Hammersmith Abstracts

Friday 6th December 2024

19th Hammersmith Multidisciplinary Endocrine Symposium 2024

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19th Multidisciplinary Endocrine Symposium, Friday 6th December
Glenister Lecture Theatre, Charing Cross Hospital

8.30am Registration & Coffee

8.55am Welcome and Introduction

Session 1: Chair: Miss Aimee DiMarco & Ms Debbie Papadopoulou (Imperial)

9.00am Advances in the management of Thyroid Eye Disease
Ms Vickie Lee (Imperial)

9.30am Radioiodine in Graves': how and why?
Dr Sairah Khan (Imperial)

9.50am Thyroid surgery for Graves' disease
Mr David Scott Coombes (Cardiff)

10.20am Case 1 (D008 The complexities of eye disease)

10.35am Case 2 (D003 A difficult management decision in a patient with an aldosterone-secreting adenoma with contralateral nephrectomy for renal cancer)

10.50am Coffee Break

Session 2: Chair: Prof Wiebke Arlt and Dr James Ahlquist

11.20am Recent advances in Adrenal Macronodular hyperplasia
Dr Lucas Bouys (Hopital Cochin, Paris)

11. 50am State of the art diagnostics and treatment of Adrenocortical cancer
Prof Cristina Ronchi (Birmingham)

12.50pm Discussion

1.00pm Lunch & Poster session

Session 3: Chair: Prof Tricia Tan

2.00pm An update on the pharmacological management of obesity
Prof Barbara McGowan (St Thomas' Hospital)

2.30pm What is the role of bariatric surgery post GLP-1 agonists?
Mr Marcus Reddy (St George's Hospital)

3.00pm Case 3 (D031 Normalisation of glycaemia in a patient with type 2 diabetes within forty-eight hours of Roux-en-Y gastric bypass surgery)

3.15pm Case 4 (D034 Now You See Me! Re-operative parathyroidectomy for a supernumerary ectopic parathyroid adenoma)

3.30pm Case 5 (D035 The sight-saving role of Bariatric Surgery in Intracranial hypertension)

3.45pm Case 6 (D030 Post adrenalectomy paranoia: non-normalising metanephrines after resection of phaeochromocytoma)
4.15pm Certificates and close

D001	Gestational weight loss in pregnancy after bariatric surgery
D002	Dopamine agonist therapy for non-functioning pituitary adenomas - first, do no harm?
D003 Case 2	A difficult management decision in a patient with an aldosterone-secreting adenoma with contralateral nephrectomy for renal cancer
D004	A potential new protocol for investigation of post-menopausal hirsutism
D005	Skeletal Complications in Gaucher Disease: Case Report and Multidisciplinary Approach to Bone Density Management in a Paraplegic Patient
D006	A Single-Centre Audit of Post Thyroidectomy Hypoparathyroidism – uncertain ability of near infrared autofluorescence technology to be cost-effective
D007	A case of difficult to treat thyroid storm in inflammatory bowel disease
D008 Case 1	The complexities of thyroid eye disease
D009	Severe osteoporosis secondary to bariatric surgery
D010	Ferinject-induced prolonged hypocalcaemia: an under recognised phenomenon?
D011	Opportunistic Fungal Infection in a Patient with Ectopic Cushing's Syndrome: A Multidisciplinary Approach to Diagnosis and Management
D012	Gestational trophoblastic disease: A rare cause of thyroid storm
D013	Refractory Hypercalcemia and Seizures in a Patient with Parathyroid Adenoma: A Case for Timely Surgical Intervention
D014	Management of carbimazole-induced liver function derangement in everyday endocrinology
D015	A working diagnosis of cerebral salt wasting in conjunction with subdural haematoma
D016	The Role of Tolvaptan in an Outpatient Setting
D017	Timely surgical intervention in a patient with pituitary apoplexy and cavernous Sinus Invasion

D018	Identification and Management of Pseudohypoparathyroidism Type 1a in a District General Hospital
D019	Navigating Acromegaly and Fertility: A Complex Case of Multidisciplinary Management and Postpartum Planning
D020	Unraveling the Complexity of SDHA Pathogenic Germline Variants in GIST: A Report of Two Distinct Cases
D021	Thyroid Storm in Acute Setting: Would You Miss It?
D022	Challenges of Managing AVP Deficiency in Pregnant Woman with Hyperemesis Gravidarum
D023	AVP deficiency secondary to herbal remedies
D024	Adrenal insufficiency secondary to skin lightening cream
D025	Molecular targets everywhere but no NHS approval to treat!
D026	Total pancreatectomy with islet auto transplantation for the treatment of chronic pancreatitis: between a rock and a hard place
D027	A case of poorly differentiated follicular thyroid carcinoma: when less is more
D028	Double Trouble: Navigating Contrast and Amiodarone-induced Thyrotoxicosis
D029	A Rare Case of Addison’s disease in Pregnancy
D030 Case 6	Post adrenalectomy paranoia: non-normalising metanephrines after resection of phaeochromocytoma
D031 Case 3	Normalisation of glycaemia in a patient with type 2 diabetes within forty-eight hours of Roux-en-Y gastric bypass surgery
D032	Adrenal Haemorrhage: Bilateral vs Unilateral
D033	A case of Congenital Adrenal Hyperplasia (CAH) in pregnancy
D034 Case 4	Now You See Me! Re-operative parathyroidectomy for a supernumerary ectopic parathyroid adenoma
D035 Case 5	The sight-saving role of Bariatric Surgery in Idiopathic Intracranial Hypertension
D036	Weight management in women with subfertility seeking assisted reproduction: a case series

D001

Gestational weight loss in pregnancy after bariatric surgery

S Ansari[1,2], M Xia[1], K Alexiadou[1], J Upton[2], A Makepeace-Smith[2], K O'Donnell[2], B Jones[3], C Yu[3], A Dornhorst[1], N Oliver[1], C Tsironis[2], S Pukayastha[2], A Ahmed[2], R Agha-Jaffar[1], B Khoo[4], T Tan[1,2]
[1] Department of Metabolism Digestion and Reproduction, Imperial College London.
[2] Imperial Weight Centre, Imperial College Healthcare NHS Trust.
[3] Queen Charlotte's and Chelsea Hospital, London, UK.
[4] Division of Medicine, University College London, London, UK.

Abstract

Introduction
Pregnancy in women with a history of bariatric surgery is becoming increasingly prevalent and is associated with risks and benefits to the mother and baby. Gestational weight gain, maternal micronutrient blood levels and glucose variability in this cohort may impact foetal growth and birthweight[1,2].

Case Report

Patient history
A 33-year-old female with a history of endometriosis and obesity was referred to the Imperial Weight Centre, she was 11 weeks pregnant. She underwent a vertical sleeve gastrectomy 11 weeks previously in Turkey. She was pregnant at the time of her operation however a pregnancy test was not performed at the time of her surgery. Pre-surgical and pre-pregnancy weight and BMI were therefore the same, 98kg and 36.4kg/m^2, respectively. There was no history of alcohol consumption, smoking or vaping. The patient was taking no micronutrient supplements at the time of referral however following initial assessment, she was commenced on Forceval capsules once-daily, Folic acid 5mg once-daily, adcal D3 one tablet twice-daily, colecalciferol 20,000IU once weekly and intramuscular B12 injections every 3 months.

Methods
The patient received monitoring of anthropometrics, body composition using bioelectrical impedance, blinded continuous glucose monitoring for 10 days (DEXCOM G6) and blood tests in each trimester. The patient received dietetic input from the Imperial bariatric team throughout pregnancy and serial foetal growth scans in the 3rd trimester at Queen Charlotte's Hospital.

Results
There was gestational weight loss of 24% (table 1), a progressive decline in fat mass and a small-for-dates foetus from the 3rd trimester onwards with moderate glucose variability and norla maternal micronutrient blood levels (according to reference ranges for non-pregnant women without bariatric surgery). Serial growth scans in the 3rd trimester showed a symmetrically small-for-gestational age foetus with normal dopplers and liquor volumes. There was no evidence of chromosomal aneuploidies or infections (e.g., CMV). Gestational age at delivery was 39 weeks with an induced vaginal birth. Birthweight and birthweight centile were 2580g and 4.6 respectively. The baby was not admitted to NICU.

Discussion
This patient was pregnant at the time of her vertical sleeve gastrectomy. Her pregnancy therefore coincided with the rapid weight loss phase after bariatric surgery resulting a gestational weight loss and a small-for-gestational age baby delivered at term. Due to the anorectic effects of surgery and the nausea of pregnancy, maintaining adequate nutritional intake in the first 12 months post-surgery can be challenging. A case series of 3 women that were pregnant at the time of bariatric surgery reported gestational weight loss of -5kg and all women delivered an SGA baby at term[2].

Conclusion
Women should avoid pregnancy after bariatric surgery until post-surgical weight loss has plateaued (i.e., after 12-18 months). Pregnant women with a history of bariatric surgery are high risk pregnancies that require specialist input from the obstetrics and bariatric teams.

References

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3. Alexiadou K, Ansari S et al. Increased glycaemic variability in pregnant women with Roux-en-Y gastric bypass compared with sleeve gastrectomy. BMJ Open Diab Res Care. 12:e003642 (2024)

D002

Dopamine agonist therapy for non-functioning pituitary adenomas – *first, do no harm?*

L Nagib, N Alavi, K Bub, B Freudenthal.
Barnet Hospital, Royal Free London NHS Foundation Trust.

Abstract

Case

An 84-year-old female presented with a 2-3 week history of visual and auditory hallucinations as well as dizziness. She reported no urinary symptoms, no cough, no fever and no recent unwell contacts. Due to her dizziness, she sustained a fall. She reported starting cabergoline two months prior to admission for treatment of pituitary macroadenoma; there were no other recent medication changes.

Her past medical history was significant for a previous non-functioning pituitary macroadenoma (NFPA), for which she had two previous transsphenoidal surgeries (2017 and 2013) and had some ongoing residual tumour for which it was felt she is too frail for further surgery. The tumour was too close to optic chiasm for consideration of radiotherapy.

She had post-operative central AVP deficiency. She also had a background of well controlled type 2 diabetes, hypercholesterolaemia, asthma and osteoporosis
Her regular medications included: cabergoline 250mcg twice a week, desmopressin 200mcg BD, atorvastatin 20mg ON, metformin 500mg BD, Colecalciferol 1,000IU once a day and lansoprazole 30mg OD.

Observations and examination normal with HR 83, BP 117/67 and apyrexial.Full blood count, renal, liver and bone profiles, inflammatory markers, and urine dip were all normal.
CT head showed no acute intracranial pathology.

Management

This lady had no obvious infective precipitant to her acute onset hallucinations, the only significant finding from her history was the new initiation of cabergoline. Her cabergoline was stopped.

Outcome

Three months later she was seen in clinic and reported prompt resolution of her hallucinations and dizziness.

Discussion

Cabergoline can be used as an adjunct to surgery to control tumour growth, as NFPA express dopaminergic receptors. As with all dopaminergic agonists common side effects include dizziness, confusion, hallucinations and impulse control disorders.

The evidence for the use of cabergoline in patients with NFPA is limited; with observational data and small sample sizes. In a study of 25 surgery naïve patients, with mean tumour size 18.6 ± 6.3 mm, cabergoline reduced tumour size in only 20% (5 patients) (mean decrease of 5.0 ± 3.0 mm)[1].

A metanalysis of 5 studies demonstrated that only 19% of post-operative patients had >20% tumour shrinkage, and 50% had no tumour progression (defined as <20% volume growth); only one of the included studies had a control group[2].

Common adverse events from cabergoline include dizziness, headaches, nausea and vomiting can affect up to two thirds of patients[3]. 17% of patients experience impulse control disorders, though this also resolves with cessation of medication[4]. There is an increased risk of cardiac valvular regurgitation in patients only at very high doses not typically used for this indication, howeverlow doses of cabergoline are safe, with extremely rare reports of significant valvular regurgitaiton[5].

Learning points

1. Cabergoline is associated with numerous side-effects including dizziness, hallucinations and impulse control disorders.
2. The use of cabergoline in NFPA is controversial; with most studies being too small to extrapolate their predicted efficacy for individual patients.
3. Patients commenced on cabergoline must be closely monitored for side- effects after initiation. Side-effects may be more likely in patients of advanced age and frailty, for whom conservative non-surgical treatment may be most likely to be considered.
4. Rapid resolution of side effects of carbergoline can be seen on cessation of medication.

References

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D003

A difficult management decision in a patient with an aldosterone-secreting adenoma with contralateral nephrectomy for renal cancer

H Imtiaz[1], J Macfarlane[2], MJ Levy[1]
[1] University Hospitals of Leicester.
[2] Cambridge University Hospitals.

Introduction
Primary hyperaldosteronism is an underdiagnosed cause of hypertension, affecting approximately 5-15% of all hypertensive patients and 25% of those with resistant hypertension. Around one-third of patients present with hypokalaemia. The aldosterone to renin ratio is used as an initial screening test, followed by CT adrenal imaging and adrenal vein sampling for lateralization and confirmation. Management options include laparoscopic adrenalectomy or medical treatment with mineralocorticoid antagonists, with newer modalities like radiofrequency ablation currently under trial in limited centres globally.

Case Report
A 68-year-old male consultant psychiatrist was referred to Endocrinology with a 30-year history of hypertension and hypokalaemia. His medical history includes a left nephrectomy for renal cancer and end-stage renal failure resulting from uncontrolled hypertension, with a subsequent renal transplant in 2019. Other comorbidities include ischemic heart disease and hypercholesterolemia. Recurrent hospital admissions with hypokalaemia revealed an elevated aldosterone to renin ratio. CT imaging identified a lipid-rich adrenal adenoma (1.3 cm, 13 Hounsfield units). Treatment with spironolactone normalized his potassium levels and controlled his blood pressure within three months. An 18F-CETO PET/CT confirmed a lesion in the right adrenal gland, consistent with an aldosterone-secreting benign adenoma. Unexpectedly, despite having a left nephrectomy, the CETO shows viableadrenal tissue on the left, which would inform decision-making. An MDT discussion with the Cambridge team is planned to decide between long-term medical treatment, radiofrequency ablation, or surgery.

Discussion
The patient's solitary kidney along with other comorbidities pose a challenge in management as surgery will result in hypoadrenalism. However, without a curative approach, there is a long-term risk of hypertensive nephropathy and renal failure in the transplanted kidney. In our patient with significant comorbidities, minimally invasive percutaneous ablation offers a more suitable treatment option, minimizing the risk of complications associated with surgery. This case highlights the complexities in managing primary hyperaldosteronism in patients with significant comorbidities and underscores the need for individualized treatment plans.

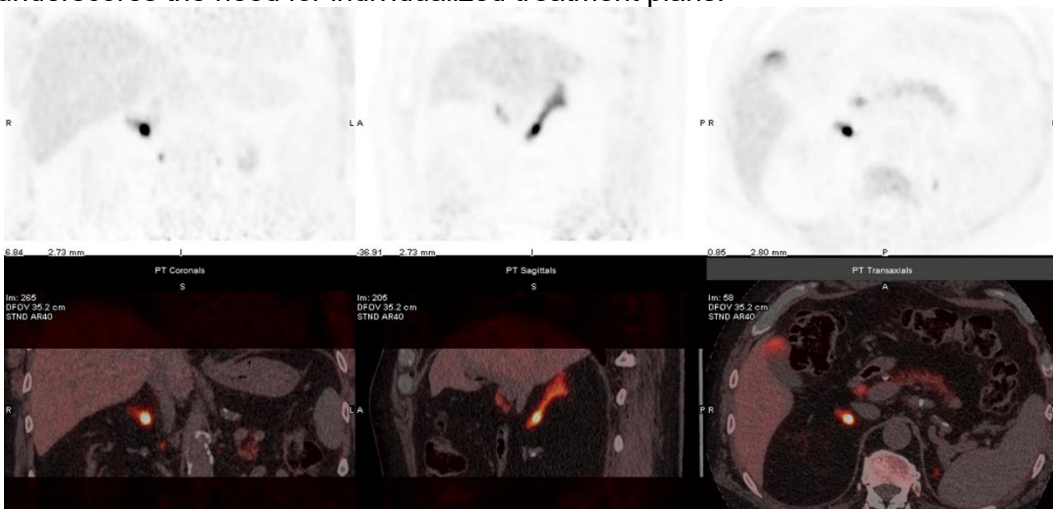

Figure 1: shows focal CETO uptake within inferior right adrenal nodule.

D004

A potential new protocol for investigation of post-menopausal hirsutism

J Sellicks[1], A Morrison[1], C Jayasena[2], MJ Levy[1]
[1] University of Leicester NHS Hospitals Trust.
[2] Imperial College London.

Introduction

Post-menopausal hirsutism is a relatively common clinical scenario, and the pathology can range from benign to malignant disease of the adrenal glands and ovaries. There are different ways to investigate this including dynamic endocrine tests, cross-sectional and functional imaging, and venous sampling. We present two cases of significant postmenopausal hirsutism with high serum testosterone levels, where use of a gonadotropin antagonist helped in both the diagnosis and treatment.

Case presentations

Case 1

A 77-year-old woman presented with a few months history of hirsutism and voice deepening. Serum testosterone was significantly elevated (14nmol/L) levels with non-suppressed LH and FSH along with polycythaemia suggested either an ovarian tumour or ovarian hyperthecosis. Pelvic MRI showed a solid enhancing lesion arising from the left adnexa. The gynecology-oncology (MDT) recommended laparoscopic bilateral salpingo-oophorectomy (BSO) and omental and peritoneal biopsies. The was reluctant to proceed with surgery and a repeat scan indicated stability in the lesion size with no suspicious features. A GnRH antagonist, Degarelix 80mg, was given to determine whether testosterone was suppressible. This caused a rapid fall in gonadotropins and testosterone levels (14nmol/L to 1.5nmol/L in 24 hours). The patient reported significant improvement in hirsutism, noting thicker hair and decreased frequency of headaches. Fatigue has reduced, and overall well-being has improved. The patient tolerated injections well and testosterone levels have consistently remained below 1 nM since initiation. She has been on treatment for 4 months, with no adverse features, and further scans have shown no growth.

Case 2

A 45-year-old female patient presented to gynaecology with amenorrhoea, hirsutism, acne and irritability. Elevated serum testosterone (LC-MS 16.2nmol/) with non-suppressed LH and FSH, together with non-suppressed adrenal DHEA and androstenedione suggested an ovarian source. 17OHP levels were normal and haematocrit was slightly elevated. Urine steroid profile showed normal adrenal metabolites consistent with an ovarian source. Cross-sectional imaging a showed solid left ovarian mass, which was non-avid on FDG-PET. A trial of Degarelix 80mg showed a rapid suppression in testosterone levels from 6.6nmol/L to 2.8nmol/L within 8 hours, and to 0.8nmol/L within 48 hours. She is being followed up by regular clinical review and biochemical assessment of androgens.

Conclusion

Gonadotrophin antagonists can be useful in confirming whether androgens have an ovarian source, in the clinical context of peri-menopausal hyperandrogenism. We suggest a protocol that might both confirm ovarian source, as well as predict whether the lesion is benign or malignant. We hypothesise that benign lesions have a good response in terms of testosterone lowering after the gonadotropin antagonist, whilst malignant tumours may not show this response. More data is needed in following up these two cases as well as trying the proposed protocol in patients with malignant ovarian tumours.

D005

Skeletal Complications in Gaucher Disease: Case Report and Multidisciplinary Approach to Bone Density Management in a Paraplegic Patient

SSC Tam[1], S Zac-Varghese[2]
[1]School of Clinical Medicine, University of Cambridge.
[2]East and North Herts NHS Trust.

Abstract

Introduction
Gaucher disease is a rare genetic lysosomal storage disorder characterised by dysfunction across multiple organs, with skeletal involvement being the most prevalent clinical manifestation. Skeletal abnormalities in Gaucher disease include impaired bone modelling, reduced bone mineral density (BMD) and increased susceptibility to bone infarction. Pathophysiologically, the accumulation of bioactive glycosphingolipids disrupts the equilibrium between osteoclast and osteoblast activity, resulting in disordered trabecular and cortical bone architecture, cortical thinning, fragility fractures and osteolytic lesions. In this case study, we present a 44-year-old patient with Gaucher disease who sustained multiple fractures following severe trauma.

Case summary
While these fractures may have occurred independently of Gaucher disease due to the traumatic nature of the event, the patient now faces secondary complications, including paraplegia, which may further exacerbate BMD loss due to immobility. The primary treatment for skeletal involvement in Gaucher disease is enzyme replacement therapy with intravenous velaglucerase alfa (VPRIV), with evidence also supporting the adjunctive use of intravenous bisphosphonates. However, given the patient's young age and potential lifelong need for osteoporosis management, the longevity and safety of bisphosphonate therapy warrant consideration. Optimising vitamin D levels, screening for secondary causes of osteoporosis, and minimising fall risk are also essential components of care. For disease monitoring, limitations in dual-energy X-ray absorptiometry (DEXA) in detecting cortical bone changes highlight the potential value of alternative assessments, such as cortical thickness index and magnetic resonance imaging (MRI). Given the complexity of this case, particularly with the additional challenge of paraplegia, we have engaged the East of England Rare Bone Network (ERBON) for multidisciplinary input. We acknowledge ERBON's ongoing role in care coordination and management for this patient.

D006

A Single-Centre Audit of Post Thyroidectomy Hypoparathyroidism – uncertain ability of near infrared autofluorescence technology to be cost-effective

T Hulf, T Sivashanmugam, S Khan, R Mihai.
Department of Endocrine Surgery, Churchill Hospital, Oxford University Hospitals NHS Foundation Trust.

Abstract

Objective
To evaluate the incidence of post-surgical hypoparathyroidism (PoSH) and to estimate the efficacy of implementing near-infrared autofluorescence (NIRAF) technology for parathyroid preservation compared to standard surgical technique.

Methods
Retrospective cohort study of consecutive unselected patients. Primary endpoints were the incidence of immediate/permanent PoSH and hypocalcaemia-related readmissions. A cost analysis compared current standard care expenses versus projected costs of NIRAF implementation.

Results
Between January 2023 and July 2024, 94 patients underwent total (n=77) or completion thyroidectomy (n=17), with (n=19) or without (n=75) lymph node dissection. Patients were routinely discharged on two weeks Calcichew supplementation, with or without Alfacalcidol, and followed up with surveillance biochemistry as indicated.

In our study post-operative PTH was in the upper half of normal range in 11 (12%) patients, the lower half of normal range in 71 (76%) patients, and undetectable in 12 (13%) patients, who were considered to have immediate PoSH. After a minimum of 4 months follow up, 3 (3.2%) patients remained with either undetectable PTH or were Calcium / Alfacalcidol dependent, two of whom had inadvertent parathyroid gland excision identified histopathologically. Three patients (3.2%) required readmission for symptomatic hypocalcaemia in the post-operative period. Incidence of immediate and permanent PoSH was not significantly related to either procedure type, lymph node dissection or underlying histopathology.

The total annual cost of managing complications of temporary/permanent PoSH, including readmissions, and long-term calcium supplementation, was estimated at GBP10,000. These costs compared favourably with the initial investment in NIRAF technology, estimated to be not less than GBP20,000, plus per-case costs exceeding GBP400.

Discussion
As only 2% of patients had permanent PoSH related to parathyroid tissue removed in the specimen, the likelihood of benefiting from routine use of intra-operative adjuncts remains questionable. Endeavouring to eliminate post-operative hypoparathyroidism, with its life altering consequences, remains a high priority for Endocrine Surgeons, however, in a high-volume unit it is unlikely to be cost effective in its current financial model.

Ethics
Quality improvement project registered with Oxford University Hospitals NHS Foundation trust.

Keywords
hypoparathyroidism, thyroidectomy, NIRAF, parathyroid preservation, cost-effectiveness.

D007

A case of difficult to treat thyroid storm in inflammatory bowel disease

K Hyams, M Sands, S Zac-Varghese.
Lister Hospital, East and North Herts NHS Foundation Trust.

Abstract

Background
Thyroid storm is a rare but potentially life-threatening exacerbation of thyrotoxicosis, requiring prompt investigation and treatment. It can lead to cardiovascular, thermoregulatory, neurological, and gastrointestinal dysfunction, and is fatal in 10-20% of cases.[1]
Patients with autoimmune thyroid disease are at risk of additional autoimmune conditions; this case highlights how co-morbidities can present challenges to effective management of thyrotoxicosis, and therefore the importance of multi-disciplinary input from various specialties.

Case History
A 25-year-old female with a background of Ulcerative Colitis and Ankylosing Spondylitis was diagnosed with Graves' disease after presenting to her GP with shortness of breath and palpitations. She was started on carbimazole and propranolol, however after 2 weeks her bowel frequency increased to 20-30 times per day. She was admitted to hospital and managed for the colitis flare with prednisolone. The carbimazole was switched to propylthiouracil (PTU) as the patient felt the carbimazole exacerbated her bowel symptoms. She was discharged home and subsequently started adalimumab. Her bowels remained poorly controlled, opening 10-15 times per day.

Around 7 weeks later, she presented to the Emergency Department with acute dyspnoea and palpitations, ongoing loose stools with blood and mucus, and a 1-week history of tremor, hot flushes, dizziness, leg weakness and fatigue. On examination she was flushed, tachypnoeic and tachycardic.

Bloods showed fT4 81.5, TSH <0.02.

The Burch-Wartofsky score was 30, indicating impending thyroid storm, and she was admitted to the Intensive Care Unit (ICU) for level 2 care (cardiovascular monitoring).

She was treated with IV beta-blocker, IV hydrocortisone and steroid enemas, cholestyramine, Lugol's iodine and the dose of PTU was increased (200mg TDS).

She was stepped down to the Gastroenterology ward 3 days later (for ongoing management of her colitis flare). Her colitis was managed with intravenous then oral and rectal steroids plus adalimumab. Her thyroid function improved with treatment of her UC; the fT4 improved to 19 after around 2 weeks in hospital. She developed a maculopapular urticarial rash likely secondary to the PTU. Definitive treatment of thyrotoxicosis was discussed, and she opted for a thyroidectomy.

Discussion
In this case, poorly controlled inflammatory bowel disease with diarrhoea and malabsorption made managing thyrotoxicosis challenging. Carbimazole was stopped after the patient suspected that it exacerbated her bowel symptoms. PTU was poorly absorbed due to ongoing bowel inflammation, and this was the likely trigger for the subsequent deterioration leading to thyroid storm.

Once her cardiovascular status had stabilised with acute management of thyroid storm, the mainstay of her management was to improve her bowel inflammation and hence improve absorption of PTU. Total thyroidectomy was not considered initially, given that lifelong levothyroxine would be needed, and reduced absorption would be problematic, risking severe

hypothyroidism.

This case highlights that multi-disciplinary teamwork is vital for patients with thyroid disease and IBD.

Points for discussion

1. How would the audience approach treatment in this case, given the potential issues with continuing medications, thyroidectomy, or radio-iodine?
2. Was carbimazole implicated in the worsening of the patient's bowel symptoms, or was uncontrolled hyperthyroidism driving this?

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D008

The complexities of thyroid eye disease

A Haddad[1], K Meeran[1], R Akshikar[2], S Partridge[3], D Peters[1], V Bravis[1], V Lee[2]
[1] Department of Endocrinology, Imperial College Healthcare NHS Trust.
[2] Department of Ophthalmology, Imperial College Healthcare NHS Trust.
[3] Department of Medical Oncology, Imperial College Healthcare NHS Trust.

Abstract

Thyroid eye disease (TED) is the major extra thyroidal manifestation of Graves' disease. Treatment decisions are based on clinical activity, severity, duration and impact on quality of life (QoL). Management requires multidisciplinary team (MDT) input in severe complicated cases.

We present a 59-year-old female patient, with no previous past medical history. She presented initially with watering eyes and no double vision, initially thought to be associated with seasonal allergies. One month later, she developed swelling of the right eye and was diagnosed with blepharitis. She was seen by an ophthalmologist, who referred her to oculoplastics. In the meantime, her GP started oral antibiotics, which improved the swelling, and, given clinical improvement, the patient cancelled her appointment with oculoplastics. She did not have any signs or symptoms of overt hyperthyroidism, and TFTs were normal at that point. A few months later, she developed severe pain in the left eye with persistent double vision. She consulted an Oculoplastic specialist, who diagnosed her with severe TED. Blood tests showed hyperthyroidism with raised TSH receptors antibodies confirming the diagnosis of Graves' disease and started antithyroid drugs. Euthyroid state was reached after a few weeks. Due to active TED, she commenced immunosuppressant therapy with 12 weeks course of intravenous methylprednisolone (IVMP) and oral mycophenolate mofetil (MMF). She remained under the care of the MDT TED clinic.

Unfortunately, after completing her systemic steroid course her TED flared, and she developed sight threatening TED. This required urgent orbital decompression surgery that initially improved her vision and provided a chance for the second line immunosuppression MMF to take effect. She then developed new painful swelling in the right eye, and completed 10 cycles of orbital radiotherapy. She also required orbital steroid injections to control her eye symptoms. This was followed by thyroidectomy, but her TED inflammation persisted, clinically and radiologically. She was therefore started on third line biological therapy with Tocilizumab. Fortunately, Tocilizumab has improved her pain, double vision and appearance. TED finally became quiescent. During this journey, the patient's QoL was tracked and was significantly impacted.

This case illustrates the importance of considering TED as and early differential diagnosis in patients that present with mild but classical signs, even when euthyroid status is confirmed. It is still unknown what the exact natural history of TED is, and whether early intervention can actually prevent the progression to sight-threatening disease. This further highlights the importance of MDT expertise in the management of patients with the condition.

D009

Severe osteoporosis secondary to bariatric surgery

R Vathenen, AR Ahmed, S Hameed, J Cox, AN Comninos*, P Behary*
*Co-senior authors
Imperial College Healthcare NHS Trust.

Abstract

Introduction
Bariatric surgery is becoming increasingly common and leads to significant benefits such as weight and cardiometabolic risk reduction. Major adverse cardiovascular events are reduced by over 40% at 4 years post-surgery (*1*). However, frequent complications include malnutrition, from malabsorption and excessive weight loss, leading to bone loss. Here we report a case of severe osteoporosis post-bariatric surgery.

Case report
A 66-year-old lady underwent a laparoscopic gastric band in 2010 for severe obesity with a body mass index (BMI) of 50.8kg/m^2 at the Imperial Weight Centre. Prior to surgery, she had a background of a previous eating disorder, gastro-oesophageal reflux disease and obstructive sleep apnoea. She had the gastric band removed due to band erosion in 2013 but this was followed by a one-anastomosis gastric bypass surgery (OAGB) in 2015, when she weighed 105 kg with a BMI of 46.6 kg/m^2. Over the next two years, she lost 38% of her body weight (weight 65 kg, BMI 29 kg/m^2). She suffered from protracted vomiting and reflux which restricted oral intake and led to severe malnutrition. Other complications included small bacterial overgrowth, severe lethargy, multiple nutritional deficiencies (thiamine, vitamin B12, magnesium, copper, selenium, zinc and vitamin D), anaemia and subsequent generalised myopathy. She required several admissions for intermittent enteral and parenteral nutrition, complicated by refeeding syndrome.

In January 2017, the OAGB was revised due to malnutrition and ongoing weight loss. During her admission for reversal surgery, she reported non-traumatic back pain and spinal imaging revealed multi-level vertebral fractures at T9-T12 and L1, which led to referral to our Endocrine Bone clinic.

Bone Health Management
Dual-energy X-ray absorptiometry (DEXA) scan showed marked osteoporosis at lumbar spine (T score of -4.8) and hips (total hips T-score -4.1). Urine N-telopeptide cross-links (NTX) were elevated at 392nmol/mmol (5-65) and pro-collagen type 1 N-terminal peptide (P1NP) at 361mcg/l (15-59), demonstrating rapid bone turnover. In addition, she was vitamin D insufficient (46.8nmol/l) with secondary hyperparathyroidism (calcium 2.34mmol/l, PTH 12.6pmol/l).

Apart from bariatric surgery, other risk factors identified for osteoporosis included a post-menopausal state, late menarche (17 years), lifelong lack of dietary calcium, maternal hip fracture and a three-month course of dexamethasone for appetite stimulation.

She was a candidate for teriparatide but she was not keen due to daily self-injection inconvenience. Denosumab was acceptable to her and this was commenced, alongside calcium and vitamin D supplementation. Urine NTX and serum P1NP were appropriately suppressed on denosumab. Bone mineral density (BMD) improved significantly and a DEXA scan at 2 years showed lumbar T-scores of -1.4 (+60.3%) and Total hips T-scores of -2.7 (+32.9%). Repeat DEXA at 5 years after initiation of denosumab showed maintenance of BMD at the spine and hips.

She remains troubled by chronic back pain but her mobility has improved and her weight has remained stable between 61kg and 65kg over the last 4 years. She continues on denosumab injections and has not sustained further fragility fractures while on treatment.
She had ongoing dietician support and nutritional supplementation and has been regularly

reviewed by specialist psychology and psychiatry services for body dysmorphia.

Discussion

This case demonstrates the potential severe detrimental impact of bypass surgery on bone health. Whilst the effects of OAGB on bone health have not been studied before, one would expect similar impact to traditional procedures such as Roux-en-Y gastric bypass (RYGB), as it leads to comparable weight loss and improvement in metabolic outcomes.

A reduction in lumbar and hip BMD, elevation of bone turnover markers, deterioration in bone microarchitecture and up to 2-fold increase in fracture risk have been described after RYGB.

Fracture risk is reported to rise approximately 2 years after RYGB, consistent with when our patient fractured (*2*). Proposed mechanisms include skeletal unloading, malabsorption especially of calcium and vitamin D, and alterations in gut hormones with potential negative bone effects, such as PYY (*3*). However, this patient's post-operative course was particularly complicated and severe, with nearly 50% weight loss (more than usually expected), significant malnutrition and a prolonged course of corticosteroids all contributing to more extensive bone loss than most patients.

Our patient required intervention with bone-specific agents and in this case, denosumab resulted in excellent improvements and no further fractures. Our patient's bone health was further aided from having a OAGB reversal to halt her catabolic state and regain/stabilise her weight via the intervention of other members of the MDT (dieticians and psychologists). Whilst the cardiometabolic benefits of bariatric surgery are invaluable, this case highlights its detrimental effects on bone health and the need to address it proactively.

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D010

Ferinject-induced prolonged hypocalcaemia: an under recognised phenomenon?

R Vathenen, J Cox, T Vakilgilani.
Imperial College Healthcare NHS Trust.

Abstract

Introduction
Hypohosphataemia is a known side effect of iron infusions, particularly Ferinject (ferric carboxymaltose), where incidence of up to 50% is reported (*1*). However, hypocalcaemia is not a well recognised phenomenon. Here we present a case of prolonged severe hypophosphataemia and hypocalcaemia secondary to Ferinject.

Case report
A 49 year old lady was referred to the medical take for incidental severe hypocalcaemia (1.74mmol/l) and hypophosphataemia (0.28mmol/l). She felt generally lethargic with no specific symptoms of hypocalcaemia. She was under investigation for suspected malignancy with a history of 20kg weight loss, back pain, headaches and vomiting. She had a background of breast cancer treated with wide local excision, radiotherapy and neo-adjuvant chemotherapy (fluorouracil, epirubicin, cyclophosphamide, docetaxel and Herceptin) 7 years previously. She had previous mild proctitis secondary to Crohn's disease. Medication history was significant for esomeprazole 40mg OD. She had received two Ferinject infusions and intramuscular vitamin D replacement one month prior. She did not receive any antiresorptive agents.

Repeat blood tests confirmed severe hypocalcaemia and hypophosphataemia, with secondary hyperparathyroidism (49.5pmol/l), normal magnesium (0.76mmol/l) and vitamin D (51.8nmol/l). Urine phosphate was inappropriately normal at 9.25mmol per 24 hours (more than 3.2mmol), demonstrating a renal phosphate leak, with undetectable urine calcium excretion (<0.59mmol/24 hours). Fibroblast Growth Factor-23 levels were requested. Extensive imaging was unremarkable: CT chest, abdomen and pelvis, MRI head and whole spine, bilateral mammogram and breast ultrasound.

She was initially managed with intravenous calcium gluconate and phosphate polyfusor infusions, then switched to oral supplementation. She was later discharged on alfacalcidol 0.5mcg daily, Calcichew 1g twice daily and sodium phosphate 16mmol twice daily. Despite this she had persistent hypocalcaemia and hypophosphataemia. This required a further admission for polyfusor infusion, and increased oral calcium and phosphate replacement. Esomeprazole was changed to famotidine in case of PPI-induced calcium malabsorption.

Hypophosphataemia was felt to be secondary to Ferinject, but a malabsorptive cause for her hypocalcaemia required consideration given previous Crohn's and low urine calcium excretion. A thorough screen was negative including a normal faecal elastase (329mcg/g), MRI small bowel and recent colonoscopy. FGF-23 returned elevated at 336RU/ml. After 3 months of treatment, she was successfully weaned off alfacalcidol. At time of writing, calcium is 2.22mmol/l and phosphate 1.17mmol/l with a PTH of 13pmol/l. She is clinically well.

Discussion
This case illustrates a severe and prolonged course of hypocalcemia and hypophosphataemia after Ferinject. Hypophosphataemia appears to be mediated through elevated FGF-23 levels, reducing intestinal and tubular phosphate absorption (*2*). Hypocalcaemia is also described in the literature (*3*). Whilst its pathophysiology is less well known, it could conceivably be similar to in oncogenic osteomalacia, given the common FGF-23 pathway. FGF-23 reduces vitamin D hydroxylation, leading to reduced intestinal and renal absorption, and this is further supported by reduced 1,25-vitamin D levels after Ferinject (*3, 4*). Further studies are required to establish the

aetiology and exact incidence of hypocalcaemia secondary to Ferinject.

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D011

Opportunistic Fungal Infection in a Patient with Ectopic Cushing's Syndrome: A Multidisciplinary Approach to Diagnosis and Management

R Minhas[1], D Peters[1], R Walls[1,2], AH Sam[1,2], F Wernig[1,2]
[1] Imperial College Healthcare NHS Trust
[2] Imperial College London

Patients with hypercortisolaemia, as first noted in Cushing's original description, are predisposed to opportunistic fungal infections due to cortisol's immunosuppressive effects. Common pathogens include *Pneumocystis jirovecci*, *Cryptococcus neoformans,* and *Nocardia* spp. (1) While most reported cases involve monomicrobial infections (2), polymicrobial infections have also been documented (3)

We present the case of a 45-year-old man with ectopic Cushing's syndrome, manifesting with central obesity, proximal myopathy, and osteoporotic spinal fractures. He had a history of iron deficiency anaemia secondary to haemorrhoids and was recently diagnosed type 2 diabetes, hypertension, and hypercholesterolaemia. Endocrine evaluation revealed hypokalaemia (potassium of 3.5 mmol/L) and hypercortisolaemia with a 24-hour urinary free cortisol level of 3876 mmol/day and a non-suppressible post- dexamethasone cortisol level of 712 nmo/L with ACTH of 105 ng/L. Salivary cortisol and cortisone were significantly raised at 42.5nmol/L and 158.4nmol/L respectively. MRI pituitary did not reveal any convincing intrapituitary abnormality.He was commenced on rivoroxaban 10mg daily for DVT prophylaxis and inferior petrosal sinus sampling (IPSS) did not exclude an ectopic source of ACTH secretion. Following IPSS, the patient was started on incremental doses of metyrapone dose reaching a daily dose of 6g to control cortisol excess.

A 68Ga DOTATATE PET/CT showed a cluster of nodules within the apical segment of the left lower lobe demonstrating low-level DOTATATE uptake, with no avid source of ectopic Cushing's syndrome elsewhere. A CT thorax was subsequently requested that showed Cluster of 3-4 pulmonary nodules in the superior segment of the left lower lobe showing minimal cavitation with no evidence of lymphadenopathy or metastatic disease. He went on to have FDG PET/CT that showed moderate to intense tracer uptake in left lower lobe lung nodules, two of which had clear central cavitation. A CT-guided biopsy suggested a fungal aetiology, with differental including *Cryptococcosis, Histoplasmosis, Coccidiomycosis,* and *Blastomycosis*.

This case highlights the importance of maintaining a high index of suspicion for opportunistic infections fungal infections in severely hypercotisolaemic patients, whether due to endogenous or iatrogenic causes. It also underscores the critical role of multidisciplinary collaboration in diagnosing and managing complex cases of ectopic Cushing's syndrome and associated complications. Early recognition and targeted treatment of fungal infections are vital to prevent severe complications, especially in immunocompromised individuals. The search for a source of ectopic ACTH production continues.

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D012

Gestational trophoblastic disease: A rare cause of thyroid storm

S Samarasinghe, T Talbot, N Sarwar, E Hatfield, K Meeran.
Imperial College Healthcare NHS Trust.

A 28-year-old female was referred to our centre following an evacuation of suspected molar pregnancy at 7/40 gestation. She had developed PV bleeding and had an evacuation of retained products of conception (ERPC) two days later. The histology was non-diagnostic. Past medical history was significant for a recent diagnosis of hepatitis A, which was conservatively managed, and a previous termination. Thirteen days following ERPC, local serum beta hCG was significantly elevated at >1,000,000 IU/L. She was referred to oncology for the management of presumed gestational trophoblastic disease (GTD). On arrival, she reported fatigue, anorexia, breast tenderness, mood swings, headaches, exertional palpitations, lower abdominal pain and PV bleeding. Observations at the time were a heart rate 141 bpm, blood pressure 108/68, oxygen saturation 100%, temperature 36.3C, and respiratory rate 19 bpm. Ultrasound pelvis showed a bulky uterus with a mixed cystic and solid mass within the uterine cavity. Admission blood tests demonstrated anaemia and biochemical hyperthyroidism with a suppressed TSH <0.01 mU/L (0.30-4.20), free T3 15.9 pmol/L (2.4-6.0), free T4 39.5 pmol/L (9.0-23.0), Hb 86 g/L (114-150), MCV 87.5 fL (83.5-99.5), eGFR >90 ml/min/1.73m^2 , ALT 45 u/L (0-34), adjusted Ca 2.28 mmol/L (2.20-2.34), phosphate 1.09 mmol/L (0.80-1.02), presenting radioimmunoassay beta hCG 2,602,046 IU/L (<5). CT chest, abdomen and pelvis showed uterine appearances in keeping with gestational trophoblastic disease, ovarian theca lutein cysts (reflecting high circulating β-hCG) and no evidence of distant metastases. The patient commenced emergency low-dose chemotherapy (Etoposide + Cisplatin) with dexamethasone. On day 1 post-EP, she developed acute dyspnoea; the echocardiogram showed hyperdynamic myocardium, and the CT chest showed new consolidation in keeping with possible ARDS or pulmonary haemorrhage. She had clinical signs suggestive of thyroid storm and was started on propranolol 40 mg TDS and propylthiouracil 200 mg QDS. She was later managed with definitive EMA-CO chemotherapy and continues to respond well to this.

There was an improvement in her thyroid function tests (below), and after two weeks, the PTU was reduced to 200 mg BD. This case of thyroid storm in the context of high-risk GTD illustrates the thyrotropic effects of β-hCG. She remains euthyroid on treatment and continues chemotherapy for GTD.

Date	TSH (0.30-4.30 mU/L)	Free T3 (2.4-6.0 pmol/L)	Free T4 (9.0-23.0 pmol/L)	Beta hCG (IU/L)
23/08/2024	<0.01	15.9	39.5	2,330,330
24/08/2024	<0.01	15.6	31.9	
02/09/2024	<0.01	6.1	21.5	2,314,877
06/09/2024	<0.01	4.8	14.6	1,457,348
16/09/2024	1.15		10.3	187,908

D013

Refractory Hypercalcemia and Seizures in a Patient with Parathyroid Adenoma: A Case for Timely Surgical Intervention

L Eltayieb, F Seif, M Bashir, D Sadulah, G Mlawa, M Dram, A Al-Ghrairi, F Hussein, A Mohamed, S Cadiz, A Can, S Reddi, K Carter.
Barking, Havering and Redbridge University Hospitals NHS Trust.

Abstract

Introduction
The clinical manifestation of severe hypercalcemia includes general lethargy, drowsiness, and confusion. Seizures as a clinical picture of severe hypercalcemia are rare.

Case
We present a case of a 75-year-old female who initially presented with nonspecific symptoms including confusion, forgetfulness, reduced appetite, and constipation over several days. Her past medical history includes parathyroid adenoma, obstructive sleep apnoea (OSA), hypertension, and diverticular disease. During her hospital admission, the patient developed twitching and generalized tonic-clonic seizures. Initial bloodwork revealed a markedly elevated serum calcium level of 3.96 mmol/L and a significantly raised parathyroid hormone (PTH) level (45). Mild renal impairment was observed with a serum creatinine level of 115 µmol/L (normal range 45–90 µmol/L). She was admitted to ITU, intubated, and initiated on aggressive treatment for hypercalcemia, including intravenous fluids and bisphosphonate therapy. Despite these interventions and the addition of cinacalcet, her calcium levels remained persistently elevated and refractory to treatment. She continued to experience twitching and seizures, which were managed with benzodiazepines and antiepileptics.

A comprehensive workup, including a CT and MRI of the head, showed mild chronic periventricular ischemic changes but no acute infarction or mass effect. An EEG displayed diffuse global cerebral dysfunction. The neurology team assessed her and attributed the seizures to metabolic encephalopathy .They have advised on correcting her electrolyte disturbances. During her ITU stay, a DVT was identified in the internal jugular and subclavian veins, for which she was started on therapeutic low-molecular-weight heparin (LMWH).

After approximately one month in ITU, her symptoms began to improve, and she was stepped down to a general medical ward. However, her hypercalcemia persisted despite continued IV fluids, bisphosphonates, and cinacalcet. Parathyroid imaging identified an oval hypoechoic lesion on the inferior lobe of the right thyroid (30x22 mm) and another on the posterolateral aspect of the left thyroid (9x4.7 mm), suggestive of parathyroid adenomas or hyperplasia. A sestamibi scan demonstrated right inferior parathyroid adenoma. She was discussed in the MDT and planned for surgery .She underwent emergency parathyroidectomy which was successful
and resulted in normalisation of calcium levels ranging from 2.20-2.56 mmol/, and her seizures resolved entirely.

Discussion
This case underscores the complexities of managing severe, refractory hypercalcemia, particularly in the context of parathyroid adenoma. The patient's presentation, marked by confusion, twitching, and potential generalized seizures, exemplifies the neurological sequelae of significant hypercalcemia. Although hypercalcemia generally reduces neuronal excitability, in more severe or rapidly progressing cases, complications such as cerebral vasoconstriction and hypertensive encephalopathy may arise, exacerbating the risk of seizures.
Electroencephalographic (EEG) findings in hypercalcemia frequently reveal fast activity, bursts of delta and theta waves, and, at critically elevated calcium levels, triphasic waves and occipital spike-slow-wave complexes.

Despite an aggressive regimen of intravenous fluids, bisphosphonates, and cinacalcet, the patient's hypercalcemia remained intractable, with persistent seizures unresponsive to medical management. The underlying etiology in this case was parathyroid adenoma, driving excessive parathyroid hormone secretion and perpetuating elevated calcium levels.

Conclusion

In conclusion, this case exemplifies the critical need for timely intervention in severe hypercalcemia, particularly when accompanied by neurological symptoms such as confusion and seizures. Despite rigorous pharmacologic management, the persistence of hypercalcemia necessitated urgent parathyroidectomy, resulting in swift normalization of calcium levels and notable clinical improvement. This case emphasizes the importance of a comprehensive, multidisciplinary approach and the pivotal role of surgical intervention when medical therapies fail particularly in the context of hypercalcemia secondary to parathyroid adenoma.

Questions for discussion

1. Have you managed a case of hypercalcemia with seizures? What was your approach?

D014

Management of carbimazole-induced liver function derangement in everyday endocrinology

C Russell, F Tilby-Jones, M Sanas.
East and North Hertfordshire NHS Trust.

Abstract
We report the case of a 36-year-old female who presented to the endocrinology clinic in early May 2024 with a 3-month history of thyrotoxicosis symptoms (anxiety, tremor, and palpitations). She has a background of asthma and anxiety and depressive disorder. Her investigations showed thyroid stimulating hormone (TSH) of <0.03 mU/L, free T4 of 30.8pmol/L, and thyroid stimulating immunoglobulin (TSI) levels of 2.56 IU/L. A diagnosis of Graves' thyrotoxicosis was made.
She was commenced on carbimazole 20mg once daily via advice and guidance. She had already been started on propranolol 10mg TDS by her GP to control her symptoms. She was subsequently seen in clinic.

Three weeks after commencing carbimazole, the patient developed new-onset paroxysmal itching. Blood tests taken at that time demonstrated an isolated elevated alkaline phosphatase (ALP) level of 175 IU/L (previously 128 IU/L in April 2024). Her bilirubin and other liver function tests (LFTs) were within normal limits. She was urgently seen again in clinic. Her abdominal ultrasound and liver screen were normal. Given that the patient's ALP was not significantly raised with no other LFT derangement, carbimazole was continued with the aim of gradually reducing her carbimazole dose whilst keeping free T3 and T4 in normal range. Her carbimazole dose was initially reduced to 20mg on alternate days.

However, the reduction in carbimazole dose resulted in the patient becoming thyrotoxic again. Thus, carbimazole dose was up-titrated to 20mg once daily.

Subsequently, her T4 normalized (free T4 of 18.5pmol/Lin August 2024). On review in September, her carbimazole was reduced to 15mg daily. On repeat blood tests performed in early November, her ALP had slightly improved, from the initial 188 IU/L to 173 IU/L.

Given that the patient's liver imaging and screen was normal, there are several possible causes that may explain the rise in her ALP levels post-carbimazole treatment.

One potential aetiology is thyrotoxicosis-induced cholestasis. The interplay between thyroid hormones, bilirubin metabolism and hepatocytes is complex and a cholestatic picture is sometimes seen in patients with thyrotoxicosis (1). Whilst various case studies have previously demonstrated this, they often show additional liver function derangements (2, 3). Furthermore, the rise in ALP was not present until the commencement of carbimazole.

Obesity may also play a role, as the patient has a high BMI and a previous history of elevated ALP levels (an isolated value of 144 IU/L in 2017), before her Grave's disease diagnosis. It has been observed that there is a possible linear relationship between BMI and ALP (4). However, her ALP prior to carbimazole commencement had been repeatedly in normal range.

Therefore, the most likely cause is carbimazole-induced cholestasis, an idiosyncratic reaction that disrupts bile excretion, raising ALP and bilirubin levels (5, 6). This explanation aligns with the timing of elevated ALP after starting carbimazole and partial improvement when the dose was reduced.
This case underscores the challenge of managing Graves' thyrotoxicosis in patients with abnormal LFTs: balancing thyroid function improvement whilst minimising liver dysfunction. Whilst existing literature often addresses severe cholestasis with thyrotoxicosis or carbimazole treatment, more research is needed on managing mild cases, a common issue in everyday endocrinology.

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D015

A working diagnosis of cerebral salt wasting in conjunction with subdural haematoma

J Choudhury, I Ilesanmi**.**
East and North Hertfordshire NHS Trust

Abstract

Introduction
Cerebral salt wasting (CSW) describes a condition of hypovolaemic hyponatraemia with elevated urinary sodium excretion, typically seen following intracranial pathology. This case describes an 86-year-old male who developed CSW following surgical evacuation of a chronic subdural haematoma (SDH), demonstrating the challenges in differentiating CSW from SIADH.

Case summary
An 86-year-old male presented to the Emergency Department following an unwitnessed fall, with subsequent left-sided weakness. Initial CT imaging revealed a right-sided acute on chronic subdural haematoma with significant mass effect, necessitating transfer to a tertiary centre for surgical evacuation.

Following repatriation to the local hospital, serial sodium measurements demonstrated a decline from an initial presentation value of 137 mmol/L to 124 mmol/L (Table 1). Initial management involved fluid restriction to <1.5L daily based on a presumptive diagnosis of SIADH. Contributing medications including amitriptyline and omeprazole were stopped, with the latter switched to famotidine. However, the patient's sodium continued to decline despite these measures.

Further investigation revealed a urinary sodium of 76.9 mmol/L and urine osmolality of 650 mOsm/kg, suggesting CSW rather than SIADH. Treatment was modified to include 0.9% saline infusion. This led to an increase to a transient increase in the level of sodium noted from 124 mmol/L to 129 mmol/L as noted in Table 1. During this period however, the patient experienced deteriorating neurological symptoms with worsening left-sided weakness, particularly marked in the lower limb. Repeat CT imaging confirmed recurrence of acute on chronic SDH with a maximal depth of 1.5 cm and 3mm midline shift, necessitating re-transfer to the tertiary centre for neurosurgical intervention. The patient was successfully transferred and underwent repeat neurosurgical intervention.

Table 1: Sodium level throughout admission – key events during admission

Date	Sodium Level (mmol/L)	Clinical Context/Interventions
15/07/24	137	Initial presentation to ED
26/07/24	128	Post-repatriation from Addenbrooke's
27/07/24	127	
28/07/24	127	Amitriptyline stopped; omeprazole switched to Famotidine
29/07/24	129	

30/07/24	126	Hyponatremia screen done: Serum osmolality: 264 Urine Na: 76.9 Urine osmolality: 650
31/07/24	127	
01/08/24	127	
02/08/24	124	Slow Na tablets stopped; Normal saline started
05/08/24	125	
06/08/24	129	Discussed with neurosurgery, due to increased neurological symptoms
07/08/24	128	

Learning points

Close monitoring of both neurological status and sodium levels is essential in managing these complex cases. Fluid restriction, while being the main treatment in SIADH, is counterproductive in CSW and would cause the patient to deteriorate further.

D016

The Role of Tolvaptan in an Outpatient Setting

B Bhatt[1,2], H Hussain[1], M Baalawi[3], A Can[1], I Ibrahim[1], G Mlawa[1]
[1] Barking, Havering and Redbridge University Hospitals NHS Trust.
[2] University College London (UCL) Medical School.
[3] Barts Health NHS Trust.

Introduction
Tolvaptan is a vasopressin-V2 antagonist, used in the treatment of hyponatremia. It functions through targeting type-2 receptors in the distal renal tubules, inhibiting the action of the hormone vasopressin. As a result, water reabsorption is reduced and excretion is increased, thus correcting the dilutional hyponatremia and balancing serum sodium levels.

Currently, Tolvaptan is most used for short-term, inpatient treatment. Due to its mechanism, Tolvaptan presents a high risk of rapid, overcorrection of serum sodium levels. Maximal change occurs within the first 24 hours, and it is therefore recommended that sodium levels are closely monitored during this period to prevent adverse events. However, there has been recent discussion into potential efficacy of this drug in the outpatient setting. We have explored a patient case where Tolvaptan was successfully provided as a longer-term treatment for symptomatic hyponatremia.

Case Report
A 72-year-old patient with a background of SIADH and left-sided adrenal adenoma had recurrent admissions to hospital since January 2023, due to acute confusion and vacant episodes secondary to euvolemic hyponatraemia (lowest Na: 119). This symptomatic hyponatraemia was treated in hospital and the patient was advised to fluid restrict to 750ml and take demeclocycline and oral sodium tablets. However, it failed to correct the sodium and has led to multiple admissions, with some requiring intensive care support. On one admission, the patient was started on Tolvaptan as an inpatient, to which she showed a good response. She was discharged with 3.75mg of Tolvaptan to be taken once a day as an outpatient, which has corrected her sodium (most recent Na: 134) and led to fewer admissions in hospital.

Discussion/ Conclusion
This case illustrates the potential role of Tolvaptan in the management of hyponatraemia in an outpatient setting. Tolvaptan offers a targeted approach by antagonising vasopressin receptors, thus promoting free water excretion without affecting sodium retention. This treatment may be particularly beneficial for patients with recurrent, symptomatic hyponatremia who fail to respond to conventional therapies. This patient's successful management with Tolvaptan suggests that it can be a valuable therapeutic option in the outpatient care of patients with SIADH, leading to better sodium control and fewer hospitalisation episodes. Further studies are needed to better define its long-term efficacy and safety in this context.

D017

Timely surgical intervention in a patient with pituitary apoplexy and cavernous Sinus Invasion

AUTHORS:
F Hussein, B Mahamud, L Eltayieb, E Shakoor, G Mlawa, F Asghar, D Sadulah, H Hussain, A Alnashrati, A Mohamed, A Al-Ghrairi, L Auguste, B Hossain, S Sawar, S Cadiz.
Barking, Havering, and Redbridge University Hospitals NHS Trust.

Abstract

Introduction
Pituitary apoplexy is a rare but potentially life-threatening emergency characterized by sudden haemorrhage or infarction of the pituitary gland, requiring immediate medical attention. The urgency is heightened when cavernous sinus invasion is present, as this significantly increases the risk of neurovascular compromise and other severe complications. We present a case of pituitary apoplexy complicated by cavernous sinus invasion, successfully managed with transsphenoidal surgery. This highlights the critical importance of early surgical intervention in achieving favourable outcomes and preventing complications such as cranial nerve damage, carotid artery injury, and cavernous sinus thrombosis.

Case Presentation
A 50-year-old male presented with a 1-day history of severe headaches, vomiting, and right-sided ptosis. Initial investigations, including blood tests and imaging, were conducted to rule out other emergencies. Laboratory results showed IGF-1 of 113 ng/mL, prolactin of 86 ng/mL, cortisol of 41 nmol/L, TSH of 3.43 mIU/L, T4 of 5.8 µg/dL, LH of 12 IU/L, and FSH of 11 IU/L. An MRI revealed a well-circumscribed pituitary mass in the right paracentral region with invasion into the right cavernous sinus, measuring 3x2x1.5 cm. The patient's medical history included excess alcohol intake and type 2 diabetes mellitus. He underwent transsphenoidal surgery without complications, leading to the resolution of right-sided ptosis. Postoperatively, he is on hormone replacement therapy with levothyroxine, testosterone and hydrocortisone.

Conclusion
Pituitary apoplexy complicated with cavernous sinus invasion warrants prompt surgical decompression to prevent neurovascular compromise of important structures as well as control bleeding within the cavernous sinus. Left untreated pituitary apoplexy with cavernous sinus invasion can lead to serious complications. The literature supports that immediate surgical decompression, coupled with hormone replacement therapy, is essential for optimal patient recovery and long-term prognosis. Hence, an multidisciplinary team approach is necessary for management of these patients.

Questions for discussion
1. Should surgical intervention be done as an emergency or electively?
2. If there is recurrence, would you advocate for further surgical intervention?
3. What is an appropriate timescale for follow-up for this patient?

D018

Identification and Management of Pseudohypoparathyroidism Type 1a in a District General Hospital

R Choudhary[1], A Ishola[2], S Zac-Varghese[3]
[1] UCL medical school, final year student.
[2] Ayoola Ishola, Cambridge University, year 5 medical student.
[3] Sagen Zac-Varghese, East and North Herts NHS Trust.

Abstract
Pseudohypoparathyroidism type 1a (PHP1a) is a rare genetic condition marked by resistance to parathyroid hormone resistance and the distinctive skeletal features of Albright's hereditary osteodystrophy. It results from inactivating mutations in GNAS gene which lead to end-organ parathyroid hormone resistance and disrupted calcium homeostasis. PHP1a is one of the most common subtypes within the pseudohypoparathyroidism group of disorders. Due to its clinically variable presentations, PHP1a is often misdiagnosed.

Here, we report a case of a 49-year-old male presenting with non-specific fatigue, confusion, and skeletal abnormalities. Mr P was referred to our endocrine service with a clinical diagnosis of hypoparathyroidism being managed with 1-alfacalcidol 2mcg. He had features of a round face, wide set eyes and, shortened 4th and 5th metacarpals. On referral, he had had no genetic confirmation of his diagnosis, however, he reported that his mother had hypoparathyroidism and had similar facial features. His main concern was multiple areas of calcific deposits in his hands, shoulders, calves and feet, some of which had been previously surgically excised. Mr P also had hypothyroidism and was treated with 150 mcg thyroxine.

Genetic testing (R293) for Albright hereditary osteodystrophy, pseudohypoparathyroidism pseudopseudohypoparathyroidism, acrodysostosis andosteoma cutis confirmed a genetic diagnosis of a GNAS-related disorder. Mr P was heterozygous for a pathogenic GNAS variant which likely was inherited from his mother.

Management includes measurement of PTH, 25-OH vitamin D and calcium at least yearly in adults and more frequently during key events such as growth spurts, pregnancy and during sickness.

Treatment of this condition is centred around correcting Calcium and Phosphate imbalances, as well as alleviating symptoms. This is primarily done through Calcium and Vitamin D supplementation. There is a slight difference in the targets of adjusted calcium compared to other forms of hypoparathyroidism and supplements should target normal levels of calcium and phosphorus whilst avoiding hypercalciuria. Establishment of the molecular diagnosis is helpful for ongoing management, screening, early recognition of complications and genetic counselling.

This case underlines the challenges of identifying PHP1a and emphasizes the need for heightened awareness of its subtle, heterogeneous symptoms to improve diagnostic accuracy and patient outcomes.

D019

Navigating Acromegaly and Fertility: A Complex Case of Multidisciplinary Management and Postpartum Planning

E Mohamed, A Theodoraki, A Wren.
Chelsea and Westminster Hospital

Abstract

A 30-year-old woman presented with primary infertility and progressive oligomenorrhea. Pituitary profile revealed an unexpected diagnosis of acromegaly. Despite only minor symptoms of sweaty palms, IGF-1 was markedly elevated (90 nmol/L, reference <39 nmol/L), and OGTT showed marked persistent GH elevation (average GH 23.3 µg/L, reference <0.4 µg/L). MRI revealed a pituitary macroadenoma invading the left cavernous sinus, a risk for incomplete resection.

Management

Pre-operative treatment with Lanreotide autogel 120 mg every 4 weeks, aiming for tumour shrinkage to optimise chances of curative resection, unfortunately did not reduce tumour size or GH/IGF-1 levels. Trans-sphenoidal surgery was performed, with near-complete resection. The post-op OGTT showed residual disease with average GH 3.25 µg/L on OGTT and persistently high IGF-1 (68.3 nmol/L). Postoperative cortisol deficiency developed, managed with hydrocortisone. Histology confirmed a sparsely granulated somato-mammotroph tumour, typically less responsive to somatostatin analogues, aligning with her pre-surgery treatment-resistance.

Despite the incomplete biochemical response, regular menstrual cycles resumed, and she wished to conceive before further definitive treatment. She rapidly conceived naturally. Pregnancy was uncomplicated, managed with hydrocortisone and frequent monitoring, resulting in a healthy term birth.

MDT Considerations Postpartum

The patient wished to breastfeed, which precluded medical therapy post-partum. Redo surgery was the preferred initial strategy, due to long term safety concerns about radiotherapy / gamma knife in young patients. Two possible foci of residual disease were seen on post-partum MRI and Methionine PET imaging was suggested at inter-regional MDT, for better surgical planning. This will be performed when the patient feels ready for further surgery. The risks and benefits of early treatment vs delayed treatment to allow a second pregnancy were discussed and will be presented.

Conclusion

Methionine PET imaging has emerged as a valuable tool in assessing residual disease in acromegaly. Balancing desire for fertility against maternal risk of ongoing GH burden requires MDT input and detailed discussion with informed and empowered patients.

D020

Unraveling the Complexity of SDHA Pathogenic Germline Variants in GIST: A Report of Two Distinct Cases

E Mohamed, D Morganstein, Terri McVeigh.
Chelsea and Westminster Hospital.

Background
Pathogenic germline variants (PGVs) in the SDHA gene are found in those with wild type GIST (gastrointestinal stromal tumors) and paraganglioma. However, the low penetrance of these variants necessitates careful interpretation to avoid over investigation. National Guidelines recommend only mutations in those with an SDHA related tumor should be acted on.

Case 1
A 60-year-old male with a background of type 2 diabetes and colonic polyps developed prostate cancer, treated by total prostatectomy. He was then admitted with upper gastrointestinal bleeding, revealing a GIST, treated with partial gastrectomy.

A strong family history of malignancies prompted a referral for genetic testing. Two mutations were identified in BRCA2 and the SDHA.

There was no clinical evidence of a paraganglioma. The GIST was identified as a PDGFRA variant on somatic testing, indicating that it was not a wild-type GIST. This was confirmed by preserved SDHA staining on immunohistochemistry. As such, no further surveillance was deemed necessary.

Case 2
A 59-year-old female had a CTPA scan in July 2022. An incidental finding of GIST was found, confirmed at endoscopic ultrasound. Her family history was positive for brain tumors and breast cancer.

She received neoadjuvant imatinib, achieving notable tumor shrinkage before surgical resection in March 2023. Somatic testing showed an SDHA mutation with 91.2% variant allele frequency, indicating loss of heterozygosity, with no other driver variants identified. The SDHA variant was confirmed as germline with tumor profiling showing LOH, supporting causality but classifying it as a wild-type variant. Although recent scans show no PPGL evidence and negative functionality, ongoing surveillance and family genetic testing are recommended.

Conclusion
The identification of SDHA PGVs in individuals with associated tumors necessitates careful assessment. Those with non-associated tumors should be regarded as non-actionable, with no surveillance of family testing required. However, those with a praganglioma or wild type GIST should be managed as other SDH gene mutation carriers.

D021

Thyroid Storm in Acute Setting: Would You Miss It?

F Hussein, S Cadiz, A Can, I Ibrahim, L Eltayeib, B Bhatt, F Seif, D Lunda-Ngandu, M Dram, G Mlawa.
Barking, Havering, and Redbridge University Hospitals NHS Trust.

Abstract

Background
Thyrotoxicosis is one of the most common endocrine disorders and its severe form can manifest as a thyroid storm in an acute setting leading to organ dysfunctions including heart failure.

Case
A 50-year-old man presented to the emergency department (ED) with a 3-week history of weight loss, palpitations, tremors, breathlessness, and leg swelling. He was initially discharged from Queens Hospital ED with furosemide as a suspected diagnosis of heart failure.

CXR on admission demonstrated clear lungs fields. An inpatient echocardiogram was not undertaken. An echocardiogram after admission demonstrated normal left ventricular function (LVEF > 55%), normal right ventricular function, mild MR and Pasp 22-27mmHg.

He was initially discharged from Queens Hospital ED with furosemide as a suspected diagnosis of heart failure.

Patient remained feeling unwell and his GP performed further blood tests including TFTs. This showed a suppressed TSH and a free T4 >100, T3 35. ALT 53, Calcium 2.72. He was subsequently referred to the Acute Medicine Department at Queens Hospital.

Upon review, there were clear symptoms of thyrotoxicosis. His lower limb swelling had improved with the furosemide prescribed previously. His Burch-Wartofsky Score was 30 suggestive of impending thyroid storm.

The case was reviewed by an endocrinologist and the following medications were commenced:

- 4g once daily Cholestyramine 2 weeks
- 30mg Prednisolone 5 days (following stat dose hydrocortisone 100mg IV in ED)
- 20mg twice daily Carbimazole
- 20mg three times a day Propranolol (following stat dose 40mg in ED)
- Furosemide 40mg once daily

He was followed up in the Endocrine clinic and his symptoms had improved

Discussion
Thyrotoxicosis is a life-threatening complication of hyperthyroidism, clinically it is manifested as thyroid storm and triggered by a secondary external event such as infection, myocardial infarction, trauma or surgery.

It is crucial to identify the underlying aetiology leading to the severe clinical manifestation of thyroid storm to start appropriate treatment.

The consequence of delayed diagnosis and lack of correct treatment can lead to further complication such as delirium, thromboembolic disease, cardiovascular collapse and eventually death.

Currently, the Burch Wartfosky point scale is used to diagnose thyroid storm. In this scale, the following are assessed: temperature, cardiovascular dysfunction, central nervous system derangements, gastrointestinal symptoms and heart failure. A score greater than or equal to 45 aligns with a clinical diagnosis of thyroid storm, and scores between 25 and 44 suggest thyroid storm as a likely diagnosis. Scores below 25 points make a diagnosis of thyroid storm unlikely.

Conclusion

Thyroid storm is a medical and an endocrine emergency and appropriate and timely treatment will ensure better patient care and outcomes.

In an emergency like acute thyrotoxicosis or thyroid storm, higher doses of antithyroid medications, beta-blockers as well as glucocorticoids can be used to return the patient to a euthyroid state.

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D022

Challenges of Managing AVP Deficiency in Pregnant Woman with Hyperemesis Gravidarum

L Eltayieb, F Hussein, A Mohamed, E Shakoor, L Auguste, D Sadulah, A Al-Ghrairi, B Mahamud, H Hussain, B Hossain, C Otigbah, A Karaca, G Mlawa.
Barking, Havering, and Redbridge University Hospitals NHS Trust.

Abstract

Introduction
Managing AVP deficiency in pregnancy can be challenging due to the physiological changes associated with pregnancy. These changes can impact both the mother and the unborn baby. Vasopressinase, an enzyme released by the placenta, acts as a degrader of vasopressin. In a healthy female, this enzyme has an insignificant effect. However, in women with incomplete AVP deficiency, the presence of vasopressinase can exacerbate the condition during pregnancy, leading to worsening polyuria and polydipsia. These women may require higher doses of Desmopressin to manage their symptoms effectively.

Case Presentation
A 35-year-old lady presented with headaches and hyperemesis gravidarum in the antenatal clinic and maternity triage unit. Nobly her background included a diagnosis of AVP deficiency following a previous miscarriage at 16 weeks. During subsequent pregnancy, she developed frank AVP deficiency. She had polyuria and polydipsia (drinking up to 20 litres/24 hrs).

The neurology team assessed and reviewed her. She had an MRI pituitary with normal findings. She was treated for gestational diabetes with metformin and she was on desmopressin for her established AVP deficiency. Blood tests since January 2017 showed sodium level ranging between 133 – 144 mmol/L with noticeably a decline in sodium level to 130 mmol/L in January 2024. Serum osmolality was found to be within the range of 275-295 mmol/kg and urine osmolality was intermediate (between 500-600 mmol/kg). The desmopressin dose was adjusted accordingly with close monitoring.

Conclusion
Pregnancy is associated with increased blood volume and glomerular distraction rate. The risk of Gestation AVP deficiency in patients with already established AVP deficiency cannot be ignored, and the dehydration is significant especially if the patient developed hyperemesis gravidarum.
In summary, managing AVP deficiency in pregnancy can be challenging and warrants close monitoring of fluids and electrolytes.
The treatment should be individualized, and the MDT approach is necessary to ensure the desired outcome.

D023

AVP deficiency secondary to herbal remedies

F Hussein, B Mahamud, L Eltayieb, E Shakoor, G Mlawa, F Asghar, D Sadulah, H Hussain, A Alnashrati, A Mohamed, A Al-Ghrairi, L Auguste, B Hossain, S Sawar.
Barking, Havering, and Redbridge University Hospitals NHS Trust.

Abstract

Introduction
Arginine vasopressin (AVP) deficiency previously known as central diabetes insipidus involves decreased release of arginine vasopressin (AVP) which could lead to polyuria. Patient usually presents with polyuria and polydipsia.

Lack of AVP can be resulted from disorders that act at one or more of the sites involved in AVP synthesis and secretion AVP deficiency is a rare disorder with prevalence of 1 in 25,000 individuals and most cases are acquired.

The most common acquired causes include autoimmune neurohypophysitis, primary or secondary tumours, infiltrative diseases (such as Langerhans cell histiocytosis and sarcoidosis), neurosurgery and head trauma.

Here we discuss a case in which AVP deficiency possibly secondary to herbal remedies.

Case Presentation
A 53 year old male was admitted with polyuria and polydipsia. Passing around 7Litres of urine daily which was disturbing his sleep.

He had no significant past medical history.

He was taking herbal remedies which including Rhodiola rosea root extract, cranberry extract, royal jelly extract and resveratrol.

One and half months earlier he also completed 14 days of ciprofloxacin antibiotics for prostatitis.

CT head showed no acute pathology.

MRI pituitary showed no acute lesions or pathology.

Bloods showed hypernatraemia of 147 mmol/L.

Urine osmolality of 162 and serum osmolality of 304, urinary sodium was<20.

He was given a trial of oral desmopressin which did not help, however when changed to intravenous desmopressin 1 microgram the patients symptoms significantly improved and was able to concentrate the urine.

Input/output chart

	Input volume (Litres)	Output volume (Litres)
Before desmopressin	9.2	9.9
After desmopressin	5.1	5

Discharged with nasal spray desmopressin 10micrograms twice daily and his symptoms continued to improve. The case was diiscussed in Neuro-pituitary MDT.

Conclusion

AVP deficiency is one of the main entities of the polyuria-polydipsia syndrome.
In most cases are acquired and commonly seen acquired causes include head injury, neurosurgery, cranial tumours and neurohypophysitis.
There have been very limited case reports on herbal remedies causing AVP deficiency such as olive extract and Solanum indicum L. There are also few cases reporting on transient diabetes insipidus in patients using quinolone antibiotics.
It is important especially in these rare cases to use an MDT approach to help manage patient's symptoms.

Questions for discussion

1. Have you come across herbal remedies induced AVP deficiency?
2. Could quinolones contribute to AVP deficiency?

D024

Adrenal insufficiency secondary to skin lightening cream

F Hussein, A Can, L Eltayieb, B Mahamud, G Mlawa,
Barking, Havering, and Redbridge University Hospitals NHS Trust.

Abstract

Introduction
Topical corticosteroids have important anti-inflammatory and immunosuppressive activity and are often prescribed for the treatment of dermatological disorders such as eczema and psoriasis. Adrenal insufficiency has been known to be a side effect for using topical steroid creams for long period of time and are therefore prescribed and monitored by the dermatologist. However, lightening creams as not regulated and are easily accessible to purchase from shops and online.

Case Presentation
35-year-old female presented with 1 year history of lethargy, fatigue and muscle cramps which had got worse in the last one week.

No infective signs. She was haemodynamically stable and apyrexic.

She has a past medical history of depression and anxiety and was taking mirtazapine medication. Her menstrual cycle is regular and has two children.

Her 9am Cortisol was found to be 7 nmmol/L and the pH, lactate and glucose was within normal range.

She denied taking oral or inhaled steroids but on further questioning it was found she has been taking a skin lightening cream call ‘Fashion fair cream’ which contains clobetasol propionate. She had been using this cream all over her body for many years.

On examination her skin had different pigmentation, thinner with more prominent veins. She had multiple striae on her arms, thighs and abdomen.

She also looked cushingoid with moon face, central obesity, buffalo hump of neck, and striae all over the body significantly on the abdomen.

Short synacthen test showed cortisol levels of 8nmol/L (0mins), 106 nmol/L (30mins) and 73 nmol/L (60mins).

She was reviewed in the endocrine clinic where she was advised to stop her topical cream and started on hydrocortisone 10mg/5mg/5mg with aim to wean down slowly

Conclusion
Long term use of topical steroid cream can cause side effects and most importantly secondary adrenal insufficiency. These are commonly prescribed for dermatological conditions such as eczema and psoriasis and are monitored closely.

However, some skin lightening creams contain steroids which the patient may not be aware of. These are easily available without prescription or monitoring from shops and online. This could lead to adrenal insufficiency if used for longer period of times. It is important to take a full history to check if they are taking topical cream, eye drops, shampoos, etc that may contain steroids. Most of the time patients are not aware of the item containing steroids. It is also important to have an MDT approach with the dermatologist and endocrinologist to help wean patient off the steroids and treat appropriately.

D025

Molecular targets everywhere but no NHS approval to treat!

NR Chander, FF Palazzo, AN Di Marco, R Gibbs, NS Tolley.
Imperial College Healthcare NHS Trust.

Abstract

Introduction
The incidence of locally invasive thyroid cancer in the UK is rising, and treatment remains challenging. Advances in the field of molecular mutations in thyroid cancer have resulted in the development of a number of targeted therapies which can improve outcomes in advanced cases.

Case report
A 53-year-old male with a three-month history of dysphagia on a background of a long-standing large right upper neck mass is presented. At examination a large right level II/III neck mass with bilateral lymphadenopathy was evident. Nasoendoscopy revealed a right vocal cord palsy.

An USS showed a 6cm right level II/III mass, inseparable from the thyroid gland and encasing the common carotid artery (CCA), in addition to bilateral pathological nodes. CT demonstrated a 210-degree contact between the CCA and the mass, with bilateral lymphadenopathy. FNAC showed PTC.

The patient was discussed at the vascular MDT and duplex ultrasound described no clear tissue plane between the mass and the CCA. FDG-PET revealed pulmonary metastases. Next generation sequencing for somatic mutations demonstrated BRAF V600E and TERT promoter variant (C228T) positivity.

The patient underwent a total thyroidectomy, bilateral selective neck dissection and carotid resection with a PTFE graft reconstruction. En bloc resection including the strap muscle, sternocleidomastoid, inferior constrictor, and cranial nerves X/XI was performed. XII was preserved. The patient made a good post-operative recovery with a safe swallow and no neurological sequelae.

Conclusion
This patient had somatic molecular mutations identified pre-operatively, for which targeted therapies are available (Dabrafenib and Trametinib). However, these are not currently licensed or funded for use in NHS practice, despite the clear potential for benefit as neoadjuvant treatment to minimise the extent and morbidity of surgery.

This case illustrates the need for an urgent review of the present NICE guidance for funding of targeted therapies for advanced thyroid cancer.

D026

Total pancreatectomy with islet auto transplantation for the treatment of chronic pancreatitis: between a rock and a hard place

H Esdaile[1], J Curney[1], N Oliver[1], A Lumb[2]
[1] Department of Diabetes and Endocrinology, Imperial College Healthcare NHS Trust, London.
[2] Oxford Centre for Diabetes, Endocrinology and Metabolism, Oxford University Hospitals NHS Foundation Trust, Oxford.

Abstract
We present the case of a 34-year-old lady who underwent a total pancreatectomy with islet auto transplantation (TPIAT) procedure, for the treatment of chronic pancreatitis secondary to gallstone disease. Pre-operative HbA1c was 32mmol/mol, and mixed meal tolerance testing demonstrated the following:

	Baseline	90 minutes
Glucose	4.5mmo/l	2375pmol/l
C-peptide	7.1mmol/l	2717pmol/l

Surgery was successful and the islet cell yield as measured by the Islet Equivalent (IEQ) was 207,000. Post-operatively she was diagnosed with Type 3c diabetes mellitus and commenced on BD Levemir insulin and Novorapid insulin 6 units with meals. She suffered with significant and prolonged nausea, anorexia, and abdominal pain. Two months following her surgery she experienced an episode of severe hypoglycaemia requiring paramedic assistance and transfer to emergency care.

At the three-month post-operative mark C-peptide was measured at 62pmol/L (paired glucose 7.9mmol/l) with an HbA1c of 77mmol/mol. She was switched to Toujeo basal insulin due to concerns about compliance, and continues to receive ongoing intensive dietary support for treatment of malabsorption and management of carbohydrate counting in the context of her diabetes. She will be discussed at the local diabetes MDT for consideration of hybrid closed loop therapy.

Chronic pancreatitis is a debilitating disease which may arise from various aetiologies, and leads to pancreatic inflammation, fibrosis and eventually, failure. Manifestations include abdominal pain, malabsorption from exocrine insufficiency, and the development of Type 3c diabetes mellitus. Few pharmacologic, endoscopic, or surgical options are available and many eventually become dependent on chronic opiate therapy to manage their pain[1]. Total pancreatectomy islet auto cell transplantation (TPIAT) was pioneered as a treatment for chronic pancreatitis in 1978. This procedure involves complete resection of the pancreas with transportal autologous islet cell transplantation. Its primary aim is to remove the visceral source of pain and cure disease, with a secondary goal of preventing brittle diabetes by preserving variable levels of endocrine function through islet cell transplantation.

Whilst a TPIAP procedure may facilitate insulin independence, rather than total pancreatectomy in which Type 3c diabetes mellitus is universal, data on outcomes is sparse, and no head-to-head trials examining endocrine function following a total pancreatectomy versus TPIAT exist[2]. Potential islet yield, unknown until pancreatectomy is performed, is the variable most consistently predictive of insulin independence. There are no reliable pre-operative predictors of post TPIAT endocrine response, and outcomes are unpredictable. Additionally, there is no consensus on the timing of the procedure in relation to disease severity. Therefore, appropriate pre-operative counselling about possible glycaemic outcomes is vital, particularly if the person with chronic pancreatitis does not have a pre-operative diagnosis of diabetes. Care for people undergoing a TPIAT is complex and will require co-ordinated multidisciplinary input, often spanning multiple centres, and involving surgeons, dieticians, diabetologists, psychologists, TPIAT specialist nurses, and pain specialists.

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D027

A case of poorly differentiated follicular thyroid carcinoma: when less is more

M Dias, NR Chander, AN Di Marco, FF Palazzo, NS Tolley.
Imperial College Healthcare Trust.

Abstract

Background
Follicular thyroid carcinomas are classified by histological features as undifferentiated (anaplastic), poorly differentiated, or differentiated carcinomas. Poorly differentiated follicular thyroid carcinoma (PDFTC) often presents as a solitary thyroid mass, with nodal and haematogenous metastases, although presentations can be atypical. PDFTC is the leading cause of morbidity and mortality among non-anaplastic follicular cell–derived thyroid cancers; therefore, prompt diagnosis and treatment, with minimal morbidity, is imperative.

Case presentation
We present the case of a 69-year-old male, who initially presented as an emergency with a 2-month history of biphasic stridor. On admission, a small palpable left sided neck mass was noted, with normal vocal cord movement. Bronchoscopy demonstrated a tracheal lesion, and so the patient underwent cold steel debulking of the lesion with cardio-pulmonary bypass, under the cardiothoracic team. Histopathology confirmed a BRAF negative PDFTC.

Subsequent imaging revealed a left retrosternal goitre, with tracheal deviation and stenosis, and an intra-luminal lesion. Metastases were noted in the lung, manubrium and C2 vertebra. Microlaryngoscopy showed tumour infiltration along a 6-8cm of the trachea, with a small foci of tumour within the trachea that was ablated with CO_2 laser.

Following MDT discussion, the patient underwent a total thyroidectomy and manubriectomy, with the aim of loco-regional control. Microlaryngoscopy was repeated, with multiple tracheal biopsies demonstrating no evidence of disease. The patient was subsequently discharged to their local centre for radioactive iodine therapy and radiotherapy.

Conclusion
This case provides valuable insight into the varied presentations of PDFTC and the importance of undertaking thorough diagnostics prior to making management decisions. Furthermore, this case highlights that in locally advanced cases, excellent local control and long-term outcomes can be achieved even with minimally invasive techniques.

D028

Double Trouble: Navigating Contrast and Amiodarone-induced Thyrotoxicosis

S Ahsan, R Ginsberg, D Mariannis, M Naik, J Cox, T Vakilgilani.
Imperial College Healthcare NHS Trust.

Abstract

Background
Distinguishing between different causes of hyperthyroidism is not always straightforward as several conditions can lead to a transient state of hyperthyroidism due to thyroiditis versus excessive endogenous thyroid hormone production. Thyroiditis is often caused by infection, drugs, post-partum and post-contrast imaging amongst other causes. Our patient demonstrates the diagnostic challenges one can face in such cases. It also highlights the importance of thorough history taking, using appropriate serological and imaging modalities as well as the multidisciplinary team approach.

Case Presentation
A 56-year-old Afro-Carribean gentleman with a background of longstanding atrial fibrillation and dilated cardiomyopathy was referred to the endocrinology clinic by his cardiologist for management of thyrotoxicosis (TSH 0.03 mU/L, FT4 29 pmol/L [9.0-23.0] and FT3 5.4pmol/L [2.4-6.0]). He was previously treated with amiodarone for 6 years which was discontinued 6 months earlier. His thyroid function tests (TFT) were within range whilst on amiodarone. Two months prior to the onset of thyrotoxicosis, he had an admission to the intensive care unit due to shortness of breath and abdominal pain and subsequently underwent CT pulmonary angiogram and contrast CT abdomen pelvis as part of the diagnostic work-up. His thyroid function test prior to the imaging was TSH 5.95 mU/L, fT4 16.4 pmol/L.

Upon endocrine review, there were no overt hyperthyroid features apart from bilateral hand tremor. He was not tachycardic (on beta blockers and digoxin). There was no goitre, no palpable nodules and no signs of thyroid eye disease. He did not have a personal or family history of thyroid disease and there was no history of recent viral illness or neck pain.

The initial impression was thyroiditis either secondary to iodine load from the contrast medium or delayed amiodarone-induced thyrotoxicosis.

Subsequent investigations revealed a fully suppressed TSH (<0.01 mU/L) with
FT4 26.5pmol/L and FT3 6.5pmol/L [2.4-6.0]), negative TSH receptor antibody and mildly elevated TPO antibodies (23 iU/ml [<5.0]). Technetium scan revealed absent uptake and ultrasound colour doppler demonstrated normal thyroid architecture and absent vascularity, both consistent with the diagnosis of thyroiditis.

Given that his heart rate was well-controlled, no steroid treatment was commenced whilst waiting for the investigations; instead he was reviewed at 6-week intervals with TFT monitoring and his thyroid function spontaneously recovered within 3 months (TSH 4.71 mU/L, FT4 17.1 pmol/L, FT3 5.3 pmol/L).

Conclusion
Initially this looked like iodinated contrast-induced thyrotoxicosis, however we questioned the role of previous amiodarone usage as a contributing factor, given the known prolonged half-life of the amiodarone. We wanted to exclude amiodarone-induced thyrotoxicosis (AIT), as this would have altered our management.

The technetium was difficult to interpret but given the colour doppler result and negative antibodies, we excluded type 1 AIT. Type 2 AIT was not possible to exclude at the time of the

investigations, however the clinical progress demonstrated that this was not the cause. We were therefore pleased to avoid unnecessary steroid treatment which could have potentially led to further cardiovascular deterioration.

The contribution of the multidisciplinary team was invaluable and this case illustrates the utility of expertise in colour doppler in such patients.

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D029

A Rare Case of Addison's disease in Pregnancy

A Roy, E Davies, P Agarwal, L Chandrarajan, A Surendran, M Bakhit.
University Hospital Lewisham, Lewisham and Greenwich NHS Foundation Trust.

Introduction
Adrenal crisis in pregnancy is rare, especially without a pre-existing diagnosis of adrenal insufficiency, yet carries a high risk of serious morbidity and mortality. Signs and symptoms are non-specific and have significant overlap with normal pregnancy symptoms, making recognition difficult. This case shows that successful outcomes can be achieved when empirical treatment with exogenous steroids is initiated on a high index of suspicion.

Case report
A 37-year-old pregnant woman, presented to the emergency department at 22+4 weeks' gestation, with non-specific symptoms of weakness, fatigue, light-headedness, abdominal pain, nausea, vomiting and fever for the last few weeks. On further history, she mentioned weight loss of about 3 kg, decreased appetite and increased pigmentation in the elbows and knees from 12 – 13 weeks of pregnancy. She had no significant medical or surgical history. Her past obstetric history included a spontaneous vaginal delivery 3 years previously and a medical termination of pregnancy. The current pregnancy was hitherto unremarkable besides increasing weakness and fatigue. On examination, her blood pressure was 81/66, which did not respond to 6 liters of fluid resuscitation. She was initially perceived to be in septic shock and was admitted to the intensive care unit. She had a positive COVID 19 swab test. Initial investigations revealed a raised WBC count at 13.1, sodium 141 meq/l and Potassium 3.6. Adrenal insufficiency was suspected, and IV hydrocortisone was started empirically. Subsequent blood tests showed a cortisol level of 75 ng/L, and ACTH of 1509 ng/L, suggesting primary adrenal insufficiency. The rest of the blood tests showed a free T4 of 14.2, and TSH of 0.99. MRI scans of the pituitary and adrenals were normal. She was admitted under the critical care team and put under the care of a multidisciplinary team involving obstetrics, Endocrinology and obstetric medicine who monitored her very closely. She improved following IV steroids, and was discharged on oral hydrocortisone with a steroid plan for vaginal and caesarean delivery. Subsequent growth scans showed a reduction in growth velocity and placental dysfunction. She underwent an elective caesarean section at 37 weeks under IV hydrocortisone cover and delivered a healthy baby. Her oral hydrocortisone doses were doubled for one week post operatively. She was started on Fludrocortisone 100 mcg OD after delivery and continued on Hydrocortisone 20mg. Adrenal antibodies are awaited.

Discussion
A literature search highlighted two similar cases and although the presenting complaints varied. Profound hypotension refractory to fluids is a common theme. Metabolic acidosis with varying degrees of hypoglycaemia is also consistent. In this case, a high index of suspicion and empirical treatment allowed for the early initiation of IV hydrocortisone which was highly effective. In retrospect she was able to identify symptoms emerging from conception, suggesting that physiological changes in pregnancy, catalysed by acute viral illness, triggered this first presentation of adrenal insufficiency.

Conclusion
Although rare, adrenal insufficiency should be considered as a differential diagnosis in pregnant patients presenting with severe hypotension refractory to IV fluids. Signs, symptoms, and biochemical markers may be subtle. Prompt treatment with IV steroids during the acute crisis is a highly effective and should be started promptly when the diagnosis is suspected

D030

Post adrenalectomy paranoia: non-normalising metanephrines after resection of phaeochromocytoma

J Agilinko, J Churiwala, R Flora, J Todd, F Wernig, FF Palazzo.
Hammersmith Hospital, Imperial College Healthcare NHS Trust.

Abstract

Introduction
The diagnosis of pheochromocytoma is established by biochemical confirmation of elevated plasma metanephrines and the localisation of an adrenal lesion via conventional and nuclear imaging. Surgical cure following adrenalectomy is typically indicated by normalisation of metanephrines. When metanephrines do not normalise postoperatively, it can generate significant concern warranting a meticulous review of all aspects of the diagnostic and therapeutic process, including preoperative imaging, biochemistry, surgical findings, histology and multidisciplinary discussion.

This report presents a case of persistent metanephrine elevation after adrenalectomy and summarises the diagnostic conundrum.

Case presentation
A 57-year-old gentleman with a history of prostate cancer - managed with 3-monthly leuprolide injections - presented with an incidental 13mm indeterminate left adrenal lesion detected on staging CT. His medical history included longstanding hypertension managed with doxazosin and phenoxybenzamine. He reported no other symptoms suggestive of adrenal disease and his family history was negative for adrenal pathology. Plasma metanephrines were found to be elevated, diagnostic of phaeochromocytoma with MIBG imaging localising a functional lesion to the left adrenal gland with no other sites of increased MIBG uptake. Following discussion in our adrenal MDT and medical optimisation, he was scheduled for prostatectomy under alpha blockade, followed by the adrenalectomy in the subsequent weeks.

However, prostatectomy had to be abandoned due to intraoperative cardiovascular instability and hypertensive episodes. A left laparoscopic adrenalectomy was expedited and was uneventful. The histology confirmed a fully excised left adrenal phaeochromocytoma with a low PASS score of 1. He made a good post-operative recovery and was pain free, but plasma normetanephrine remained elevated. Genetic tests were negative for hereditary phaeochromocytoma and paraganglioma syndromes. Further MDT discussions and review of preoperative imaging led to the decision to carry out a gallium-68 DOTATATE PET/CT which did not show any pathological tracer avidity to explain elevated metanephrines.

The urology team initially deferred further surgery pending resolution of the “phaeochromocytoma” status but eventually carried out a robotic-assisted prostatectomy without intraoperative complications.

Discussion
Plasma metanephrines provide a sensitive marker for assessing biochemical cure after surgery. Persistent elevation following resection of a confirmed adrenal phaeochromocytoma suggests alternative sources or contributing factors when imaging excludes other metanephrine-secreting lesions. Potential causes of falsely elevated metanephrines include factors such as caffeine, cigarette smoking, blood sampling conditions, and pharmacological interference [1,2] Medications such as paracetamol, sympathomimetic drugs e.g., phenoxybenzamine or leuprolide, can affect catecholamine metabolism and interference with liquid chromatography assays potentially contributing to false positive results. [3]

The search for the source of this patient's elevated metanephrines continues …

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D031

Normalisation of glycaemia in a patient with type 2 diabetes within forty-eight hours of Roux-en-Y gastric bypass surgery

E Brachimi, A Fardanesh, A Abdelhamid, S Hameed, S Purkayastha.
Imperial Weight Centre, St Mary's Hospital, Imperial College Healthcare NHS Trust

Abstract

Background
The incidence and prevalence of type 2 diabetes continue to increase. In the UK, almost five million people are living with the condition, which costs the NHS ten percent of its annual budget in drug and other treatment costs. In some cases, achieving good glycaemic control cannot be achieved through lifestyle or pharmacotherapy interventions. These patients may significantly benefit from metabolic (bariatric) surgery, in particular Roux-en-Y gastric bypass (RYGB).

Case History
A fifty-seven-year-old woman presented to the bariatric clinic with a weight of 135 kg and a body mass index of 50 kg/m^2. The patient had a seven-year history of type 2 diabetes. Despite attempts to optimise her diabetes medication regimen with both oral and glucagon-like-peptide-1 (GLP-1) agonist medications, adequate blood glucose control could not be achieved either due to medication intolerance, lack of pharmacological efficacy or both and HbA1c was 80 mmol/mol.

The patient underwent RYGB and was placed on a subcutaneous insulin sliding scale as per usual post-operative protocol. The insulin requirement was low, and serial point of care glucose measurement confirmed capillary blood glucose (CBG) in the normal range. The sliding scale was stopped and normoglycaemia persisted. The patient was discharged three days after surgery on oral Metformin and Sitaglitpin with a view to stopping these medications if home CBG measurements show normoglycaemia.

Learning Points
After years of sub-optimal glycaemic control, normoglycaemia was achieved within forty-eight hours of bariatric surgery. This effect occurs prior to weight loss and reflects a post-operative elevation in gut hormones such as GLP-1. Usually prior to bariatric surgery, the HbA1c is reduced through intensive pre-operative pharmacological management which did not prove effective in this patient and a decision was taken to treat her diabetes with RYGB. Bariatric surgery is a highly efficacious and cost-effective treatment for type 2 diabetes, particularly in cases unresponsive to intensive pharmacotherapy. As with the management of all conditions, type 2 diabetes also fits into the treatment paradigm of conservative, medical, surgical and bariatric surgery should be considered as part of a mutli-disciplinary treatment strategy in patients with glycaemia that is unresponsive to conservative and medical interventions.

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D032

Adrenal Haemorrhage: Bilateral vs Unilateral

P Agarwal, A Roy, A Khanam, Mohamed Bakhit
University Hospital Lewisham, Lewisham and Greenwich NHS Trust.

Abstract
Adrenal haemorrhage is a rare but clinically significant condition that can affect one or both adrenal glands. Common underlying causes include trauma, sepsis, anticoagulant therapy, and adrenal tumours. This condition is critical, as it can precipitate adrenal insufficiency or crisis. Here, we examine cases of bilateral versus unilateral adrenal haemorrhage, emphasising key differences in their aetiology, clinical presentations, and management strategies.

Case 1
70-year-old male with hypertension, previous stroke, atrial fibrillation (not on anticoagulation), and COPD - presented with flank pain, urinary sepsis, and refractory hypotension. Abdominal CT showed bilateral adrenal thickening, with a dedicated adrenal CT confirming bilateral adrenal haemorrhage.

9am cortisol 61 nmol/L, ACTH 14 ng/L, aldosterone <60 pmol/L, and renin 16.8 nmol/L/h, indicating primary adrenal insufficiency, with normal sodium and potassium (144 mmol/L and 5.0 mmol/L, respectively). 1 mg overnight-dexamethasone-suppression cortisol was normal (35 nmol/L). Hydrocortisone therapy was initiated. Repeat imaging at five months showed resolution in adrenal haemorrhage. He remains under endocrine follow-up with persistently low aldosterone and is maintained on hydrocortisone and fludrocortisone, with further tests planned to evaluate adrenal reserve.

Case 2
An 82-year-old male on anticoagulation and heart failure medications presented with a fall and hypotension. Abdominal CT revealed unilateral adrenal haemorrhage, with a dedicated adrenal CT showing a lesion consistent with hematoma. 9am cortisol was 319 nmol/L, sodium 140 mmol/L, and potassium 4.4 mmol/L, indicating adequate adrenal function. Though clinically stable, he requires close endocrine follow-up to assess lesion functionality and exclude malignancy, with repeat imaging scheduled in three months to confirm resolution.

Discussion
Both cases explore the challenges in diagnosing adrenal haemorrhage due to its nonspecific symptoms and emphasise the need for targeted imaging and endocrine evaluation. Unilateral haemorrhage with no adrenal insufficiency often stabilises with conservative management; whereas bilateral haemorrhage needs vigilant monitoring and potential lifelong adrenal steroid cover.

D033

A case of Congenital Adrenal Hyperplasia (CAH) in pregnancy

D Papadopoulou[1,2], N Primrose[1], K Meeran[1,2], S Jarvis[1,2]
[1] Imperial College Healthcare, London, United Kingdom.
[2] Imperial College, London, United Kingdom.

Abstract
Congenital adrenal hyperplasia (CAH) is the commonest inborn endocrine disorder due to single-gene defects in the various enzymes required for cortisol biosynthesis with a reported incidence of 21-Hydroxylase deficiency of 1:12000 live births and milder non-classic CAH affecting 1:2500.[1] It is well recognised that management of CAH is challenging with many unmet clinical needs and a requirement for future deep phenotyping of the condition.[2] Knowledge around the optimal management of CAH during pregnancy remains limited. Here we share the experience of a CAH pregnancy which highlights the need for multidisciplinary care within a specialised maternal medicine centre.

This is the case of a 26-year-old lady diagnosed with classical salt wasting CAH at 3 months of age following a crisis. This was her third pregnancy; she had received pre-pregnancy genetic counselling before her second pregnancy and her partner was negative for the CAH genetics (i.e., 21 hydroxylase deficiency). During the pregnancy, she required close monitoring at the Obstetric Medicine clinic (from 10 weeks' gestation) with joint input by Endocrinology and Obstetricians. She received close Endocrine Specialist nurse input providing education around sick day rules, the emergency hydrocortisone injection and steroid card/medical ID. She received steroid replacement of Hydrocortisone 10mg at 9am and 15mg at 9pm in a reverse circadian fashion and fludrocortisone 100 micrograms daily. This pregnancy was complicated by persistent symptoms of nausea (despite anti-emetics) and postural dizziness. In the late second trimester in response to postural dizziness a trial of fludrocortisone up-titration was undertaken for 1 week with a resolution of her symptoms and stable biochemistry. Her usual endocrinologist was also kept informed of progress. Closer to term, a clear delivery plan focused on intrapartum steroids was made with the multidisciplinary team input (including Endocrinologists, Obstetricians, Endocrine Specialist Nurse and midwives) and the patient received a copy. At 38 weeks, she underwent spontaneous vaginal delivery with a healthy baby boy, and she remained on her current dosing with endocrine follow up planned postpartum.

This case presented, provides a deeper understanding of the CAH patient journey during pregnancy. Deciphering the common symptoms of pregnancy versus CAH symptoms requires meticulous attention to the clinical history, clinical assessment alongside the limited biochemical measurements available in pregnancy. Perceived patient confidence in her antenatal care was enhanced by maintaining communication with her usual Endocrinologist and provision of continuity with her Endocrine specialist nurse from 10 weeks gestation.

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D034

Now You See Me! Re-operative parathyroidectomy for a supernumerary ectopic parathyroid adenoma

JJ Churiwala[1], J Agilinko[1], F Kaplan[2], S Morley[3], FF Palazzo[1]
[1] Department of Endocrine & Thyroid Surgery, Hammersmith Hospital, Imperial College Healthcare NHS Trust, UK.
[2] Department of Diabetes & Endocrinology, Lister Hospital, East and North Hertfordshire NHS Trust, UK.
[3] Department of Radiology, University College Hospital, University College London Hospitals NHS Foundation Trust, UK.

Abstract
Background: Primary hyperparathyroidism is caused by parathyroid glands that can usually be localized. However unlocalized parathyroid disease on first line imaging occurs in up to 17%. The incidence increases as the biochemical severity and gland size decrease. The cure rate in index parathyroidectomy of unlocalized disease remains high in specialist centres. However, despite a meticulous approach to neck exploration in such cases around 4% may not be cured – compared to 2% of localized disease (1). We describe a case of persistent hyperparathyroidism due to a parathyroid adenoma in a very unusual location. A comprehensive literature review was undertaken to draw comparisons with other publications examining this rare occurrence.
Case Presentation: A 67-year-old woman was referred to endocrine surgery with a diagnosis of primary hyperparathyroidism (PHPT) and osteoporosis. First line imaging with US and MIBI scintigraphy failed to localise an adenoma. The patient underwent a 4-gland neck exploration. While both eutopic right parathyroid glands were normal on inspection, no symmetrical parathyroids or classic ectopics on the left could be identified, despite an extensive search. A left hemithyroidectomy was performed for presumed intrathyroidal parathyroid adenoma. The calcium failed to normalise and the PHPT persisted which prompted further imaging. The ^{18}F-choline PET-CT failed to convincingly localize the offending adenoma but the subsequent 4D CT identified the culprit in the left pyriform fossa. A re-operative parathyroidectomy led to biochemical cure.
Discussion: Pyriform fossa parathyroid adenomas are exceedingly rare with just 15 reported in the world literature to the best of our knowledge (2). The embryologic basis for this event is postulated to be a failure of separation of the superior parathyroid primordium from the pharyngeal wall and subsequent retropharyngeal migration into the pyriform sinus primordium. The identification of such an ectopic gland is highly unlikely. Additional imaging is invariably required to identify this parathyroid of interest and plan a targeted surgical approach to achieve cure.

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D035

The sight-saving role of Bariatric Surgery in Idiopathic Intracranial Hypertension

M Abdel-Malek, R Aggarwal, L Brolly, S Scholtz, A Pambakian, M Wilson, A Ahmed, T Tan.
Imperial College Healthcare NHS Trust, London, United Kingdom.

Abstract
Idiopathic intracranial hypertension (IIH) classically occurs in women with obesity and is characterised by raised intracranial pressure. Weight loss leads to reduction in intracranial pressure. We present a case of a 35-year-old female who was urgently referred to Imperial Weight Centre. She had recently undergone revision of a right ventriculoperitoneal shunt for longstanding IIH which had not resolved her high-pressure symptoms and continued to require therapeutic lumbar punctures (LPs) for worsening headache and vision loss.

She had a BMI of 37.6kg/m^2 (107.3kg) with no history of childhood obesity or binge eating disorder. Over the last decade, her weight had gradually increased from a 70kg baseline following two pregnancies and concurrent deterioration in her mental health (post-natal psychosis, post-traumatic stress disorder). The long-term use of Quetiapine, and also Mirtazapine, were additional contributors to weight gain given the orexigenic effect with increasing her hunger. She was also on Levothyroxine for previous hypothyroidism. Previous attempts for weight loss intervention included a gastric balloon where she achieved 14kg loss, but it was removed due to pancreatitis and she regained weight thereafter.

Her case was discussed at the Imperial Weight Centre MDT and bariatric surgery was recommended versus pharmacotherapy in view of previous pancreatitis. She underwent a laparoscopic sleeve gastrectomy shortly after multidisciplinary review (pre-operative weight 108kg) with no immediate post-operative complications. Three months later, she achieved 16.3% weight loss (92.1kg) and reported fewer headaches with less frequent therapeutic LPs required. She unfortunately required a laparoscopic cholecystectomy for pancreatitis secondary to gallstones after presenting with sudden, severe epigastric pain and amylase of 2,431unit/L.

This case illustrates the significant impact of obesity on IIH and the important role of weight loss in clinical management. In this case, the weight loss intervention prevented vision loss, reduced hospital admissions for repeat LPs and neurosurgery. Quality of life and mental health also improved, and the patient can now look forward to resuming her daily activities. In addition to weight loss surgery, we now have pharmacotherapy options e.g. Wegovy, which patients suffering with IIH will significantly benefit from and their accessibility should be implemented as part of the treatment pathway.

D036

Weight management in women with subfertility seeking assisted reproduction: a case series

M Abdel-Malek, C Izzi-Engbeaya, J Upton, L Tweedlie, S Hameed, S Scholtz, H Chahal, T Tan.
Imperial College Healthcare NHS Trust, London, United Kingdom

Abstract
The impact of obesity on fertility and pregnancy is well-established. The literature remains conflicting around optimal BMI threshold and weight loss strategy on reproductive outcomes. We describe a series of twenty-six women with obesity that were planning a future pregnancy or seeking assisted fertility services. They were initially reviewed for weight management at Imperial College Healthcare between September 2020 and September 2022. Clinical case information was retrieved from documentation on electronic patient records up to November 2023. The median age was 31 years (range 25 to 41) and mean baseline weight 112.9kg (SD +/-21.7kg). Polycystic ovarian syndrome was the aetiology for subfertility reported in 76.9% (*n*=20) with pelvic or unknown cause in the remainder (*n*=1 and *n*=5, respectively). Other metabolic comorbidities noted included dyslipidaemia (38.5%,*n*=10), type 2 diabetes (23.1%,*n*=6), pre-diabetes (15.4%,*n*=4) and fatty liver disease (11.5%,*n*=3).

By September 2022, half of the twenty-six patients (*n*=13) had been initiated on a GLP-1 analogue (semaglutide *n*=10; liraglutide *n*=3). At this point in time, for those receiving a GLP-1 analogue of various duration (3 to 24 months), the percentage change from baseline weight ranged from 2.88% to -20.2% (3.5kg to 21kg) with a mean loss of -9.38kg or -8.23% in body weight (SD +/-6.55kg and +/-5.92%, respectively). 42.3% reported use of orlistat and three patients had already undergone bariatric surgery prior to initial review. By November 2023, a further three patients had undergone a sleeve gastrectomy and three patients were awaiting planned weight loss surgery. Eight patients had proceeded to have a natural pregnancy or received access to assisted conception after meeting BMI eligibility.

Our case series demonstrate the clinical need for special input around weight management for women with overweight or obesity seeking fertility support. A clearer treatment pathway will support existing NHS guidelines around BMI eligibility criteria to access assisted reproduction. Current limitations include the limited supply of GLP-1 pharmacotherapy, long waitlist for bariatric surgery and the need to avoid pregnancy for 12-18 months thereafter. There is a need to address obesity-related subfertility at an earlier age, which will also reduce the risk of pregnancy complications secondary to metabolic comorbidities.

Research to compare different steroids for replacement in patients with adrenal insufficiency.

Funded by NHS National Institute for Health Research

At present, there is no evidence in support of any of the different steroids used in patients with primary or secondary adrenal failure.

Although the primary reason for the use of prednisolone once daily instead of hydrocortisone at Charing Cross was the huge difference in price in 2014 (Amin et al, 2014), it has become clear over the last five years that many patients prefer prednisolone once daily. The price of hydrocortisone has now fallen, but patients who have switched to once daily prednisolone are very reluctant to switch back and preliminary data is encouraging (Smith et al, 2017; Choudhury et al 2019). In addition, the DREAM study suggests that late doses of hydrocortisone may affect clock genes and might also have an effect on the immune system and hence thrice daily hydrocortisone might be harmful, especially an evening dose (Muller et al, 2018). It is thus possible that in addition to being cheaper and more convenient, when used at the correct dose, prednisolone has fewer undesirable effects than hydrocortisone thrice daily. The plasma profile of once daily prednisolone matches the natural circadian rhythm of plasma cortisol levels better than any other steroid (Williams et al, 2016).

At present, there is no evidence to support the use of either drug. We really need data to inform our future practice and to this end the NIHR have adopted three trials, one of which you can take part in through your NIHR Clinical Research Network.

Once your site is enrolled, the study is very simple although it needs someone to lead each site. Patients need to be stably replaced on either hydrocortisone or prednisolone for the preceding four months. When seen, the locally available markers of steroid exposure such as bone turnover markers, glucose, Hba1c and lipids, weight, BMI, waist and hip measurements, blood pressure and the results of an SF36 well-being questionnaire need to be recorded. They are then switched to an equivalent dose of the alternate drug for a further four months and the data collected again at the end of that period. Checking for an appropriate dose can be supported by prednisolone levels if desired (Sharma 2023).

On the back of these studies, there was a launch of 2.5mg and 5mg tablets of hydrocortisone, which was associated with a fall in the price of hydrocortisone.

If you would like to take part in this study, please e-mail steroids@imperial.ac.uk

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IMPERIAL

UNIVERSITY OF CAMBRIDGE

OPTIMAL: Advancing Adenoma Detection in Cushing's Disease

Study Title: OPTIMAL: Osilodrostat therapy and 11C-methionine **P**E**T** to **IM**prove corticotroph pituitary **A**denoma detection and **L**ocalisation in patients with Cushing's disease

Sponsor: Imperial College London

Background: Cushing's disease is caused by corticotroph pituitary adenomas that produce excessive cortisol. Localising these adenomas with standard MRI can be difficult.

- **11C-methionine PET/CT:** An emerging imaging technique for identifying functional pituitary adenomas undetectable by MRI.
- **Osilodrostat**: A potent steroidogenesis blocker. By lowering cortisol and increasing ACTH, it may enhance adenoma visibility on 11C-methionine PET/CT.

What is the OPTIMAL?
A multicentre observational study assessing radiological changes in presumed corticotroph adenomas. Patients undergo standard MRI and 11C-methionine PET/CT before and after a period of osilodrostat therapy.

- **Primary Outcome:** Change in 11C-methionine PET/CT uptake (SUVmax) pre- and post-treatment.
- **Secondary Outcome:** MRI changes pre- and post-treatment.

Eligibility:
Adults with newly diagnosed or recurrent Cushing's disease and no clear surgical target on MRI.

Why Join?
- Collaborate on innovative research to enhance adenoma detection in patients with Cushing's disease.
- Contribute to advancements in diagnosis and treatment for patients facing this challenging condition.

Contact Us:
Interested in collaborating or referring patients? Contact us for more details and to join this study.

Zin Htut: z.htut23@ic.ac.uk

Florian Wernig: fwernig@ic.ac.uk