitary source, rather than ectopic or adrenal.

B008

Partial hypopituitarism with unknown aetiology followed by spontaneous recovery: a case report.

T Chopra, A Qureshi, A Haddad. Diabetes & Endocrinology, Northwick Park Hospital, London

Hypopituitarism may be due to primary pituitary or hypothalamic identifiable aetiology, and usually patients stay on hormonal replacement therapy for the remainder of their life. We report a 58-year-old gentleman with partial hypopituitarism of unknown aetiology who recovered spontaneously within ten months of diagnosis.

Our patient presented initially to Emergency Department with sudden onset vomiting, generalised weakness, and visual blurring. He reported a 3-month history of severe frontal headaches, a 1-week history of ataxia and chronic erectile dysfunction. He denied any history of head trauma. He did not have anosmia or a family history of any endocrinopathies. He had a history of Type 1 Diabetes which was controlled with basal bolus regime. He worked in a museum and denied any use of steroids, opiates, or recreational drugs. On admission, he was noted to be hypotensive with low capillary glucose. There was evidence of bitemporal superior quadrantanopia and ataxia with no other focal neurology elicited.

Initial bloods were normal apart from low sodium (127mmol/L) with a normal potassium (4.8mmol/L). CT head was unremarkable. Sodium levels decreased to 120mmol/l over the next few days. A hyponatremia screen revealed a low morning cortisol (110nmol/l), low TSH (0.04mIU/L), low T3 (3.0pmol/L) and low T4 (6.0pmol/L), with raised urine osmolality (361 mOSmol/Kg) and raised urinary sodium (60mmol/L). A full pituitary profile showed low levels of LH (<1IU/L) and low testosterone (<0.7nmol/L) with a borderline low FSH (1.9iU/l), normal IGF-1 (18.1nmol/L) and normal prolactin (199mIU/L). A short SynACTHen test confirmed low basal cortisol (174nmol/L) but an adequate response with ACTH (30 min cortisol 693nmol/L and 60 min cortisol 776nmol/L). An MRI head and pituitary were normal, with discussions at the radiology MDT confirming no pituitary lesions.

Given findings of partial hypopituitarism of unknown aetiology; he was commenced on hydrocortisone, levothyroxine & Nebido. His symptoms improved and he was discharged from hospital. Over the next few months, he remained asymptomatic with good compliance to medications. Ten months following initial diagnosis, it was noted that testosterone levels were supratherapeutic (28.5nmol/l) with suppression of FSH and LH (<1IU/L) and his T4 levels were raised (28.5pmol/L) with a supressed TSH (0.02mIU/L). Due to the noted partial recovery in pituitary profile, a 9am cortisol was repeated (310nmol/L) with a short synACTHen test showing an adequate basal cortisol (340nmol/L) and ACTH (25.5ng/L) with good response (30 min cortisol 522nmol/L and 60 min cortisol 557nmol/L). This represented a complete spontaneous resolution of his partial hypopituitarism. Hydrocortisone, levothyroxine and Nebido were stopped. The patient continues to remain asymptomatic with normal TFTs and gonadotrophins.

In conclusion, it is uncommon for partial hypopituitarism to spontaneously recover within few months of diagnosis, so it is very important to re-assess hormonal replacement to avoid overreplacement especially in cases with no clear aetiology where the MRI should be always discussed in radiology MDT.

B009

Analysing the hormonal characteristics of men self- administering hormonal therapy to facilitate their withdrawal from anabolic androgenic steroids

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Background: Anabolic-androgenic steroids (AAS) are highly potent testosterone-like substances. Some men use AAS to boost muscle bulk or enhance athletic performance. AAS are known to suppress endogenous testosterone and abrupt withdrawal causes hypogonadal symptoms, including low mood and sexual dysfunction. AAS users have procured hormonal therapies, such as selective oestrogen receptor modulators and human chorionic gonadotrophin, with the aim to restore endogenous testosterone and lessen withdrawal symptoms. This self-directed treatment is known as post-cycle therapy (PCT).

Objective: Evaluate the association of PCT use and other characteristics with reproductive hormones in men stopping AAS.

Methods A retrospective clinical audit of men who underwent a post-AAS assessment at a harm reduction clinic in Glasgow from 2015-2022. User characteristics, patterns of AAS use and PCT use were recorded. Reproductive hormones were compared in men who took PCT (n=466) and men who did not take PCT (n=147).

Results: PCT was used in 76% of men. Men using PCT had a significantly greater total testosterone compared with no PCT use (11.2$\pm$0.6, no-PCT; 12.8$\pm$0.4, PCT; p=0.024). A higher proportion of men with normal total testosterone ($\geq$ 10nmol/L) was seen in the PCT group compared with the no-PCT group (49%, no-PCT; 59%, PCT; p=0.036). A greater number of AAS used was associated with lower total testosterone (p<0.01) and LH (p<0.01).

Conclusions: This is currently the largest study of men withdrawing from AAS, in an area with some of the highest substance misuse rates in the country. These results suggest an association between PCT use and reproductive hormonal recovery from AAS-induced hypogonadism. Prospective studies are needed to confirm the strength of this association. Men taking more AAS were found to have lower total testosterone, suggesting repetitive exposure to highly supraphysiological levels of androgens may result in chronic gonadal dysfunction. Overall, this data has important implications for the formation of a supportive management plan for men withdrawing from AAS.

B010

Neutropenia in a Patient Taking Thionamides

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[1] Ealing Hospital, London North West University Healthcare NHS Trust.
[2] Northwick Park Hospital, London North West University Healthcare NHS Trust.

A 47 year old lady with paranoid schizophrenia, asthma and moderate learning disability presented in 2019 with subclinical hyperthyroidism with a TSH of <0.03 mIU/L (0.34 - 5.6) and fT4 of 16.5 pmol/L (7.5-21.1). Her radioisotope thyroid uptake scan was suggestive of toxic multinodular goitre (MNG), so she was started on carbimazole as treatment. Prior to treatment she was given written information from the British Thyroid Foundation about the side effects of carbimazole. In October 2022, she presented acutely with a 1 week history of productive cough, fever and myalgia. She was found to be neutropenic (WBC 2.7, Neutrophils 1.0) so was treated for neutropenic sepsis. She was biochemically euthyroid on admission Her white cell count improved by itself after carbimazole were stopped. She was not a suitable candidate for radioiodine as she lived with 24 hour carers and it was thought that she would be unable to comply with radiation protection guidelines. She was therefore referred for thyroidectomy, which was successfully performed on 3 November 2022. Preoperatively she was given propranolol and Lugol's iodine.

A rare but serious side effect of Carbimazole is agranulocytosis, which is reversible if recognized and treated accordingly. The incidence of Carbimazole induced agranulocytosis is 0.3–0.6% and it has a mortality of 21.5%. Both direct bone marrow toxicity and immune-mediated responses have been described. Drug-induced agranulocytosis usually occurs within 1–2 months of starting carbimazole but the onset can be delayed. Treatment includes broad-spectrum antibiotics and granulocyte-colony stimulating factor in selected patients. Carbimazole should immediately be stopped to prevent further bone marrow damage. These patients will then need definitive treatment for thyrotoxicosis, usually with radioactive iodine or surgery. Although our lady was also on aripiprazole, her neutropenia improved once carbimazole was stopped. Our lady successfully had a thyroidectomy after multidisciplinary discussion between endocrinology, nuclear medicine, psychiatry and our thyroid surgeon.

B011

Transient hypophosphatemia secondary to iron infusion

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Hypophosphatemia is commonly missed due to nonspecific signs and symptoms. It can cause muscle weakness, confusion, white blood cell dysfunction and disrupt cardiopulmonary systems. Three main mechanisms of hypophosphatemia are shifts from the extracellular to intracellular compartment, increased renal excretion and decreased intestinal absorption. Here we report a case of symptomatic hypophosphatemia post ferric carboxymaltose (Ferinject) infusion.

A 42-year-old lady with longstanding ulcerative colitis on vedolizumab injection and mesalazine attended the Emergency Department with palpitations, nausea and fatigue. She also suffered from iron deficiency anaemia secondary to menorrhagia and had received intravenous Ferrinject six days prior. Her blood tests were unremarkable apart from low phosphate level (0.39mmol/L). Electrocardiogram revealed sinus rhythm with atrial ectopics.

Over eight days, she required three IV phosphate infusions and oral phosphate replacement. Within two months, serum phosphate returned to normal (1.0 mmol/L) with resolution of symptoms. During this time, a 24-hour urine phosphate collection highlighted an inappropriately high level of 17.05mmol/24hrs, with a high fractional phosphate excretion of 25.8%. Further investigations showed a low vitamin D (37.6nmol/L), normal 1,25 OH vitamin D (133nmol/L), normal fibroblast growth factor 23 (FGF-23) (51RU/mL) and normal retinol binding protein/creatinine ratio (7.6ug/mmol). These tests exclude proximal renal tubular injury and tumour-induced osteomalacia as a cause and the normal FGF-23 indicated resolution of the transient pathology.

It is suggested that iron infusions cause hypophosphatemia by increased FGF-23, which reduces phosphate reabsorption in the proximal tubules. Severe hypophosphatemia is infrequent, but a potentially serious and reported complication. It can occur three days post infusion and last up to eight weeks which is significant given the increased use of iron infusions within ambulatory care. Further education and monitoring post infusion should be implemented, and assessment of renal tubular phosphate handling to identify the cause.

B012

Highlighting Multidisciplinary Interdependence - Post Operative Hypoadrenalism following Unilateral Adrenalectomy

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Day one post-operative early morning cortisol testing is the standard protocol at ICHT for all patients undergoing a unilateral adrenalectomy, to assess for post-op adrenal insufficiency. An audit on adrenalectomy cases performed at Hammersmith Hospital was carried out to assess the following: 1. The adherence to this standard. 2. The proportion of patients with post-operative low cortisol measurement. 3. The proportion of patients with new post-op hypocortisolism requiring steroid replacement. 156 adrenalectomy cases from 1/1/19 to 1/9/22 were identified, with data extracted including 9am cortisol, the requirement for steroid replacement post op, the indication for surgery and the functional status/histology of the excised adrenal gland. Low cortisol was defined as 9am cortisol <160nmol/L.

Day 1 post-operative early morning cortisol testing was carried out in 125 patients (80.1%), with low cortisol identified in 41 (26%) patients. However, only 16 (10%) patients required steroid replacement postoperatively. Pre-op hypercortisolism was identified in 32 (20.5%).

Sub-group analysis revealed that pre-op hypercortisolism was associated with a reduced mean cortisol (159.8 vs 253.5, p=0.0004). Interestingly however, an analysis of the 41 cases of low post-op cortisol revealed that 15 (36.6%) received 3.3mg-6.6mg IV dexamethasone intraoperatively, resulting in post-op axis suppression. The majority of these patients were clinically well, however one result masked a true adrenal insufficiency requiring steroid replacement.

Dexamethasone is a useful agent in the anaesthetic management of postoperative nausea and vomiting (PONV). Adrenal surgery represents a rare subset of procedures where the administration of exogenous glucocorticoids can hinder the recognition of post-operative morbidity. Operative data alongside a combined surgery/endocrine anaesthetic discussion concluded that a multi-layered approach to increase the awareness of this interaction would be a useful strategy to reduce the use of intraoperative dexamethasone in adrenal surgery, including written and verbal notification at the time of surgery booking and during pre-operative checklists, as well as targeting the dissemination of this information through local channels.

We hope this audit exemplifies the interdependence of the different branches of the endocrine MDT, demonstrating the value of interdisciplinary communication as a catalyst for the delivery of quality patient care.

B013

A case of diabetes insipidus unmasked by initiation of steroids

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Clinical presentation: 77 year old male was admitted with hypotension and collapse. GCS 8/15, hypernatraemia and complaining of polyuria and polydipsia.
Treated for urosepsis with acute kidney injury and required HDU admission for vasopressor support. Past medical history of epilepsy, atrial fibrillation, COPD, brain tumour 2011 with histology suggestive of lymphoma.

The patient had a recent admission 2 weeks earlier for feeing unwell, vomiting, shortness of breath and cough. He was treated for community acquired pneumonia and was also found to have hypovolaemic hyponatraemia of 116 and hypothyroidism.

During this first admission his 9 am cortisol was 42, serum osmolality 241, urine osmolality 276, urine sodium < 20. Short Synacthen test showed adrenal insufficiency.

He was started on steroids and levothyroxine.

During this admission whilst treating for urosepsis and acute kidney injury his steroid dose was doubled as per sick day rule.

Investigations: Serum osmolality was raised 338 with urine osmolality of 272 and urine sodium of 110. MRI pituitary: Abnormal FLAIR high signal intensity in the hypothalamic region involving the optic chiasm, optic tracts, hypothalamus with oedema extending into the medial aspects of both thalami.

Multidisciplinary Management: Endocrinology diagnosed with diabetes insipidus and started on desmopressin. Neurology advised LP (normal), EEG and increase anti-epileptic medication. Haematology advised CT CAP to look for peripheral lymphadenopathy due to history of suspected CNS lymphoma. This showed new onset parenchymal lesions and small groundglass changes.

Neuro-Onc MDT advised for biopsy to help with diagnosis of lymphoma however, patient expressed wishes not for further biopsy, invasive procedures, or chemotherapy.

Neurosurgical team did not think that proceeding with brain biopsy will be in the patient's best interest.

Conclusion: Central diabetes insipidus may be unmasked by initiation of steroids and has been reported in case reports of hypopituitarism where diabetes insipidus may be masked by adrenal insufficiency without clinical symptoms initially but unmasked once started on steroids. This case also reflects the importance of multidisciplinary teams in the management of patient care and most importantly taking patient wishes in consideration to give the most appropriate treatment in according to patient's best interest.

B014

Severe Hyponatremia secondary to NSAIDs

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We present a case of a 24-year-old woman initially admitted with palpitations and chest pain, likely due to myopericarditis. On admission, she was noted to be hypertensive (blood pressure of 173/124) which was later attributed to her use of the combined oral contraceptive pill (OCP). She was treated with antihypertensive medications, initially amlodipine, which was then replaced with a regime consisting of nifedipine, ramipril, and clonidine. Prior to admission, she had been taking ibuprofen 400mg TDS for seven days, and from admission the dose was increased to 600mg TDS to alleviate her pain which continued for three days post admission.

On admission, her serum sodium was 135 mmol/L. It began to fall reaching 120 mmol/L and 111 mmol/L on days two and three of admission respectively. Her urine was inappropriately concentrated, with a paired urine sodium 128 mmol/L and osmolality 617 mOsm/kg. Her serum osmolality was low at 250 mOsm/kg. She had a generous afternoon cortisol of 1030 nmol/L and a normal thyroid function test (TSH 1.59 milliunit/L). Syndrome of Inappropriate Anti-Diuretic Hormone (SIADH) was diagnosed with ibuprofen as a rare causative drug. Ibuprofen was stopped. She initially remained clinically euvolaemic and was treated with strict fluid restriction at 500ml/24 hours, but she subsequently became hypovolaemic with a further drop in her sodium to 109 mmol/L necessitating admission to the intensive care unit, where she received two boluses of 2.7% hypertonic saline and her sodium improved to 125 mmol/L. She was continued on fluid restriction which normalised her sodium to 136 mmol/L on her discharge from hospital. She has remained well on follow-up with no recurrence of hyponatremia. Over the next 3 months following discharge, all her antihypertensive medications were discontinued successfully after cessation of OCP.

Hyponatremia remains the most frequent electrolyte disturbances seen in hospitals and frequently is the manifestation of SIADH. Drugs are often the culprit, and this case highlights that ibuprofen can be a rare cause of severe acute onset drug-induced SIADH. A likely mechanism for this is that inhibition of prostaglandins in the kidneys potentiates the action of ADH, impairing the excretion of free water.

B015

Abnormal LFT and Hypercalcaemia due to Thyrotoxicosis

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Introduction: Hypercalcaemia and liver injury are now well-recognized as a complication of thyrotoxicosis. It is the purpose of this paper to record a case of hypercalcaemia and abnormal LFTs secondary to thyrotoxicosis. The changes were corrected by treatment of thyrotoxicosis.

Case: A 26-year-old woman with no medical history presented to ED due to pain in the abdomen and pelvis. A blood test showed high serum Ca level and deranged LFTs ALP. CXR and abdominal X-ray were normal.

ED treated her hypercalcaemia with IV Fluids. Abdominal and Pelvic US scan was also unremarkable (normal liver). However, low TSH, high FT4, and low PTH were noted, and she was referred to the endocrine clinic. She was found to be thyrotoxic with palpitations, weight loss and insomnia. Hypercalcaemia and Abnormal LFTs secondary to thyrotoxicosis were diagnosed and anti-thyroid treatment was commenced. 5 months later, FT4 improved, hypercalcaemia was normalised and LFTs improved.

Discussion: This case report demonstrates the importance of considering thyrotoxicosis as a cause of hypercalcaemia, as well as deranged LFTS. The mechanism of liver dysfunction in thyrotoxicosis is not very clear. Upadhyay et al suggested T3-induced hepatocytes apoptosis as a possible explanation. Thyrotoxicosis can be associated with congestive cardiac failure explaining the deranged LFTS. Managing thyrotoxicosis should be a priority which in turn will lead to the resolution of both hypercalcaemia and deranged LFTs (Clark et al 2010)

B016

Interesting survivor of Di-George syndrome

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DiGeorge syndrome, also known as CATCH 22, is a common condition caused by genetic microdeletion. It occurs in about 1 in 3000-6000 births with equal prevalence among both genders.

The symptoms include cardiac anomalies, short stature, learning and behaviour problems, cleft lip or palate, speech and hearing difficulties, and hormone problems like underdeveloped parathyroid glands.

A case report illustrating untreated hypoparathyroidism in a 47-year-old woman with DiGeorge syndrome is presented. It is a documentation of a patient who has a single child, does not take her supplements for hypoparathyroidism and has amenorrhea.

This case sheds light on one of the varied ways the syndrome might present and the management of the patient so far with no treatment for her hypoparathyroidism or amennhorea.

B017

Thyroiditis presenting with abnormal LFTs, and weight loss

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Introduction: Hyperthyroidism has been known to be associated with abnormalities of serum liver chemistry.

Case: A 34 year old man presented to the emergency department at Queens Hospital with non-significant medical background and had a 4 week history of sore throat, dry cough, palpitation, drenching night sweats, unintentional weight loss, and fever. He was initially seen by his GP and completed 2 courses of antibiotics with no improvement. Then presented to the Endocrine clinic he was given a trial of carbimazole 5mg on alternate days, and propranolol 10mg TDS with no improvement. He responded well to prednisolone 30mg OD.

Investigations	Normal Range (Units)	At 1st presentation	After Steroid
TSH	0.27 - 4.2 mU/L	<0.1	2.04
Free T3	3.1 - 6.8 pmol/L	21.3	4.7
Free T4	12 - 22 pmol/L	>100	15.2
ESR	0-22 mm/hr	>130	90
CRP	<5	116	7
ALP	30 - 130 IU/L	325	103
ALT	0-50 IU/L	274	36
All Serology	-	Negative	Negative
Septic Screen	-	Negative	Negative
COVID-19 (3times)	-	Negative	Negative

CXR-NAD

Ultrasound of the thyroid showed thyroiditis.

Uptake scan-reduced uptake in the left upper pole region. The technetium thyroid uptake at 20 minutes is within normal limits at 2.7%

Discussion: Thyroiditis usually presents with neck pain and fever. This patient presented with the feature of sepsis. This case report adds to the emerging evidence of deranged liver enzymes associated with Thyroiditis. This patient had an increase in liver enzymes following the diagnosis of thyroiditis. The patient improved after a course of steroid therapy. It can be concluded that hyperthyroidism causes alteration in liver enzymes, and therefore, it is recommended that liver function tests be interpreted in hyperthyroidism.

B018

Tachy Tea: The perils of a Kalamitous Cuppa

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We present the case of a 46 year old lady who was admitted to hospital with a history of vomiting, palpitations and weakness, which culminated in a collapse. She had been symptomatically unwell for some time with palpitations, nausea and weakness predominating. She denied any significant past medical history with the exception of chronic constipation for which she was taking occasional lactulose. Initial investigations showed a serum potassium of 2.4 mmol/L, mild hyponatremia and a normal serum magnesium. Her venous gas demonstrated the following: pH 7.52, bicarbonate 35.1mmol/L and chloride 95mmol/L suggesting possible gastrointestinal electrolyte loss. Biochemistry pre-dating her symptoms indicated no preceding concerns with potassium levels. She had a normal electrocardiogram, unremarkable MRI brain, a normal pituitary profile and aldosterone:renin ratio on admission. She was discharged after her potassium was normalised and prescribed omeprazole and Movicol with a plan for further outpatient investigations.

At her follow up appointment she was still experiencing weakness and had felt generally unwell since discharge. She had an oesophagogastroduodenoscopy that showed gastritis with a positive H pylori: she was treated with antibiotics. A 24-hour tape demonstrated occasional ventricular ectopics. Repeat electrolytes on the day showed a low serum potassium of 2.5 mmol/L with a normal sodium and magnesium. She was re-admitted to hospital. Subsequent tests were more indicative of a renal tubular potassium leak: a spot and 24 hour urine collection demonstrated inappropriately elevated potassium levels. On this admission she indicated that she had been drinking over-the-counter “Slimatee” on most days for months, which was recommended by her community pharmacist for constipation. Her urine was tested for diuretics and found to be positive for hydrochlorothiazide and furosemide. Following cessation of Slimatea, her potassium at follow up was normal (4.2mmol/L).

Many herbal teas and alternative medicines, particularly those advertised for weight loss, contain ingredients, which have diuretic or laxative properties. The harmful effects of overuse of these remedies are well documented and include hepato-/nephrotoxicity, hypertension, and hypokalaemia, which can lead to life threatening cardiac arrythmias. This case highlights the importance of taking an accurate medication history and serves as a reminder to both clinicians and patients that remedies bought over-the-counter can have health consequences just as potent as those on prescription.

B019

A rare case of a functioning adrenal oncocytoma masquerading as adrenocortical carcinoma

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Oncocytomas are rare tumours of the adrenal gland, accounting for 1.8% of adrenal tumours. They are usually non-functional and benign, and incidentally detected on imaging. However around 30% of adrenal oncocytomas are functional and 20% are malignant, thereby warranting investigation and management.

We present a case of a 34-year-old female with no past medical history, with a longstanding history of undiagnosed right upper quadrant pain. After an episode of right loin pain she underwent an ultrasound scan, which demonstrated a 3.5cm cyst at the upper pole of the right kidney with some debris. Following this she underwent a CT and MRI, both of which characterised this as a 5cm mass arising from the right adrenal gland. This was also found to demonstrate intense FDG avidity on PET scan. A panel of bloods were performed which showed normal plasma metanephrines, a raised aldosterone of 1795pmol/l with unsuppressed renin of 8.3pg/ml and autonomous cortisol secretion, with a cortisol of 321nmol/l and ACTH <1.5ng/l after overnight dexamethasone suppression testing. Multi-disciplinary team meeting review concluded that this was highly likely to be an adrenocortical carcinoma (based on the FDG-PET and functionality) and recommended urgent adrenalectomy.

The patient therefore subsequently underwent a laparoscopic adrenalectomy, at which a well circumscribed tumour was identified and excised in its entirety. The patient's recovery was uncomplicated but delayed by post-operative nausea and she was therefore discharged home on the third post-operative day. Her post-operative cortisol level was >200nmol/l on both the first and second post-operative day, and so she was discharged without the requirement for prednisolone. Histology from the specimen returned showing a 4.5cm tumour that was identified histopathologically as a completely excised oncocytoma. Subsequent functional testing at 2 months showed a normal aldosterone and cortisol.

This represents a rare case of a hormone-secreting adrenal oncocytoma and highlights the need to consider this diagnosis whilst performing diagnostic work-up for adrenal tumours. Due to its uncommon nature, there is limited literature on this topic - the aim of this case report is therefore to contribute to and summarise the existing literature regarding this rare diagnosis.

B020

Covid-19 vaccination and new diagnosis adrenal insufficiency: cause or accelerated presentation?

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We present the case of a 37-year-old female, with a background of childhood asthma and bronchiectasis, who presented to accident and emergency with vomiting, dizziness and symptomatic hypotension. She described a 6-week prodrome of lethargy and weight loss, which were exacerbated following her first dose of the AstraZeneca Covid-19 vaccine. Further history revealed recurrent steroid use as a child. On examination, she appeared tanned and slim with mild pigmentation to her gums. Her biochemistry was as follows: sodium 115mmol/L, potassium 3.9mmol/L, random cortisol <28nmol/L and ACTH of 36.6 ng/L. She was managed for an adrenal crisis in intensive care and responded well to parentral steroids. Pituitary profiling was otherwise satisfactory, and adrenals were unremarkable on MRI: her adrenal cortex antibodies were positive. Following discharge, she remains well on hydrocortisone and fludrocortisone supplementation and is aware of sick day rules.

Vaccines have infrequently been associated with precipitation of an adrenal crisis. The AstraZeneca Covid-19 vaccine is a recombinant vaccine and utilises the SARS-CoV-2 spike protein to generate an immune response. It is plausible, that the immune response or side effects of the vaccine including fever, gastrointestinal symptoms and physiological stress could have accelerated the presentation of an adrenal crisis in this patient. At present, case reports of adrenal crisis following the Covid-19 vaccine are rare. Cases described are in patients with an established diagnosis of adrenal or pituitary insufficiency who present acutely (<24 hours) following vaccination. Interestingly, this patient was not known to have adrenal insufficiency prior to her admission and her symptoms progressed gradually from vaccination to admission. Once steroid replete, she tolerated the second dose of her AstraZeneca Covid-19 vaccine well. Clinicians should be mindful of the systemic effects a novel vaccine may have and consider adrenal crisis in patients who present critically unwell in order to reduce the risk of morbidity and mortality in this cohort.

B021

The Role of Fluconazole and Cinacalcet in the management of CYP24A1 mutation Induced Hypercalcaemia

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Introduction: There are many causes of hypercalcaemia including parathyroid adenoma, malignancy, drugs, myeloma and sarcoidosis. Rare causes of hypercalcaemia include genetic causes should be considered. Hypercalcaemia due CYP24A1 mutation (idiopathic Infantile Hypercalcaemia) although rare should be considered as differential diagnosis after ruling out other causes of hypercalcaemia.

Idiopathic Infantile hypercalcaemia is a condition first described by Fanconi et al. and Lightwood in the early 1950s. Patients classically present within the first year of life with profuse vomiting, irritability, constipation, poor weight gain and nephrocalcinosis.1,2 Although rare, the condition is an important cause of failure to thrive, and can be lethal during the acute phase of hypercalcaemia.3

There are many causes of hypercalcaemia including parathyroid adenoma, malignancy, drugs, myeloma and sarcoidosis. Rare causes of hypercalcaemia include genetic causes. Hypercalcaemia due CYP24A1 mutation (Infantile Hypercalcaemia) although rare should be considered after ruling out other causes of hypercalcaemia.

Long-term management involves placing patients on low calcium and vitamin D-free diets.19 Patients with persistent hypercalciuria and recurrent nephrolithiasis can be treated with thiazide diuretics, which serve to reduce urinary calcium excretion.20

Here we report a case of male patient who presented with resistant hypercalcaemia and recurrent renal stones. Investigations over a 10-year-period were unable to identify a cause for these symptoms. Genetic testing revealed CYP24A1 mutations which confirmed a diagnosis of IIH.

Case report: 72 years old man was referred to Endocrine clinic with hypercalcemia. He was known to have recurrent admission with severe hypercalcemia and recurrent renal stones requiring hospital admission. His blood test revealed detectable/normal PTH 2.3 and fluctuating level of levels of hypercalcaemia. He was under Urology team for recurrent urinary tract infections, previous microscopic haematuria and renal stones, high cholesterol, type 2 diabetes. He had Ultrasound parathyroid and sestamibi scan with negative results, CT chest abdomen, pelvis as well as PET were normal. His current medication includes metformin 1g twice a day, losartan 50mg once daily, allopurinol 300mg once at night, linagliptin 5mg once a day, folic acid 5mg once a day, atorvastatin 20mg once a day, alendronic acid 70mg once a week. The patient subsequently underwent genetic testing which was positive for CYP24A1 mutation. He was started on fluconazole 50mg once a day and cinacalcet 30mg twice with normalisation of calcium level.

The patient most recent blood test the calcium levels were 2.52 and 2.56 which is within normal level.

Discussion: The prevalence and incidence of the condition has not been quantified, although these are estimated to be very low, with 200 cases occurring over 2 years in the United Kingdom during the 1950s.4 The introduction of routine vitamin D supplementation in fortified milk and other infant products during this period led to a significant increase in the numbers of cases. This phenomenon was reversed when the dose of Vitamin D supplementation was reduced on advice from the British Paediatric Association. It was therefore suggested that the condition might result from a hypersensitivity to Vitamin D, although it was unclear whether its pathogenesis was due to excess activation or deficient inactivation of Vitamin D.4

A distinction has subsequently been made between cases of infantile hypercalcaemia due to Williams-Beuren syndrome and Lightwood type, or idiopathic infantile hypercalcaemia (IIH). The former is a syndrome that manifests a complex phenotype consisting of characteristic facies and supravalvular aortic stenosis, and is associated with an early and severe presentation of infantile hypercalcaemia.5 The latter, which is the focus of this case report, presents with milder symptoms.2

The link between CYP24A1 mutations and IIH has been recently established. 6 Vitamin D is converted to its active form by 25-hydroxylase in the liver followed by 1α-hydroxylase in the kidney. This active form, 1,25-dihydroxyvitamin D 3 is inactivated by 24-hydroxylase, an enzyme encoded by the CYP24A1 gene on chromosome 20. 7,8 Inactivating mutations or deletions in CYP24A1 result in higher circulating active vitamin D, which binds to the vitamin D receptor to upregulate serum calcium levels.

Anti-fungal agents such as fluconazole inhibit 1α-hydroxylase and 25-hydroxylase involved in activating vitamin D, and have been used to treat IIH with good effect.13–15
Our patient responded well to the combination of fluconazole and cinacalcet, which helped to reduce his calcium level.

Question to panel
What would you do differently in managing such patient with rare condition?

B022

Peculiar numbers requiring clinical correlation.

R Pereira, A Kapoor, M Yee. St Mary's Hospital, Imperial College Healthcare NHS Trust, London.

Automated immunoassay platforms are currently the preferred method for the measurement of thyroid function test in clinical laboratories[1]. However, immunoassays are susceptible to various forms of interference, thyroid stimulating hormone (TSH), free thyroxine (T4) and free triiodothyronine (T3): macro-TSH, biotin, anti-streptavidin antibodies, anti-ruthenium antibodies, thyroid hormone autoantibodies, and Heterophile antibodies that can result in erroneous clinical decisions[1].

We present a case of a 68-year-old lady that was referred to the Endocrinology team following the incidental finding of deranged thyroid function results after routine bloods. Other co-morbidities include systemic lupus erythematosus (SLE) and chronic bladder inflammation. She is only on Hydroxychloroquine. On consultation, the patient denied any symptoms of hyperthyroidism - No symptoms of palpitations or tremors. No symptoms related to her neck, did not notice any goitre or pain. No ocular symptoms. She does not have a family history of thyroid related disorders.

Bloods test, free T4 >64.4pmol/L, have been exceedingly elevated but unsuppressed TSH 2.36miliunits/L (0.30 – 4.20) and normal free T3 of 5.1pmol/L. Her sex hormone binding globulin (SHBG) - 49nmol/L (30-100) which was also normal. These results were measured using Abbott Alinity Assay. We questioned about T4 Interference and samples were sent to the Royal Brompton for further analysis using the DELFIA method which showed free T4 levels were significantly different 13.8 pmol/L and 15.3 pmol/L (reference range: 7.9-14.4).

The DELFIA TSH assay is a robust two-step method against binding protein abnormalities. Repeat samples using this method showed no evidence of interference. This case highlights that great caution should be applied to the individual case when interpreting thyroid function test. Hence it is prudent to seek external assessment whenever biochemical or clinical discrepancies arise.

Reference:

Favresse, J. Burlacu, MC. Maiter, D. and Gruson, D. (2018). 'Interferences With Thyroid Function Immunoassays: Clinical Implications and Detection Algorithm'. Endocrinology

B023

Are SGLT2 inhibitors over-prescribed in primary care without appropriate risk-stratification

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We present a case of 52 year old lady with background of T2DM treated with Metformin and Empagliflozin. Her background history includes carcinoma of unknown primary with lung, liver and peritoneal involvement on triplet chemotherapy; hypothyroidism and hypercholesterolaemia.

She presented with vomiting and poor oral intake as well as a high anion gap metabolic acidosis (pH 7.086; ketones 4.9; potassium 3.68; bicarbonate 8.4; base excess of minus 24.7; lactate 1.25; glucose 19.6; calculated anion gap: 22.9mmol/L). She was hypovolemic on admission with normal heart sounds and her abdominal examination was unremarkable.

Her blood test results include Hb 141g/L; wcc 13 x 109/L; neuts 12.1 x109/L, CRP 207.8mg/L; ALT 187units/L; AST 337units/L; ALP 1686unit/L; Bil 36umol/L; Na 133mmol/L; K 3.3mmol/L; eGFR 58ml/min/1.73m2 (previously more than 90) and HbA1C 68mmol/mol.

She was treated with a fixed rate insulin infusion and broad spectrum antibiotics for sepsis. Her Metformin and Empagliflozin was withheld due to her renal impairment and she was initiated on SC Abasaglar instead. Her acidosis resolved with discontinuation of SGLT2i treatment and an in-patient magnetic resonance cholangiopancreatography revealed extensive soft tissue surrounding the portal vein, cystic duct, common hepatic duct and common bile duct associated with indeterminate liver lesions; suspicious for an underlying cholangiocarcinoma. Discussion at Upper GI MDT was recommended.

Discussion: SGLT2i are prescribed as either first line or adjuvant treatments to other anti-diabetic therapies in UK. SGLT2i treatment is associated with an enhanced risk of euglycaemic Ketosis unless risk stratification was achieved prior to prescribing these medications. A review completed by the EU medicine regulators identified that DKA is a rare (occurring in 1 in 1000 and 10,000 patients) but life threatening side effect of SGLT2 inhibitors. SGLT2 inhibitors should only be prescribed after careful patient selection.

It is advised that SGLT2 inhibitors should not be commenced in patients with either i) low beta cell function reserve as evidenced by low serum C-peptide, latent autoimmune diabetes, or history of pancreatitis; ii) conditions leading to reduced food intake or severe dehydration; iii) surgery; iv) alcohol abuse and v) sudden reduction or increased insulin requirements. We advocate for an increased awareness particularly in the primary care setting where these medications are mainly prescribed that these patients are risk stratified appropriately prior to initiation of these therapies to avoid these life threatening complications.

References: SGLT2 inhibitors: updated advice on the risk of diabetic ketoacidosis - GOV.UK (www.gov.uk)