Hammersmith Abstracts

18th Hammersmith Multidisciplinary Endocrine Symposium 2023

Hammersmith Hospital 18th Multidisciplinary Endocrine Symposium

Programme Friday 1st Dec 2023

Wolfson Conference Centre

Hammersmith Hospital, London

8.30am Registration & Coffee.

8.55am Welcome and Introduction.

Session 1: Chair: Prof Tricia Tan & Dr James Ahlquist

9.00am Thyroid Disease in Pregnancy.
Rochan Agha-Jaffar (Imperial)

9.30am Paediatric Endocrine Surgery – Just small adults?
Mr Tom Kurzawinski (UCH)

9.50am Day Case Thyroidectomy.
Helen Doran (Salford Royal Hospital)

10.20am **C012** Recurrent thyrotoxicosis in pregnancy (Rabia Arfan)

10.35am **C017**: A challenging case of metastatic struma ovarii with rare BRAFG469A mutation (Luke Boyle)

10.50am Coffee Break.

Session 2: Chair Dani Power and Fausto Palazzo

11.20am Surgical Strategies in Syndromic Phaeochromocytoma.
David Scott Coombes (Cardiff)

11.50am International Guest Lecture
New algorithms in the treatment of aggressive thyroid cancers.
Prof Jan Zedenius (Karolinska Institute, Stockholm)

12.50pm Discussion

1.00pm LUNCH and POSTER session.

1.00pm **LUNCH and POSTER session.**

Session 3: Parathyroid Session. Preeshila Behary and Anna Crown

2.00pm Syndromic primary HPT – when to test and what to test for?
Alex Comninos (Imperial)

2.30pm **C030:** A case of hyperparathroidism – jaw tumour syndrome (HP-JTS): parathyroid carcinoma and hungry bones: **Dr James Pittaway (Barts)**

2.45pm Reoperative parathyroidectomy – lessons learned from 20 years at HH
Fausto Palazzo (Imperial)

3.15 pm **C028:** Persistent primary hyperparathyroidism cured by diagnostic FNA:
Dr Jamal Dirie

3.30pm Primary Hyperparathyroidism in pregnancy
Aimee DiMarco (Imperial)

4.00pm Clinical Cases: Tales from the Endocrine MDT.

4.00pm **C006**: The Role of Surgery in Managing Hypercalcemia in Pregnancy Furhana Hussein.

4.15pm **C002**: Addisonian Crisis as first presentation of Thyrotoxicosis.

4.30pm **C029**: The importance of under-treating adrenal failure in order to allow normal adrenal function following unilateral adrenalectomy for Cushings syndrome

4.45 **C009**: Multidisciplinary Management of Multiple Endocrine Neoplasia Type 1 (MEN1) A Case Report

5.00pm **Certificates & Close**

C001	A retrospective cohort study investigating the role of thyroidectomy in a multi-ethnic cohort of patients with Thyroid Eye Disease (TED)
C002	Addisonian Crisis as first presentation of Thyrotoxicosis
C003	A case of severe hypothyroidism and abnormal liver function post Pembrolizumab therapy
C004	Management of the patient with Prolactinoma during pregnancy
C005	A potential diagnosis of cerebral salt wasting in absence of acute intracranial pathology
C006	The Role of Surgery in Managing Hypercalcemia in Pregnancy
C007	Revisiting the Water Deprivation Test: Unveiling the Significance in Diagnosing Arginine Vasopressin Deficiency
C008	Severe Hyponatraemia Leading to the Diagnosis of Acute Intermittent Porphyria
C009	Multidisciplinary Management of Multiple Endocrine Neoplasia Type 1 (MEN1) A Case Report
C010	MEN-2A - Challenges in management - A developing country perspective
C011	Thyrotoxicosis with Immune Mediated Neutropenia
C012	Recurrent thyrotoxicosis of pregnancy
C013	Protracted hypocalcemia After the Administration of Denosumab in a Patient with Prostate Cancer with bony metastasis
C014	Should we keep treating all endocrine conditions in end of life
C015	Thyroid storm and NSTEMI a rare and dangerous association
C016	A rare coexistence of Acromegaly with metastatic Adrenocortical carcinoma

C017	A challenging case of metastatic struma ovarii with rare BRAFG469A mutation
C018	A patient with pituitary macroadenoma and brain abscess: Treatment challenge
C019	Cushing's, PCOS or just the drugs? Difficulties in interpreting dynamic testing of the Hypothalamo-Pituitary-Adrenal axis
C020	Through the Looking Glass: Unravelling an Endocrinopathy through a change in vision.
C021	MDT approach toward Thyrotoxicosis and Carbimazole induced Hepatitis
C022	A case of severe symptomatic hungry bone syndrome, would vitamin D replacement have prevented it?
C023	Mental Health Association with Grave`s Thyrotoxicosis is rare but not uncommon
C024	How is the calcium normal?
C025	Severe hypercalcaemia secondary to CD4+ T cell lymphoma
C026	Substance abuse unmasking the underlying illness
C027	Megacolon in Multiple Endocrine Neoplasia Type 2B: an acute on chronic presentation
C028	Persistent primary hyperparathyroidism cured by diagnostic FNA
C029	The importance of under-treating adrenal failure in order to allow normal adrenal function following unilateral adrenalectomy for Cushings syndrome
C030	A case of hyperparathroidism – jaw tumour syndrome (HP-JTS): parathyroid carcinoma and hungry bones

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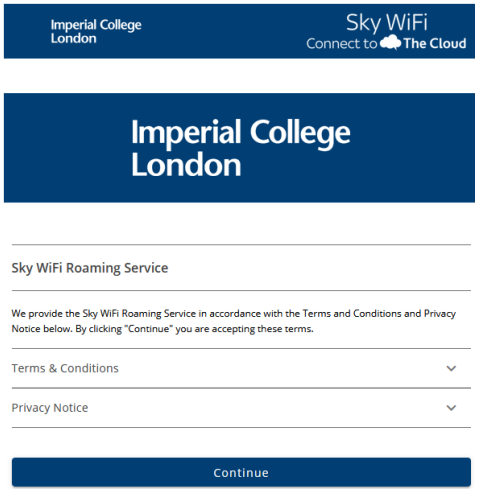

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C001

A retrospective cohort study investigating the role of thyroidectomy in a multi-ethnic cohort of patients with Thyroid Eye Disease (TED).

Daisy Metcalf, Nour Houbby, Natalie Man, Malik Moledina, Vickie Lee. Imperial College Healthcare NHS Trust.

Background:
90% of Thyroid Eye Disease (TED) is associated with Grave's Hyperthyroidism (GH). The benefit of thyroidectomy on TED remains controversial in the peer-reviewed literature. Our aims are to evaluate the indications, and endocrine and TED outcomes for thyroidectomy in our TED cohort.

Methods:
Retrospective collection of demographic data, endocrine diagnosis status, TSH receptor (TRAb) antibody levels and treatment as well as TED parameters (Clinical Activity Score and immunosuppression treatment), indications for thyroidectomy, intra- operative data, complications and outcomes for consecutive patients >18 years attending the MDTED service. Statistical analysis was carried out using SPSS.

Results:
13% patients (75/568) underwent total thyroidectomy of which 51% (38/75) received immunosuppression treatment for TED. Endocrine and eye records were available for 46 patients. 96% (44/46) had Graves' disease and 2 Hashimoto's disease. 98% were female with mean thyroid onset age of 39 years (range 21-72 years). 52% (14/27) were current or ex-smokers. 91% (30/33) were biochemically euthyroid at time of thyroidectomy. Indications for thyroidectomy: 30% (14) unstable control on medical treatment, 17% (8) to improve TED control; 35% (16) post GH relapse, 11% (5) facilitation of pregnancy; 17% (8) declined/allergy/relapse radio-iodine treatment, suspected tumour (n=1) and co-existing primary hyperparathyroidism (n=1). N=11 unknown indication. Histological examination of excised thyroid showed 20% incidence papillary carcinoma (n=6/30). 37% (17/46) experienced no post-operative complications and the most common complications were transient hypoparathyroidism (n=9) and transient hypocalcaemia (n=5). Other complications included voice problems (n=4), permanent hypocalcaemia (n=1), permanent hypoparathyroidism (n=2), neck swelling (n=2), neck pain (n=2), wound infection (n=3) and post-op bleed (n=1). One patient required re-operation.

T4 values recorded in 19 patients significantly increased post-operatively compared to preoperative values (12.8 vs 14.5, *p=0.016). TRAb levels significantly decreased post-operatively (11.45 vs 7.32, *p=0.036) in the 6 patients with pre and post-operative TRAb levels. CAS scores and TSH did not significantly change between pre-thyroidectomy, immediately post thyroidectomy and in the six months post thyroidectomy (p=0.320 and p=0.339 respectively).

Conclusion:
17% of our cohort were recommended for thyroidectomy to improve their TED control. Our study demonstrates a clinically significant reduction in TRAb and increase in T4 levels post-operatively but no significant change in TED parameters.

C002

Addisonian Crisis as first presentation of Thyrotoxicosis.

Tanya Chopra, Nael Alavi, Lydia Nagib, Thomas Beaumont, Asmaa Ali, Shamaila Zaman, Mark Cohen. Barnet Hospital, Royal Free London NHS Trust.

Cushing's syndrome and endogenous steroid production may dampen the hypothalamic-pituitary-thyroid axis and mask autoimmune diseases. We report a 62-year old lady who presented with an Addisonian crisis secondary to new onset thyrotoxicosis, following an adrenalectomy for Cushing's syndrome.

A 62-year-old lady had previously been diagnosed with Cushing's syndrome caused by a 2.8cm benign adrenal adenoma and had undergone a unilateral left adrenalectomy in 2019. She also had a past medical history of ulcerative colitis treated with a total colectomy with a resulting ileostomy, hypertension treated with ramipril, stage 3 chronic kidney disease with Type 4 renal tubular acidosis and previous AV block for which she had a pacemaker inserted recently. She had no significant family history of autoimmune disease. She was on hydrocortisone 10/5/5mg following the adrenalectomy and was also subsequently started on fludrocortisone 100mcg once a day for Type 4 renal tubular acidosis.

She presented to the Emergency Department with a 2-day history of generally feeling unwell with flu-like symptoms and a 1-day history of vomiting. She stated that she was unable to follow the sick day rules and had not received any steroids in the last day. Prior to this, she reported that she had been systemically well in herself; there were no symptoms of weight loss, fatigue, palpitations, change in mood or visual changes. She did notice a slight increase in stoma output and some heat intolerance. On examination, there was evidence of exophthalmos and a lid lag, but examination was otherwise unremarkable.

On admission, she was noted to be profoundly hypotensive and tachycardic. Initial blood tests showed marked hyperkalaemia (6.9mmol/l) and mild hyponatremia (131mmol/l) with a significant acute kidney injury (creatinine 551umol/l and urea 25.1mmol/l) and metabolic acidosis (pH 7.11, base excess –15.1mmol/l and bicarbonate 12.7mmol/l).

She was promptly treated with IV fluids and IV hydrocortisone (100mg initially then 50mg 6 hourly). Medical treatment for hyperkalaemia was commenced; calcium gluconate and insulin/dextrose were given and ramipril was stopped. She was reviewed regularly by the ITU team with a normal CT abdomen and pelvis carried out which ruled out any acute surgical pathology and alternative causes of AKI. She significantly improved with treatment, becoming haemodynamically stable and her serum potassium normalised.

Given findings of tachycardia and exophthalmos, thyroid function tests were sent which confirmed thyrotoxicosis (TSH < 0.01munit/l, T4 41.3pmol/l and T3 6.1pmol/l). Her TSH-receptor antibodies were positive (5.51unit/l), confirming Grave's disease. She was commenced on carbimazole 20mg twice a day and propranolol 40mg twice a day. She continued to improve and was discharged on carbimazole and hydrocortisone. It was felt that prior to admission, the undiagnosed thyrotoxicosis may have resulted in a heightened stress response with increased hepatic metabolism of hydrocortisone[1], precipitating the Addisonian crisis.

In conclusion, treatment of Cushing's Syndrome results in the resolution of hypercortisolaemia which may precipitate the development of autoimmune diseases such as Grave's Disease[2]. In addition, thyrotoxicosis may act as precipitant for Addisonian crisis and therefore, should be excluded if there is clinical suspicion.

References:

1. Fredette, M. E. and Topor, L. S. (2018) 'Graves' Thyrotoxicosis Leading to Adrenal Decompensation and Hyperandrogenemia in a Pediatric Patient with Salt-Wasting Congenital Adrenal Hyperplasia', *Case Reports in Endocrinology*. Hindawi, 2018, pp. 1–3. doi: 10.1155/2018/2359205.
2. Petramala, L. *et al.* (2018) 'Autoimmune diseases in patients with Cushing's syndrome after resolution of hypercortisolism: Case reports and literature review', *International Journal of Endocrinology*, 2018. doi: 10.1155/2018/1464967.

C003

A case of severe hypothyroidism and abnormal liver function post Pembrolizumab therapy.

Bhavini Bhatt [1,2], Rasha Sharafeldin [2], Lubna Etbinah [2], Gideon Mlawa [2].
[1] University College London Medical School.
[2] Queen's Hospital Romford, Barking, Havering & Redbridge Hospitals NHS Trust.

Introduction:
Immune checkpoints regulate immune responses and maintain self-tolerance within the body, functioning through both stimulatory and inhibitory T-cell activity. In cancers, immune checkpoint proteins on malignant cells disrupt the immune response and stimulate cancerous cell growth.

Immune checkpoint inhibitors are a class of immunotherapy drugs which target checkpoint pathways such as programmed cell death-1 (PD-1) and cytotoxic T lymphocyte antigen-4 (CTLA-4). However, the mechanism of these drugs can cause patients to experience immune-related adverse events, affecting different organs with varying severity. A patient with endocrine specific complications as a result of immunotherapy is discussed below.

Case report:
A 65-year-old male presented with a history of T4 N3 M0 squamous cell carcinoma of the right lung, for which he was treated with pembrolizumab. Subsequently, the patient developed immunotherapy induced hepatitis and severe hypothyroidism post immunotherapy. The patient was admitted into hospital with deranged LFTs, including amylase levels reading 367 U/L. Additionally, the patient displayed elevated TSH levels of <100 mU/L and low free T4 levels of 1.1 pmol/L. Inpatient treatment included intravenous methylprednisolone and the discharge treatment plan composed of levothyroxine 50mg daily and prednisolone 60mg daily.

Discussion/conclusion:
This patient experienced hepatoxicity and thyroid disfunction due to an adverse reaction to the immune checkpoint inhibitor. These conditions were successfully managed through corticosteroids and hormone replacement therapy. The patient's TSH and T4 levels have been monitored since this event, with the most recent results indicating stabilised results. From an endocrine perspective, it is evident that there is a need for TSH and T4 levels to be regularly measured in patients on immunotherapy treatments, in accordance with current guidelines, to improve the diagnosis and management of patients presenting with immune induced thyroid disfunction.

C004

Management of the patient with Prolactinoma during pregnancy.

Z Jahanzeb, S Hafeez, O Ishtiaq, U Yousaf, M Kamin, M Jabeen. Shifa International Hospital and Foundation Trust, Pakistan.

Introduction:
Prolactinomas are the most prevalent functional pituitary tumours. Managing prolactinomas during pregnancy can be challenging. The main concern is possible tumour enlargement during pregnancy. This case highlights how preventable complications can arise from a delay in diagnosis and treatment.

Case History:
A-34-year-old female with 23 weeks gestation, presented to our endocrine clinic with intermittent headaches, progressive blurring of vision over 1 month, more over the right eye than the left.

Detailed history revealed that she was treated for primary infertility locally as she was unable to conceive despite trying for last 3 years. She was treated with low doses of cabergoline over last 12-18 months by obstetrics team which was stopped when her pregnancy test was positive. No previous prolactin levels were available for review. She had an MRI brain done locally just prior to conception which demonstrated 1.7 cm pituitary macro adenoma without any compression on optic chiasm.

She was investigated further with MRI pituitary with contrast and full pituitary profile. MRI pituitary showed expanded sella with interval increase in size of the significant pituitary lesion. It measured 1.8 x 2x 3.1 cm (AP x TR CC). Pituitary profile revealed:

Prolactin 3981 ng/ml (4.7-23.3)
TSH 1.82 microIU/ml (0.4-4.2)
Free T4 0.93 ng/dl (0.7-107)
9 AM Cortisol 12.30 mcg/dl (6.2-19.4)
Urine osmolality 247 mosm/kg (random specimen 50-1200).

At this point diagnosis of pituitary prolactinoma was made, surgical option was discussed with patient and neurosurgical team to restore vision. She underwent Trans-sphenoidal surgical removal of the tumour within next few days. Her vision improved significantly post-surgery and the prolactin had dropped to 967 ng/ml. She has been started on Cabergoline 250 mcg twice weekly with close monitoring under obstetric, endocrine and surgical team. Her expected date of delivery is in December 2023**.**

Questions for discussion:
1. Would you manage this patient differently?
2. At what point should surgery be considered an option vs medical treatment?
3. Is it safe to withhold medical treatment for breast feeding?

C005

A potential diagnosis of cerebral salt wasting in absence of acute intracranial pathology.

Jean-Luc Daurat, Robert Connolly, Sagen Zac-Varghese. East and North Herts NHS Trust.

Introduction:
Cerebral salt wasting (CSW) describes a contentious diagnostic entity involving hypovolaemic hyponatraemia and an elevated urine sodium, with a positive clinical response to intravenous fluid therapy. CSW is typically associated with acute cerebrovascular events, most commonly subarachnoid haemorrhage. This case describes a 58-year-old woman found to be acutely hyponatraemic with a raised urine sodium.

Case summary:
A 58-year-old woman presented to the Emergency department following a fall at home. After workup in the emergency department including measurement of serum electrolytes, she was discharged home with no concerning features having been found.

Five days following discharge, the patient re-presented to the emergency department with worsening drowsiness and was found to have a serum sodium level of 119.

Initial treatment in A&E resus involved fluid rehydration. Following 1 L of normal saline, she deteriorated and had a seizure. It was assumed that she had SIADH initially as her response to initial fluid rehydration was a marked clinical deterioration.

CT head revealed no acute intracranial event though a left occipital bone fracture was reported, with agreement from the orthopaedic team for conservative management.

Osmolalities revealed hypotonic serum and hypertonic urine. She was promptly admitted to the intensive care unit (Table 1, pink), and placed on a fluid restriction of <1.5L/day. The serum sodium level showed a small improvement over the following three days before plateauing at approximately 125mmol/L. At this point, a diagnosis of cerebral salt wasting was considered given that the patient had no further improvement. Treatment with 0.9% NaCl was commenced, and the following four days saw recovery of sodium levels to physiological range without complication. Once clinically and biochemically stable, the patient was discharged, and serum sodium levels remained normal at day 11 post-discharge.

Table 1: sodium and fluid balance monitoring throughout inpatient admission

Date	Serum Na (mmol/L)	Urine Na (mmol/L)	Serum Osm (mmol/kg)	Urine Osm (mmol/kg)	Fluid input (mL/24hr)	Urine output (mL/24hr)	Fluid balance (mL/24hr)
24/11	141						
29/11	119	232	255	637			
	121	200	253				
	117						
30/11	118				1916	1840	+74
01/12	120				1022	3100	-2098
	121						
02/12	125				1080	3720	-2640
03/12	124						
04/12	134	30.4	274	261	2500	2400	-100
	132						
05/12	136						
06/12	137						
07/12							
08/12	138						
19/12	143						

KEY: admitted to intensive care unit, admitted to intensive care unit and fluid restricted to <1.5L/day

Discussion:

1. Do the audience consider that cerebral salt wasting was a possible diagnosis in this case? Or that the diagnosis was SIADH that resolved as the patient recovered from the initial head trauma?
2. What significance, if any, do the audience place on the initial urine sodium level of 232?

Learning points:

The key learning point from this case is to monitor the response to treatment and to re-evaluate the initial diagnosis if the response treatment is unexpected.

C006

The Role of Surgery in Managing Hypercalcemia in Pregnancy.

Furhana Hussein, Man-Yan Cheung, Mariana Dram, Belayet Hossain Gideon Mlawa.
Queen's Hospital, Barking, Havering and Redbridge University Hospitals NHS Trust (BHRUT).

Introduction:
The leading cause of hypercalcemia in pregnancy is primary hyperparathyroidism[1]. Hypercalcemia in pregnancy due to parathyroid adenoma may lead to both fetal and maternal complications and therefore surgical intervention (parathyroidectomy) may be required in the second trimester.

Maternal complications may include constipation, nephrolithiasis, pancreatitis, pre-eclampsia and depression[1-2].

Fetal complications may include pre-term delivery, fetal growth restriction, severe neonatal hypocalcemia, hyperparathyroidism, tetany and death[1-2].

Case 1:
A 35 years old 12/40 weeks pregnant lady was admitted with 6 weeks history of nausea and vomiting. Whilst inpatient she had ongoing nausea and vomiting with general lethargy.

Blood test showed: Na+ 137mmol/L, K 3.4mmol/L, urea 2.4 mmol/L, creatinine 61µmol/L, ALP 79 iu/L, ALT 32 iu/L, albumin 36 g/L, bilirubin 10 µmol/L, Corrected ca2+ 3.50mmol/L, phosphate 0.71mmol/L, magnesium 0.70mmol/L, PTH(parathyroid hormone) 15.2 pmol/L, TSH 0.50 mIU/L, Free T4 9.5pmol/L. 24hour urinary calcium was elevated at 17.5 mmol/24 h

Her medications include folic acid 5mg once daily, metoclopramide 10mg three times a day and Pregnacare.

She was initially admitted and treated with intravenous fluids and re-admitted a week later as symptoms persisted with ongoing vomiting. She was discussed in an MDT with the surgical team, and she underwent parathyroidectomy surgery at 14/40 gestation week.

Case 2:
A 33-year-old female was referred to the Endocrinology clinic during her 15th week of pregnancy. She initially complained of body aches, generally feeling unwell and intermittent nausea and vomiting. She visited her GP who performed blood tests including serum bone and thyroid profiles. This revealed a high adjusted serum calcium of 3.27 millimoles/litre (mmol/L), phosphate of 0.79 mmol/L, a high PTH of 9.4 pmol/L, and normal vitamin D level of 83. T (FT4) level of 35.1 pmol/L, (TSH) of less than 0.01 mIU/L. 24-hour urine calcium result was elevated at 11.1 millimoles. She was discussed in an MDT with the surgical team and underwent parathyroidectomy at 19/40 gestational week.

Discussion:
The symptoms of hypercalcemia are similar to non-pregnant patients. Primary hyperparathyroidism may be associated with adverse outcome in the fetus and neonate. Maternal complications rates may be as high as 67% and Fetal complications 80%. Some studies have reported complication of fetal/neonatal mortality of 30% [1].

Management of hypercalcemia in pregnancy due to primary hyperparathyroidism includes medical and surgical options. Medical therapy is limited, and the surgical intervention of parathyroidectomy should be performed in the 2nd trimester. Therefore, it is important to apply an MDT approach involving Endocrinologist, surgeons, and psychological support.

References:

1. Gehlert J, Morton A. Hypercalcaemia during pregnancy: Review of maternal and fetal complications, investigations, and management. Obstet Med. 2019 Dec;12(4):175-179. doi: 10.1177/1753495X18799569. Epub 2018 Dec 11. PMID: 31853257; PMCID: PMC6909300.
2. Appelman-Dijkstra, N.M., Ertl, D.A., Zillikens, M.C. *et al.* Hypercalcemia during pregnancy: management and outcomes for mother and child. *Endocrine* **71**, 604–610 (2021). https://doi.org/10.1007/s12020-021-02615-2

C007

Revisiting the Water Deprivation Test: Unveiling the Significance in Diagnosing Arginine Vasopressin Deficiency.

Yash Akkara, Kavita Narula, Kate Lazarus, Sirazum Choudhury, Niamh Martin, Karim Meeran. Imperial College London NHS Trust.

A 37-year-old gentleman presented to the hospital 20 days post-operatively with severe thirst and polyuria after a recent trans-sphenoidal endoscopic resection of a pituitary macroadenoma, without any pre-operative or peri-operative history of frequent urination or polydipsia. He had been discharged on glucocorticoid replacement (prednisolone 4mg once daily). His initial blood test showed sodium of 144 mmol/L with a normal venous glucose.

The patient was then referred for a water deprivation test. This showed a peak urine osmolality of 334 mOsm/L, with plasma osmolality and sodium reaching 308 mOsm/L and 147 mmol/L respectively. DDAVP was given early due to 3.2 kg of weight loss, following which the patient's urine osmolality reached 653 mOsm/L. An arginine-copeptin test was also performed, showing a below-normal result of 2.6 pmol/L (normal range >3.8).

Following this clinical presentation and investigations, a diagnosis of arginine vasopressin deficiency (AVP-D) was made, and he started desmopressin (2x10 mcg intranasal puffs). At routine endocrine follow-up, he reported he rarely used desmopressin and denied any symptoms of AVP-D. A repeat water deprivation test was performed, which showed first-phase urine osmolality peaking at 725 mOsm/L, with plasma osmolality and sodium reaching 295 mOsm/L and 143 mmol/L respectively. Following desmopressin administration, urine osmolality rose to 989 mOsm/L. In view of this normal response to water deprivation, it was recommended that he discontinued desmopressin.

In patients presenting with acute and unprecedented AVP-D following pituitary surgery, which in certain cases can develop weeks after the initial procedure, it is vital to follow up regularly regarding treatment to identify whether they may have regained their ability to produce arginine vasopressin[1,2]. Unnecessary DDAVP regimens in patients with normal water balance can induce hyponatraemia and other complications[3].

References:

1. Tomkins M, Lawless S, Martin-Grace J, Sherlock M, Thompson CJ. Diagnosis and Management of Central Diabetes Insipidus in Adults. J Clin Endocrinol Metab. 2022;107(10):2701-15.
2. Nemergut EC, Zuo Z, Jane JA, Jr., Laws ER, Jr. Predictors of diabetes insipidus after transsphenoidal surgery: a review of 881 patients. J Neurosurg. 2005;103(3):448-54.
3. McCarty TS, Patel P. Desmopressin. StatPearls. Treasure Island (FL)2023.

C008

Severe Hyponatraemia Leading to the Diagnosis of Acute Intermittent Porphyria.

Diba Debnath, Praveena Vankayalapati, Rabail Mustafa. Wexham Park Hospital, Frimley Health Foundation Trust and West Middlesex Hospital, Chelsea and Westminster Hospital NHS Foundation Trust

A 23-year-old woman with a background of Crohn's disease and anxiety and depression presented to the emergency department with new onset confusion, blurred vision, upper and lower limb weakness and an unsteady gait. She was also experiencing abdominal pain, constipation and vomiting. CT head showed no acute abnormality. In the past week, there had been two previous hospital attendances: the first due to back pain where MRI spine excluded cauda equina, the second due to abdominal pain, constipation and urinary retention with CT abdomen and pelvis showing no acute findings.

On this third presentation, admission blood tests showed severe hyponatraemia with serum sodium of 112 mmol/litre. Given neurological involvement, hypertonic saline was started, and the patient was transferred to the high dependency unit for close monitoring. She was clinically dehydrated due to recent vomiting and continued on 0.9% sodium chloride for twelve hours and medications contributing to hyponatraemia including the proton-pump inhibitor *Lansoprazole* and serotonin and noradrenaline re-uptake inhibitor *Venlafaxine* were switched to alternative agents. Paired sodium studies revealed a low serum osmolality at 242 mOsm/kilogram, urine osmolality of 209 mOsm/kilogram and urine sodium of 35 mmol/litre. Random serum cortisol level of 896 nmol/litre excluded adrenal insufficiency and her thyroid profile was also normal with TSH 0.58 mIU/litre, T4 19.2 pmol/litre and T3 4.1 pmol/litre. Serum sodium improved to 122 mmol/litre after 24 hours then further to 131 mmol/litre after 48 hours and within 72 hours was within the normal range where it remained during the remainder of the admission. Review of historical sodium trends revealed no previous hyponatraemia except for a moderate hyponatraemia of 125 mmol/litre seen two days prior to this admission.

Given recurrent presentation with abdominal pain, new neurological symptoms and acute severe hyponatraemia, a porphyria screen was sent. On day five of the admission the national porphyria laboratory called to relay that a *Porphobilinogen:creatinine* ratio of 53.4 umol/mmol was found, highly suggestive of Acute Intermittent Porphyria (AIP). The patient was started on intravenous *Haem Arginate* under the guidance of clinical biochemistry and discharged after three doses. Further sodium levels since diagnosis and ongoing follow-up have shown no more episodes of hyponatraemia. This case highlights how the relatively common electrolyte disturbance of hyponatraemia can unmask rare diagnoses such as AIP; especially given low serum sodium is present in around one-fifth of patients experiencing an acute porphyria attack. The mechanism for hyponatraemia is usually driven by inappropriate anti-diuretic hormone secretion.

C009

Multidisciplinary Management of Multiple Endocrine Neoplasia Type 1 (MEN1): A Case Report.

Vivek Ramburuth, Andrew Lim, Jeyanthy Rajkanna. Peterborough City Hospital, North West Anglia Foundation Trust.

Multiple endocrine neoplasia type 1 (MEN1) is a genetic disease characterised by a predisposition for parathyroid, pancreatic neuroendocrine, and pituitary tumours. We report a patient with MEN1 who presented with recurrent urinary tract stones that started at the age of twenty-two.

Blood investigations revealed severe hypercalcaemia and a markedly elevated serum parathyroid hormone. A technetium-99m sestamibi scan demonstrated focal uptake in the left lower of the thyroid, suggestive of functional parathyroid adenoma. Given the onset of recurrent urolithiasis and parathyroid adenoma at a young age, MEN1 syndrome was suspected. This was confirmed by a positive genetic test for the MEN1 mutation.

He was thereafter screened for MEN1-associated tumours and investigations lead to the detection of a probable pancreatic neuroendocrine tumour. This was based on radiological findings of a 10 mm pancreatic lesion and biochemical findings of raised serum gastrin and chromogranin A levels. In terms of management, he underwent a left hemithyroidectomy and parathyroidectomy. Regarding the pancreatic lesion, he underwent endoscopic ultrasound-guided radiofrequency ablation.

Postoperatively, his calcium levels normalised, and he was offered a program of combined clinical, biochemical and radiological screening for early detection of MEN1-associated tumours. Predictive genetic testing was also offered to his first-degree relatives.

This case highlights the complexity of managing patients with MEN1 and the importance of a multidisciplinary team consisting of endocrinologists, gastroenterologists, oncologists, endocrine surgeons, radiologists, and clinical geneticists.

C010

Men-2A Challenges in Management – A Developing Country Perspective.

Shahroz Azhar[1], Zainab Atiq[2], Sidra Safdar[3], Ali Raza Naqvi[4.]

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A 27 yearold female, presented to secondary care hospital with palpitations, intermittent headache and hypertensive urgency since 1 year, managed as per guidelines in emergency . Investigations were requested to rule out causes of secondary hypertension. Doppler abdominal ultrasound showed Bilateral adrenal hyperplasia with normal flow indices and 24hr urinary free metanephrines were raised 1076.92 ug/day (normal less than 350 ug/day). She was then referred to PKLI hospital Urology and Endocrinology Department. In addition to above symptoms, she also informed that her mother had thyroid disorder and she died of heart disease. Her maternal uncle also had bilateral adrenalectomies for phaeochromocytoma fifteen years ago. She is married and is a mother of two children, youngest is 11 months old.

Investigations revealed Normal thyroid function tests, Ultrasound neck showed normal thyroid gland, Raised calcium 12.5mg/dl (normal 10.5mg/dl), Raised PTH= 318.3 Pg/ml (normal 68.3pg/ml) , Raised calcitonin 37.5 pg/ml (normal less than 5.8) and normal prolactin 6.2 ng/ml (normal 27 ng/ml),serum 25-0H VIT D 45.8ng/ml (normal 20.01—100 ng/ml). Parathyroid planner imaging with SPECT showed scintigraphic evidence of hyperfunctioning parathyroid tissue at the inferior pole of right lobe of thyroid gland. CT with adrenal protocol showed right adrenal gland is enlarged and contains an enhancing lesion measuring 41mm x 28mm with precontrast attenuation of 46HU, attenuation arterial phase enhancement of 134 HU and absolute washout of 40%,similarly another enhancing lesion measuring 20x18mm is noted arising from left adrenal gland with non-contrast attenuation of 49HU,arterial phase attentuation of 104 HU and absolute washout of 58% with no locoregional lymph nodes. PET-CT-DOTA scan was not done due to financial constraints and technical difficulties but discussed with radiologist they were certain that it was bilateral phaeochromocytoma.

Since patient was symptomatic with biochemical and radiological evidence of Phaeochromocytoma, she was started on Alpha and Beta blockers before surgery. Her case was then discussed in MDT meeting, recommended for bilateral adrenalectomies, genetic testing, total thyroidectomy and parathyroidectomy. Perioperative and post operative management was done as per recommended guidelines. She then underwent Bilateral Robotic adrenalectomy and started on replacement corticosteroids (10mg +5mg +5mg) and Fludrocortisone (100mcg/day). On her followup, we have discussed her regarding possible thyroidectomy and parathyroidectomy. Her clinical and biochemical profile are consistent with MEN-2A, unfortunately we could not able to perform genetic studies due to financial constraints. Her followup up urinary free metanephrines were within normal limits at 7th post op day. She was reviewed by our local psychiatrist.

Today i received the histopathology report of bilateral adrenalectomy sample, macroscopic cut section reveals lobulated tumour with yellow appearance arising in both adrenal glands. Microscopic findings suggests encapsulated neoplasm arising in the adrenal gland

and composed of cells with basophilic cytoplasm and variable salt and pepper nuclei, the cells are polygonal with numerous areas of spindled appearance, some cells have markedly pleomorphic nuclei ,these cells are arranged in Zellbellen pattern with some diffuse areas of capsular invasion is seen however there is no extension into extraadrenal tissues or vessels,no areas of necrosis is seen. Immunohistochemistry showed :

Synaptophysin : Positive

Chromogranin :Positive

Ki 67: 1-2%

S100:Highlights supratenticular cells

Reticulin :Highlights Zellbellen pattern

Phaechromocytoma of adrenal gland scaled score : 6

1. Now question is should we go for total 4 gland parathyroidectomy or do just right inferior selective parathyroidectomy?
2. This case shows the challenges of managing complex endocrinology disorder in developing countries.

C011

Thyrotoxicosis with Immune Mediated Neutropenia.

Rabia Arfan, Ben Turner. Basingstoke & North Hampshire Hospital.

Case history:
46 years old man was referred to Endocrine in Feb 22 with symptoms of weight loss, chest pain and palpitations, he was noted to have hyperthyroidism on blood result with FT3>30.8 mu/l (3.1-6.8), FT4 82 pmol/l (11-22) and suppressed TSH. He said that his symptoms were triggered by his covid 19 booster vaccination in Nov 21, after which he was investigated for his chest pain with negative CTPA and was diagnosed with pericarditis.

It was not until Feb 22 when his TFTs were checked and was referred to Endocrine. He had marked tremors with mild goitre on examination with no eye dysthyroid signs. He was started on carbimazole 40 mg once a day. His TSH receptor antibody came back raised at 2.7 IU/L (0-0.4) with thyroid peroxidase antibody of >1300 u/ml (0-60). He was also noted to have raised ALT since Jan 22 with normal bilirubin and ALP which after initial rise to carbimazole, started settling down with negative liver autoimmune screen. His corrected calcium was mildly raised at the time of diagnosis but then became normal. He continued to report itching of his skin since his covid vaccine which was mainly after shower and did not respond to improvement in his thyroid function. His Thyroid function gradually started improving but repeat blood test in May showed neutropenia of 0.76 which was normal before his treatment therefore his carbimazole was stopped with a diagnosis of carbimazole induced neutropenia although he was asymptomatic for neutropenia.

For ongoing hyperthyroidism, he was urgently referred to nuclear medicine department in Southampton Hospital for Radioiodine therapy which he received on 9th May 22. His thyroid function test was monitored via nurse led virtual thyroid clinic locally. He developed swollen eyes after RI therapy and was reviewed by ophthalmology, he did not have proptosis or restriction of ocular motility, he was discharged from their clinic. His Thyroid function stayed slightly above normal with FT3 of 6.6-8 pmol/l and FT4 of 22.2 pmol/l with suppressed TSH few months after radioiodine therapy and his neutropenia persisted when he was referred to haematologist who investigated him with immunoglobulins/electrophoresis/Jak 2/Bone marrow aspirate and all of his test came back negative with conclusion that he has got unexplained neutropenia which is likely immune in origin.

He was also diagnosed with aquagenic pruritis which stays for an hour after shower, he was given antihistamine and topicals for his condition. There was discussion about need for GCSF in case of any procedure/surgery in view of neutropenia. Repeat radioiodine therapy was also discussed with him as his TFTs stayed above normal but conservative approach was taken with stable thyroid function, his symptoms were managed with propranolol along with ongoing monitoring of TFTs. Finally, 17 months after radioiodine therapy he became euthyroid (TSH 1.5 mu/l and normal FT4/FT3) and his Neutrophil improved to 1.4.

Discussion:
It was interesting case of thyrotoxicosis triggered by severe immune response which warranted multidisciplinary approach for management. Although he was treated as carbimazole induced neutropenia but it did not resolve for many months after stopping carbimazole and finally settled with resolution of thyrotoxicosis. It is not clear whether his aquagenic pruritis was associated with Graves but certainly was part of autoimmune response.

C012

Recurrent thyrotoxicosis of pregnancy.

Rabia Arfan. Basingstoke & North Hampshire Hospital.

Case History:
23 years old lady was referred to Endocrinology clinic with hyperthyroidism in Feb 2010, she was 16 weeks pregnant when reviewed in clinic and had clinical symptoms of hyperthyroidism including palpitations and tremors. She denied symptoms of neck pain or swelling. This was her first pregnancy; she did not have prior history of thyroid dysfunction or family history of thyroid disorder. On examination she had mild lid lag with tremors to outstretched hands and a heart rate of 120 beats/min. She had a non-tender thyroid goitre on palpation with no bruit. Her reflexes were brisk with no myopathy. Her blood result showed TSH <0.03 mu/l, FT4 43 pmol/l and FT3 of 16 pmol/l. She was started on Propylthiouracil (PTU) 100 mg BD with ongoing follow up in Endocrine clinic. Her TSH receptor antibody and thyroid peroxidase antibody came back negative. She was monitored during pregnancy, and she needed PTU therapy throughout her pregnancy, dose of which was reduced as per her response. She continued her PTU 50 mg on alternate days for 3 months post-partum when it was stopped, and she remained euthyroid. She became pregnant again in June 2011 with clinical and biochemical hyperthyroidism when she was managed with PTU again during her pregnancy and 3 months post-natally. Her thyroid function stayed normal until she became pregnant again for her third baby in June 2017 with same course of management for thyrotoxicosis of pregnancy. Her ESR stayed 15-16 during pregnancy and went back to normal (2) once her thyroid function improves after her pregnancy. Her thyroid ultrasound was consistent with thyroiditis but did not have thyroid uptake scan. She was not keen for thyroidectomy and radioactive iodine was not an option with her young Kinds. Her iodine intake was not excessive. She was discharged when was re-referred last year with her fourth pregnancy and hyperthyroidism but unfortunately suffered from missed miscarriage in early weeks before she was seen in Endocrine clinic. She has got 3 children and is currently under our joint antenatal clinic for 5th Pregnancy and hyperthyroidism. She is aware of course of her treatment.

Discussion:
The most common cause of thyrotoxicosis in pregnancy is gestational transient thyrotoxicosis (GTT), which occurs from the stimulatory action of human chorionic gonadotropin (HCG) on the TSH receptor. Other aetiologies to consider in the differential diagnosis of thyrotoxicosis during pregnancy include subtypes of overt hyperthyroidism, such as Graves ‘disease, toxic multinodular goitre, and toxic adenoma, as well as thyroiditis and exogenous thyroid hormone use. In addition, a rare cause of thyrotoxicosis during pregnancy is trophoblastic disease. This is a case of recurrent thyrotoxicosis of pregnancy due to thyroiditis needing treatment during pregnancy and early postnatal period.

C013

Protracted hypocalcemia After the Administration of Denosumab in a Patient with Prostate Cancer with bony metastasis.

Saira Yousaf, Peter Winocour, Sagen Zac-Varghese. Lister Hospital, East and North Hertfordshire NHS Foundation Trust.

Background:
Denosumab is a potent antiresorptive agent used for the treatment of osteoporosis and the prevention of skeletal-related events in patients with bone metastasis from solid tumors including prostate cancer. It can be associated with severe hypocalcemia requiring prolonged hospitalization for intravenous calcium treatment.

Case report:
We present the case of a 67-year-old gentleman who was diagnosed with micro acinar adenocarcinoma of prostate Gleason score 4+5=9. Bone scan confirmed extensive bony disease involving hip, spine, pelvis, and right shoulder. He was commenced on androgen deprivation therapy (Abiraterone) and denosumab 60mg subcutaneously every 2 weeks. He had blood tests eight days after the first dose of denosumab which showed low calcium and phosphate. Hence, he was referred to acute medical department with severe hypocalcemia necessitating a prolonged hospitalization. On admission corrected serum calcium was 1.9 mmol/L (reference range 2.20-2.60) and low phosphate levels of 0.33 mmol/L (reference range 0.8-1.50). Alkaline phosphatase was high at 3002 IU/L (reference range 44–147) and Vitamin D3 levels were adequate at 56.7 ng/mL (adequate above 50 ng/mL), magnesium levels were normal at 0.98 mmol/L (reference range 0.7-1.00) and PTH levels were high at 42.29 pmol/L (reference range 1.6-6.9). He required daily intravenous calcium infusion along with phosphate replacement for 9 days. He was discharged on oral calcium carbonate/colecalciferol (1500mg/400IU) three times a day, and 1 alfacalcidiol 0.5 mcg daily. He re-presented 6 days later with peri-oral tingling and blood suggested recurrent hypocalcemia and hypophosphatemia, hence had further intravenous calcium infusions and then continued to have intravenous calcium infusions weekly for another few weeks. He was later closely followed up in endocrine clinic to monitor calcium levels which normalized in approximately 6 months.

Conclusion:
This case highlights the fact that patients with uncontrolled osteoblastic skeletal metastases from prostate cancer, denosumab treatment can lead to severe, potentially life-threatening hypocalcemia. Denosumab associated risk of hypocalcemia can be reduced by ensuring the adequate control of underlying disease activity and optimizing modifiable risk factors which include serum calcium, vitamin D, renal failure, and bone turnover. Patients receiving denosumab should be appropriately counselled about symptoms of hypocalcemia and offered calcium and vitamin D supplementation prior to receiving Denosumab with dose adjustments based on biochemistry post infusion.

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C014

Should we keep treating all endocrine conditions in end of life?

A Karaca, B Mahamud, A Mohamed, L Etbinah, Z Khan, G Mlawa. Barking, Havering and Redbridge University Hospitals NHS Trust.

Background:
Decisions regarding endocrine care at the end of life can be complex and challenging. What should be the goals of this care, when and where appropriate to deprescribe or cease the treatment?

Here we present our patient with vasopressin insufficiency (VI) (previously known as diabetes insipidus) due to metastatic lung cancer in palliative care, and our experience in the approach in her last days of life.

Case summary:
A 55-year-old female patient presented to A&E with fever, diarrhoea and abdominal pain. As she was feeling unwell prior admission, she missed her regular desmopressin tablets for few days, therefore, her sodium levels at admission were found to be 188mg/dl, CRP: 394.

Antibiotics and supportive treatments were commenced. As well as strict fluid balance monitoring.

She was started on iv desmopressin and her sodium levels were decreasing, however, next days she developed fluctuating confusion, following that she deteriorated due to sepsis and didn't respond to medical treatment.

Medical team decided on end of life care. However, ceasing desmopressin raised a question, was it ethical?

We did not find any guidance on VI treatment in the end of life and we knew that considering her hypernatremia, withdrawal of this medication could accelerate her death. So, we decided to keep the medication. She passed a week later.

Discussion:
Unfortunately, there is no national guidelines or advice specific to management of VI in the end of life. However, tailoring treatment according to patients' wishes and condition might be appropriate, because without desmopressin, patients with VI likely to die with 'endocrine-driven mechanism of death' within hours or days.

This hormone is life-sustaining and likely to provide dramatic symptom relief, unlike growth hormone and gonadotropins, which may be ceased in the end of life.

We suggest that patients with VI, especially, with severe hypernatremia can be treated with desmopressin in the end of life, but there is no accepted consensus on this.

Keywords; Vasopressin insufficiency; End of life;

Reference:
1. Management of endocrine conditions at the end of life. Anne de Bray, Jon Tomas, Neil Gittoes, Zaki Hassan-Smith. https://doi.org/10.12968/hmed.2020.0096

C015

Thyroid storm and NSTEMI: a rare and dangerous association.

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1. Barking, Havering and Redbridge University Hospitals NHS Trust
2. University College London (UCL) Medical School

Background:
Thyroid storm is a rare condition with prominent cardiovascular manifestations including tachycardia, atrial fibrillation, heart failure, and myocardial infarction (MI). The condition is frequently overlooked and under-recognized in emergency departments due to its rarity.

Case summary:
A 51-year-old female, previously diagnosed with Graves disease, presented with epigastric pain, vomiting and chest pain. Following hospital admission, she was found with rapid atrial fibrillation (AF), ST-segment depression and elevated troponins; 129 (<14ng/L) concerning for non ST elevated MI (NSTEMI).

Hence, she was seen by cardiologist and started on dual anti platelet therapy (DAPT). Echocardiography demonstrated normal systolic function.

Thyroid function tests at admission showed profound elevated thyroid hormone, and suppressed thyroid-stimulating hormone consistent with thyroid storm (TSH<0.01, fT4>100(11.9-21.6))

Following initiation of propylthiouracil, stress-dosed steroids and beta blockers treatment her rapid AF and ST-depression and related symptoms were resolved. Her thyroid hormones were dropping as well as her troponin levels.

She was discharged on medical treatment with a 4 week follow up with endocrinology and cardiology clinic.

Discussion:
New rapid AF and high troponin levels are a rare presentation of thyroid storm and should be suspected in patients with thyroid disease background. In literature there are cases with thyroid storm initially presented as STEMI and NSTEMI, however, with normal coronary arteries following percutaneous coronary intervention. As thyroid storm, if unrecognized, is often fatal, prompt diagnosis and treatment are life saving.

Keywords: Thyroid storm; Myocardial infarction; NSTEMI;

Reference: Brown J, Cham MD, Huang GS. Eur Heart J Case Rep. 2020 Nov 12;4(6):1-5. doi: 10.1093/ehjcr/ytaa414. PMID: 33442653; PMCID: PMC7793194.

C016

A rare coexistence of Acromegaly with metastatic Adrenocortical carcinoma.

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Adrenocortical carcinoma (ACC) is a rare endocrine malignancy often with an unfavourable prognosis. The coexistence of acromegaly and ACC is quite rare. Here we describe a case of metastatic ACC in a patient who had Acromegaly treated with surgery, radiotherapy, and medical management.

A 38-year-old patient was referred to endocrine department in 2018 to investigate for change of facial appearance and he was biochemically confirmed as having Acromegaly with 33 mm pituitary macroadenoma with bilateral cavernous invasion. He had Transsphenoidal adenectomy and histology revealed somatotroph adenoma of partially granulated type with Ki 67 index of 3-5 %. Post-operatively, there was biochemical recurrence of acromegaly and panhypopituitarism with residual tumour in left cavernous sinus. Therefore, he was commenced on Lanreotide 120 mg monthly injections. His IGF1 failed to control on Lanreotide, and he was offered 25 sessions of pituitary radiotherapy in 2019. Subsequently he was started on Pegvisomant injections in 2021 due to suboptimal IGF1 control and IGF1 was well controlled.

2 years later in 2023, While he is on Pegvisomant he complained progressive lower limb swelling and weight gain with deranged liver function tests. Pegvisomant, Lanreotide and hydrocortisone were discontinued. Detailed evaluation revealed non supressed plasma cortisol (1593 nmol/L) and very high urine cortisol (>5000 nmol/L) and supressed ACTH. Abdominal imaging revealed right adrenocortical mass with metastasis, Inferior Vena cava tumour thrombus on with bilateral pleural effusion and ascites. He was initially planned for surgical excision followed by aggressive chemotherapy, however as his general condition deteriorated. A repeat CT scan of the adrenal showed rapid tumour progression. MDT agreed that the case was inoperable and palliative care is the best option. He was initiated on Mitotane and Methyropane with Hydrocortisone cover. He was initiated on Clexane for IVC thrombus, unfortunately ended up with tumour bleeding resulted further deterioration and death. Extensive evaluation for genetic studies revealed negativity including AIP, MEN1, CDC73, CDKN1B, PRKAR1A, RET, VHL.

Acromegaly is associated with a greater morbidity and higher incidence of tumours, possibly due to the permissive role of elevated GH and IGF-I levels. Adrenal lesions seem more frequent in acromegaly than in the general population, but no single factor (GH/IGF-I levels or disease duration) predicts them. Certain genetically inherited conditions are associated with aggressive pituitary tumours and ACC. Negative results of frequently mutated gene profile opens path for more research on novel genetic associations.

C017

A challenging case of metastatic struma ovarii with rare BRAF^{G469A} mutation.

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[6] Head & Neck Surgical Oncology Services, Guy's and St Thomas' NHS Foundation Trust

In June 2022, a 63-year-old woman was referred to gastroenterology on the 2WW pathway for urgent assessment of a six-month history of abdominal bloating, dysphagia and reflux symptoms. She had a background of right-sided salpingo-oophorectomy (2008), a benign breast lump being followed up with serial mammograms and was prescribed omeprazole 40mg OD. Upper GI endoscopy was normal apart from mild reflux oesophagitis.

A contrast CT abdomen and pelvis demonstrated a 2cm nodule medial to the caudate lobe of the liver and a 1.5cm pleural-based nodule at the right lung base. Following discussion in the Upper GI MDT, a liver MRI further characterised the 2x2cm lesion separate to the caudate lobe; she underwent an endoscopic biopsy of this lesion which was suggestive of metastatic thyroid carcinoma (positive immunostaining for TTF-1 and thyroglobulin). Both FDG then DOTATATE PET CT imaging revealed low-grade but focal uptake in a para-oesophageal lymph node and in soft tissue medial to the caudate lobe. No further DOTATATE avid nodules were seen elsewhere – the right lower lobe lung nodule did not accumulate DOTATATE and there was no evidence of uptake in the thyroid. Serum thyroglobulin was 43.1 µg/L with no interfering anti-thyroglobulin antibody. Thyroid USS and FNA showed a benign 6x8mm U2 nodule, confirmed as Thy2.

Her case was then discussed in the central thyroid cancer MDT where a diagnosis of struma ovarii papillary thyroid carcinoma was considered; the outcome included a recommendation that histology from her previous gynaecological surgery in 2008 be obtained. Review of histology from the right ovary and fallopian tube confirmed a large cystic teratoma (weighing 1368 gm) with well-formed thyroid tissue of fetal origin which showed thyroglobulin and TTF-1 marker positivity on immunohistochemistry.

In November 2022, the patient underwent resection of the mediastinal node via VATS, followed by an exploratory laparoscopy and laparoscopic right crus lymph node excision in December 2022. Histopathology from the para-caval node was similar to the previous atypical thyroid lesion within the mature teratoma of the right ovary, consistent with a diagnosis of metastatic struma ovarii.

Although the metastatic disease has progressed slowly, after careful consideration the patient had a total thyroidectomy with level VI neck dissection in February 2023 (no evidence of neoplasia on histology), followed by 3.7 GBq 131-Iodine in May 2023. She has now been established on Levothyroxine 125mcg OD, and close surveillance is ongoing. A specimen sample from the right crus metastasis was sent for both DNA and RNA next-generation sequencing, which detected a BRAF (Gly469Ala) variant.

Discussion:
Struma ovarii is a rare ovarian germ cell tumour and comprises only 1.4-2.7% of ovarian tumours. 5-15% of teratomas contain thyroid tissue. The vast majority of struma ovarii are benign, with malignant disease seen in fewer than 5-6% of cases – papillary carcinoma being the most common. The $BRAF^{G469A}$ mutation in this case has been reported only once previously. As struma ovarii is rare, MDT working is vital especially as consensus guidelines on the management of metastatic disease are lacking, and relatively little is known about the molecular profile of these tumours and resultant effects on disease severity and progression.

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C018

A patient with pituitary macroadenoma and brain abscess: Treatment challenge

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1. Benjamin Mkapa Hospital
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Macroadenomas are often asymptomatic, however, the common clinical manifestations include galactorrhoea and menstrual abnormalities. Although rare, these tumors have the potential to exhibit invasive behavior, penetrating the skull base and leading to subsequent thinning and bone defects. The bone defects serve as the direct pathway of pathogens to the brain.

A 33-year-old woman presented with an 18-year history of amenorrhoea which was noted 3 years after menarche. Three weeks before admission, she started to experience headaches, and body malaise with subsequent progressive weakness of the right upper and lower limbs, confusion, convulsions, and loss of consciousness. She had the normal visual field. Following laboratory investigations, she was found to have elevated prolactin levels with low LH and FSH levels. The brain MRI revealed the presence of pituitary macroadenoma, brain abscess, and sphenoid sinusitis. Serum electrolytes and other anterior pituitary hormones were within normal ranges. She was given IV dexamethasone and carbamazepine. The abscess was successfully evacuated, and she was kept on Meropenem and vancomycin while awaiting culture results.

Additionally, she has been prescribed cabergoline at a dosage of 0.25mg twice weekly. Currently, her right-sided weakness and headache have improved, and she can now walk without support.

C019

Cushing's, PCOS or just the drugs? Difficulties in interpreting dynamic testing of the Hypothalamo-Pituitary-Adrenal axis.

Ella Davidson, Sophie Jones, Vassiliki Bravis. Imperial College Healthcare NHS Trust.

Initial Presentation (2019):
A 41-year woman was found to have a pituitary lesion on a CT Head performed for investigation of memory problems. She reported depression, undetermined weight gain and memory problems for several years and amenorrhoea since the removal of her Mirena coil. Her medical history included severe depression and fibroids. She was taking amitriptyline, pregabalin, sertraline, tapentadol and tramadol. She was normotensive and normoglycaemic, without any examination findings suggestive of pituitary hormone excess. She weighed 80kg (BMI 32.8kg/m^2). A baseline 9am pituitary profile is below and was normal, but she failed overnight and low-dose dexamethasone suppression tests. A late night salivary cortisol measurement was raised but 24hr urinary free cortisol (UFC) was normal. An open MRI Pituitary demonstrated a macroadenoma of the pituitary stalk. She was discussed in the Pituitary MDT; a putative diagnosis of ACTH-dependant Cushing's disease was made, and Inferior Petrosal Sinus Sampling (IPSS) was planned.

Paired Cortisol & ACTH (random)	333 nmol/L (160-550) & ***39.2 ng/L*** ↑		**HPA axis**	
	407nmol/L (160-550) & ***53ng/L*** ↑		Overnight DST 9am cortisol	***332nmol/L*** (<50) ↑
9am Cortisol	407 nmol/L (160-550)		ACTH	Not done
9am ACTH	***53 ng/L*** (<30) ↑		Low dose DST cortisol T=48	***349 nmol/L*** (<50) ↑
Prolactin	352 mU/L (100-500)		ACTH	Not done
LH	2.8 U/L (2-14)		24hr UFC	192 nmol/day (50-270)
FSH	7.2 U/L (1.5-10)		Late night salivary cortisol	***4.6 nmol/L*** (<2.6) ↑
IGF-1	23.9 nmol/L (7.5-35)		Late night salivary cortisone	***22.5 nmol/L*** (<18) ↑

Post-operative Course (2020-2021):
Investigations were interrupted due to the COVID-19 pandemic. The patient did not attend IPSS, and travelled to Iran where she underwent pituitary surgery, which was reported to have removed an uncomplicated adenoma. Unfortunately, no operation report, histology or slides are available. Post-operatively, in the UK, a short-synacthen test showed baseline ACTH 29.7 ng/L and cortisol 268 nmol/L, and peak cortisol 670 nmol/L. 3 UFC measurements were normal, demonstrating biochemical cure of her Cushing's disease.

Post-op open MRI showed a thickened infundibulum, probably representing ectopic pituitary tissue. However, her symptoms of depression and amenorrhoea persisted. Clinically, she still did not have examination features of Cushing's syndrome, and her weight remained stable. HPG axis testing demonstrated undetectable oestradiol with low gonadotrophins, which rose adequately in response to a Gonadotrophin simulation test, suggesting functional hypothalamic amenorrhoea. A diagnosis of PCOS was also considered and she commenced metformin.

HPA axis			**HPG axis**	
24 UFC	211 nmol/day (<270)		LH	***0.8 U/L*** (2-14) ↓
24 UFC	190 nmol/day (<270)		FSH	1.6 U/L (1.5-10)
24 UFC	193 nmol/day (<270)		Oestradiol	***<100 pmol/L*** (>190) ↓
Short Synacthen Test Cortisol T=0	268 nmol/L		SHBG	***22 nmol/L*** (30-100) ↓
T=60	670 nmol/L (>550)		AMH	1.5 pmol/L
SST baseline ACTH	29.7 ng/L (<30)		GnRH Test T=60	LH 10.7 U/L (>10) FSH 4 U/L (>2)

Further follow-up (2022):
She was referred for a second opinion to the Reproductive Endocrinology clinic. Testing one year later showed a high midnight cortisol and failure to suppress on a low dose dexamethasone test. HPG axis testing showed a raised DHEAS, now detectable oestrogen, and no LH:FSH imbalance. A pelvic ultrasound demonstrated normal size ovaries, and adrenal imaging demonstrated a benign-looking 11mm nodule of the left adrenal. Supra-clavicular fat pads, central adiposity, proximal myopathy and facial hirsutism were noted, but there was no bruising or striae, and her weight had fallen to 75kg (BMI 30.8kg/m^2). Visual field testing remained normal. The case was discussed in the adrenal and pituitary MDTs and the possibilities of recurrence of Cushing's disease, PCOS, and an adrenal source of hyperandrogenism were all considered.

HPA axis			**HPG Axis**	
9am Cortisol	251 nmol/L (160-550)		LH	1.0 u/L (2-14) ↓
9am ACTH	27.5 ng/L (<30)		FSH	2.3 u/L (1.5-10)
Midnight cortisol	***199 nmol/L*** ↑		Oestradiol	336 pmol/L
Inpatient LDDST T48 cortisol	***172 nmol/L*** (<50) ↑		DHEAS	***11.3 umol/L*** (0.8-6.9) ↑
Midnight salivary cortisol	***2.7 nmol/L*** ↑ 2.1 nmol/L (<2.6)		SHBG	***18 nmol/L*** (30-100) ↓
Midnight salivary cortisone	14.7 nmol/L 15.1 nmol/L (<18)			

2023:
The patient subsequently revealed that she had been taking hormone-replacement-therapy (HRT), obtained in Iran. She returned to clinic in 2023 having stopped HRT, amitriptyline, tapentadol and tramadol (remaining on sertraline 50mg OD only). She displayed leg muscle wasting and a moon-face, but her weight continued to fall to 67kg (BMI 27.5kg/m^2). She had developed pigmented cystic acne lesions on her face.
All investigations were repeated: On low-dose dexamethasone suppression test, cortisol failed to suppress but DHEAS declined and 24hr urinary free cortisol measurements were suppressed, reassuring us against a malignant adrenal tumour or recurrence of Pituitary Cushing's. Now off HRT, a repeat pelvic ultrasound showed an enlarged left ovary, and oestradiol and Testo:SHBG ratio were raised, suggestive of likely PCOS. CT pituitary showed no interval change, with a persistent mild thickening of the pituitary infundibulum. Midnight cortisol levels are still pending.

Following a low-dose dexamethasone suppression test:			**HPG Axis**	
Baseline ACTH	***36.8 ng/L*** (<30) ↑		LH	3.3 U/L (2-14)
T=0 cortisol	481 nmol/L (160-550)		FSH	2.4 U/L (1.5-20)
T=48 cortisol	***300 nmol/L*** (<50) ↑		Oestradiol	1585 pmol/L
24hr UFC	76 nmol/day <18 nmol/day (<164nmol/day)		SHBG	21 nmol/L (30-100) ↓
DHEAS T=0	***12.6 nmol/L*** (0.8-6.9) ↑		Testo:SHBG ratio	7.1 (1-6) ↑
DHEAS T=48	***7.7 umol/L*** (0.8-6.9) ↑			

Conclusions:
We postulate that psychotropic and opiate medication caused suppression of the HPG axis at the hypothalamic level. The removal of these, and stopping of HRT, have unmasked biochemical and ultrasound evidence of likely PCOS. We postulate that severe depression continues to cause false positive cortisol suppression tests.

Questions for Discussion:
1. Did this patient ever have Cushing's disease?
2. Do the tests support a recurrence of Cushing's disease?
3. Did the patient always have PCOS?
4. How would you proceed?

Take-home Messages:
This case highlights the importance of doing the right endocrine tests at the right time and in the right sequence. The lack of IPSS caused significant diagnostic uncertainty following surgery because we were never completely confident of the initial diagnosis of Pituitary Cushing's disease. There are ACTH results not done at the time of dynamic endocrine testing, which has created a weakness in the results narrative. The importance of a detailed drug history when performing endocrine testing is also highlighted by the differences in HPG axis testing and US pelvic interpretation when the patient was on hormone-replacement therapy. Opiates and psychotropic medication can have substantial effect on the HPA and HPG axis.

C020

Through the Looking Glass: Unravelling an Endocrinopathy through a change in vision.

Mariana Nalmpanti, David Hope, John Vekinis, Janmohamed, Wladyslaw Gedroyc, Rashpal Flora, Florian Wernig, Aimee Di Marco, Rochan Agha-Jaffar**.** Imperial College Healthcare NHS Trust.

A 19-year-old male with no past medical history was referred to the medical team by the ophthalmology team due to a 7-day history of reduced visual acuity and optic disc appearances suggestive of grade 4 hypertensive retinopathy. On assessment he had signs of malignant hypertension (blood pressure 220/99mmHg) with associated tachycardia. Subsequent metanephrine levels were significantly elevated (Normetadrenaline: 150,256pmol/l, Metadrenaline 8,335pmol/l, 3-methoxytyramine 1314pmol/l). An overnight dexamethasone suppression test showed an unsuppressed morning cortisol of 439nmol/l; Imaging demonstrated an 11cm isolated T2 hyperdense left sided adrenal lesion, with appearances consistent with a malignant phaeochromocytoma.

A staging Ga-68 DOTATE scan confirmed this and excluded distant disease.
The case was discussed at the Neuroendocrine Tumour MDT and was referred to Endocrine surgery team. The patient was jointly managed by endocrinologists and surgeons in clinic and ambulatory care, initially with phenoxybenzamine 10mg once daily, which was slowly up-titrated based on blood pressure response to a daily dose of 40mg daily (divided doses): beta blockade was later initiated. The patient subsequently underwent a successful retroperitoneoscopic left-sided adrenalectomy within twelve weeks of his initial referral.

One month post-operatively, the patient was well with no residual symptoms and urinary metanephrines had normalised. Histology of the lesion revealed tumour with a nested architecture with polygonal lesional cells showing mildly pleomorphic round/oval nuclei. Immunohistochemistry showed that the lesional cells were positive for synaptophysin, chromogranin and CD56 with a Ki67 index of 1%. Extra-adrenal extension or vascular invasion could not be reliably assessed and therefore the adrenal mass had a PASS score of 6. Genetic panels were negative (SDHB, SDHD, VHL, RET, SDHC, SDHA, SDHAF2, TMEM127, MAX, FH, MEN1) and although this is a probable sporadic case of phaeochromocytoma, long-term follow-up will be required and repeat genetic studies with an expanded panel could be considered in the future.

This is an interesting case of a less classical presentation of a phaeochromocytoma, which can present with variable symptoms, and should be considered in young patients with persistent hypertension and/or ophthalmic signs of hypertension. Biochemical follow up is important to ensure both adrenergic and cortisol axes normalise. The case also highlights the importance of multiple disciplines involved in patient care from presentation, diagnosis, operative and post-operative management.

C021

MDT approach toward Thyrotoxicosis and Carbimazole induced Hepatitis

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Hull University Teaching hospital NHS Foundation Trust.

Abstract:
54 years old gentleman with background of treated B cell lymphoma presented to his practitioner with symptoms of thyrotoxicosis. His thyroid work up confirmed positive thyroid receptor antibodies, and hence treated as Graves' disease.

He was started on carbimazole by his practitioner. After two weeks of initiation of treatment with carbimazole, he presented to his GP with yellowish discoloration of skin, nausea, vomiting and passing dark coloured urine. He was found to have remarkably deranged liver function tests indicating acute liver injury warranting urgent MRCP. MRCP ruled out any obstructive pathology. Non Invasive Liver Screening was unremarkable. Carbimazole was stopped.

Patient was started on high dose propranolol and cholestyramine to optimise the symptoms of thyrotoxicosis. Gastroenterology team was consulted and patient was started on ursodeoxycholic acid with a trial to improve the Liver Functions and reduce the level of bilirubin. Liver function tests were monitored regularly which showed sluggish improvement in weeks. Liver biopsy showed acute cholangitis and cholestatic hepatitis suggesting likely drug induced liver injury. He was started on Enteral feeding to support with nutrition during the phase of acute liver injury. Team of gastroenterologists, endocrinologists and dietitians were involved to devise a holistic management plan of the patient to further facilitate the eligibility of patient to have thyroidectomy eventually.

C022

A case of severe symptomatic hungry bone syndrome, would vitamin D replacement have prevented it?

Konstantinos Bourgalis Michalopoulos. Imperial College Healthcare NHS Trust.
Abstract

Introduction:
Hungry bone syndrome is a frequent complication post parathyroidectomy, usually presenting as mild hypocalcaemia.

Case presentation:
A 60-year-old man, with a history of kidney transplantation, presented with paresthesia involving the limbs and perioral area, 3 days post subtotal parathyroidectomy for tertiary hyperparathyroidism. Bloods showed adjusted calcium: 1.62, total Calcium:1.58, iCa: 0.7, Mg: 0.46. Pre-operative bloods showed ALP: 197, Adj Calcium: 2.29 and vitamin D levels <10. The patient was on cinacalcet prior to surgery but not on vitamin D replacement. Treatment with multiple boluses of IV 10% calcium gluconate were given with unsustained effect. Given the degree of hypocalcaemia (Adj calcium 1.41), he was admitted to HDU for a continuous calcium gluconate infusion (50ml of 10% calcium gluconate over 10-12 hours) via central line. The patient stayed in hospital for a total of 10 days receiving continuous IV and oral treatment with calcium gluconate and alfacacidol until his calcium levels normalized and symptoms were controlled.

Discussion:
HBS is very common in renal patients post parathyroidectomy with incidence of up to 85% in some studies[1]. Predictors prior to surgery are high ALP and PTH levels (indicating increased bone turnover), younger age and size of the adenoma[2]. A study showed no correlation between preoperative 25(OH)D deficiency and HBS in PHPT[4]. Although vitamin D supplementation has been shown to decrease PTH in PHPT patients[3], replacing Vit D before surgery has not been shown to reduce the incidence of the syndrome[4,5] . Interestingly, prolonged and high-dose calcitriol loading did not decrease the incidence in 45 patients with secondary HPT and ESRF[6.] However, administration of colecalciferol or alfacacidol pre surgery is a common practice as many experts believe it may help HBS treatment by promoting calcium absorption.

Cinacalcet is commonly used in this population as it can reduce PTH levels. It has, however, rarely been associated with an HBS like state with severe hypocalcaemia in case reports[7]. There are no studies investigating cinacalcet administration to prevent hypocalcaema in the setting of parathyroid surgery.

Conclusion:
HBS is a very common complication of parathyroidectomy that can result in severe hypocalcaemia and prolonged hospital admission as with this case. A multidisciplinary approach, including endocrine surgeons, nephrologists and endocrinologists, is needed both prior to surgery to identify those at risk and after to properly manage hypocalcaemia. Given the paucity of current data, more research is needed to identify approaches that could prevent hungry bone syndrome.

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C023

Mental Health Association with Grave`s Thyrotoxicosis is rare but not uncommon.

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Hyperthyroidism causes irritability, insomnia, anxiety, restlessness, fatigue, impairment in concentrating and memory. These symptoms can occur sporadically or lead to Psychosis, depression and mania.

Mania and delirium have been reported with more than 1 % of patient with hyperthyroidism. Moreover, anxiety, and panic attacks have been reported to occur in patients with thyrotoxicosis**.**

The association between thyroid disorders and mental health have been documented in the literature.

Case presentation:

1. A 30-year-old was admitted to the mental unit after being sectioned. As part of the investigation, he had a thyroid function test while inpatient and in the mental health unit and it showed he was thyrotoxic with an FT4 of 81.8 and a TSH of <0.01. He was confused and paranoid and initially, he refused to take his antithyroid medications. His BP was 128/79, his heart rate of 125, his weight was 66.9 kg. After review in the clinic, he agreed to take anti-thyroid medications and was started on Propylthiouracil 200 mg twice a day and to take Propranolol 20 mg three times a day. He has clinically improved with the improvement in the thyroid blood test. His most recent blood test showed FT4 of 44 and TSH<0.01 with improvement in symptoms

2. A 44-year-old was seen in the clinic with palpitations, sweating, and weight loss. She had a past medical history of panic attacks and anxiety. She was very stressed at work and unable to cope and was described by family members that she had mood swings and sometimes getting easily upset. She was found to be thyrotoxic and started on anti-thyroid medications. She was poorly compliant with her medications, and she had radioactive iodine therapy and is currently euthyroid on levothyroxine. She apologized for previously erratic behaviour and said she did not realize how her overactive thyroid negatively affected her physical and mental health.

Discussion:

Thyrotoxicosis is reported to be associated with anxiety, restlessness, depression, emotional lability, and lack of sleep. These symptoms may overlap with the symptoms of mental health problems such as bipolar disorders, and other mental illnesses. Although NICE does not mandate routine checks of TFTS in patients presenting with acute psychosis due to low yield. However, from this case that checking TFTS in patients presenting acutely with mental health issues.

C024

How is the calcium normal?

Kaenat Mulla, Patrick McGown, Florian Wernig. Imperial College Healthcare NHS Trust.

A 25-year-old lady with a background of MEN2a was referred to the endocrine team during her first pregnancy. She was initially diagnosed with bilateral phaeochromocytomas in Romania in 2018 after presenting with paroxysmal headaches and hypertension and underwent laparoscopic bilateral adrenalectomies.

Subsequently, she was diagnosed with MEN2a (RET gene mutation: RET exon 11 C1901 G2a, p.CYs634Tyr) as the index case in her family, and three months later underwent prophylactic total thyroidectomy and total parathyroidectomy. Three other family members have since been confirmed as MEN2a.

On referral to the endocrine team, she was 21 weeks pregnant and taking hormone replacement in the form of Hydrocortisone (10, 5, and 5mg), Fludrocortisone 100mcg od, Levothyroxine 150mcg od, Adcal 1000mg od, and Calcitriol 0.25mcg od.

Our patient became hypercalcaemic in the second trimester. She was advised to stop Calcitriol and adcal. After cessation, she remarkably had normal levels of adjusted calcium for the remainder of her pregnancy, despite low/undetectable levels of both PTH and PTHrP.

Literature review states that there is an increase in serum calcitriol levels in the latter half of the pregnancy. The increase in calcitriol can be regulated by other pregnancy hormones, which are normal in hypoparathyroidism, such as prolactin, oestrogen, and placental growth hormone. In case calcitriol dose is not reduced or stopped then in combination with elevated serum levels, there will be increase in calcium absorption and bone resorption, which result in hypercalcaemia. Calcitriol levels drop during breastfeeding. Her calcium level dropped post-delivery and she was restarted on supplementation.

During the last trimester, our patient was diagnosed with pre-eclampsia (PET). Interestingly calcium supplementation in pregnancy may help prevent hypertension; therefore, reducing the chances of PET.

Six months post-partum her calcium levels remain in optimal range (low-normal) on calcitriol 0.5 mcg od and calcium 1000mg od.

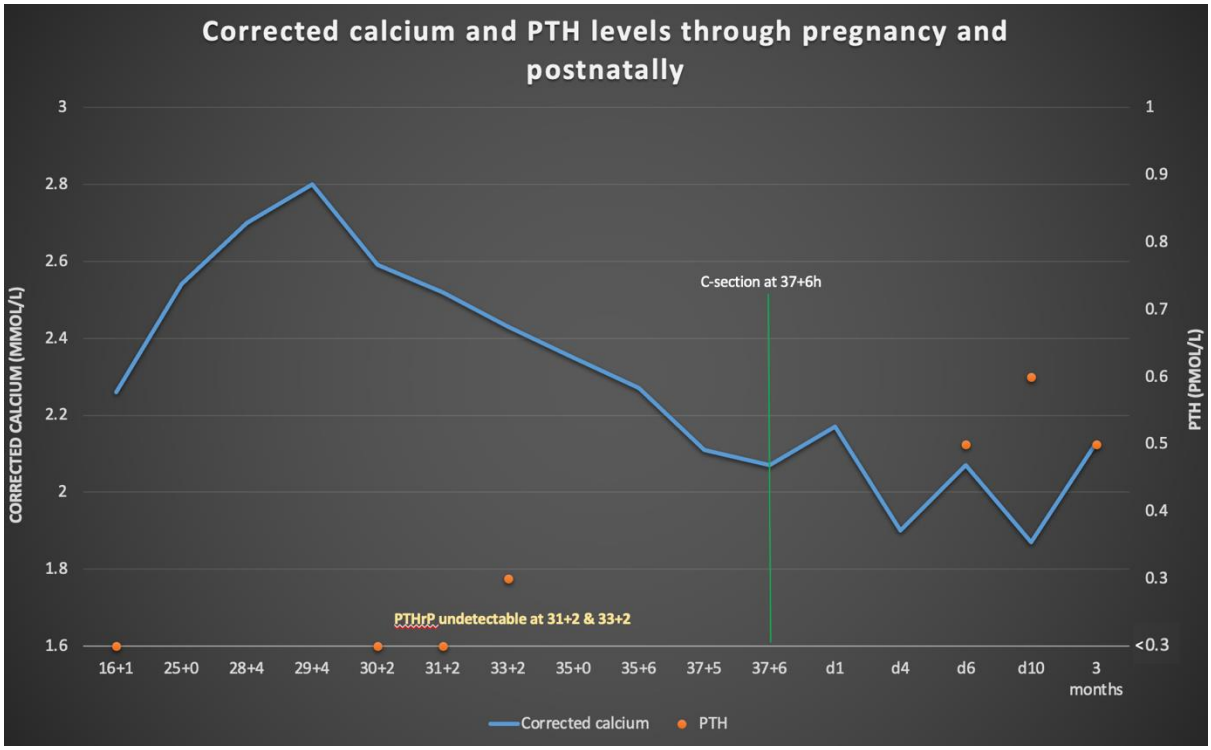

Figure 1, shows the trend of her adjusted calcium, PTH and PTHrp during pregnancy and after.

C025

Severe hypercalcaemia secondary to CD4+ T cell lymphoma.

K Jethwa, B Wang, O Oboh, M Abeywickrema, K Michalopoulos, M Siddiqui, S Khan, F Bahowairath, E Hatfield. Charing Cross Hospital, Imperial College Healthcare NHS Trust

A previously well and independent 82-year-old gentleman, with a history of hypertension, anaemia and previous pacemaker insertion, was admitted in 2023 with new, worsening confusion and recent weight loss.

He had constipation and nocturia, but no previous history of nephrolithiasis or bone pain. On admission, his blood tests showed; adjusted calcium 3.9mmol/L, phosphate 1.03mmol/L, magnesium 0.57mmol/L, vitamin D 60.6nmol/L, parathyroid hormone (PTH) 1.6pmol/L, haemoglobin 131g/dL, creatinine 126μmol/L, alkaline phosphatase 169U/L and normal PSA measurement. Further subsequent investigations included a normal angiotensin converting enzyme (ACE) level (26IU/L), a normal serum protein electrophoresis and light chain assay, with no signs to indicate multiple myeloma. He was given acute treatment with intravenous (IV) fluid resuscitation, pamidronate, and IV magnesium replacement.

Despite this, his hypercalcaemia remained refractory, and bisphosphonate therapy was repeated with zoledronic acid, and ongoing IV fluid replacement increasing to four litres per 24 hours. Fluid management was complicated by hypernatraemia and hypokalaemia. A CT chest, abdomen and pelvis showed small volume mediastinal, hilar and retroperitoneal lymph nodes of uncertain significance. Pending a follow-up PET CT scan, serum calcium levels responded to ongoing fluid replacement and returned to normal limits (2.65mmol/L). Biopsies were taken from a right inguinal lymph node, identified on PET CT.

Pending biopsy results, he developed gross peripheral and pulmonary oedema, and a worsening acute kidney injury (creatinine 380μmol/L) secondary to acute tubular necrosis, from hypercalcaemia and excessive sodium chloride, and cardiorenal decompensation. Biopsy results would later confirm a diagnosis of CD4 positive T cell lymphoma.

This case highlights the complexities of managing severe hypercalcaemia, especially in the elderly population, with this case requiring input from multiple disciplines – endocrinology, haematology, radiology, histopathology and renal medicine.

Although the precise mechanism of action remains unclear, adult T cell lymphomas are thought to cause hypercalcaemia through the accumulation and activation of osteoclasts, possibly through overexpression of RANKL. This case report looks to highlight the collaborative nature of managing complex patients with unclear aetiologies and summarise the literature for the mechanisms by which T cell lymphomas contributes to hypercalcaemia.

C026

Substance abuse unmasking the underlying illness.

Muhammad Shoaib Zaidi. King Saud University Medical City, Riyadh, KSA).

Introduction:
Thyroid storm can be precipitated by various factors, including trauma, thyroid and non-thyroid surgeries, abrupt discontinuation of anti-thyroid medications, infections and parturition. It can also be drug-induced e.g., amiodarone, iodine (used in contrast and as radioiodine), salicylates. It has also been reported as a consequence of sympathomimetic substance abuse, like cocaine, amphetamine, pseudo-ephedrine.

Clinical Case:
A 40 years old Saudi married male soldier, a heavy smoker and with a previous history of Hyperthyroidism, that was diagnosed outside, 3 years ago (had discontinued his medications and had lost follow-up for 1 year). He was a social alcohol drinker and was taking occasional amphetamine and cannabis (had consumed alcohol 3 weeks back, cannabis, 1 week before and amphetamine,2 days prior to the presentation).

The patient had presented to the ER on 19th February 2023 with 2 months history of progressive weight loss, enhanced apprehension, palpitations, sweating, tremors and heat intolerance for 7 days, with worsening of the afore-mentioned symptoms for 3 days. Rest of the systemic review- unremarkable. No travel history, extra-marital relations. Family and past history - insignificant.

Exam- BP 155/90 mmHg, Pulse 120/m, regular, RR 20/m, O2sat 98% (on room air), afebrile. BMI 23.39 kg/m^2 . Anxiety & restlessness + Fine tremors + Thyroid-diffusely enlarged, non-tender. No bruits. Exophthalmos, lid-lag, pretibial myxedema- absent. Skin-warm and moist. Nicotine-stained fingers. Otherwise rest of the general and systemic exam - insignificant.

Results:
Investigations-(19.02.23) TFTs-[TSH 100(12-22pmol/l), FT4 0.005(0.25-5mIU/l)]. Thyroid Abs (Anti TG Abs-1582.87(0-100 units), Anti TPO Abs-108.52(0-60 units). Other significant labs-high ALP 184(40-129 IU/l), Urine amphetamine positive >500 ng/ml, Urine cannabinoids-positive >100ng/ml, Serum ethanol & other toxicology screening negative. ECG- sinus tachycardia,120/m. Chest film-unremarkable.

US Thyroid - Diffuse severe heterogeneous thyroid gland likely related to underlying diffuse thyroid disease / thyroiditis. Tc-99 uptake scan-diffusely increased radiotracer uptake with no definite cold or hot nodules. Tc thyroid uptake = 15% (N = 0.5 - 4.0%), findings in keeping with Graves' disease.

Our patient was diagnosed to have thyroid storm(Thyroid storm Point scale score 40. Definite Thyroid storm) He was managed with Carbimazole, Propranolol & Hydrocortisone, under the supervision of Endocrinology team. The patient got improved was discharged home on 26th February, 2023. A follow-up with Endocrinology was given.

CONCLUSION:
Our case exemplifies the need of emphasizing upon the patient, a regular follow-up and compliance to the antithyroid medications, which could have prevented the thyroid storm. This case also draws attention to look for any precipitating factors for the thyroid crisis, like amphetamine intake.

C027

Megacolon in Multiple Endocrine Neoplasia Type 2B: an acute on chronic presentation.

Alexandra Cann, Mariana Abdel-Malek, Julian Walters, Tricia Tan. Imperial College Healthcare NHS Trust.

Multiple Endocrine Neoplasia Type 2B (MEN2B) is the rarest of the MEN syndromes and is inherited in an autosomal dominant fashion. It is characterised by early onset medullary thyroid cancer, phaeochromocytoma and extra endocrine features such as mucosal neuromas, marfanoid body habitus and ganglioneuromatosis of the gastrointestinal tract. We describe an acute on chronic episode of intestinal ganglioneuromatosis that presented in late adulthood which was jointly managed by the endocrinology, gastroenterology, acute medical and general surgical teams at Imperial College NHS Trust.

Case Presentation:
A 60-year-old female presented with an unexplained weight loss history of 20kg in one year, on a background of known MEN2B and previous thyroidectomy for which she was under regular surveillance follow up. She described feeling unwell reporting symptoms of anorexia, bloating, nausea and constipation which prompted further evaluation. Her biochemistry at the time revealed a raised erythrocyte sedimentation rate, normocytic anaemia, suppressed thyroid stimulating hormone, vitamin B12 and folate deficiency. An upper gastrointestinal endoscopy was normal and a colonoscopy showed left colon dilatation with normal looking mucosa, albeit inadequate bowel prep.

Further imaging with FDG PET did not reveal a cause for the weight loss with stable appearance of the glomus tumour since the previous scan three years beforehand. She was subsequently commenced on empirical treatment in gastroenterology clinic for small intestinal bacterial overgrowth with oral antibiotics in addition to iron, vitamin B12 and folate replacement. Over the following months she had some minor symptomatic improvement.

Four months later, however, she was acutely admitted from the Endocrinology clinic with postural hypotension, reduced appetite, continued weight loss and poor bowel opening. On abdominal examination, she had a palpable mass with audible bowel sounds and X-Ray imaging showed severe faecal loading. She was initially managed with Macrogol, Senna and Phosphate enemas as well as a nasogastric tube and Klean Prep.

Despite these interventions, a CT scan one week later demonstrated an increase in colonic distension due to constipation and faecal impaction with a risk of stercoral perforation. The surgical team advised conservative management in view of the widespread neuroganglioma involvement throughout the bowel. She proceeded to have a manual evacuation and, in the week following, she was discharged with a regular bowel habit on Movicol and Bisacodyl. Her gastrointestinal symptoms have since remained stable, sometimes fluctuating between constipation and diarrhoea which the patient manages herself and she reports a good quality of life.

Discussion:
The extra endocrine features of MEN2B can be life-threatening, and although ganglioneuromas are benign tumours, they can exert detrimental effects on the normal physiology of surrounding structures. In this case, the dysfunction led to severe constipation which culminated in bowel obstruction and risk of perforation. Given the

associated morbidity and mortality, this case illustrates the importance of promptly recognising the gastrointestinal manifestations of MEN2B syndrome complicated by intestinal ganglioneuromatosis. The multidisciplinary team approach in this case- between endocrinologists, gastroenterologists, general medics and surgeons- was also crucial to ensuring a good patient outcome and conservative management proved to be an effective treatment option.

C028
Persistent primary hyperparathyroidism cured by diagnostic FNA

J Dirie, J Agilinko, M Kedrzycki, E Collins, A Di Marco, F Palazzo

Introduction

Persistent primary hyperparathyroidism is defined as hyperparathyroidism that is not cured by initial operation, and occurs in approximately 5% of patients after a parathyroidectomy. [1] Such cases require a meticulous review of all aspects of their previous care from the confirming the diagnosis and reviewing the imaging, operation notes, and histology.

In cases where second line imaging does not secure localisation it can be necessary to perform invasive tests such as venous sampling or a needle aspiration for PTH of a lesion of interest. [2]

We present one such case with a peculiar result.

Case discussion

We present the case of a 62-year-old female who was referred to our service with persistent hyperparathyroidism after 4 gland exploration. During her initial operation, a left parathyroid gland had been excised without normalisation of the biochemistry - the second ipsilateral gland could not be found.

Post-operative blood tests suggested persistent hyperparathyroidism along with significant neuro-cognitive symptoms. After re-confirming the diagnosis, we opted to arrange a Choline-PET CT, which demonstrated an unusually located warm area posterior to left submandibular gland.

MDT discussion concluded that more information was required and an FNA for PTH was performed. The PTH from the lesion corresponding to the warm area came back significantly elevated (230.34pmol/L). Re-operative parathyroidectomy was scheduled but on repeat blood tests 2 weeks post aspiration, her PTH and Calcium had normalised so surgery was postponed. At 1 year follow up, she remains cured and reports improvement of her symptoms.

Conclusion

Diagnostic FNA is a useful tool in some challenging cases of persistent HPT. It is exceptional that the diagnosis is followed by immediate cure.

It is presumed that the FNA may have caused the adenoma to undergo arterial or venous infarction by creating a capsular haematoma. Past attempts to do this therapeutically have been less successful in the long term but this experience may justify revisiting this technique.

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C029

The importance of under-treating adrenal failure in order to allow normal adrenal function following unilateral adrenalectomy for Cushings syndrome

Kavita Narula, Kate Lazarus, Asjid Qureshi, Fausto Palazzo, Karim Meeran

A 26-year-old woman presented to the Endocrine clinic with features of Cushing's syndrome (CS). Investigations revealed a non-suppressible cortisol on a low dose Dexamethasone suppression test with a suppressed ACTH, and two positive 24-hour urine cortisol collections, indicating CS. CT imaging revealed a 3.4cm left adrenal adenoma. Following a left adrenalectomy, she was discharged on once daily Prednisolone 3 mg. She was well for a year except for one occasion where she reported forgetting to take her Prednisolone and started vomiting. She did not seek medical help. This resolved after she had her Prednisolone dose the following day.

On review a year later, a Short Synacthen test (SST), showed a suboptimal response (Table 2). As we were certain that the right adrenal was intact, we planned to gradually reduce the prednisolone dose, to assess for recovery of endogenous cortisol production. Administering a replacement dose of glucocorticoids during the perioperative period helps to preserve normal physiological function, while also reducing potential risks of adrenal crisis immediately postoperatively.

While corticosteroid therapy is commonly used for patients with CS undergoing adrenalectomy, there is no clear consensus on the best approach. A general strategy involves administering steroids both intraoperatively and postoperatively, starting with intravenous administration followed by oral administration, and then gradually tapering the dosage to allow recovery of the HPA axis and endogenous cortisol production. There is limited evidence how best to do this.

We have found use of a Prednisolone tapering regimen alongside clinical assessment can help the weaning process. She is now off Prednisolone. Patients with adrenal CS have reduced cortisol secretion due to negative feedback on the HPA axis. This leads to a decrease in ACTH release. Reduced ACTH stimulation to both the adrenal cortex surrounding the lesion and the contralateral, often atrophic adrenal gland, results in adrenal insufficiency and need for glucocorticoid replacement therapy. However, patients undergoing unilateral adrenalectomy will have adrenal reserve in the contralateral adrenal gland and should be able to recover endogenous cortisol production. This can be stimulated by deliberately reducing the dose of prednisolone to aid ACTH secretion. Life-long glucocorticoid replacement therapy may be avoided in such patients.

Dates & Prednisolone dose	Baseline cortisol at 0 minutes (nmol/L)	Cortisol at 30 min (nmol/L)	Cortisol at 60 min (nmol/L)	ACTH (ng/L)
02/12/22 – Prednisolone 2 mg	187	189	218	130
11/10/22 - Prednisolone 2 mg	125	137	135	76.4
27/07/22 - Prednisolone 3 mg	59	76	81	78.5

Table 2: SST on different doses of steroid

Week	Mon	Tues	Wed	Thurs	Fri	Sat	Sun
0	3	3	3	3	3	3	3
1	3	3	3	2	3	3	3
2	3	2	3	3	2	3	3
3	3	2	3	2	3	2	3
4	2	3	2	3	2	3	2
5	2	3	2	2	3	2	2
6	2	2	2	3	2	2	2
7	2	2	2	2	2	2	2
8	2	2	2	1	2	2	2
9	2	1	2	2	1	2	2
10	2	1	2	1	2	1	2
11	1	2	1	2	1	2	1
12	1	2	1	1	2	1	1
13	1	1	1	2	1	1	1
14	1	1	1	1	1	1	1
15	1	1	1	0	1	1	1
16	1	0	1	1	0	1	1
17	1	0	1	0	1	0	1
18	0	1	0	1	0	1	0
19	0	1	0	0	1	0	0
20	0	0	0	1	0	0	0

C030

A case of hyperparathroidism – jaw tumour syndrome (HP-JTS): parathyroid carcinoma and hungry bones

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A 47-year-old Tanzanian gentleman presented to clinic at our department with history of 2 stone weight loss, musculoskeletal symptoms, osmotic symptoms and epigastric pain. His only medical history of note was of a surgical resection of a left maxillary ossifying fibroma 16 years previously.

Baseline investigations had revealed hyperparathyroidism, with a serum corrected calcium >3.0mmol/L (2.15-2.65), PTH >168pmol/L (1.6-6.9) and vitamin D3 30nmol/L (80-120). Initial management was with ergocalciferol and further investigations arranged. He did not attend numerous clinic appointments and represented three years later to clinic with 2x4cm very firm solitary nodule in the left anterior triangle of his neck and corneal calcifications on fundoscopy. Biochemistry revealed residual profound hyperparathyroidism with a serum corrected calcium 3.40mmol/L (2.15-2.65), PTH 263pmol/L (1.6-6.9) and vitamin D3 12nmol/L (80-120). Neck ultrasonography followed by CT scan identified 3.3 x 2.5 cm mass related to the upper left pole of the thyroid with evidence of extrathyroidal extension posteriomedially into the para-oesophageal region. Nuclear medicine SESTAMIBI imaging identified the mass as likely parathyroid.

He proceeded to surgery with presurgical management of 300,000units IM ergocalciferol, hydration with intravenous fluids and cinacalcet titrated to 60mg bd. The mass was incompletely excised and showed histology consistent with parathyroid carcinoma with Ki 67 of 5-10%. Post operatively he required intensive care for severe hypocalcaemia secondary to hungry bone syndrome. On his recovery he had external beam radiotherapy to the neck and US revealed no evidence of recurrent tumour. He was referred for genetic testing and he was found to have a CDC73 deletion consistent with HP-JTS.

He was discharged and again lost to follow up but represented two years later with symptomatic hypercalcaemia (serum corrected calcium 4.22mmol/L and PTH 149.2pmol/L) requiring denosumab after failure of conventional treatment. Neck imaging was normal but a target for possible PTH production was identified on MRI spine as a soft tissue mass in T8. This mass was avid for FDG and MIBI. Biopsy revealed PTH +ve cells with Ki 67 of 10% consistent with disease recurrence. He underwent left lateral thoracotomy, T8 corpectomy and metastatic debulking with cage reconstruction in November 2022. This was followed by further external beam radiotherapy to T1-3 vertebrae for new deposits in June 2023.

We present a case of parathyroid carcinoma in a gentleman with genetically confirmed HP-JTS. He remains on 2 monthly denosumab infusions for ongoing PTH driven hypercalcaemia.