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Society for Endocrinology: Endocrine Update 2017

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Society for Endocrinology: Endocrine Update 2017

Obesity Update

Oral Communications

OC1

Which test should the bariatric physician use to test for postprandial hypoglycaemia – prolonged oral glucose tolerance test versus mixed meal test?

George Tharakan[1], Preeshila Behary[1], Werd Al-Najim[1], Harvinder Chahal[1], Alexander Miras[1], Ahmed Ahmed[2], Stephen Bloom[1] & Tricia Tan[1]
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Background
An increasingly recognized complication of Roux-en-Y Gastric Bypass (RYGB) surgery is the development of postprandial hypoglycaemia (PPH). However, there remains no agreed standard on how to diagnose this condition. Commonly used tests include a prolonged oral glucose tolerance test (POGTT) and mixed meal tolerance test (MMTT). Little is known regarding how these tests compare in the post-bariatric population.
Methods
Ten patients who had confirmed PPH on CGMS underwent both a POGTT and MMTT on two different days, separated by 1 week. For both tests, volunteers attended fasted, and had regular venous sampling for glucose, insulin, GLP-1, GIP and glucagon. For the MMTT, Ensure Plus (13.8 g of protein, 10.8 g of fat, 44.4 g of carbohydrates, 330 kcal, 220 ml, Abbott, Maidenhead, UK) was used. For the POGTT, 75 g of anhydrous glucose was used.
Results
Seven of the 10 patients had biochemical hypoglycaemia during the POGTT (70% sensitivity) compared to only two during the MMTT (20% sensitivity). Consistent with this, there was a significantly lower glucose nadir during the POGTT (Mean $\pm$ S.E.M.; glucose nadir 3.64 ± 0.27 in POGTT versus 2.68 ± 0.18 in MMTT ($P < 0.0002$)). Counterintuitively, although the peak insulin response was higher in during the POGTT, there was a significantly higher peak incretin response (both GLP-1 and GIP) during the MMTT. Whilst there was a trend for glucagon to decrease during the POGTT, in the MMTT, there was a rise prior to the glucose nadir.
Discussion
This comparison of two commonly used provocation tests for PPH demonstrated that the POGTT was more sensitive at detecting hypoglycemia. However, the POGTT can be considered less physiological than the MMTT as it contains only one carbohydrate. The protein and fat contained in the MMTT more closely reflects a normal meal and this may account for the differences in both incretin response as well as postprandial glucagon concentration.

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OC2

Cardiac risk stratification in bariatric patients: a step to minimise heartache

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Introduction
Morbid obesity has serious associated co morbidities and is an independent risk factor for ischaemic heart disease. Obesity together with low functional status and exercise tolerance makes pre-operative cardiac assessment difficult and patients with few cardiac risk factors are often referred for cardiac assessment. This can not only be burdensome to services and prolongs waiting times for surgery but can also cause patient anxiety and poor compliance.
Background
The revised cardiac risk index (RCRI) is a validated risk stratification tool for predicting the risk of major cardiac events in the non-cardiac surgical setting. Multiple factors like type of surgery, history of ischaemic heart disease, history of congestive heart failure, history of cerebrovascular disease,preoperative treatment with insulin and preoperative serum creatinine (>2 mg/dl) stratify individuals into four categories (I, II, III and IV), the risk of cardiac events increases with each category (0.4, 0.9. 6.6, and 11% respectively).
Aim
The aim of this study is to assess whether the number of referrals to cardiology could be reduced by applying RCRI, yet still capture all of the pre-operative cardiac therapeutic interventions. We also studied the impact of cardiology referral on waiting times, development of cardiac complication whilst awaiting surgery and associated symptomatology of patients requiring cardiac intervention.
Methods
Between 2005 and 2014, a cohort of 1040 patients that had been evaluated for weight loss surgery was identified. Retrospective analysis of the clinical records was undertaken. Referrals to cardiology at this time were based on clinical judgement. Data collected included: RCRI, referral to cardiology, symptomatology, cardiac investigations and interventions, waiting time, morbidity and mortality.
Results
Out of 1040 patients, 868 patients were not referred to cardiology and did not require any cardiac intervention (0.0%). 172 (16%) were referred to cardiology, nine of the 172 patients (5%) required cardiac intervention; of which, six patients (20%) belonged to RCRI category III and IV ($n=30$) compared to three patients (2%) in category I and II ($n=142$), making patients in category III and IV significantly more likely to receive cardiac intervention ($P<0.01$). Waiting time for surgery was significantly higher in cardiology referral group (Median: 240 days) compared to non-cardiology group (Median: 0 days, $P<0.01$). Four patients (2%) in cardiology referral group developed myocardial Infarction whilst waiting for review (including 1 death = 0.5%), which was significantly higher than those not referred to cardiology ($n=0$, $P<0.01$). Of all the patients requiring cardiac intervention ($n=9$), chest pain (alone or in combination with previous cardiac history or SOB) was the strongest associated symptom requiring cardiac intervention ($n=8$, $P<0.01$). This together with RCRI III and IV consisted of positive predictive value of 66.66% in this studied population.
Conclusions
Cardiology referrals significantly increase waiting time. Cardiac interventions are more likely in patients with RCRI III and IV. Application of RCRI together with symptom of chest pain can make a good risk stratification tool for cardiac assessment in bariatric patients. Limiting cardiology referrals predominantly to this group would substantially reduce waiting time, cardiac referrals and development of complications as a result.

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OC3

The early improvement of glycaemia following RYGB can be mimicked by a Very Low Calorie Diet in obese volunteers with diabetes

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Introduction
Improvement in glycaemia is observed early after Roux-en-Y Gastric Bypass surgery (RYGB) in patients with diabetes. Commonly cited mechanisms to account for these changes include the elevation of gut hormones, increase in bile acids levels, changes in the gut microbiota and calorie restriction. Calorie restriction is believed to play a role in improving hepatic insulin sensitivity and reduce hepatic glucose output early after RYGB, in the context of non-significant weight loss.
Aim
To compare the effect of RYGB on glycaemia at two weeks post-operatively versus a Very-Low-Calorie diet (VLCD) of 800 kcal/day at matched weight loss.
Methods
Eleven obese volunteers with diabetes treated with either diet or single oral hypoglycaemic agent, and due for RYGB were recruited from the Imperial Weight Centre. A separate matched cohort of seventeen volunteers was recruited and given a VLCD of 800 kcal/day (Cambridge diet). Both groups were studied before and at 2 weeks after the intervention. Weight and body composition were measured using a Bio-impedance scale (Tanita BC-418MA). Fasting glucose and insulin blood levels were measured and an index of insulin resistance calculated using the HOMA-IR model. Data is presented as Means $\pm$ S.E.M.. A Student unpaired *t*-test was used for group comparison.
Results
The RYGB and VLCD cohort were well matched for age, BMI and glycaemia (Fasting glucose; RYGB: 9.3 ± 0.6 mmol/l, VLCD: 8.2 ± 0.7 mmol/l, $P=0.3$). Weight loss was comparable across the two groups at two weeks (RYGB: 5.5 ± 0.4%, versus VLCD: 4.9 ± 0.4%, $P=0.3$) as were changes in body composition (fat mass RYGB -2.8 ± 0.5 kg versus VLCD -2.5 ± 0.6 kg, $P=0.7$ and free fat mass RYGB -4.7 ± 1.2 kg versus VLCD -3.0 ± 0.5 kg, $P=0.1$). Similar changes in fasting glucose (-2.3 ± 0.5 mmol/l versus -2.1 ± 0.4 mmol/l, $P=0.8$), fasting insulin (-5.4 ± 1.2 mU/l versus -6.9 ± 1.5 mU/l, $P=0.5$) and HOMA-IR (-0.87 ± 0.2 versus -1.1 ± 0.2, $P=0.5$) were also observed following RYGB and VLCD respectively.

Discussion
Our data is consistent with the argument that calorie restriction is a key mediator of the early 'anti-diabetes' effects of RYGB. However, as the very low calorie restriction after RYGB is a temporary phenomenon, other mechanisms (for example elevations in gut hormone secretion or changes in gut microbiota) must supervene at later stages.

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OC4

One year efficacy, safety and tolerability outcomes of endoscopic proximal intestinal exclusion therapy using the Endobarrier device: institution of the UK's first National Health Service Endobarrier service for type 2 diabetes and obesity

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City Hospital, Birmingham, UK.

Introduction
Our institution leads a UK, multicentre, randomised controlled trial (REVISE-Diabesity ISRCTN00151053) investigating the interaction of Endobarrier therapy, a 60 cm endoscopically implanted proximal intestinal liner, with glucagon-like peptide-1 drug therapy.
Aims
To evaluate whether acquired experience could translate into establishment of an effective and safe NHS Endobarrier service in patients with diabesity.
Methods
We initiated: i) an NHS Endobarrier service for patients with suboptimally controlled type 2 diabetes and obesity ii) a secure online registry to monitor outcomes.
Results
Since service initiation (October 2014), 46/118 (39.0%) referred have been accepted for treatment. Twenty-five patients have so far completed 1-year's Endobarrier treatment (age 51.9 ± 7.5 years, 56.0% male, 44.0% Caucasian, diabetes duration 12.0(8.5–21.0) years, 60.0% insulin-treated, BMI 41.1 ± 8.9 kg/m^2). Mean(±S.D.) HbA1c fell by 24.8 ± 25.0 mmol/mol from 81.2 ± 24.7 to 56.4 ± 11.5 mmol/mol ($P < 0.001$), weight fell by 16.3 ± 10.0 kg from 118.5 ± 27.7 to 102.2 ± 28.2 kg ($P < 0.001$), systolic blood pressure from 137.7 to 126.0 mmHg ($P = 0.006$) and ALT from 32.0 ± 20.5 to 17.4 ± 9.8 U/l ($P < 0.001$). In the 15 patients on insulin median (IQR) total daily insulin dose reduced from 104(60–135) to 12(0–65)units ($P = 0.003$) with 6/15 (40%) discontinuing insulin. Two patients had gastrointestinal haemorrhage having not complied with mandatory avoidance advice. There was one liver abscess. Early removal led to resolution in these cases. 93.8% patients would be extremely likely to recommend this service to friends and family.
Conclusion
This inaugural NHS service demonstrates Endobarrier to be highly effective in patients with refractory diabesity, with high patient satisfaction levels and an acceptable safety profile. As endoscopy units are ubiquitous, our service could be readily disseminated, with the registry useful for on-going monitoring nationwide.

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OC5

Copper deficiency presenting as subacute common peroneal nerve palsy post-duodenal switch surgery

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Background
Gastric reduction duodenal switch involves restrictive and mal-absorptive aspect, removing approximately 70% of stomach and most of the duodenum. Surgical alteration leading to malabsorption of macronutrients is achieved with varying results of weight loss but resulting nutritional side effects are under-recognised. Issues in nutritional complication leading to neurological complications like peripheral neuropathy are increasingly recognised. We discuss a case of subacute common peroneal nerve palsy as a result of copper deficiency.
Aim
To recognise the key features of non-compressive nerve palsy in post-bariatric surgery as a result of copper deficiency.
Case Report
A 40-year old male presented post-operatively with unilateral lower leg pain and weakness made worse by a progressive sensory ataxia with occasional steatorrhea. Four months prior to this he underwent a gastric reduction duodenal switch surgery for severe obesity (pre-operative weight 155 kg and BMI of 55). Patient achieved a rapid weight loss of 79 kg. His comorbidities include type 2 diabetes mellitus in remission and obstructive sleep apnoea. On clinical examination, he had a unilateral foot drop with concurrent deficiency in serum copper and caeruloplasmin levels of 13 mg/dl (normal range 15–30 mg/dl). His other biochemical investigations including iron, zinc (12.8 mg/dl; range 11.5–18.5 mg/dl), calcium, vitamin B12 and vitamin D were all normal with good concordance to routine oral supplements. He denied exogenous excess zinc ingestion. Nerve conduction studies were unable to be performed as he was lost during follow-up.
Conclusion
Copper deficiency must be increasingly common as growing numbers of bariatric gastrointestinal surgery. Copper deficiency should be considered as a differential in investigating for non-compressive neuropathy with low serum copper and caeruloplasmin levels. Screening for copper should be considered for patients who undergo bariatric surgery.

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OC6

Complexities cause considerable confusion in confirming a case of Cushing's (after gastric bypass surgery)

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Case history
A 26 year old female with severe gastro-oesophageal reflux disease was seen in our service for assessment prior to roux-en-Y gastric bypass (RYGB) surgery for this condition. She had additional history of obesity, hypertension, insomnia, cholecystectomy for gallstones and anxiety-depression. At age 18 her weight was 85 kg, BMI 28 kg/m^2. She gained 40 kg over the subsequent 8 years and in clinic was 123 kg, BMI 41.1 kg/m^2 with a gynaecoid pattern (waist to hip ratio 0.8). HbA1c was elevated at 6.8% and metformin commenced. She had no features suggestive of endocrine disorders. RYGB surgery was performed in May 2015, but weight loss was disappointing, with a nadir weight of 111 kg reached in February 2016, and her diabetes failing to resolve. This was accompanied by severe upper abdominal pain and worsening psychiatric symptoms including self harm. Repeat examination revealed violaceous abdominal striae. Clinical suspicion of cortisol excess was sufficient to warrant formal exclusion.
Investigation results
An abdominal CT scan to investigate her pain did not find a cause. Intra-abdominal fat volume did not appear high and her adrenals were normal with the exception of a subcentimetre adenoma on the left.
An in-patient low-dose dexamethasone suppression test (LDDST) was conducted, with cortisol and ACTH markedly failing to suppress (nadirs of 559 nmol/l and 40 ng/l respectively). Urinary free cortisol (UFC), however, was only modestly elevated at 565 nmol/day. Both cortisol and ACTH fully suppressed following an 8 mg overnight dexamethasone suppression test (< 5 ng/l and 35 nmol/l respectively). Pituitary MRI was equivocal, showing a possible small hypoenhancing lesion.
Discussion
The moderate pre-test suspicion, discrepant mean cortisol and UFC, lack of enlarged adrenals and relatively normal intra-abdominal fat lead to continued diagnostic uncertainty. The effect of RYGB on oral drug absorption, particularly lipophilic drugs such as dexamethasone, add an additional layer of difficulty to the already complex task of correctly diagnosing Cushing's. Alternative investigative options include the use of an equivalent-dose intravenous low-dose dexamethasone suppression test (to circumvent any potential malabsorption) or scrutiny of serial at-home midnight salivary cortisols.

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OC7

Pregnancy after bariatric surgery: a single-centre retrospective cohort study

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Background
The number of women seeking bariatric surgery in order to improve their fertility is increasing. Current guidelines recommend that pregnancy should be delayed for at least 1 year post-surgery. However, there are limited data regarding pregnancy outcomes post-surgery, particularly for sleeve gastrectomy.
Methods
We performed a single-centre, retrospective cohort study of pregnancies in women post-bariatric surgery. Surgical and maternity records were reviewed and maternal and fetal outcomes examined.
Results
Ninety-seven pregnancies in 69 women were documented over an 8-year period. Forty-one women had undergone a Roux-en-Y gastric bypass and 28 a sleeve gastrectomy. There were 79 pregnancies with a known outcome; 18 women were lost to follow-up during pregnancy. 32.9% of pregnancies occurred in the first post-operative year. Pregnancies conceived in the first post-operative year were associated with a higher miscarriage rate compared to pregnancies after this time (50% vs 30.4%, $P<0.05$). A pre-conception body mass index (BMI) of >35 kg/m^2 also associated with an increased miscarriage rate (22.8% vs 50%, $P<0.05$). Caesarean sections rates were similar in pregnancies conceived before or after the first post-operative year and in women with a BMI higher or lower than >35 kg/m^2. During pregnancy, two women developed gestational diabetes (1.9%) and there were no cases of gestational hypertension. Three women had a post-partum haemorrhage (2.9%). With regards to fetal outcomes, mean birth weight was 3.11 ± 0.1 kg and mean gestational age at birth 39.5 ± 0.4 weeks. Birth weight was not affected by duration from surgery or pre-conception BMI. There were two cases of severe fetal developmental abnormalities. Intrauterine growth restriction was seen in two pregnancies, both in the same woman.
Conclusion
Our data support current recommendations to delay conception until 1 year after surgery, however larger data collections are required to build a robust evidence base and allow guideline development.

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OC8

Assessing significant risks surrounding bariatric surgery in a patient with Emotionally Unstable Personality Disorder

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Case History
A 55 year old woman with a BMI of 57.4 was referred to our service to assess her suitability for bariatric surgery given her psychiatric surgery. Her problems with weight began aged 8 when she was sexually abused. She saw a doctor aged 14 who prescribed amphetamines for her weight, and states her problems with drugs as an adult (LSD, cannabis, amphetamines) stemmed from this. She has a diagnosis of EUPD-borderline type, and was prescribed quetiapine which had kept her relatively stable. She had a history of bingeing and purging but had not done so for 25 years. She picks her skin every day as a type of self-harm and eats the scabs. She has a history of four suicide attempts: three were overdoses and one was when she set fire to a boat with her inside it in 2011. At the time of initial assessment she had begun CBT for agoraphobia and was in the process of switching from quetiapine to aripiprazole.
Investigations
Mental state examination was in keeping with a diagnosis of EUPD, with labile mood. She reported hearing the voices of dead people since childhood, but had no other symptoms of psychosis. She reported intermittent suicidal ideation, but had a clear plan that she would definitely kill herself if she was not allowed to have bariatric surgery.
Management
Collateral psychiatric history was obtained from her GP and a plan made to review her after finishing the CBT. At review she had shown significant willing to improve her health by stopping smoking, improving her diet, switching her antipsychotic and engaging with CBT. She had been discharged by her community mental health team (CMHT) due to her stable mental health. However she continued to state that she would kill herself if she were not allowed bariatric surgery, and had drunk two glasses of wine prior to the appointment. It was felt that on balance she should be allowed surgery, but that there were significant inherent risks in either decision.
Discussion
There is evidence suggesting that patients who actively self-harm should not be allowed bariatric surgery due to an association with risk of relapse post-operatively. It was felt that in this case, her mental state had been stable for 5 years, and she had demonstrated enduring commitment to improving her maladaptive behaviours in other ways. However this case was laden with significant risks whichever decision was made.

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Poster Presentations

P1

Weight loss and associated improvements in cardiometabolic risk factors with liraglutide 3.0 mg in the SCALE Obesity and Prediabetes randomised, double-blind, placebo-controlled 3-year trial

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Aims/objectives
Obesity and prediabetes are risk factors for developing T2D. 5–10% weight-loss can reduce risk of developing T2D by >50%. The 3-year part of this phase 3 trial investigated effects of liraglutide 3.0 mg, as adjunct to diet + exercise, in delaying onset of T2D over 3 years, body-weight and cardiometabolic risk factors in adults with obesity or overweight with comorbidities, and diagnosed with prediabetes at screening.
Methods
Individuals (BMI $\geq$30 kg/m^2, or $\geq$27 kg/m^2 with $\geq$1 comorbidity) were randomised 2:1 to once-daily subcutaneous liraglutide 3.0 mg (n=1505) or placebo (n=749) and advised on a 500-kcal/day deficit diet and 150-min/week exercise. Efficacy data are observed means, with last-observation-carried-forward (LOCF) imputation. (NCT01272219).
Results
Baseline characteristics were (mean ± s.d.): age 47.5 ± 11.7 years, 76.0% female, weight 107.6 ± 21.6 kg, BMI 38.8 ± 6.4 kg/m^2. With continued treatment over 160 weeks, estimated time to onset of diabetes was 2.7 times longer with liraglutide 3.0 mg than with placebo (95% CI, 1.9–3.9, P<0.001), corresponding to a HR of 0.2. Based on the Kaplan-Meier plot of cumulative probability of a diagnosis of diabetes, 3% of patients in the liraglutide group vs 11% in the placebo group were diagnosed with diabetes by week 160 while on treatment. More individuals on liraglutide (66%) vs placebo (36%) regressed to normoglycaemia by week 160 (OR 3.6 [3.0;4.4], P<0.001). Individuals on liraglutide 3.0 mg lost more weight than on placebo (6.1% vs 1.9%; ETD −4.3% [95%CI −4.9;−3.7]), accompanied by greater mean reductions in waist circumference (ETD −3.5 [−4.2;−2.8] cm), SBP (ETD −2.8 [−3.8;−1.8] mmHg), triglycerides (ETD −6%[−9;−3]) and high-sensitivity C-reactive protein (ETD 29% [−34;−23]) (all P<0.001). Mean pulse increased with liraglutide 3.0 mg vs placebo (ETD 2.0 [1.2;2.7] beats/min, P<0.0001). AE incidence was 94.7% with liraglutide 3.0 mg vs 89.4% with placebo, SAEs 15.1% vs 12.9%. Adjudicated major adverse CV events (non-fatal MI, stroke, cardiovascular death) were low overall (0.19 vs 0.20 events/100 patient-years-of-observation for liraglutide 3.0 mg vs placebo).
Conclusion
Liraglutide 3.0 mg for 3 years, as an adjunct to diet + exercise, was associated with lower risk of T2D and greater weight loss, and improved cardiometabolic risk factors compared with placebo.

DOI: 10.1530/endoabs.48.P1

P2

Effect of increased endogenous glucose levels within Type 2 diabetes on cellular lipid profiles

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Background & Aims
ATP binding cassette subfamily G1 (ABCG1) is involved in mediating cholesterol efflux and modulating cellular lipid homeostasis. It is influenced by endogenous glucose and our aim was to examine the relationship between ABCG1 expression and lipid profiles within the increased glucose levels of subjects with Type 2 diabetes (ODM).
Methods
RNA was extracted from visceral fat collected from subjects undergoing abdominal surgery (bariatric and routine non-acute, non-malignant conditions). 30 subjects were categorised as non-obese (NO, n=10), obese (O, n=10) or ODM (n=10). Samples were examined using Real-Time qPCR and $2^{-\Delta\Delta Ct}$ data analysis in order to determine gene expression changes. Lipid profiles were measured using a colorimetric assay on the Randox Daytona Plus™.
Results & Conclusions
All three subject groups were well matched with weight/BMI being the only significant differences between clinical parameters. There was no overall change in the expression of ABCG1 when comparing the O to the NO group. Reduced expression of ABCG1 was observed in ODM compared to O and the NO group (ABCG1 = 2.0-fold and 2.4- fold decrease, respectively). An increase in plasma glucose correlate with the reduced expression of ABCG1 (P<0.05). Data suggests that this is having a knock on effect on lipid profile markers. Within the ODM there is a significant decrease in plasma levels of total cholesterol, HDL and LDLs (P; <0.05, <0.05, <0.01 respectively). This decrease in plasma lipids is independent of prescribed statin use. Therefore, hypothesised to be the result of a reduction in cholesterol export from the cell and consequently cellular lipid retention.

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P3

Hyperinsulinemic hypoglycaemia after bariatric surgery: Is it commoner than anticipated?

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Introduction
With the increasing incidence of obesity worldwide, there is growing demand for bariatric surgery which carries its own risks. Post Bariatric hyperinsulinemic hypoglycaemia is rare but yet challenging condition to manage.
Case Presentation
In this paper we are describing the case of a 54 years old female who presented with severe hypoglycaemia symptoms 10 months post her RYGB surgery. She underwent an oral glucose tolerance test through which she developed hypoglycaemia symptoms and was found to have a blood sugar of 1.7 mmol/l after 150 minutes of the glucose load with an insulin level of 28.7 mIU/l (2.0–25.0) and a C-Peptide level of 3.15 nmol/l (0.34–1.8) confirming a diagnosis of hyperinsulinemic hypoglycaemia. Different non-operative approaches were tried to help controlling her hypoglycaemic episodes with good response achieved by the combination of proper patient education, dietary modifications, Acarbose and Octreotide injections. As a reflection of this case, we have conducted a retrospective case series review for 422 patients who underwent a bariatric operation in James Cook Hospital–Middlesbrough from 2012 to 2016. 18 patients were confirmed to have hyperinsulinemic hypoglycaemia where different treatment options were tried to control the symptoms.
Conclusion
The incidence of sever hypoglycaemia occurring post bariatric surgery might be commoner that appreciated. Early recognition and patient involvement in the management are crucial to prevent life threatening consequences.

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P4

Night-blindness and neurological sequelae post bariatric surgery

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Background
Bariatric surgery is considered the most effective tool to manage the growing pandemic of obesity related health disorders. Lack of regular surveillance following bariatric procedures can put patients at risk of developing serious micro-nutritional deficiencies and related complications.
Case report
A 51-year-old lady presented to the bariatric services in 2015 with long standing neurological symptoms including memory and cognitive impairment, agitation, loss of dexterity, clumsiness, headaches, tiredness and poor night vision. She complained of pins and needles affecting her hands and feet. She was vomiting intermittently and complained of steatorrhea. She had undergone a Scopinaro gastric bypass procedure 25 years previously but had not been under regular follow-up and was not taking nutritional supplements. She had undergone a thyroidectomy for Grave's disease 3 years previously and was on Thyroxine replacement, her pre-operative weight was 160 kg (BMI 51.1) and her current weight was 96.9 kg (BMI 30.9). Prior to this, she had been referred to several specialist services, including gastroenterology, haematology, and dermatology for investigations of these symptoms and had been treated for anxiety and depression. She presented to the neurologists in 2013 who found no neurological deficit on examination. She was thoroughly investigated with imaging including

CT and MRI brain, MRI of the spinal cord and EEG and no significant abnormalities were detected. The Cerebral perfusion scan suggested the possibility of early Alzheimer's disease. The neurologists eventually carried out a nutritional assessment, to include iron, ferritin, folate, vitamin B12, vitamin D and she was found to be severely nutritionally depleted. She was started on iron, vitamin D, vitamin B12 and folate with some improvement in her neurological symptoms. Additional tests through our bariatric service found her to be deficient in vitamin A, Vitamin B12, copper, zinc and selenium. She was replaced with those and maintained on multivitamin replacement with marked improvement of her symptoms of night vision and neuropathy.
This case highlights the importance of life-long nutritional replacement post-bariatric surgery. Malabsorptive Bariatric procedures may affect absorption of several nutrients including fat-soluble vitamins (A, D and E) and other minerals including zinc and copper. If neurological sequelae are present, thiamine, vitamin B12 and copper should be assessed. Malabsorption of thyroxine may also affect thyroxine replacement which needs to be carefully monitored in these patients.

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P5

Oral glucose tolerance test should be selectively performed to confirm reactive hypoglycaemia in post-bariatric surgery patients

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Background
Bariatric surgery has contributed to a large spectrum of presentations of hypoglycaemia. Dumping syndrome caused by food reaching the duodenum rapidly is associated with abdominal pain, bloating and diarrhoea as well as vasogenic symptoms of tachycardia and flushing. Hypoglycaemia may occur as a late sign of dumping 1–3 hours after a meal. In contrast, post-gastric bypass reactive hypoglycaemia is thought to result from discordance between blood glucose circulation and insulin secretion. We report a case of reactive hypoglycaemic via oral glucose tolerance test (OGTT) in the absence of classical features of dumping syndrome immediately post-carbohydrate load.
Case report
A 59-year old lady was referred with light-headedness on and off for 3 years coinciding with tingling in her back, arms and lower limbs. These intermittent episodes last for up to 10 minutes, without any seizures. There is absence of clinical features of dumping syndrome up to 30 minutes after meals. She had a roux-en-Y gastric bypass surgery 7 years ago with co-morbidities including ischaemia heart disease, hypertension, hypercholesterolaemia, and arthritis. OGTT was performed by administering a 75 g oral glucose load, during which she had a nadir venous glucose of 2.2 mmol/l confirming the diagnosis of reactive hypoglycaemia. She had no features of dumping syndrome. Incidentally she had low zinc levels, 10.1 µmol/l (normal range 11.5–18.5 µmol/l) but normal vitamin B12, folate, ferritin, iron, vitamin D and lipid profile. Her gut hormones including glucagon, vasoactive intestinal peptide, chromogranin A, somatostatin and gastrin were all within normal limits. With dietary modification and dietician advice she managed to control her symptoms.
Discussion
Since Mayo report by Service *et al*. in 2005, it is hypothesised that post-prandial hypoglycaemia may be due to endogenous hyperinsulinaemia from abnormal islets, as a result of nesidioblastosis.
Conclusion
OGTT for reactive hypoglycaemia should be performed in a monitored environment. However, we should be cautious on patient selection for OGTT due to increasing reported incidence of adverse effects (64.8%) and hypoglycaemia (14.8%) during test in post-bariatric surgery patients.

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P6

Review of process of care for bariatric surgery in a specialist weight management centre

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Background
Whilst bariatric surgery is cost effective in the management of severe or complex obesity, successful outcomes necessitate specialist multidisciplinary pre-operative and post-operative care. The report by the National Confidential Enquiry into Patient Outcome and Death (NCEPOD), *Too Lean a Service? A review of the care of patients who underwent bariatric surgery* (2012) made critical recommendations for the process of care.
Aims
To assess the delivery of pre-operative and post-operative care for patients undergoing bariatric surgery in a single centre.
Setting
Specialist weight management service in a university teaching hospital.
Methods
We performed a retrospective observational analysis of the elements of pre-operative and postoperative care for patients who underwent bariatric surgery during a two-year period between 31st July 2010 and 31st July 2012.
Results
A total of 334 patients had bariatric surgery which included 231 (69%) gastric bypass, 86 (26%) sleeve gastrectomy, 13 (4%) gastric banding and 4 other procedures.
Pre-operatively, 98% had documented evidence of multidisciplinary team review at a one stop clinic that comprises bariatric specialist nurse, bariatric dietitian, bariatric physicians and bariatric surgeons. All patients underwent sleep studies for obesity-induced hypoventilation and obstructive sleep apnoea and 84% had documented pre-bariatric psychological assessment and counselling. All the patients had preoperative evaluation involving clinic review and blood tests, electrocardiogram, pulmonary function tests and anaesthetic assessment if required.
Postoperatively, 97% of patients had documented multidisciplinary ward rounds comprising bariatric surgeon, bariatric physician, specialist nurse and specialist dietician prior to discharge. During the first week after discharge patients received phone calls from the bariatric specialist nurse (95%) and specialist dietician (54%); 97% of patients were reviewed in a specialist nurse-led outpatient clinic at median 7 weeks after surgery. Further dietetic review was offered to 81% of patients and 68% attended. Specialist medical annual review was arranged for 97% of patients, of which 45% attended during the study period.
Conclusions
We report near universal levels of documented evidence of concordance with multidisciplinary elements of pre-operative care. Attendance for standardised long-term post-operative medical care should be improved but challenges include a wide geographical catchment area and the need to take time off work for appointments.

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P7

Setting a weight loss or clothes size goal following laparoscopic adjustable gastric banding: Does this impact on outcomes?

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Background
Setting a goal helps individuals achieve their long-term aims. A goal that challenges an individual, but at the same time is achievable, is likely to be attained. Candidates for laparoscopic adjustable gastric banding (LAGB) may have different long-term goals, which may include achieving a particular weight loss or clothes size.
Methods
39 individuals undergoing LAGB were prospectively monitored for 5 years. Participants were weighed, had a 1:1 interview, and completed three standardised psychometric questionnaires (CARVAL/CARSAL, DAS-24) at 7 time points; pre-LAGB, 6 months post-surgery, and annually until 5 years post-operatively. Prior to analysis the sample were grouped based on their pre-LAGB goal; weight loss ($n=23$) or clothes size ($n=16$). Repeated-measures ANOVAs were used to explore potential differences between groups.
Results
Psychometric measures demonstrated a positive difference in CARVAL, CARSAL and DAS-24 scores over the 5 years, but no differences were observed between the weight and clothes goal groups. Similarly, measures of weight, total weight lost, and excess body weight loss demonstrated a positive difference over

the 5 year period, but no difference was observed between the weight and clothes goal groups.
Conclusion
Outcomes following LAGB appear to be positive regardless of the long-term goal an individual chooses. If clinicians are aware of an individual's long-term goal following surgery, advice and support may be tailored to help them to achieve this goal. Knowing the individual's goal is important, as this will help clinicians identify and manage unrealistic long-term goals.

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P8

Perioperative nutritional status, supplementation and monitoring in patients referred for bariatric surgery: Closing the audit loop

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Background
Nutritional deficiencies are frequently seen in bariatric patients, and typically become increasingly common and resistant following bariatric surgery. Local, national and international guidelines recommend routine blood tests and lifelong supplementation protocols after surgery. Local practice was initially audited for 2013. The purpose of this re-audit was to assess the impact of changes made in the department and to assess concordance with current recommendations.
Methods
Using electronic patient record systems and clinic letters, data were extracted for 137 patients who underwent gastric band, bypass and sleeve gastrectomy procedures in 2014. Data on pathology results, timings of samples and nutritional supplementation were recorded and compared to 2013 audit data.
Results
For bypass patients, a greater percentage of patients underwent postoperative monitoring blood tests at the 6 and 12 month time points relative to the initial audit, although once again the proportion of patients undergoing monitoring fell at each successive time point. More patients were discharged following surgery with Forceval multivitamin and a combined calcium and vitamin D supplement. Fewer patients were discharged with iron (26%). Despite the fact that fewer than 10% of bypass patients were receiving the recommended 3 monthly vitamin B12 injections in the first year after surgery, less than 10% of patients were found to be B12 deficient. Low Vitamin D was a common preoperative finding and for each of the three surgical procedures greater than 85% of patients were insufficient or deficient. In general, vitamin D deficiency became less common over time postoperatively, presumably due to increased surveillance and replacement in the early postoperative period. Monitoring of ferritin, folate, vitamin B12 and vitamin D was below 80% at all time points.
Conclusion
Whilst there have been increases in monitoring blood tests and provision of postoperative nutrient supplements after bariatric surgery, there is still room for improvement in compliance with local guidelines. A closer interaction between the bariatric service and Primary Care could support these vital elements of postoperative care and also allow more comprehensive audit and research in this area.

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P9

Comparison of the impact of Roux-en-Y gastric bypass and sleeve gastrectomy on 2-year post-operative glycaemic outcomes in people with type 2 diabetes: the role of weight-loss

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Background & aims
Data comparing the effect of Roux-en-Y gastric bypass (RYGB) and sleeve gastrectomy (SG) on type-2 diabetes (T2D) outcomes and the relationship between weight-loss (WL) and T2D remission are limited. Thus, we undertook a retrospective study of patients with obesity and T2D who underwent RYGB or SG and examined the relationship between 2-year (2y) post-surgery T2D and WL outcomes.
Material & methods
Patients who underwent RYGB (107) or SG (103) as a primary procedure were included in the study and followed up to 2y post-surgery. The DiaRem score, a validated pre-surgery T2D remission score utilising HbA1c, age, glycemic medications and insulin usage, was calculated for each patient. Combined T2D remission (CR = total + partial) was defined according to the 2nd Diabetes Surgery Summit (DSS-II) Consensus group criteria (HbA1c $<6.5\%$/48 mmol/mol for ≥ 12 months without T2D medications). Multivariate adjustment analysis was used to correct for baseline confounding factors and odds of CR were tested categorizing %WL in quintiles, continuous scale and 5%WL groups.
Results
Pre-surgery the RYGB group had a lower BMI ($43.1+6.3$ vs $48.2+7.8$, $P<0.001$), greater insulin usage (70% vs 30%, $P<0.05$) and higher DiaRem score ($8.3+5.2$ vs $6.0+4.4$, $P<0.001$). At 2y post-surgery the %WL was higher in the RYGB compared to the SG [26.6% vs 20.6%, $P<0.001$]. CR was associated with younger age, female gender, higher BMI and lower pre-surgery DiaRem scores. RYGB patients had 175% higher odds of CR compared to SG, ($P=0.012$). This association became non-significant when adjusted for WL ($P=0.157$). For every 5%WL the odds of CR increased by 61% ($P<0.001$). This association remained after adjusting for gender, BMI and surgery type.
Conclusion
In conclusion, at 2y, RYGB led to greater %WL accompanied by superior T2D outcomes compared to SG. %WL plays a major role in determining glycaemic improvements.

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P10

Change in body mass index (BMI) after highly active antiretroviral therapy among hiv patients in kano, Northwestern Nigeria

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Background
Highly Active Antiretroviral Therapy (HAART) has improved the health and wellbeing of people living with HIV, but at the same time, it causes excessive weight gain through abnormal fat distribution (lipodystrophy). Overweight and obesity have implications on the cardiovascular status of these patients. We aimed to determine the change in BMI after the commencement of HAART and the factors associated with this change.
Methodology
It was a longitudinal prospective study. One hundred and eighty HIV patients that met HAART criteria were recruited before the commencement of therapy. Their weight, height, waist and hip circumferences, blood pressure and laboratory investigations were done. Six months into HAART the anthropometric and laboratory parameters were repeated. Only data of 150 participants were available at the end of the study.
Results
The mean age of the participants was 35.7 ± 10.0 years, and 64% of them were females. Mean BMI pre-HAART was 19.4 ± 5.9 kg/m^2 while post-HAART mean BMI was 24.0 ± 6.0 kg/m^2 ($P<0.000$). Before commencement of HAART, 46.0% of the participants were underweight, 40.0% of normal weight, 10.0% overweight and 4.0% obese. After initiation of HAART, 12.7% were underweight, 55.3% of normal weight, 16.7% overweight and 15.3% obese. The factors associated with increased BMI were impaired fasting glucose, Diabetes, Insulin resistance, raised triglyceride, low HDL, increased waist circumference and waist-hip ratio and metabolic syndrome ($P<0.05$). There was a statistically significant association between development of obesity and increased CD4 cell count ($P=0.007$).
Discussion
Exposure to HAART causes stabilization of weight in the majority of the participants and in others they became overweight and obese. This finding is similar what was found in other short-term studies that looked at weight changes following HAART initiation in other parts of the world. Duration of HAART and type of regimen has no effect on this weight change. Weight gain causes metabolic derangement which can cause cardiovascular problems in these patients.
Conclusion
Exposure to HAART causes weight gain and its attendant complications in HIV patients. There is the need for adequate metabolic follow-up for these patients.

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P11

Reduction in the risk of developing type 2 diabetes (T2D) with liraglutide 3.0 mg in people with prediabetes from the SCALE Obesity and Prediabetes randomised, double-blind, placebo-controlled trial

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Background
The 3-year part of this trial investigated the effect of liraglutide 3.0 mg, as an adjunct to diet + exercise, in delaying onset of T2D (primary endpoint) in adults with prediabetes and obesity (BMI $\geq$30 kg/m^2) or overweight ($\geq$27 kg/m^2) with comorbidities.
Methods
Participants were randomised 2:1 to once-daily subcutaneous liraglutide 3.0 mg or placebo plus 500 kcal/day deficit diet and 150 min/week exercise. Efficacy data are observed means, with last observation carried forward for missing values. Clinicaltrials.gov ID: NCT01272219.
Results
Of 2254 randomised individuals with prediabetes (age 47.5 $\pm$ 11.7 years, 76.0% female, weight 107.6 $\pm$ 21.6 kg, BMI 38.8 $\pm$ 6.4 kg/m^2, mean $\pm$ S.D.), 1128 completed 160 weeks (52.6% on liraglutide, 45.0% on placebo). At Week 160, mean weight loss (WL) was 6.1% with liraglutide vs 1.9% with placebo (estimated treatment difference $-$4.3% [95%CI $-$4.9;$-$3.7], $P<0.0001$). Comparing liraglutide and placebo, 49.6% vs 23.7% of individuals achieved $\geq$5% WL (estimated odds ratio [OR] 3.2 [2.6;3.9]) and 24.8% vs 9.9% achieved $>$10% WL (OR 3.1 [2.3;4.1]), both $P<0.0001$. Based on the Kaplan-Meier plot of cumulative probability of a diagnosis of diabetes that takes censoring into account, 3% of patients in the liraglutide group vs 11% in the placebo group were diagnosed with diabetes by week 160 while on treatment. With continued treatment over 160 weeks, the estimated time to onset of diabetes was 2.7 times longer with liraglutide than with placebo (95% CI, [1.9;3.9], $P<0.001$), corresponding to a hazard ratio of 0.2. Liraglutide was generally well tolerated. Gallbladder-related events (2.9 vs 1.2/100 patient-years of observation [PYO]) and confirmed pancreatitis (0.29 vs 0.13 events/100 PYO) were low, but more frequent with liraglutide.
Conclusion
Liraglutide 3.0 mg for 3 years, plus diet + exercise, was associated with lower risk of T2D and greater weight loss compared with placebo.

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P12

Role of peri-operative psychiatric assessment and intervention in managing the obese patient presenting for bariatric surgery: A complex case of post-bariatric surgery addiction transfer

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Objective
A case study that demonstrates the importance of thorough psychiatric assessment, intervention and follow-up in morbidly obese patients presenting for Bariatric surgery and risk of addiction transfer.
Background
Positive correlation between psychiatric disorders and obesity is documented. Cases of addiction transfer have been reported complicating outcome post Bariatric Surgery.
Case description
A 29 year old Caucasian female with a BMI of 51 kg/m^2 and background of previous episodes of depression, anxiety and previous paracetamol overdose referred for Bariatric Surgery. She had history of binge eating and alcohol dependence both were not active at time of review. She has been assessed by psychiatric team and was deemed suitable for surgical intervention hence had Roux-en-Y Gastric Bypass surgery.
2 weeks postoperatively she had relapse of binge eating and purging and was unable to comply with recommended vitamins and minerals supplements. She reported feeling 'out of control', had started binge-drinking and developed a compulsive-shopping habit. She also reported episodes of 'wild behaviour' where she would engage in sexual activity with strangers. Unfortunately her financial situation had spiraled out of control and she had been evicted from her home after falling into arrears. She was living with her mother and had been unable to attend appointments due to financial constraints. She was re-referred to psychiatry for urgent assessment. She failed to attend further reviews but re-engaged 3 months later when she called to inform the dietitians she was pregnant. Under the care of the specialty midwife team she was able to bring her alcohol intake under control but continued to binge eat throughout pregnancy since she felt this was her only coping mechanism. She was found to have multiple nutritional deficiencies mid-pregnancy which required urgent correction. She eventually delivered a healthy baby but continued to require support from social services and family in caring for her child. Most recently having sought a second psychiatry opinion she was diagnosed with bipolar disorder and was started on treatment.
Of note is that postoperatively she achieved up to 60% loss of excess body weight which was relatively maintained at 2 years follow-up (56.1% excess body weight loss) but unfortunately despite her ongoing difficulties she reported not having adequate psychological support through her journey.
Conclusion
Addiction transfer is a documented risk after gastric bypass surgery in vulnerable candidates. High vigilance is required in patients with prior history of alcohol dependence and eating disorders even if no longer active. Bariatric surgery can help losing weight but might complicate mental illness. Psychological follow-up and support should be a routine in managing patients at high risk post Bariatric Surgery.

Keyword: Obesity, Addiction transfer, Bariatric surgery, Psychiatry
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P13

Insulin and glucose homeostasis 5 years after bariatric surgery

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Background
Literature suggests that whole glucose excursion, rather than plasma glucose concentration at a point, provides more information about glucose tolerance. The glucose area under the curve (AUC) is an index of whole glucose excursion after glucose load. We sought to investigate changes in insulin and glucose homeostasis, including the glucose AUC, 5 years after bariatric surgery.
Method
A non-randomised prospective study of 18 participants with T2DM undergoing bariatric surgery [12 females, mean age 50.4 $\pm$ 6.4 years, mean body mass index 55.3 $\pm$ 14.1 kg/m^2, median duration of diabetes 29 months]. Serial measurements of glucose, insulin and C-peptide were performed during the 75-g oral glucose tolerance test pre-operatively and 5 years post-operatively. The glucose AUC were examined at 30, 60 and 120 minutes.
Results
Significant reduction (baseline vs 5 years) in 2-hr plasma glucose (2 hr-PG) [13.4 (10.1–16.4) vs 8.4 (6.0–12.1) mmol/L, p = 0.007]; HbA1c [7.5 $\pm$ 1.7 vs 6.4 $\pm$ 1.4%, p = 0.001]; fasting C-peptide [1.3 $\pm$ 0.5 vs 0.7 $\pm$ 0.5 nmol/L, p = 0.004]; 2-hr C-peptide [3.2 $\pm$ 1.6 vs 1.9 $\pm$ 1.4 nmol/L, p = 0.033]; and improvement in HOMA%S [log transformed (1.5 $\pm$ 0.2 vs 1.8 $\pm$ 0.4, p = 0.02)] were observed. Fasting plasma glucose (FPG) showed non-significant reduction at 5 years [7.6 (5.8–9.4) vs 6.5 (5.6–9.1) mmol/L, p = 0.136]. There were no changes in the median glucose $AUC_{0\text{-}30}$ 4.5 (3.9–6.1) vs 4.5 (3.4–6.5), $AUC_{0\text{-}60}$ 10.9 (9.4–15.0) vs 10.4 (8.5–15.2) and $AUC_{0\text{-}120}$ 18.4 (14.2–22.9) vs 18.7 (11.7–21.5), baseline vs 5 years, respectively.
Conclusion
The traditional glycaemic markers (2 hr-PG and HbA1c) suggest improvement in glucose homeostasis 5 years after bariatric surgery. However, the glucose AUC measures suggest otherwise.

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P14

Clinical outcomes of bariatric surgery in a specialist centre

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Background
Bariatric surgery is the most successful treatment for patients with severe and complex obesity.
Aims
To assess clinical outcomes after bariatric surgery in a specialist centre.
Setting
Specialist weight management service in a university teaching hospital.
Methods
We performed an observational analysis of clinical outcomes for patients who underwent bariatric surgery during the period 31st July 2010 and 31st December 2010. All patients had assessment of their weight and body mass index (BMI), nutritional status, co-morbid conditions and medications. The results at the 36-month follow up are reported.
Results
A total of 76 patients (63 women) had bariatric surgery (61 gastric bypass, 13 sleeve gastrectomy and 2 gastric banding). The follow up rate at median 36 months after surgery was 90% (68 patients). Patients achieved significant reduction in BMI (Table 1). Significantly fewer patients were on hypoglycaemic and antihypertensive medications and continuous positive airway pressure therapy (CPAP). No changes were observed in the numbers of patients taking anti-depressants and analgesics. Levels of haemoglobin, serum iron, folate and vitamin B12 were similar or significantly higher than baseline.
Conclusion
At a median follow up of 36 months, bariatric surgery was associated with significant reduction in weight and resolution or improvement in co-morbid conditions without compromising serum levels of key micronutrients.

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Table 1 Clinical outcomes before and at median 36 months after bariatric surgery.

Outcome measures	Before BS	36 months after BS	P (Paired t-test)
Median BMI (kg/m^2)	52	35	$<$0.0001
Regular medicines (number of patients)			(Fisher's exact test)
Antidiabetic drugs	20	9	0.0351
Analgesics	36	28	ns
Antihypertensives	33	14	0.0011
Antidepressants	20	17	ns
CPAP	27	5	0.0001
Blood results (Median)			(Unpaired t- test)
Haemoglobin A1c (mmol/mol)	44	36	$<$0.0001
Haemoglobin (g/L)	133	136	ns
Serum Iron (μmol/L)	10.5	13.5	0.0008
Serum Ferritin (μg/L)	50	113	0.0403
Serum vitamin B12 (ng/L)	319	652	$<$0.0001
Serum folate (μg/L)	6.9	12.1	ns

CPAP, Continuous positive airway pressure therapy; ns, non-significant

Society for Endocrinology NATIONAL CLINICAL CASES FRIDAY 27 JANUARY

National Clinical Cases

Oral Communications

O1

40 years of hypoglycaemia and an adrenal mass

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Case History
A 69-year-old gentleman was admitted having lost consciousness. This episode was preceded by typical hypoglycaemic symptoms which, in retrospect, he had experienced for four decades with increasing frequency and severity over the past year. He was hypertensive. There was no family history of endocrinopathy or diabetes.
Investigations
A supervised inpatient fast was undertaken along with cross-sectional and radio-isotope imaging.
Results and Treatment
Hypoglycaemia (glucose 1.8 mmol/l) occurred following an overnight fast. Corresponding insulin and c-peptide measurements were inappropriately detectable – consistent with endogenous hyperinsulinaemic hypoglycaemia. Serum calcium and cortisol were normal.
Abdominal imaging revealed a large locally invasive left supra-renal mass with associated lymphadenopathy which was both FDG- and MIBG-avid. There was also diffuse mild FDG uptake throughout the pancreas without an anatomical correlate.
These unexpected imaging findings prompted further investigations. Chromogranin A and B and urine normetadrenaline were all elevated – consistent with a tumour of neuroendocrine origin.
We postulated that this tumour might be releasing a secretagogue that was stimulating pancreatic insulin release. Analysis of incretin hormones at the time of hypoglycaemia revealed a markedly and inappropriately elevated GLP-1 in combination with an undetectable neurotensin (co-secreted with GLP-1 from the gut). This is suggestive that the observed hyperinsulinaemia was mediated by tumoural GLP-1 production.
Hypoglycaemia was eliminated by somatostatin analogue therapy. Whilst awaiting surgery he represented with weight loss and worsening abdominal pain. Repeat imaging showed disease progression such that surgery was no longer felt to be in his best interests. He underwent chemotherapy with stable disease after four cycles.
Conclusions and points for discussion
We describe a unique case of symptomatic hyperinsulinaemic hypoglycaemia mediated by tumoural GLP-1 release from a malignant MIBG and FDG-avid adrenal lesion.

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O2

Metastatic pituitary carcinoma in an *SDHB* mutation positive patient

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Case history
A 63-year-old female presented with bi-temporal hemianopia. Pituitary MRI demonstrated a macroadenoma with suprasellar extension. Her medical history included a glomus tumour of the right ear treated with external beam radiotherapy (EBRT) 25 years previously. She had no evidence of pituitary hormone abnormality and had normal urinary metanephrines levels. She underwent transsphenoidal surgery with total resection and full recovery of her visual fields. Immunohistochemistry demonstrated a null cell pituitary adenoma. Recurrence developed 5 years later.
Investigations
Histology showed an atypical null cell tumour, Ki67 was 10%, MGMT methylation negative, with unusual features of widespread cytoplasmic vacuolisation and negative SDHB staining. Genetic testing revealed a heterozygote pathogenic missense *SDHB* mutation.
Results and treatment
On follow-up MRI a new extra axial cerebellar lesion at the foramen magnum was visible, with extension into the left hypoglossal canal and an intradural extra medullary cervical lesion at the level of C3/4. Both were initially thought to be metastatic deposits from the previous paraganglioma, but histology of the cerebellar lesion demonstrated a metastatic deposit from a pituitary carcinoma, composed of large extensively vacuolated cells; with positive immunostaining for cytokeratin MNF116, LMW cytokeratin, synaptophysin and pituitary transcription factors SF-1, and negative for pituitary hormones, TTF1, EMA and S-100 protein.
Conclusions
Pituitary carcinoma is a very rare phenomenon, accounting for only 0.1% of all pituitary tumours. *SDHB* mutations are known to have a higher risk of developing aggressive and malignant paragangliomas, with figures reported as high as 30%; thought to be due to the higher prevalence of extra adrenal paragangliomas. Other tumours such as GIST, renal cell carcinoma and thyroid papillary carcinoma are recognised in these patients; as well as links to pituitary adenomas. However, here we describe, the first case of pituitary carcinoma in a patient with a germline *SDHB* mutation.

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O3

Compensated hyperthyrotropinaemia due to partial loss-of-function mutation in TSH receptor gene

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Case history
A clinically euthyroid 7-year-old boy was noted to have a persistently elevated TSH 7.35–14 mU/l (NR 0.27–4.2) and normal FT_4 15.0 pmol/l (NR 10-24) with negative anti-thyroid peroxidase antibodies. Thyroid ultrasonography revealed a eutopically-located thyroid gland of normal size. Following commencement of levothyroxine, he developed insomnia, irritability and headaches, resulting in cessation of treatment. Growth and development proceeded normally, however sequential thyroid hormone measurements over 7 years revealed failure to suppress TSH fully despite high-normal FT_4 concentrations and further trials of levo-T_4 replacement.
Investigations
Re-evaluation off medication aged 16 years revealed an elevated TSH 20.1 mU/l (NR 0.4–4.0) in the setting of a normal FT_4 14.3 pmol/l (NR 9–20) and FT3 6.0 pmol/l (NR 3.0–7.5). In addition, his serum calcium and PTH were normal. Direct sequencing of the TSH receptor gene (TSHR) was undertaken following exclusion of assay interference.
Results and treatment
A heterozygous, loss-of-function TSHR mutation (c.122G>C, p.Cys41Ser) was identified, supporting a diagnosis of partial TSH resistance. Cys 41 is located in the extracellular domain of the receptor and substitution with serine has previously been shown to result in impaired binding of TSH to the TSHR *in vitro*. This molecular diagnosis provided justification for withholding further levo-T_4 treatment, and the patient subjectively improved.
Conclusions and points for discussion
Loss of function mutations in TSH receptor gene are associated with a spectrum of phenotypes. The degree of resistance is dictated by the severity of receptor functional impairment, with partial forms showing a heterogeneous hormonal and clinical profile. Levo-T_4 replacement should be instituted in severe resistance but there is a paucity of evidence to guide treatment in partial TSH resistance cases. Available literature suggests that these individuals may not require treatment but should nonetheless continue sequential biochemical evaluation.

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O4

Something to make you twitch: an interesting case of severe hyponatraemia

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Case History
A 69-year-old lady was admitted to her local district general hospital with a history of confusion, memory disturbance and focal twitching affecting her upper

limbs and face. She had a past medical history of type 2 diabetes mellitus, hypertension, hypercholesterolaemia and ischaemic heart disease. Her medications included gliclazide, pioglitazone, atorvastatin, bisoprolol, aspirin and omeprazole. She had never smoked and was teetotal. Neurological examination on admission did not demonstrate any obvious cranial nerve abnormalities or lateralising signs.

Investigations

Admission bloods revealed a severe hyponatraemia of 114 mmol/l (133–146) and low magnesium of 0.54 mmol/l (0.66–0.99). Serum osmolality was low at 270 mOsmol/kg (275–295), urine osmolality was 408 mOsmol/kg (80–1200), and urine sodium was 24 nmol/l. Serum cortisol was appropriate at 884 nmol/l. A vasculitic screen was negative. CSF analysis was completely normal. A CT scan of the chest, abdomen and pelvis showed endometrial thickening but no other significant abnormality. CT head showed multifocal white matter low density lesions at the grey-white matter interface, and MRI brain showed widespread bilateral multifocal areas of increased T2 signal intensity.

Results and treatment

Voltage-gated potassium channel (VGKC) antibody titres were raised with positive LG-I IgG. She was treated with intravenous Methylprednisolone 1 g daily for 3 days followed by a reducing dose of prednisolone (starting dose 60 mg daily). On this regime her MOCA score increased from 7/30 to 13/30 and her sodium rose to 132 mmol/l on discharge from hospital. Subsequently her cognitive function returned to normal relatively quickly and she has remained seizure-free.

Conclusions and points for discussion

Hyponatraemia is the commonest electrolyte disorder affecting hospitalised patients. The clinical triad of poor memory, brachiofacial seizures and severe hyponatraemia is consistent with a VGKC-associated limbic encephalitis. Hence it remains an important differential diagnosis in patients presenting with neurological disturbance and unexplained hyponatraemia. Early recognition of this condition is vital as timely, aggressive immunotherapy may allow reversibility of the disease process and thereby halt neurocognitive decline, as evidenced in our case.

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O5

Hypercalciuric hypocalcaemia – potential pitfalls and a novel treatment option

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The calcium-sensing receptor (CaSR) is a G-protein coupled receptor expressed in the parathyroid gland and kidneys. Loss of function mutations of the CaSR result in familial hypocalciuric hypercalcaemia (FHH). Rarer, gain of function mutations of the CaSR result in hypercalciuric hypocalcaemia and are inherited in an autosomal dominant pattern. The phenotype varies from asymptomatic individuals, to profound hypocalcaemia. We present a severely affected individual whose case highlights the potential pitfalls in treatment of hypercalciuric hypocalcaemia and a novel therapeutic approach with a continuous parathyroid hormone (PTH) infusion. A 16 year old, Caucasian female was referred to our unit with severe hypocalcaemia and frequent fits. She was born at 31 weeks gestation and aged 2 days old was noted to be hypocalcaemic, hypomagnesaemic and hypoparathyroid. The patient's mother had hypocalcaemia since childhood and an initial diagnosis of familial hypoparathyroidism was made. Genetic analysis aged 4 years identified a heterozygous missense CaSR mutation and she was diagnosed with hypercalciuric hypocalcaemia. Her medication included sandocal, magnesium, alfacalcidol and bendrofluazide. Biochemistry: Corr Ca++1.4–2.9 mmol/l, creatinine 101 μmol/l. Renal ultrasound:early nephrocalcinosis. A trial of subcutaneous PTH 1–34 20 μg bd and cessation of alfacalcidol/bendrofluazide, resulted in improved hypercalciuria, but persistent hypocalcaemic fits. She was thus trialled on a continuous PTH infusion, via an insulin pump (omnipod, unlicensed use) at 1.3 μg/h (concentration 125 μg/ml) resulting in a stable serum calcium, with no fits for 1 year. She was converted to a Medtronic pump due to supply issues and developed hypercalcaemia, requiring a reduction in PTH to 0.735 μg/h (concentration 35 μg/ml). This case demonstrates the potential severity of hypercalciuric hypocalcaemia and the very difficult balance between avoiding hypocalcaemic seizures and hypercalciuria-associated nephrocalcinosis. It highlights the importance of avoiding vitamin D in this condition, which worsens hypercalciuria and nephrocalcinosis. Our patient has impaired renal function and her mother has end stage renal failure. This case demonstrates the dramatic clinical improvement and smaller PTH doses that can be achieved with a continuous PTH infusion. It also shows the potential variability in PTH delivery between different insulin pumps.

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O6

Using Kisspeptin to assess GnRH function in an unusual case of primary amenorrhoea

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Case history

An 18-year-old Caucasian female presented with primary amenorrhoea after her younger sister aged 16 years old started menstruating. She had a normal childhood and progressed through puberty normally, with no past medical history or family history. BMI was 22.6 with a normal examination including secondary sexual characteristics (Tanner Stage 5) and no signs of hyperandrogenism.

Investigations and Results

Bloods showed an elevated LH:FSH ratio, low oestradiol with borderline raised testosterone, similar to results found in polycystic ovarian syndrome (PCOS). Other pituitary function and pelvic ultrasound were normal and Karyotype was 46XX. DEXA scan showed a lumbar Z-score −1.0 and wrist X-ray fused bones. She had a withdrawal bleed post progesterone, indicating normal uterine function. A GnRH stimulation test confirmed normal pituitary gonadotroph functioning and 8-h blood sampling every 10 min for LH showed normal LH pulsatility but an elevated LH baseline and pulse amplitude. Hypothalamic GnRH function was tested by administering IV kisspeptin, a neuropeptide that stimulates GnRH neurons to secrete GnRH. She responded normally by raising gonadotrophins (surrogate for GnRH secretion). Therefore, with normal investigations and hypothalamic pituitary function but raised LH, the most likely diagnosis was of PCOS presenting with primary amenorrhoea.

Treatment

The oral contraceptive was given to provide oestrogen and progesterone replacement for regular withdrawal bleeds as well as to achieve peak bone mass.

Conclusions and points for discussion

PCOS has been shown to present with primary amenorrhoea. Although our patient is not the classical phenotype, a subset of patients are lean with little or no signs of hyperandrogenism and so should be considered in the differential diagnosis of primary amenorrhoea. An early and prompt diagnosis of PCOS is important, as up to 30% of these women are predisposed to glucose intolerance and obesity. Interestingly the subgroup presenting with primary amenorrhoea display a higher incidence of metabolic dysfunction. Kisspeptin has the potential to form a novel diagnostic tool for assessing hypothalamic GnRH function. Confirmation of intact GnRH function helps consolidate a diagnosis in primary amenorrhoea and gives an indication of future fertility.

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O7

Hypertestosteronemia and primary infertility due to an extragonadal germ cell tumor of the anterior mediastinum

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A 26-year-old Caucasian male presented to the joint infertility outpatients clinic with primary infertility. His medical history included hypertrophic cardiomyopathy (HCM) due to genetically confirmed *MYH7* sarcomere protein mutation, treated with implantable cardioverter-defibrillator while his partner was a healthy 24-year-old Caucasian nulliparous female. Initial investigations showed hypertestosteronemia (Testosterone: >51.0 nmol/l) and azoospermia, hence the couple was referred for endocrine review. During consultation, he reported hoarseness of voice, hypersexuality, and increased hair distribution over the past 3 years. He denied ever having used anabolic steroids and was only on amiodarone to ameliorate arrhythmias from his known HCM.

On examination he was hirsute with bilaterally small testes. A testicular/scrotal ultrasonography was unremarkable. Subsequent investigations revealed elevated testosterone (52.9 nmol/l), b-hCG (900 IU/l) and suppressed FSH and LH: < 1 IU/l. The provisional diagnosis of an extragonadal germ cell tumor (EGCT) was made and whole body contrast enhanced CT revealed a 7×6×5 cm mass of the anterior mediastinum without further disease dissemination.

Due to his HCM and reduced EF: ~35% he was not eligible for neo-adjuvant treatment with bleomycin-etoposide-cisplatin (BEP), in view of the increased risk for cardiotoxicity. He was instead referred for transthoracic resection of the tumor, which he had uneventfully with R0 resection margins. Immediately following excision of the mediastinal mass, his testosterone substantially dropped (Testo: 1.0 noml/l) confirming that the mediastinal mass was the source of β-hCG driven testosterone hypersecretion. Histopathology revealed a mixed primarily seminomatous (95%) with minor teratomatous (5%) component EGCT.

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O8

Time to change the focus with a new treatment for primary aldosteronism

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Case history
We report a 56-year-old man who was referred to the Endocrinology Clinic with hypokalaemic hypertension. He had a history of bladder cancer 9-years previously, treated with surgery, chemotherapy and BCG therapy. At referral, he was taking Amlodipine 10 mg and Doxazosin 8 mg twice daily with average home blood pressure readings of 160/90 mmHg.
Investigations
Aldosterone to renin ratio (ARR), taken on Doxazosin with potassium 3.6 mmol/l was 3900 (renin 0.2 nmol/l per h and aldosterone 780 pmol/l). Overnight dexamethasone suppression test was normal, excluding co-secretion of cortisol. CT adrenal demonstrated a 9 mm left adrenal adenoma. He underwent adrenal vein sampling, which lateralised to the left adrenal with appropriate suppression on the right, which was concordant with imaging of a left adrenal adenoma. The Adrenal MDT agreed that he should be referred for a left adrenalectomy and he was listed for a left retroperitoneoscopic adrenalectomy.
Results and treatment
At surgery, he had significant perinephritis related to previous BCG therapy and decision to perform a partial adrenalectomy was made. In the postoperative period, he remained hypokalaemic and hypertensive. At day 14 in the Endocrine Clinic, blood pressure was 150/90 mmHg on Amlodipine 10 mg, potassium 3.9 mmol/l on Sando-K 2 tablets bd and ARR 1460. His histology showed normal background adrenal architecture and a benign cortical adenoma. However, postoperative imaging demonstrated a partial left adrenalectomy and a 6 mm nodule in the lateral limb of the left adrenal still visible. The treatment options at this point for persistent primary aldosteronism (PA) were re-do surgery, long-term medical therapy or radiofrequency ablation (RFA). He underwent RFA.
Conclusions and discussion
Post-RFA, he was able to discontinue potassium supplements and antihypertensive therapy. His potassium was 4 mmol/l, renin 0.8 nmol/l per h, aldosterone 70 pmol/l and ARR 88; suggesting cure from PA. The evidence base shows that CT guided-percutaneous RFA is an effective and safe treatment option for PA. However, at present there is insufficient evidence for valid comparison with surgery for resolution of PA and hypertension. As our case demonstrates, it is a justifiable alternative for patients who are unfit or reluctant for surgery.

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O9

Retroperitoneal fibrosis presenting with panhypopituitarism

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Case History
A 68-year-old gentleman with hypertension and diet-controlled type 2 diabetes presented in September 2015 with weight loss, fatigue, low libido and cold intolerance.
Investigations
Blood results demonstrated secondary hypothyroidism (TSH 0.59 mU/l (reference range 0.35–5.00), free T4 8.3 pmol/l (ref 9.0–21.0)), hypogonadotrophic hypogonadism (testosterone 1.0 nmol/l (ref 10.0–36.0), FSH 1.5, LH 1.1) and a modestly elevated prolactin (795 mU/l (ref <400)). Short Synacthen test revealed baseline cortisol of 87 nmol/l rising to 376 nmol/l after 30 min. Serum angiotensin converting enzyme and ferritin were normal. MRI pituitary with contrast revealed normal appearances of the pituitary gland.
Results and treatment
Initial working diagnosis was panhypopituitarism of unclear aetiology. He was treated with oral hydrocortisone, levoT_4 and Testogel, with good symptomatic improvement. Six weeks later, he re-presented with a swollen left leg. Doppler ultrasound excluded DVT. To exclude underlying malignancy, CT thorax, abdomen and pelvis was performed. This confirmed extensive inflammatory-looking tissue within the abdomen and pelvis, involving the left ureter and iliac vessels resulting in hydroureteronephrosis. Radiological appearances and subsequent biopsy were in keeping with retroperitoneal fibrosis. A diagnosis of IgG G4-related disease (IgG4 RD) was made on the basis of his clinical presentation, radiological appearances and biopsy findings. Serum IgG4 levels in this patient were within normal limits. He was commenced on high-dose oral prednisolone. Repeat imaging has demonstrated a modest reduction in the inflammatory material around the left ureter, distal aorta and iliac vessels. He remains well and continues on prednisolone at a dose of 5 mg once daily.
Conclusions and points for discussion
IgG4 RD is a collection of disorders characterized by tissue infiltration with IgG4-positive plasma cells and CD4 + T lymphocytes, accompanied by fibrosis. It may affect one or more organs, and in this case manifests as retroperitoneal fibrosis and hypophysitis. Lymphadenopathy is often present, alongside weight loss in those with multiorgan disease.
Retroperitoneal fibrosis is common has been associated with hypopituitarism in a number of case reports of IgG4 RD. The relationship is poorly understood, but the underlying pituitary disease is thought to be related to hypophysitis. Diagnosis is based on characteristic histopathological features on biopsy. Serum levels of IgG4 are only elevated in 60–70% of patients. Most patients respond well to glucocorticoid treatment.

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O10

Broken bones and blindness – a rare cause of osteoporosis

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Case History
A 37-year-old gentlemen was referred to metabolic bone clinic due to recurrent recent fragility fractures. He had suffered several fractures throughout childhood, and these had continued to occur into his adult life. He was blind since birth in his left eye. He felt that this had contributed to his fractures due to falls and other accidents. There had been no other major health problems in the past. His brother had a similar eye condition. There were no other risk factors for osteoporosis. There was nothing of significance to find on examination.
Investigations and results
DXA scan- Spine L1-L4 T-score −3.6, Left hip T-score −2.4. CTX 0.24 μg/l (normal), P1NP 34 μg/l (normal). Spine X-rays: T11 grade 1 fracture and T_4 grade 2 fracture.
No other secondary cause of low bone mass identified (normal levels of testosterone, parathyroid hormone, vitamin D, calcium, phosphate and coeliac antibodies).
Discussion
This is a case of a young man with seemingly unexplained multiple fragility fractures and low bone mass. The clue to the aetiology for low bone mass was the nature of the gentleman's hereditary eye condition. The eye problems are caused by a condition known as Familial Exudative Vitreoretinopathy (FEVR). FEVR is a rare genetic disorder affecting retinal angiogenesis that can cause progressive visual loss. FEVR may be mediated by mutation in LRP5 coding for the LRP5 transmembrane receptor. LRP5 plays a key role, alongside Frizzled protein, in the Wnt signalling pathway, which has effects on cellular proliferation, adhesion and migration. Importantly, Wnt signalling is also known to regulate bone mass. Therefore, it is the defect in this pathway that is the common denominator for this gentleman's blindness and bone problems. We speculate that upcoming new drugs that target Wnt signalling in osteoporosis, such as Romosuzumab (a Sclerostin inhibitor), may be particularly beneficial in patients with low bone mass associated with FEVR.

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Poster Presentations

CP1

Here Comes The Zebra!

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Case history
A 22-year-old woman presented with a week's history of abdominal pain and constipation. further history revealed episodes of tachycardia during childhood but no cause at that time was identified. On examination she had a sinus tachycardia, her abdomen was mildly tender and the rest of her clinical examination was completely unremarkable, including blood pressure, and there were no clinical findings consistent with Addisonian crisis.
Investigations
Sodium was found to be 104 mmol/l. Biochemistry confirmed a diagnosis of SIADH and she was referred to endocrinology. In view of the history of abdominal pain and tachycardia, and normal synacthen test, although a rapid urine dipstick test was negative a diagnosis of acute intermittent porphyria was considered.

Table 1

	D1	D2	D3	D4	D5	D6
Serum Sodium *133–146 mmol*/l	106	103	105	112	122	131
Urine Sodium	202					
Urosmolality	767					

Table 2

Total Porphyrin Creatinine Ratio *0–40 nmol/mmol creat*	984
Porphobilinogen Creatinine Ratio *0.0–1.5 umol/mmol creat*	49
Porphobilinogen	509
Porphobilinogen Creatinine Ratio (*0.0–1.5 umol/mmol creat*)	57.9

Treatment
Her serum sodium was monitored meticulously with careful administration of hypertonic 5% saline. Although acute porphyria was suspected she was not treated with haem arginate. Biochemical confirmation of AIP was made some days after the acute episode She was discharged 8 days after admission with a normal serum sodium and a list of safe drugs to avoid further episodes. A precipitating cause for the acute episode was not identified. A genetic confirmation of AIP was also made (c.912G>C, p(Gln304His) in hydroxylmethyl bilane synthase gene. She is undergoing follow up the National Porphyria Clinic.
Conclusion
Acute intermittent porphyria (AIP) is a very rare disease which can present with hyponatraemia and a wide range of nonspecific symptoms. In view of its rarity it is not often seen apart from in MRCP questions and as clinicians we may not be confident in its management. Severe hyponatraemia can be challenging to manage and hypertonic saline needs to be given carefully in a high dependency area. We will highlight the pertinent diagnostic and management issues of both AIP and severe hyponatraemia.

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CP2

Scratch below the surface: you never know what you might unearth
A case of suspected Di-George at 64 years of age

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Case history
We present the case of a 64 year old gentleman diagnosed with hypoparathyroidism following presentation with acute hypocalcemia post thoracic aneurysm surgery for aortic dissection. The etiology was felt to be a complication of thoracic surgery with possible disruption of blood supply to the parathyroid glands.
Deeper investigation at a subsequent review revealed persistent hypocalcaemia predating surgery by over 10 years. This was on the background of considerable mental health history diagnosed as obsessive compulsive disorder with compulsive spending. At clinic review he also had facial characteristics that raised the suspicion of an inherited genetic abnormality of Di-George syndrome as a unifying diagnosis. In retrospect, this gentleman had a chronic Type B aortic dissection on an aneurysmal ascending aortic arch, a classical cardiovascular manifestation of Di-George Syndrome. This case marks the oldest reported age at diagnosis for DiGeorge syndrome in the literature.
Investigations
Serum Calcium, PTH, CT-aorta, cytogenetic testing.
Results and treatment
Late diagnosis of Di-George resulted in the need for total aortic arch replacement (frozen elephant trunk technique), significant psychiatric morbidity and alfacalcidol replacement.
Conclusions and points for discussion
This case highlights the need to challenge established diagnoses. There is a clear need to review historical biochemical trends, and join together diagnoses made by disparate speciality teams – *a key role for the modern endocrinologist!*

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CP3

Vitamin B very strong – complex endocrine dysfunction

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A 40 years old man with a diagnosis of Adrenoleukodystrophy (ALD) was referred for evaluation of symptoms suggestive of hypogonadism. He had a past medical history of ALD associated adrenal insufficiency and osteoporosis. He took regular hydrocortisone and a trial medication, MD1003.
Following review, post clinic blood tests revealed a testosterone 24.5 nmol/l (7.6–31.4), LH 8.8 IU/l (1.7–8.6) and FSH 1.5 IU/l (1.5–12.4). Thyroid function tests had been reported urgently with an elevated FT4 > 100 pmol/l (12–22), FT3 12.3 pmol/l (4.6–6.8) and a suppressed TSH 0.02 mIU/l. The patient was not clinically thyrotoxic, had no goitre and no opthalmopathy. Assay interference was suspected and so the sample was sent to the supra-regional assay service laboratory (Cambridge) to be analysed by DELFIA immunoassay. The results confirmed assay interference with a FT4 19.5 pmol/l (10–19.8), FT3 6.8 pmol/l (3.5–6.5) and a normal TSH 1.23 mIU/l (0.35–5.0). The likely causative agent was the trial medication, MD1003. The MD1003 – Biotin European AMN Trial is a study of ultra-high dose biotin in patients with ALD. Biotin interference, particularly with thyroid function testing, has been highlighted recently and may occur in samples analysed by Roche cobas immunoassays used in our institution. Typically, this may cause positive interference in competitive immunoassays (FT4 and FT3) and negative interference sandwich immunoassays (TSH).
Given biotin interference was likely to be an issue in all of our immunoassays, we turned our attention to his gonadotrophins axis. Analysis by a different immunoassay (Siemens Centaur) confirmed likely assay interference with a testosterone of 13.1 nmol/l, LH 30.7 IU/l (1.5–6.3) and FSH 13 IU/l (1.0–10.1). He is now being considered for testosterone replacement.
This case highlights – i) The importance of detailed history and clinical assessment where laboratory tests are discordant with the clinical picture. ii) The need to recognised biotin interference in Roche cobas immunoassays, which if missed, can lead to unnecessary referrals; investigations; treatment or treatment delays. iii) Interference may affect multiple immunoassays, either raise or lower hormone levels and cause marked (thyroid function) or mild discrepancies (gonadotrophins).

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CP4

A novel approach to the investigation of an atypical adrenal lesion: ^{11}C-metomidate PET-CT combined with ^{18}F-FDG PET-CT in a rare case of adrenal Hodgkin's lymphoma

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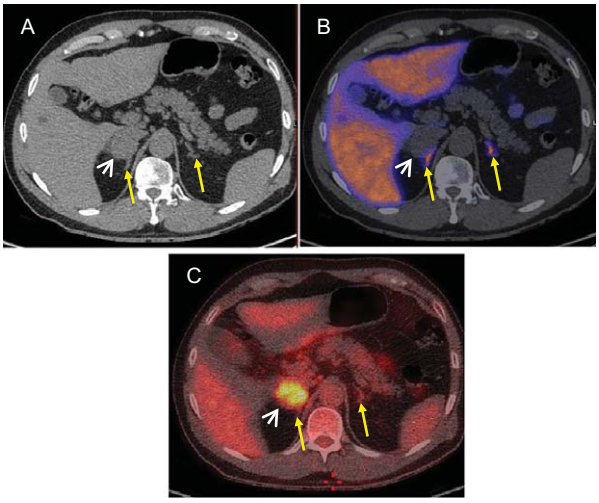
Case history
A 66-year-old man presented with a 1 month history of a productive cough, in the context of a 20 pack-year smoking history. CT chest revealed minor bronchiectasis and an incidental 3 cm right adrenal lesion. He was referred to the endocrine clinic for further evaluation. On questioning, he reported no symptoms suggestive of adrenal hormone hypersecretion and, aside from a short history (<1 month) of sweating episodes at night, he described no other constitutional symptoms. Physical examination was unremarkable, with no stigmata of endocrine disease.
Investigations
Routine biochemistry, including renal and liver function and full blood count, returned normal results. Plasma metanephrines were not elevated, and the plasma aldosterone: renin ratio was unremarkable. Serum cortisol suppressed to 27 nmol/l following a 1 mg overnight dexamethasone suppression test and plasma ACTH was not suppressed. DHEAS was normal. Further imaging was arranged to characterise his adrenal lesion.
Results and treatment
The adrenal nodule was considered indeterminate on the original CT chest, with Hounsfield units (HU) of 45. A dedicated adrenal scan demonstrated a 45 mm irregular right adrenal lesion, indicating a 50% increase in size in just 8 weeks (**Panel A**, white arrowhead denotes lesion, yellow arrows indicate normal adrenal tissue). Triple phase studies revealed an attenuation value of 40HU at baseline, 62HU at 1 min following contrast and 52HU on 10 min delayed imaging (washout 45%), suggesting the lesion was not a benign adenoma. No separate primary tumour was identified on staging CT. ^{11}C-metomidate, which binds CYP11B1 and B2 in adrenocortical tissue, showed no uptake by the mass, but demonstrated normal tracer distribution within the left adrenal gland and preserved uptake in remaining normal right adrenal tissue adjacent to the mass (**B**). In contrast, ^{18}F-FDG-PET revealed high tracer uptake in the right adrenal mass and in 6 mm retrocaval and 11 mm aortocaval nodes, but low uptake at sites corresponding to the normal left adrenal and residual remaining normal right adrenal gland (**C**). An open adrenalectomy was performed and a 75×50×35 mm right adrenal lesion was resected. Histology and immunohistochemistry confirmed a compressed normal adrenal gland surrounded by, and partially infiltrated by, a firm white lesion comprising predominantly CD4 and CD8 positive T lymphocytes, with larger atypical cells staining for CD30, PAX5 and EBR, and negative for ALK and CD45. These appearances are characteristic for classical Hodgkin's lymphoma (HL - nodular sclerosing type). The FDG-avid retrocaval nodes also demonstrated HL.



Conclusions and points for discussion
This unusual case provides the opportunity to discuss:
i) the approach to investigating an adrenal incidentaloma,
ii) demonstration of the potential utility of the novel paradigm of combining ^{11}C-metomidate and ^{18}F-FDG PET-CT to distinguish between primary adrenal tumours and other benign or malignant lesions and
iii) one of the first descriptions of primary adrenal Hogdkin's lymphoma.

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CP5

Pasireotide: successful treatment of a resistant case of Acromegaly

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Case history
A 26 year old lady presented in 1999 with a 3 year history of worsening headache, increased sweating, excessive tiredness and progressive enlargement of hands and feet. She was otherwise well and had no family history of pituitary tumors, hypercalcaemia or renal stones. On examination she had typical facial features of acromegaly and acral enlargement. Her blood pressure was normal and visual fields were full.
Investigations
Oral Glucose Tolerance Test (OGTT) confirmed Acromegaly (nadir GH: 772 mU/L). Biochemistry suggested excessive disease burden (IGF-1 −1710 μg/l, mean GH >600 mU/l). Other hormone testing showed, secondary hypogonadism (LH<0.5, FSH <0.37, Oestradiol <50 pmol/l), elevated prolactin of 796 mU/l and hypothyroidism (TSH 10.8 IU/l, free T4 of 6 pmol/l). MRI scan showed a 4×2×3 cm pituitary macroadenoma with suprasellar extension and right sided cavernous sinus invasion.
Treatment
She underwent Transsphenoidal Pituitary Surgery (TSS) of the pituitary tumor. Histology revealed moderate amounts of sparsely granular eosinophilic cytoplasm. Immunohistochemistry was positive for growth Hormone and negative for other pituitary hormones.
Postoperatively, Acromegaly remained uncontrolled with an IGF -1 of 1474 μg/l and mean GH 228 mU/l. An MRI of the pituitary revealed residual tumor in the cavernous sinus and close to the optic chiasm. The patient underwent external beam fractionated radiation five months after surgery. Over the following years she was treated with Octreotide LAR up to 30 mg every 2 weeks and Lanreotide autogel up to 120 mg every 2 weeks. Lanreotide was also combined with Cabergoline 1 mg twice per week. The lowest IGF-1 level achieved was still >600 μg/l.
In 2004, she was treated with Pegvisomant which resulted in some control of the disease, IGF-1 levels dropped from above 1000 to 340 μg/l. Pegvisomant had to be stopped as the patient developed abnormal liver function tests. In 2010, the patient had Stereotactic radiosurgery targeting tumor within the cavernous sinuses as a form of debulking. She was also part of a trial with antisense therapy to the GH receptor.
Over 15 years none of the treatments were able to control the disease burden and the patient remained highly symptomatic with headaches, fatigue, and osteoarthritis.
In October 2015, the patient was treated with Pasireotide 40 mg four weekly. Within a month of starting treatment the IGF-1 levels dropped and remained within the normal range for age (103–310 μg/l). Pasireotide is well tolerated, the patients symptoms have greatly improved she is now back to work and riding her bike. The patient has not developed glucose intolerance. In view of the good response to Pasireotide, somatostatin receptor (SSR) subtyping was done. This revealed positivity for SST5 and SST2a subtypes.
Conclusion
This case demonstrates that there are patients with severe acromegaly resistant to first generation SRLs' that can be well controlled on Pasireotide.

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CP6

The long search for an occult ectopic ACTH-producing tumour

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Case history
Ectopic adrenocorticotropic hormone (ACTH) production accounts for 10–20% of all endogeneous Cushing's syndrome. The ideal treatment is curative surgery of the underlying tumour. In difficult cases bilateral adrenalectomy is an option. We report a 58-year-old woman with an aggressive ectopic Cushing's syndrome that required bilateral adrenalectomy undertaken in 1989 at age 32-years.
Investigations
At 16 years post adrenalectomy, two right lung nodules became apparent on subsequent surveillance imaging. Over a 10-year period, these two foci on CT and Octreotide imaging demonstrated only marginal interval growth. The lung multidisciplinary team therefore advised against surgical intervention in 2006.
Results and treatment
However, in 2015, these two foci increased in size with growth noted in proximity to the main vessels and bronchi. She was therefore referred for surgery and underwent a right lung wedge resection. Histology was consistent with two bronchial typical carcinoids with clear resection margins and stained positive for ACTH. Post-operative imaging (CT chest, abdomen and pelvis, and gallium DOTATATE) demonstrated surgical lung changes, but no evidence of new or recurrent DOTATATE avid disease.
Conclusions and discussion
Since surgery, her tan significantly decreased. ACTH fell from a preoperative level of 1465 ng/l to 12 ng/l postoperatively with reduction in pigmentation, suggesting cure from ectopic ACTH-secretion. Ectopic ACTH-secreting tumours present challenges and require careful clinical, biochemical, radiological and pathological investigations. As this case demonstrates, ectopic ACTH-producing tumours can be extremely difficult to localise and often require multiple modalities of imaging. At present, there is no accepted frequency for imaging in occult ACTH-producing tumours. Our patient had an occult and indolent tumour that took almost two decades to present but has now had curative surgery.

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CP7

Postpartum diagnosis of a phaeochromocytoma: A lucky escape!

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A 34-year old lady presented in the postpartum period following her 3rd pregnancy with severe hypertension. Her first two pregnancies were 3 and 5 years previously where she delivered via elective Caesarean section without any complications. During this pregnancy which was a twin pregnancy, her antenatal care was mainly unremarkable but on specific questioning, she reported increased sweating for a period of 4 months. Her blood pressure was noted to be normal during her antenatal care. When she was 35 weeks pregnant, she became unwell with significantly increased sweating. On admission to the labour ward, her blood pressure was 194/149 mmHg and urine dipstix revealed 2+ proteinuria. A diagnosis of severe pre-eclampsia was made and she was treated with Nifedipine and Magnesium Sulphate. She had an emergency Caesarean section with delivery of a healthy set of twins. Post operatively, her blood pressure remained high necessitating IV Labetolol, Amlodipine and Ramipril. An ultrasound identified a 4.1 cm lesion in the right adrenal bed which was subsequently confirmed by MRI. Blood tests showed a normal dexamethasone suppression test and renin/aldosterone ratio. Two 24 h urine collections confirmed significantly elevated urinary metadrenaline levels at 38.8 µmol and 46.2 µmol respectively. She was commenced on phenoxybenzamine which was gradually titrated up and then referred for laparoscopic adrenalectomy.
Phaeochromocytomas in pregnancy are exceedingly rare with a frequency of 0.002% of all pregnancies. Although the fetus is not directly affected by the catecholamines, this condition can have potentially disastrous consequences for the mother and fetus as maternal hypertension can lead to placental insufficiency and placental abruption. In a recent systematic review looking at 77 case reports of phaeochromocytomas in pregnancy, 80% were diagnosed during pregnancy with the remaining within 1 month postpartum. Survival rates are improved if the diagnosis is made during the antenatal period. There was no definite advantage noted in proceeding with tumour removal during the second trimester.
We present a rare case of a phaeochromocytoma diagnosed in the postpartum period with a positive outcome for both mother and fetus.

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CP8

Hypothyroidism in a patient dependent on total parenteral nutrition

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Case history
A 30-year old female presented with a 10-month history of enlarging neck mass, fatigue and weight gain. Assessment by her GP found her to have a large smooth goitre and biochemical hypothyroidism. The patient had a history of intestinal failure secondary to mitochondrial disorder. As a result of intestinal failure she was entirely dependent on parenteral nutrition and was intolerant of any oral intake, with venting of her stomach to reduce pain. She was referred to endocrinology for consideration of intravenous thyroid hormone replacement. It was noted that her anti-TPO Abs were negative, and the aetiology of her hypothyroidism unclear. Subsequent enquiry revealed that 18 months prior to presentation the TPN regimen had been changed with the removal of the vitamin and mineral supplement, Additrace. Thus the patient was not receiving iodine in her feeding regimen. Vitamin supplementation was recommenced with adequate iodine. The patients goitre reduced in size and TFTs returned to normal within 2 months. Thyroid hormone replacement was not required.
Investigations
TSH, fT4, fT3, and TPO Abs were measured.
Results and treatments
Initial TFTs showed TSH 8.2 mIU/l (0.2–4.2) and fT4 5.9 pmol/l (11.0–22.0). TFTs were repeated with anti-TPO Abs and these showed TSH 25.0 mIU/l, free T4 3.1 pmol/l and TPO Abs <28 u/ml. Free T3 was within normal limits. The patient received treatment with Additrace. It contains a number of trace elements and vitamins, including iodine. The patients daily iodine supplementation was 1 umol once Additrace was commenced. After iodine supplementation was added to the TPN regimen, TSH normalised to 1.3 mIU/l within 2 months of treatment. The goitre resolved completely.
Conclusions and points for discussion
Iodine deficiency is widespread in the developing world and is a leading cause of hypothyroidism, though rarely seen in developed countries. There are few cases in the medical literature of endemic goitre and hypothyroidism in adults receiving TPN. This case highlights the importance of iodine supplementation in patients with no oral intake, and the importance of considering iodine deficiency within the differential diagnosis of hypothyroidism.

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CP9

Searching for the cause of high HCG in a man

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We present a 50-year-old man who was referred to endocrine clinic with painful gynaecomastia of 3 months duration. He was waiting hip replacement. He had history of a lump in his left breast 9 years ago. He had USS and FNA. He was treated with some tablets for a month and discharged from breast clinic. He had no other past medical history. He worked as a physical trainer to metropolitan police. He did not smoke or drink and was on no medication. He had never used recreational drugs. His BMI was 26 kg/m^2.
Examination and USS confirmed bilateral gynaecomastia. USS of the testes was normal with a small hydrocele on the left side. His blood tests showed FSH of <0.1 (1–10 IU/l), LH <0.1 (2–9 IU/L), testosterone 36.2 nmol/l (6.68–25.70), oestradiol 354 pmol/l (99–192), SHBG 44 noml/l (20.6–76.7) and his HCG was 250 IU/l (0–2). Ct Thorax, abdomen and pelvis was reported to be normal. His whole body bone scan was normal. His Repeat HCG in 6 weeks was 1265 and subsequently in 3 months increased to 3756. He had NM Whole body FDG PET CT which showed suspicion of an

aggressive lesion in the anterior mediastinum with metastatic deposits in the lung. Mediastinal biopsy showed no unequivocal evidence of malignancy and a panel of immunohistochemical stains was not contributory. He had elective Lt anterior mediastinotomy + VATS. Anterior mediastinal mass biopsies confirmed choriocarcinoma. He was referred for further treatment to Royal Marsden hospital.

Beta-human chorionic gonadotropin (β-hCG) is normally produced by syncytiotrophoblasts of the placenta and may also be secreted by germ cell neoplasms. An increase of serum hCG concentration in a male patient often suggests malignant neoplasms with a trophoblastic element. Common examples include classic seminoma with syncytiotrophoblast-like giant cells, combined germ cell tumour, and choriocarcinoma. Non-gestational choriocarcinomas typically arise from gonadal organs but they may originate in extragenital sites such as the mediastinum, retroperitoneum, pineal gland, liver, gallbladder, and urinary tract. Ectopic secretion of β-hCG is associated with a poorer prognosis.

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CP10

Bladder paraganglioma presenting in pregnancy as an incidental mass on a first trimester scan

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Case history

This 32-year-old lady was found to have a 23 × 17 mm bladder mass incidentally identified on a first trimester scan and confirmed with renal tract ultrasound. Urine collections showed mildly raised normetadrenaline 4.32 umol/24 h (normal range 0–3.0), plasma normetanephrine 1339 pmol/l (120–1180), no metastases were identified on MRI and genetic screening was negative. The patient had two previous normal pregnancies and deliveries. BP in the current pregnancy was mildly elevated at 10 weeks gestation (140/71 mmHg) but remained normal during pregnancy and she was otherwise asymptomatic. Despite normotension low dose alpha blockade with doxazosin was used to try to prevent marked BP fluctuations and in preparation for delivery, however the patient was non-compliant due to side effects. A multidisciplinary decision was taken to induce labour at 41 weeks with alpha blockade prior to labour given caesarean section would require significant bladder manipulation. The patient attended in spontaneous labour at 40+5, an epidural was sited and labour progressed normally. Two minutes after delivery the mother experienced sudden severe headache and BP was recorded as 151/62 mmHg. Headache and BP improved without treatment and syntocinon was given (rather than syntometrine which includes ergotamine and can cause hypertensive crises). Mother and daughter were discharged on day 2 post delivery with the patient on prazocin 5 mg bd. She is shortly to be seen by endocrinology and urology for planned resection of the paraganglioma.

Prevalence of paraganglioma in the urinary bladder is $<1\%$ and those associated with pregnancy are extremely rare. Recommended management is based upon case reports, small case series and expert opinion with traditional recommendation that vaginal delivery is best avoided due to risk of hypertensive crisis from active labour. Although bladder paragangliomas often present with hypertension and symptoms of catecholamine excess our case is consistent with a small case series highlighting pregnancies with paragangliomas may be at lower risk of adverse outcome than those with phaechromocytomas (*Wing JCEM 2015*). This rare case exhibits a positive outcome due to early recognition, investigation and initiation of treatment with a multidisciplinary approach.

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CP11

A malignant looking renal mass is not always renal cancer

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A 58-year-old female presented to Urology clinic after an abdominal ultrasound revealed a right sided mass. She had a 9 year history of hypertension which had been difficult to control despite treatment with six agents. She also had a history of Type 2 Diabetes Mellitus and Hyperlipidaemia. CT Thorax/Abdomen/Pelvis reported a 20 cm right renal mass with areas of calcification and following MDT discussion the diagnosis of renal cell carcinoma was made. An open right radical nephro-adrenalectomy was performed with no reported intra-operative complications. However, her post-operative period was complicated by recurrent episodes of hypotension and acute kidney injury. She received multiple courses of antibiotics and required two admissions to Critical Care. She developed type 2 respiratory failure secondary to fluid overload and had a brief spell of non-invasive ventilation. Echocardiogram revealed a new regional wall motion abnormality suggesting a perioperative myocardial infarction. Her hypotension episodes continued despite stopping her antihypertensives and she also developed hypoglycaemia. Morning cortisol was 170 nmol/l, prompting the initiation of Hydrocortisone. She was eventually discharged after 48 days and subsequent dynamic testing demonstrated a profoundly inadequate response to Synacthen stimulation. Histological analysis of the extracted mass reported a normal kidney and adrenal gland within a malignant neuroendocrine carcinoma. This stained strongly with synaptophysin and negatively for chromogranin A, however the presence of vasoactive amines suggested it had arisen from a pre-existing functioning phaeochromocytoma. Functional imaging after discharge showed no evidence of active disease or metastasis. We present this rare and fascinating case of a malignant neuroendocrine tumour encapsulating a normal kidney and adrenal gland that was originally diagnosed as a renal cell carcinoma. It is likely the sudden reduction in circulating catecholamines following tumour excision significantly contributed to her unstable post-operative period and resultant prolonged admission. Her pre-existing hypertension and Type 2 diabetes have now resolved and she remains off the medications. This case raises the question of whether patients with a renal mass in the context of resistant hypertension should be screened for phaeochromocytoma during their pre-operative assessment.

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CP12

An incidental pituitary tumour: blood, fret and no tears

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Case history

A 73-year-old female with a background of hypertension presented to the acute physicians with a 4 month history of progressive headaches. The headaches did not have any 'red flag' features. A non-contrast CT scan revealed an intrasellar lesion. She was referred to endocrinology for further management. Her history did not suggest hypopituitarism, Cushing syndrome or acromegaly. There were no visual field defects and her eye movements were normal.

Investigations

Biochemical investigations revealed normal sodium, prolactin, IGF-1, FT4 and TSH levels. A Short Synacthen test was also normal. A non-contrast MRI of her pituitary showed a well-defined, 15 mm lesion on the left side of the pituitary fossa of indeterminate signal on both T1 and T2 sequences. An intrasellar aneurysm was suspected, hence the imaging was followed up with a magnetic resonance angiogram. This confirmed an aneurysm of the anterior communicating artery, within the sella.

Results and treatment

The aneurysm was treated endovascularly with a flow diverting stent across the neck of the aneurysm. A follow-up MRI scan 5 months later showed the lesion was now hyperintense on T1 MRI, in keeping with a thrombus within the aneurysm.

Conclusions and points for discussion

Incidentally discovered intrasellar lesions are common, with a prevalence ranging between 1 and 30% in imaging and autopsy series. The differential diagnoses include neoplasia, cysts, inflammatory, infectious or vascular lesions. Intrasellar aneurysms, which mostly originate from the internal carotid artery are rare causes of intrasellar lesions, with a recent systematic review identifying only 40 such lesions in the published literature. Aneurysmal rupture is a presenting feature in 15% of cases. Others present with headaches, visual field deficits or endocrinopathies, of which hyperprolactinaemia or hypogonadism are common. Importantly, 20% of intrasellar aneurysms can coexist with an adenoma. Our patient has done well following her flow diverting stent and her aneurysm is likely to involute within 12–18 months. Intrasellar aneurysms presenting as pituitary lesions deserve a high level of suspicion, as missing these can be catastrophic. Endovascular repair is now the treatment of choice for most of these lesions.

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CP13

ACTH-dependent Cushing's syndrome unmasked following transphenoidal surgery for Acromegaly – the rare coexistence of dual endocrinopathies

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Case history
We describe a rare case of a 59-year-old woman, whose hypercortisolism was unmasked following transphenoidal surgery for Acromegaly. She presented to the Endocrine Clinic in 2006 with acromegalic features and MRI revealing a pituitary macroadenoma 20×18×18 mm. Repeat dynamic evaluation showed inadequate GH suppression (initially normal), raising the possibility of early rumbling Acromegaly. 0900 h serum cortisol was 287 nmol/l. She was started on Cabergoline, later switched to Lanreotide but underwent transphenoidal surgery in 2015 due to evolving visual defects. Histology revealed a sparsely granulated somatotroph adenoma, ACTH negative with Ki67 < 1%. Postoperative serum cortisol was noted > 1000 nmol/l on discharge.
A month later, she attended outpatients with typical Cushingoid facies, new onset diabetes mellitus, hypertension with severe hypokalaemic alkalosis and significant proximal myopathy and was admitted acutely.
Results and treatment
Random serum cortisol > 2000 nmol/l, with failure to suppress on overnight, low and high dose dexamethasone suppression. ACTH was elevated at 118 ng/l. She was initiated on Metyrapone and Octreotide with clinical improvement a few days later. Repeat MRI pituitary revealed significant residual pituitary tumour burden with new bilateral avid adrenal hyperplasia on FDG-PET (normal 6 months earlier), with persistent incidental non FDG-avid lung and thyroid lesions. She unfortunately became septic whilst awaiting transfer to the local tertiary centre and died from a stroke.
Conclusions and points for discussion
This case highlights the rare coexistence of two endocrine pathologies with Cushing's syndrome being unmasked following discontinuation of Lanreotide and the importance therewith of close endocrine surveillance in postoperative pituitary patients in the acute, medium and long-term phases. Prompt early management of active Cushing's is imperative to minimize significant morbidity and mortality. The true source of her excess ACTH causing hypercortisolism (pituitary or ectopic) remains a discussion point as post-mortem findings were inconclusive.

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CP14

Oligo-amenorrhoea – a triple whammy?

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Case history
A 20-year-old female first presented to our endocrine clinic in 2013 with a 6-month history of feeling faint, palpitations, weight loss and oligo-amenorrhoea. She was found to have autoimmune thyrotoxicosis with a fT3 of 15.2, fT4 43.3 and TSH < 0.05. Her TPO antibodies were strongly positive. She was subsequently commenced on Carbimazole 20 mg once a day and was biochemically euthyroid within 6 months. Interestingly, however, she continued to lose weight and remained oligo-amenorrhoeic. Her BMI was now 18 (weight 46 kg).
Investigations
Her initial pelvic ultrasound did not show polycystic ovaries. Serum testosterone was 1.2 mmol/l, LH 0.9 U/l, FSH 3.7 U/l, oestradiol 161 pmol/l, prolactin 124 mU/l with 0900 cortisol and remaining pituitary profile within normal range. MRI pituitary was unremarkable. Her TFTS remained within normal range off the Carbimazole. DEXA scan: T-score −0.2 at lumbar spine, −0.1 at left hip.
Results and treatment
She was diagnosed with anorexia nervosa by the specialist eating disorders team. With their treatment and support, she gradually gained weight from 46 kg in January 2014 to 54.3 kg in April 2015, 65 kg in September 2015 and now 73 kg in 2016 (BMI 30). However despite normalisation of her low body weight, spontaneous periods did not resume. Her repeat pelvic ultrasound in October 2015 showed ovaries that were a little bulky with several small peripheral follicles and were thought to be polycystic in appearance with a reverse FSH:LH ratio (FSH 7.1 U/l, LH 9.3 U/l, oestradiol 255 pmol/l, Prolactin 132 mU/l, TFTs normal) suggestive of PCOS. She has no immediate plans to start a family and is taking an oral contraceptive pill at present, which is giving her regular withdrawal bleeds. She has been given lifestyle advice to help her regain a normal weight.
Conclusion and points for discussion
This case highlights multiple diagnostic and treatment challenges in a young patient with oligo-amenorrhoea. It is proposed that her amenorrhoea may originally have been due to Graves' disease, was subsequently due to her persistent low body weight (hypothalamic amenorrhoea secondary to anorexia nervosa), and ultimately, after gaining excess weight, due to exacerbation of underlying polycystic ovary syndrome – a clinical conundrum?

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CP15

Pituitary apoplexy presenting with hypopituitarism and a generalised tonic clonic seizure

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Case history
A 28 year old male presented following his first generalised tonic clonic seizure in the context of a headache with mild photophobia, nausea and vomiting. He was haemodynamically stable, euvolaemic and had no focal neurological deficit, but was mildly disoriented. He had reported cannabis and MDMA use 8 days prior to his presentation. The patient was profoundly hyponatraemic with a serum sodium of 108.
Investigations
Serum osmolality was 223, urinary osmolality 676 and urinary sodium 55. Bloods revealed hypopituitarism, with hypocortisolaemia (8am cortisol 64), hypothyroidism (TSH 0.33, T4 7.1), and hypogonadtrophic hypogonadism (LH 0.4, FSH 2.2, testosterone 0.6). ACTH was less than 1, and IGF-1 and prolactin were normal. An MR pituitary revealed a large sella mass, with an expanded sella turcica and compressed optic chiasm. Visual acuity and fields were intact. Pituitary antibodies were negative and serum ACE normal.
Results and treatment
The patient was managed conservatively for hypopituitarism secondary to a non-haemorrhagic pituitary apoplexy (PA) complicating a pre-existing pituitary lesion of unknown aetiology. He received fluid therapy, including hypertonic 1.8% saline, intravenous hydrocortisone, levothyroxine replacement and empirical IV ceftriaxone. Due to a rapid rise in serum sodium, desmopressin was introduced to control the serum sodium. Once stabilised, the patient was discharged on oral hydrocortisone, thyroxine and desmopressin. Subsequent MR pituitary scans revealed a reduction in the size of the pituitary lesion, with resolution of the chiasmal compression. Minor high T1 signal was noted, suggestive of a resolving intralesional haemorrhage.
Conclusions and points for discussion
Numerous pathologies from neoplasia and infiltrative conditions to granulomatous disease may be implicated in the aetiology of hypopituitarism. The expanded sella turcica suggested an element of chronicity, consistent with a longstanding pituitary lesion. The patient was clinically well-androgenised, however, highlighting that an acute decompensating insult, such as an apoplexy, precipitated the presentation. MDMA use may have contributed to the clinical picture: it can produce hyponatraemia by stimulating the secretion of antidiuretic hormone, by acting on aquaporin 2 channels in the collecting duct to increase fluid retention and by producing neurogenic primary polydipsia.
PA, defined as infarction or haemorrhage typically into an underlying pituitary adenoma, has a wide spectrum of manifestations. It classically presents with a sudden onset severe headache with nausea, vomiting, impaired consciousness, ophthalmoplegia and haemodynamic instability. It can, however, present subacutely or remain clinically silent. Initial management is supportive with intravenous fluids, corticosteroids and replacement of deficient hormones. After initial stabilisation, patients with severely impaired visual acuity, persistent visual field deficits or impaired consciousness may be considered for neurosurgical decompression.

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CP16

Multi-drug-resistant hyperprolactinaemia – a rarity or a rising entity?

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Case history
A 22-year-old female first presented to our endocrine clinic in 2008 with a six-month history of galactorrhoea and irregular menses.
Investigations
She had hyperprolactinaemia (2401 mIU/l), a negative macroprolactin screen and her pituitary MRI scan demonstrated a 4-mm microadenoma. Her cannulated prolactin levels were >1500 mIU/l. She had reverse FSH:LH ratio hence the possibility of PCOS was considered. Her pelvic ultrasound did not show polycystic ovaries. TFTs, IGF-1, cortisol and remaining pituitary profile were within normal range.
Results and treatment
Cabergoline was commenced and gradually increased to 2 mg twice a week because of a poor response to therapy. Other than one serum prolactin of 486 mIU/l, her prolactin levels all remained >1000 mIU/l. She always reported good concordance with medication. Resistance to drug therapy was confirmed by admitting the patient to hospital where she received medication under supervision and despite this, her serum prolactin did not decline. She was thereafter switched to Bromocriptine in 2012 and titrated to a maximum dose of 15 mg with no biochemical or clinical response. Treatment with Quinagolide (up to 150 mcg od) was also tried. This was poorly tolerated (headaches and nausea) and again unsuccessful in lowering serum prolactin levels. Pergolide was discussed, but was not tried. She has had two further MRI scans in 2010 and 2015 that did not demonstrate a pituitary microadenoma. The patient is currently taking no dopamine agonist therapy, her latest prolactin is 1656 mIU/l, she menstruates four times a year and continues to experience galactorrhoea. She would like to conceive and has been referred to a fertility specialist.
Conclusion and points for discussion
A subset of patients with hyperprolactinaemia, due to a prolactin secreting pituitary tumour, are resistant to dopamine agonist therapy. Resistance is believed to be mediated by loss of pituitary D2 receptors and this may occur in micro- and macroadenomas. A reduction in tumour size (as in our case), but failure to normalise serum prolactin levels has been described. Treatment options in such cases could include transsphenoidal surgery or radiotherapy. Our case further highlights these treatment challenges particularly in a young patient trying to conceive.

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CP17

Water retention: not always the presenting symptom of heart failure

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Case history
An 80-year-old female presented with progressive leg and facial swelling, postural dizziness, generalised lethargy and reduced mobility. She suffered with chronic kidney disease (stage 3), rheumatoid arthritis and hypertension and had undergone partial thyroidectomy. Examination revealed marked bilateral leg oedema to the sacrum, facial oedema, an ejection systolic murmur, normal JVP and a clear chest.
Investigations and results
Investigations showed serum sodium 124 mmol/l (133–146), creatinine 70 umol/l (55–110), albumin 32 g/l (35–50), BNP 44.7 ng/l (<266), and normal liver function and glucose. Urinalysis showed trace proteinuria; urine protein:creatinine ratio 79 mg/mmol (<20). Echocardiography showed mild aortic stenosis, preserved ejection fraction and no evidence of diastolic dysfunction. Heart failure, nephrotic syndrome and hypoalbuminaemia were thus excluded. Computed tomography showed no masses. Mild, but not adequate, rise in serum sodium was achieved with intravenous diuresis. Endocrine tests showed fT4 8.0 pmol/l (9–23), TSH 2.28 milliunit/l (0.3–4.2), Oestradiol <70 pmol/l, LH <0.5 IU/l, FSH 0.5 IU/l, Prolactin 1,473 milliunit/l (100–500). Short synacthen test showed baseline cortisol 269 nmol/l, ACTH <5.0 ng/l and peak cortisol 450 nmol/l, inadequate in the presence of intercurrent urinary tract infection. MRI of the pituitary showed an expanded pituitary fossa containing homogenously enhancing preserved pituitary tissue within the floor and lateral margins of the sella, raising the possibility of a previous haemorrhagic nodular adenoma or intrasellar craniopharingioma.
Treatment
A diagnosis of panhypopituitarism with partial loss of the HPA axis was made. She was started on hormone replacement with Levothyroxine after acute hydrocortisone replacement on the ward. Serum sodium normalised to 134 mmol/l. Hydrocortisone is to be taken only in the context of any acute illness in the future.
Discussion
Oedema is a rare sign of panhypopituitarism. Several case reports have reported non-specific symptoms of central (and peripheral) cortisol deficiency in the elderly, but review of the literature found no cases where oedema was the sole presenting symptom. The diagnosis should be considered in cases of unexplained oedema, which ensues in the context of inability to excrete free water due to hypocortisolaemia.

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CP18

An elusive parathyroid gland

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We describe a case of a male who presented to a rheumatologist with hypercalceamia at the age of 22 in 1995. Investigations were incomplete and he was lost to follow up. He was referred to a general surgeon in 2002 as another blood test had showed hypercalcaemia of 2.8 mmol/l (2.2–2.6), parathyroid hormone 9.5 pmol/l (1.6–6.9). A spot urine calcium/creatinine excretion ratio was 0.014. It was felt he probably had primary hyperparathyroidism and he was managed conservatively but he then underwent surgical neck exploration at James Paget University Hospital in April 2004 following an episode of ureteric colic (presumed nephrolithiasis); histology did not show any parathyroid tissue. Calcium remained elevated post operatively.
He was referred to another centre in 2007 where an ultrasound, sestamibi scanning and CT scan did not show any evidence of parathyroid pathology. Subsequent venous sampling showed PTH 127 pmol/l in the midline inferior thyroidal vein. Further imaging was inconclusive but in 2009 he underwent a left hemithyroidectomy and thymectomy. Calcium remained elevated post operatively. He underwent further imaging including a contrast MRI scan in 2012 which suggested the possibility of a nodule in the upper mediastinum adjacent to the brachiocephalic artery. He was offered cinacalcet but declined. Bone densitometry was within normal limits. He was lost to follow up.
He then visited a clinic in Florida, USA where he had an operative procedure where some thyroid rest tissue was resected but no parathyroid gland identified. However, a sestamibi scan suggested uptake in the mediastinum. He was then referred to Hammersmith Hospital where a 4D CT scan showed a possible low mediastinal intrathymic ectopic parathyroid gland. Venous sampling showed a 10-fold concentration of PTH in the samples from the thymic vein. He has recently undergone an excision of the ectopic parathyroid gland via a thoracoscopic approach and we would hope to present the outcome of this.
In summary, we presented a patient with a history of primary hyperparathyroidism of over 20 years who had three unsuccessful operations. This was complicated by kidney stones, neurocognitive symptoms, lethargy and aches and pains.

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CP19

Hypercalcaemia: an uncommon cause

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Case history
An eight-year-old white male presented to his General Practitioner with a six-month history of cough and malaise. Blood tests revealed isolated hypercalcaemia (4 mmol/l). He was referred to the Children's Emergency Department for urgent management. On examination, uveitis and lymphadenopathy were noted. Repeat blood tests confirmed hypercalcaemia. Despite hyperhydration and Furosemide, the serum calcium remained persistently elevated.
Investigations

A chest radiograph revealed widespread bilateral fine nodularity. A high-resolution, non-contrast chest computed tomography scan showed multiple tiny nodular opacities with bilateral patchy areas of ground-glass opacification within the lungs. A lymph node biopsy exhibited granuloma formation and serum angiotensin converting enzyme level was elevated (>120 u/l).
Results and treatment
A diagnosis of Sarcoidosis was made and the child was started on prednisolone for Sarcoidosis-related hypercalcaemia.
Conclusions and discussion
Sarcoidosis is a multi-system disorder characterised by non-caseating granuloma. It has a spectrum of clinical manifestations and is known the 'great masquerader'. It is often encountered in the second to fourth decades of life and is rare in childhood. Hypercalcaemia and/or hypercalciuria are common endocrine manifestations of Sarcoidosis.
Hypercalcaemia is a recognised endocrine manifestation of Sarcoidosis. Recognition of this rare cause of hypercalcaemia is a challenge for clinicians. Sarcoidosis is rarely seen in childhood, but should be considered in patients presenting with refractory hypercalcaemia. High-dose corticosteroid is the treatment of choice for Sarcoidosis-induced hypercalcaemia.

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CP20

A case of late presentation of radiation-induced hypopituitarism

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Fifty-five years old patient with background of type 2 DM, referred by the GP with low serum Cortisol levels (<50), when was investigated for his ongoing symptoms of tiredness with low energy level. He also noticed reduced sexual drive and erectile dysfunction worsening over years along with reduced shaving frequency, to twice a week and sparse hair growth on his body. He denied any trouble with his peripheral vision or any significant headaches, but did feel dizzy on occasions He maintained a good glycemic control since his diagnosis was made around 10 years, with metformin alone. He denied using any anabolic steroids, and had a short course of oral steroids for his acute severe asthma around 10 years ago.
Past medical history
Other than Type 2 DM he had surgical removal of his left maxillary bone tumour around 20–30 years earlier with enucleation of his left eye followed by radiotherapy sessions. Examination was significant for sparse body hair distribution,low testicular volume and enucleated left eye but no visual field defect on confrontational method of his right eye.
Results and treatment
Pituitary Functions IGF1: 4.6 (NR 7.0–25.6) FSH: 0.8 LH: <0.2, TSH: 0.01: Prolactin: 301(NR 86–324) Testosterone: 5.2 (NR 9.9–27.8) Short synecthen test: 9:00 AM Cortisol < 50.0 Post ACTH: 163 (inadequate response) MRI of pituitary: Pituitary atrophy. In view of his biochemical findings of Pan hypopituitarism he was commenced on oral hydrocortisone, levothyroxine and topical testosterone gel in replacement doses.
Conclusion and points for discussion
This case demonstrates the importance of considering late onset hypopituitarism in the patients who received radiotherapy for the tumours of the adjacent organs. In the central nervous system the Hypothalamus pituitary axis acts an importance unit which is sensitive to the effects of radiations and its exposure put these patients at great risk of developing hypopituitarism. The timely identification and management of this condition will help reducing the morbidity and mortality associated with this condition.

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CP21

A challenging case of hypercalcaemia due to primary hyperparathyroidism in an elderly patient successfully treated with non-elective surgical inpatient management

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An 86-year old man was admitted to hospital with gradual decline in mobility and leg weakness. Co-morbidities included atrial fibrillation, hypertension and osteoarthritis. The adjusted serum calcium on admission was 3.42 mmol/l; bone profile showed phosphate 0.56 mmol/l, alkaline phosphatase 140 U/l, albumin 34 g/l, parathyroid hormone 21.4 pmol/l. He was vitamin-D replete (68 nmol/l) with normal renal function. The hypercalcaemia was not thought to be due to any medications. Urgent treatment was commenced with 45 mg disodium pamidronate intravenously and 4 hourly intravenous fluid replacement. Further investigations included a negative myeloma screen, normal serum ACE, unremarkable plain film chest X-ray, 24-hour urine calcium output 2.5 mmol/24 h (2.5–7.5 mmol/24 h). Parathyroid ultrasound scan identified a nodule posterior to the right thyroid lobe measuring 1.4×1×2.7 cm with a concordant focus of delayed washout on nuclear medicine parathyroid uptake scan. Pamidronate therapy led to a reduction of calcium to 2.98 mmol/l over 4 days, but the effect was short lasting, necessitating a second dose of intravenous pamidronate. However, the calcium again increased to 3.14 mmol/l within 4 days and Cinacalcet 30 mg daily was commenced. Due to persistent hypercalcaemia requiring continuous intravenous fluid replacement and not responding to medical therapy, an urgent surgical referral was made to the Surgical Endocrine unit at our linked tertiary centre. He was transferred to the tertiary unit and had a parathyroidectomy which revealed a large adenoma of the right superior gland. The left side was not explored due to convincing findings on the right and a sufficient drop in intraoperative PTH (20–4 mmol/l). He was transferred back to our hospital for further rehabilitation and recovered well, being discharged shortly thereafter. Adjusted calcium levels remained <2.6 mmol/l, PTH 3.8 mmol/l and no further intravenous fluids, bisphosphonate or calcimimetics were required. This uncommonly encountered case highlights non-malignant hypercalcaemia resistant to medical therapy and successful non-elective inpatient parathyroidectomy as part of the armamentarium for treatment of patients, even the very elderly, who are either symptomatic or requiring continuous intravenous fluid replacement. This is in contrast to the majority of patients with hypercalcaemia, managed in an outpatient setting, where there are clear guidelines indicating surgical management. Early parathyroid adenoma localisation studies and discussion with the Endocrine Surgical team should be considered if concerns are raised over resistant hypercalcaemia secondary to primary hyperparathyroidism, to facilitate definitive treatment and optimise outcomes.

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CP22

Diagnostic dilemma of a lady with hirsutism and male pattern baldness

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Forty-three years old lady with background of hypertension was seen in the GP surgery with hirsutism noticed over years. She was referred to the gynaecologist for further work up who in turn discharged her back suggesting possibility of PCOS in view of her biochemical and clinical findings but with no radiological evidence of ovarian cysts.
Her PMHx was significant for mild asthma though rarely requiring inhaled steroids.
Previously she was investigated for the cause of hypertension with all of her biochemical results reported as negative. She remained on triple agents for blood pressure control.
Her symptoms and signs gradually progressed, prompting her to seek medical attention form the local GP surgery from time to time (2011–2016).
On her ultimate referral, to the endocrinology clinic, she was investigated for the cause of hirsutism and male pattern baldness along with her coexisting hypertension.
Investigations and treatment
Overnight dexamethasone suppression test (DST): 9 AM Cortisol: 533 Post Dose: 581, Low dose DST: Plasma ACTH: < 0.1 Baseline Cortisol: 583 Post 48 h: 590, plasma aldosterone: renin: normal, urine catecholamine: normal LH FSH TFTs normal, MRI ABDO: solid mass measuring about 2.6 cm in diameter arising from the left adrenal. A diagnosis of adrenal Cushings was made and a diagnosis of adrenal Cushings was made and she was commenced on Metyrapone until Laparoscopic left adrenalectomy. Post procedure she was started on cortisol replacement and no longer required three antihypertensive drugs.
Conclusion and points for discussion
One of the rear but important cause of Cushing syndrome (endogenous) is adrenal tumour which is ACTH independent in nature with intact HPA where the Cortisol excess is solely of adrenal origin. This case highlights the importance of appropriate investigations for the patients with hirsutism as well as hypertension > The diagnostic dilemma lingering on for years in this young with an obvious endocrine condition which remained uncovered for years putting her at great risk of morbidity and mortality associated with this condition.

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CP23

Lithium-induced diabetes insipidus

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Case history
A 76-year-old female presented following an unwitnessed. On arrival she was confused, dehydrated, complaining of nausea, constipation and abdominal pain. Bloods tests revealed serum calcium of 3.6 mmol/l. She had a history of bipolar disorder and had been taken off lithium due to suspected lithium-induced hyperparathyroidism. Whilst an inpatient, she had a sudden drop in consciousness (Glasgow Coma Scale 3). She was hypotensive, dehydrated and had an average urine output of 130 ml/h. She was intubated, ventilated and admitted to the Intensive Care Unit. Serum sodium and calcium were 157 and 3.54 mmol/l, respectively. She remained polyuric and was investigated for diabetes insipidus.
Investigations
Computed tomography (CT) on admission showed age-related atrophy with nil acute changes. Magnetic resonance imaging (MRI) of the head showed moderate chronic small vessel disease and evidence of a small old infarct in the left cerebellar hemisphere, but nil acute findings. A repeat CT head following the acute deterioration did not reveal any new features. A lumbar puncture was performed.
Results and treatment
Cerebrospinal fluid analysis was unremarkable. A collateral history revealed a history of polydipsia and polyuria prior to admission. A diagnosis of lithium-induced nephrogenic diabetes insipidus was made. She was managed with high dose desmopressin, as well as a trial of indometacin and amiloride.
Discussion and conclusion
We present a case of lithium-induced nephrogenic diabetes insipidus. Desmopressin, diuretics and non-steroidal anti-inflammatory drugs have been used to manage nephrogenic diabetes inspidus related to lithium use. Nephrogenic diabetes insipidus has been seen to persist despite discontinuation of lithium. Patients taking lithium should be monitored for signs of electrolyte disturbance. Diabetes insipidus should be considered in patients presenting with polyuria and polydipsia of a background of lithium use.

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CP24

Multiple endocrine neoplasia type 1 presented with diarrhoea, vomiting and peptic ulcers

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Multiple parathyroid tumors causing hyperparathyroidism are the most common manifestation of multiple endocrine neoplasia type 1 (MEN1), displaying almost 100 percent penetrance by age 40–50 years. In most cases, it is the initial manifestation of MEN1.
We report a case of young lady presented to hospital with diarrhoea and vomiting and diagnosed to have multiple peptic ulcers with severe oesophagitis. Her other biochemistry was consistent with primary hyperparathyroidism. The possibility of MEN1 was considered. On taking detailed family history, she had a strong family history of primary hyperparathyroidism with complications. She underwent genetic testing which confirmed MEN1 mutation. Her family members were screened for familial MEN-1 and two of her siblings revealed mutation in MEN1 gene. Patient later had three parathyroid glands removed along with unremarkable thymic tissue and small lymph nodes. Histology confirmed parathyroid hyperplasia with no evidence of malignancy. She was later referred to tertiary care hospital where she was further investigated for possibility of neuroendocrine tumor. CT pancreas revealed multiple foci of hypervascularity in tail of pancreas consistent with neuroendocrine tumor. She had surgery done for neuroendocrine tumor.
Histology confirmed well-differentiated neuroendocrine tumor.
We concluded that it is important to take a detailed family history in patients with hyperparathyroidism as it may indicate the possibility of a familial disorder like MEN1. It is also important to consider the possibility of MEN1 in a patient presenting with primary hyperparathyroidism if presenting at a younger age, multiple gland disease and recurrence after successful subtotal parathyroidectomy.

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CP25

Two unusual cases of abnormal male sex hormone profile

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Case history
Two cases:
(1) NM 55-year-old man with no erectile dysfunction, normal libido, but low energy levels for 6 months. **PH** Hemithyroidectomy for benign nodule, CML with Philadelphia chromosome positive, vitamin D deficiency. Peripheral vascular disease secondary to kinase inhibitors.
DH Sildenafil, aspirin, ibuprofen, MagnaPhate, Vitamin D3 1000 units, Levothyroxine 50 mcg, Imatinib 400 mg started 10/2015
SH Non-smoker. Drinks 20 units/week
(2) KB 64-year-old man with normal energy levels, normal libido, but had erectile dysfunction since thrombotic stroke 2005 resulting in left sided hemiparesis
DH Folic acid, aspirin, lansoprazole, simvastatin
SH No alcohol, smokes 15/day
Investigations
Table illustrates blood results that were confirmed with a further test.

Patient no.	Testo-sterone	LH	FSH	9 AM cortisol	TSH	Prolactin	IGF-1	HbA1C
1	6.6(↓)	2.9	5.9	527	6.4	119	14.6	36
2	42.6(↑)	28.9(↑)	13(↑)	444	<0.02[a](↓)	322	19.7	–[b]

[a]Free T4 was16.3 suggesting subclinical hyperthyroidism.
[b]Plasma glucose 5.8.

Results and treatment
Patient 1 demonstrates an inappropriately normal LH/FSH for a low testosterone level, suggesting a possible pituitary/hypothalamic problem. MRI pituitary was normal but showed an incidental lacunar infarct cerebellum. Patient was initiated on testosterone replacement.
Patient 2 has an inappropriately high FSH/LH for a high testosterone level. This was consistent with different assays.
Conclusion and points of discussion
The first patient has biochemical results that illustrate hypogonadotrophic hypogonadism with normal pituitary gland. Therefore, the possibility of imatinib causing this abnormality has to be explored and discussed. There are many endocrine side effects of kinase inhibitors.
The results of the second patient are suggestive of un-inhibited elevated LH and FSH levels given the high levels of testosterone.
We would like to open these two cases for discussion.

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CP26

Rapid preoperative preparation of patients with thyrotoxicosis

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Thyroidectomy is a definitive form of treatment for patients with hyperthyroid Graves' disease particularly those who are noncompliant with or have serious side effects to the antithyroid drugs, have very large goitres, refuse 131I therapy or, have moderate to severe ophthalmopathy. The risk of perioperative thyroid storm is usually higher following an acute event such as surgery, trauma, or infection. Thus, patients with thyrotoxicosis presenting for surgery should ideally be made biochemically and clinically euthyroid before surgery in order to reduce this risk. Conventional preoperative preparation for surgery includes antithyroid drugs and iodine administration. This often takes months to render patients euthyroid. Far more rapid control of thyrotoxicosis can be achieved by the oral administration of lithium given in combination with corticosteroids, antithyroid drugs and b-blockers. We describe two such cases with successful and uneventful perioperative outcome.

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Clinical Update

Workshop A: Disorders of the hypothalamus and pituitary

WA1

Macroprolactinoma: Challenges in management

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Case
A 73 year old man was seen in eye clinic with 5 month history of visual problems. He was diagnosed with bilateral glaucoma and early Cataracts. His vision was not improving despite eye drops and new glasses thus cranial imaging was requested. He also complained of feeling off balance and tiredness. He denied headache or galactorrhoea. Examination: Bitemporal hemianopia (L > R). Investigations: CT head scan – Pituitary Macroadenoma 3.6×3.1×3.8 cm, with displacement of Infundibulum in optic chiasm. Prolactin of 114 655 mU/l (No evidence of macroprolactin), LH < 1.0 U/l, FSH 1.1 U/l, Testosterone 0.9 nmol/l. TSH 2.39 (0.35–4.5 mU/l), FT_4 11 (10–25 pmol/l), FT_3 4.6 pmol/l (3.0–7.0 pmol/l), cortisol 469 nmol/l, ACTH 13 ng/l. He was subsequently referred to the endocrine clinic. A diagnosis of macroprolactinoma was made in April 2016 and he was commenced on cabergoline 0.5 mg twice weekly. Testosterone was not replaced as it was thought that it would improve with lowering of prolactin levels. Three months post treatment (July'16), he reported no significant change in energy levels or vision. Prolactin had improved to 829 mU/l. MRI Pituitary in Aug'16 showed no change in size of adenoma. Scans were discussed at the pituitary MDT in Aug'16 where it was felt that there might be two separate lesions – pituitary adenoma and possible meningioma. He was urgently referred to neurosurgeons and ophthalmologists but a week later (beginning of September 2016) he presented with progressive visual field and acuity loss with worsening headache. He was diagnosed to have pituitary apoplexy and underwent transsphenoidal debulking of pituitary tumour. Post-operatively he had a stormy course with CSF leak -requiring repair, meningitis and hydrocephalus requiring a VP shunt. He continues on cabergoline 0.5 mg twice weekly and not on any hormone replacement therapy. His latest prolactin is 244 mU/l and his vision has improved slightly.
Discussion points:

i) Pituitary apoplexy has rarely been reported in context of cabergoline therapy.
ii) Approach to patients with discordant response to DA agonists' therapy.
iii) Pre-existing visual conditions can hamper the assessment of pituitary induced visual loss.

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WA2

A case of dopamine agonist resistant macroprolactinoma

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31 year old lady was referred to the endocrine clinic by her GP in August 2008. She had 1 year history of galactorrhoea, 4 months history of irregular periods and intermittent short lived severe headache in the right temporal area without visual disturbance. Her investigation showed a prolactin level of 900 mmol/l, subsequent MRI scan confirmed the presence of right-sided pituitary adenoma of around 1 cm in size with evidence of recent haemorrhage in to it and normal other pituitary function. She was started on Cabergoline 0.25 mg twice a week in September 2008. Developed unpleasant psychological symptoms although multiple attempt were made to adjust the dose there was a ceiling of Cabergoline dose above which she experienced subtle symptom which meant full medical control was not possible. The tumour itself was slightly unusual in that it seemed to extend down through the floor of the pituitary into the sphenoid sinus through a bony defect with repeated evidence of haemorrhage in to it on follow up MRIs. Ongoing headache with intolerance to high dose dopamine agonist dictated surgical intervention. She underwent trans sphenoidal surgery in March 2014. Off dopamine agonist since and currently she is 35 weeks pregnant after spontaneous conception with minimal symptom during pregnancy.

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WA3

Intolerance to dopamine agonists and the challenges of treating pituitary lactotroph macroadenomas in pregnancy

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A 36 year old lady presented to the endocrine clinic with a 4 month history of secondary infertility, amenorrhea, galactorrhoea and headaches. She had stopped breast-feeding her 5th child 18 months previously and was not taking any antidopaminergic drugs. She had a normal neuro-ophthalmic examination, including full visual fields to confrontation with a red pin. Her prolactin was elevated at 5690 mU/l and MRI pituitary confirmed a 17 mm partially cystic macroadenoma which was approaching, but not contacting the optic chiasm.
She was commenced on cabergoline 0.25 mg twice weekly, but was unable to tolerate this due to nausea and vomiting. She was therefore changed to bromocriptine 1.25 mg od, which resulted in restoration of menses. She returned to clinic 7 weeks pregnant and was advised to remain on bromocriptine throughout her pregnancy. She discontinued her bromocriptine at 13 weeks gestation due to nausea, vomiting and dizziness and failed to attend subsequent appointments. She had a normal vaginal delivery following induction at 38 weeks gestation.
She represented to the endocrinology clinic 5 months post-partum with persistent galactorrhoea and amenorrhoea having discontinued breast-feeding 2 months earlier. Repeat MR imaging showed the pituitary adenoma to be 13 mm. She was commenced on quinagolide 75 µg od and subsequently conceived and stopped her medication. She was admitted to hospital at 33 weeks gestation with worsening headache and a non-contrast pituitary MRI showed growth of the adenoma to 18 mm, touching the left optic nerve. Visual fields remained intact. She was commenced on bromocriptine 1.25 mg od and had an elective caesarean section and tubal ligation at full term with hydrocortisone cover.
Quinagolide was re-commenced post-partum, but was discontinued due to nausea and vomiting. She was therefore referred for trans-sphenoidal surgery. She underwent an uncomplicated resection of the lactotroph macroadenoma, resulting in normalisation of prolactin post-op and preservation of anterior pituitary function.
This case highlights the potential difficulties in treating pituitary lactotroph adenomas in cases of dopamine agonist intolerance.

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WA4

Management of microprolactinoma in pre-pregnancy and pregnancy

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Case history
A 39 year old lady was referred to the endocrinology clinic with difficulty conceiving, after screening bloods revealed an elevated prolactin (1816 miu/l)). There was no past medical history or family history of note and she took no regular medications. She denied headache, visual disturbance or galactorrhoea. Systemic examination was normal.
Investigations
Elevated prolactin was confirmed on a cannulated sample (1435 miu/l). Further analysis excluded macroprolactin. Her pituitary profile and basic bloods were otherwise unremarkable. MRI revealed a 6mm pituitary tumour, consistent with a microprolactinoma. Humphrey visual field testing demonstrated normal visual fields.
Treatment
Our patient was started on bromocriptine 1.25 mg bd, uptitrating to 2.5 mg bd after 2 weeks. Prolactin levels have improved, but she is yet to conceive.
Discussion
Microprolactinomas are common in the endocrinology clinic, and frequently seen in young female patients desirous of pregnancy. Dopamine agonists restore ovulation in up to 90% of such cases, but there remains considerable debate about drug choice and dose. Bromocriptine is usually favoured in these patients, given the extensive experience with this drug in early pregnancy. However, cabergoline appears to offer equivalent safety, along with a superior efficacy, dosing regimen and side effect profile. Serious consideration should be given to cabergoline as initial treatment in this population, particularly in older patients attempting pregnancy who would benefit from conceiving sooner rather than later.

Once pregnancy has been achieved, the approach is less controversial. Risk of tumour enlargement is only 2% with microprolactinomas, so there is widespread consensus that dopamine agonists should be withdrawn. Subsequent follow up should focus on clinical evaluation and visual field testing if indicated, as opposed to serial prolactin levels which do not correlate with tumour enlargement in pregnancy.

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WA5

Pituicytoma – Lessons from anabolic steroid abuse?

Amber Khan & Malcolm Littley
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A 34 year old male, previously healthy nightclub doorman presented with 2 years of reduced libido, fatigue, hot flushes and reduced beard growth. He had fathered three healthy children. He admitted to previous use of veterinary anabolic steroids up until 2 years ago. He had normal secondary sexual characteristics, no features of pituitary hypersecretion, normal visual fields but reduced testicular volumes (8 ml).

Biochemical investigation showed growth hormone, gonadotrophin and corticotrophin deficiency. Serum prolactin and thyrotrophin reserve were normal. Urinary screening for anabolic steroids was negative. Treatment with Hydrocortisone and subsequently testosterone was commenced. Magnetic resonance imaging showed a 15 mm lesion (hyperintense on T2 weighted studies) arising from the hypothalamic region. Biopsy demonstrated a pituicytoma with positive staining for S100 and Epithelial Membrane Antigen. Unfortunately, the patient died as a result of haemorrhage from the tumour after the biopsy.

This case demonstrates the importance of undertaking full assessment of pituitary function in hypogonadal patients, even when there is an apparently obvious cause. Subsequent imaging and biopsy revealed an unusual type of hypothalamic-pituitary tumour.

Some anabolic steroids can be extremely long acting, particularly those normally used in veterinary practice. This may confound accurate diagnosis. Anabolic steroids have been reported to be mitogenic in both human and animal models. Affected tissues include bone, bone marrow, pancreas and liver. There is some evidence that this may be mediated through the androgen receptor and induction of growth factors. We were unable to find previous reports of pituitary or other intracranial tumours in association with anabolic steroid use.

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WA6

Amenorrhoea and hyperprolactinaemia

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Case history

A 19 year old lady presented with amenorrhoea, fatigue and headaches. She had normal pubertal growth spurt with an appropriate height for mid-parental height. Her menarche was at age 17, another bleed at 18 but amenorrhoeic since. There was right sided galactorrhoea only on expression. In her teens she had an eating disorder, periods of self-harm and low mood. No treatment except for St. John's Wort occasionally. No visual disturbance was present.

Investigations

Biochemical investigations and pituitary MRI.

Results and treatment

Hyperprolactinaemia (12 225 munits/l), IGF1 24 pmol/l, LH 2.7 units/l, FSH 4.3 units/l, oestradiol 67 pmol/l, testosterone 2.5 nmol/l, cortisol 653 nmol/l, free T_4 13.2 pmol/l, corrected calcium 2.23 mmol/l.

Pituitary MRI showed 17×15×17 mm left sided partially cystic macroadenoma within the fossa.

Patient was started on treatment with cabergoline, titrated up to 0.25 mg three times a week. Her prolactin level on this treatment was 3017 munits/l. However cabergoline had a mildly negative effect on her mood and therefore treatment was stopped. A week after her prolactin level was 6156 munits/l.

We postulated that the mood lowering effect of cabergoline might be dose related and therefore restarted treatment with cabergoline 0.25 mg every 2 weeks, with an intention very gradually to increase the dose and monitor any adverse symptoms. There was a ceiling of cabergoline dose above which she developed adverse symptoms, but the maximum tolerated dose was continued prior to pituitary surgery in order to try and optimise the surgical target. Transphenoidal surgery resulted in mild compensating transient diabetes insipidus, managed conservatively and resolved spontaneously. Hyponatremia followed diabetes insipidus, which resolved with fluid restriction. ACTH-cortisol axis following surgery was intact and prolactin level was undetectable. There was no cerebrospinal fluid leak. Patient had continuous nose bleed from right nostril, for which she received ENT review, nasal packing for 24 h, tranexamic acid, and amoxicillin and clavulanate, which led to resolution.

A year from surgery patient is feeling slightly tired but is otherwise well, only occasional occipital headaches relieved with simple analgesics. Amenorrhoea persists. Prolactin 11 munits/l, LH 12 units/l, FSH 6.3 units/l, TSH 2.53 munit/l, free T_4 17.7 pmol/l, random cortisol 326 nmol/l, and sodium 141 mmol/l.

Conclusions and points for discussion

We describe a case of macroprolactinoma discovered following investigation of amenorrhoea, initially treated with cabergoline, stopped due to adverse effects, and then with transphenoidal surgery after medical preparation with the maximum tolerated dose of cabergoline to optimise the surgical target. Ongoing management questions include whether persisting amenorrhoea requires further investigation and whether macroadenoma discovered at a young age requires any genetic testing. The effects of cabergoline on mood in patients with a history of low mood and its dose-dependent nature merits further discussion.

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WA7

CSF leak with dopamine agonist

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A 48-year-old gentleman was admitted to AMU overnight in January'12 from eye casualty with weeks of worsening visual disturbances followed by rapid deterioration – complete loss of vision in the right eye and a temporal hemianopia on the left side but no headache.

Urgent hormone profile revealed elevated prolactin 53 988 mU/l, low testosterone of 3.3, SHBG 22, LH 2, FSH 3.1, IGF1 292, satisfactory SST (0 min cortisol 213 with ACTH 10 and 30 min cortisol 630), FT_4 11, TSH 1.2. MRI pituitary showed 'A macroadenoma (40×31×26 mm) compressing the optic chiasm. No evidence of apoplexy.' He was started on Cabergoline 500 μg once a week and discharged. He came back to endocrine clinic 3 days later with improved vision and doubled Cabergoline dose. Seen in Pituitary MDT in 2 weeks as he had developed quite significant CSF leak from the left nostril a week ago. He underwent joint Neurosurgical and ENT procedure to repair the leak.

Reviewed in April'12 – feels tired and lack of libido (prolactin 1500, testosterone 4.4). Started on testosterone gel. MRI pituitary showed reduction in macroadenoma (28×28×28 mm) with no significant optic chiasm compression. Last seen in pituitary MDT in February'16. His prolactin is 810 and normal vision. The MRI shows mainly post-surgical packing of the sphenoid fossa with no suggestion of a problematic tumour mass with a prolactinoma.

This case highlight that sudden shrinkage of the macroprolactinoma with dopamine agonist can leave a hole in the dura leading to CSF leak. The patient needs to be warned about this and manage accordingly.

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WA8

Aggressive prolactinoma

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A 50 year old male presented with left visual field loss, afferent pupillary defect and reduced libido in 1999. Initial pituitary profile showed prolactin 130 000 mu/l (55.4–276), and testosterone was 5 nmol/l (10–27.6). MRI pituitary showed giant

pituitary macroadenoma compressing chiasm. He was started on **Cabergoline** with subsequent improvement in visual fields, prolactin levels and size of adenoma on subsequent MRI. His disease remained stable on low dose cabergoline until 2003 when his follow-up MR showed some increase in size of adenoma. His cabergoline dose was increased and his follow-up MR showed stable appearances until 2007 when he presented with horizontal diplopia, and right sixth nerve palsy. MR showed progression in the size of adenoma with cavernous sinus invasion and erosion of floor of pituitary fossa. He was given **Radiotherapy** along with cabergoline treatment. His tumour size remained stable with some improvement in his prolactin levels. In 2010, he presented again with right ptosis, cranial nerve III and VI palsy, rising prolactin and growing pituitary macroadenoma. Treatment with **Quinagolide** initiated with no clinical response and later **Trans-sphenoidal debulking** of tumour was performed. Histology showed high Ki67 (22%) tumour staining for prolactin. He developed partial hypopituitarism and was started on hormone replacements. His tumour increased in size on further MR scan in 2011 with corresponding rise in prolactin. He was treated with six cycles of **Temozolamide** which resulted in reduction of tumour size and prolactin levels. In 2013, his follow-up MR showed progression in size with rise in prolactin, so he was re-challenged with temozolamide and radiotherapy as surgical intervention was not possible due to tumour invading the cavernous sinus and carotids. In 2015, **Transethmoid/Transsphenoid** decompression performed in view of further tumour growth and severe headaches on follow-up. MRI showed extensive tumour invasion to right cavernous sinus, Cranial nerve III, IV and VI, right orbital apex, greater wing of sphenoid, Meckel's cave. Six months later he presented with dysphagia and blurring of vision and MR showed progressive growth of tumour compressing the brain stem. He underwent further surgical **Debulking with palliative intent** and post-operatively developed hydrocephalus requiring **VP shunt**.
Discussion points

i) Is this a malignant pituitary tumour?
ii) What are other treatment options given the resistance to DA and multi-model treatment?
iii) Shall we continue with Cabergoline?

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Workshop C: Disorders of the thyroid gland

WC1

Thyroid resistence

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Abstract learning objective
Resistance to thyroid hormone is a rare condition caused by tissue refractiveness to the effects of circulating thyroid and may be misdiagnosed as hyperthyroidism. This syndrome is characterized by elevated circulating thyroid hormones, and unsuppressed TSH levels. Although most patients are euthyroid, rarely they may present with clinical hyperthyroidism, if the Pituitary gland is more insensitive than other tissues to thyroid hormones.
In this study we present a case of Graves disease on a background of thyroid resistance.
Case
54 year old lady who suffered from loss of weight and diarrhoea for two years, with a family history of two of her sisters having been diagnosed with thyroid resistance, had clinical evidence of thyrotoxicosis. TFT's showed TSH <0.03, $FT_3 > 30.8$, TRAB 0.8, Sonography revealed diffuse alteration of echogenicity and increased vascularity suggestive of Graves disease and a small nodule 8mm nodule in right lobe. Patient was treated with Propranolol 40 mg BD and carbimazole 30 mg and subsequently reduced to 20 mg a day. After 6 months of treatment TSH (2.34) normalised with FT_3 10.1 and FT_4 29. At this stage a NM scan was performed to see if the thyroid nodule was toxic, it showed a small diffuse goitre, diffuse symmetrical uptake throughout, alpha subunit was normal (1.15). In this case report we emphasized the importance of timely diagnosis, and therefore various inappropriate treatments were avoided.

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WC2

A multidisciplinary approach in the management of a challenging Grave's ophthalmopathy case

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A 44 year-old female presented to endocrine outpatient with 1-year history of 4 stone weight loss, heat intolerance, insomnia along with diplopia and sore and watery eyes. Her medical history includes IgG4 disease and rheumatoid arthritis, treated with prednisolone that had been stopped 6 months previously.
On examination, she was clinically hyperthyroid with a resting tremor, tachycardia and sweaty palms. Eye examination showed bilateral proptosis and chemosis worse on the left eye, diplopia and marked color vision loss in the left eye. She had a small, smooth palpable goitre.
Thyroid function tests revealed FT_4: 58.9 pmol/l TSH: <0.01 mU/l and FT_3: 13.2 pmol/l and positive TSH receptor antibodies. Treatment was commenced with carbimazole, propranolol and prednisolone for her ophthalmopathy. Urgent MRI of the orbits showed symmetrical enlargement of the extra ocular muscles and crowding of orbital apices, requiring urgent ophthalmology review and referral for external beam radiotherapy (EBRT).
She underwent urgent orbital decompression of the left eye as her vision deteriorated to light perception only. A repeat CT of the orbits following her operation still showed marked bilateral proptosis and crowding of the orbital apices with no improvement in her vision, so further decompression was performed to both eyes but this was delayed as she had uncontrolled thyrotoxicosis, which required admission for conversion of carbimazole to propylthiouracil and intravenous steroids. She had significant improvement of her visual acuity postoperatively.
Thyroid MDT discussion subsequently recommended definitive treatment for her Grave's disease with a total thyroidectomy, as she was still requiring very high doses of carbimazole and radioiodine is contradicated due to the severe ophthalmopathy. She also had EBRT to stabilize her ophthalmopathy.
This case of severe Graves' ophthalmopathy refractory to steroids, complicated by fluctuating thyroid control, highlights the importance of a multidisciplinary approach to avoid severe life-changing complications such as blindness, which can happen rapidly and potentially non reversible if delayed.

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WC3

Abstract Withdrawn.

WC4

A difficult to manage eye disease

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A 61-year-old ex-smoker with a background of chronic obstructive pulmonary disease, bilateral cataracts and advanced retinitis pigmentosa presented in April 2014 with a 5 months history of feeling generally unwell and weight loss. He was found thyrotoxic with TSH suppressed to less than 0.01 mU/l, free T_4 of 38 pmol/l and free T_3 of 26 pmol/l. On examination there was tunnel vision bilaterally and diplopia in all directions with no evidence of thyroid eye disease. He was initiated on carbimazole 20 mg once a day. The impression was of thyrotoxicosis and a strongly positive thyroid receptor antibody confirmed Graves' disease. He responded well to the treatment and gained weight. His thyroid function continued to improve and the dose of carbimazole has been gradually reduced.

In September he developed a foreign body sensation particularly in the left eye and pain on eye movement. There was conjunctival injection and periorbital oedema, mild proptosis, bilateral chemosis, lid oedema and erythema. He was prescribed topical lubricants and referred to ophthalmology who diagnosed moderate orbitopathy. Given difficulties in fully assessing his vision owing to his retinitis pigmentosa related optic neuropathy he underwent a MRI scan of his orbits which confirmed thyroid ophthalmopathy with active inflammation of the extraocular muscles and bilateral straightening of the optic nerves, suggestive of nerve stretching. In March 2015 he developed a significant flare up of his thyroid eye disease and was offered an intravenous methylprednisolone (six doses administered weekly). He did not respond well to this and subsequently was considered for orbital radiotherapy. He received a course of low dose radiotherapy – 20 Gy/12F. On follow up there was symptomatic benefit and was able to use less strong prisms. His vision was 6/24 in both eyes. 18 months since initiating carbimazole treatment he was receiving 5 mg daily and it was advised to further reduce it to 5 mg alternate days to prevent relapse of Graves' disease. Once confirmed the thyroid eye disease was inactive his carbimazole was stopped and he was discharged from the endocrinology clinic. In summary, we presented a difficult to manage case of thyroid eye disease complicated by concurrent illness requiring multidisciplinary approach to ensure patient's best outcome.

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WC5

Amiodarone induced thyrotoxicosis (AIT) type 1

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Case history
81-year-old female patient admitted for fracture neck of femur was referred to endocrinology for high T_4. Clinically she was euthyroid with mild thyroid eye disease.
PMH
Atrial fibrillation Post CABG 30.09.15, Hypothyroidism since 1983 (started on Eltroxin by GP for weight gain and tiredness though patient never had biochemical evidence of hypothyroidism), OA of spine 2007, T2DM, Asthma.
Drug history
Amiodarone 200 mg od since 30.09.15, Aspirin, Metformin, Levothyroxine 75 µg od, Adizem XL, Inhalers.
Investigations
FT_4 31.9 pmol/l (6.5–17.0) TSH 0.03 (0.35–4.94) FT_3 3.7 (4.2–6.7) TPO antibodies negative. USG thyroid showed MNG with increased vascular flow. ECG: Sinus rhythm.
Results and treatment:

	Pre amiodarone	Post amio	CBZ start	30 mg	20 mg	10 mg
	31.08.15	18.11.15	23.05.16	06.07.16	10.10.16	19.12.16
T4	18.8	42.6	31.9	24.2	9.0	8.9
TSH	0.02	0.07	0.03	0.06	0.79	5.27

In view of underlying MNG and thyroid eye disease, diagnosed as amiodarone induced thyrotoxicosis (AIT) type 1 and initially commenced on carbimazole 30 mg daily and her levoT_4 and amiodarone both were stopped. Her carbimazole dose has been tapered slowly to 10 mg as her latest T_4 8.9 and TSH 5.27. She continues to be asymptomatic.
Conclusions and points for discussion
Though in our patient there were indicators towards the type of AIT, most of the time the management of AIT is challenging due to the difficulty in diagnosing the type of AIT. If there is a diagnostic dilemma, it is better to start steroids and anti-thyroid medications and assess the response to decide further management.

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WC6

Relapsed Graves' thyrotoxicosis following total thyroidectomy 20 years earlier

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We report a 55 year old non-smoker with a history of Graves' disease diagnosed in 1990 at age 29 years old. Due to poor compliance to therapy, she underwent a total thyroidectomy within 1 year of diagnosis. She remained well controlled on thyroid hormone replacement for over 20 years with Levothyroxine 100 µg daily. However, in the 2 years before referral to the Endocrine Clinic, she had difficult to treat hypothyroidism with persistent over-replacement; at the time of referral she was taking Levothyroxine 25 µg daily. She reported no symptoms of over-replacement. She had no evidence of thyroid associated orbitopathy. Taking Levothyroxine 25 µg daily, TSH remained suppressed at <0.01 mIU/l, Free T_4 14.5 pmol/l and Free T_3 4.3 pmol/l. Thyroid autoantibodies were both positive: thyroid peroxidase 285 unit/mL and TSH receptor 2.9 unit/ml. Ultrasound of the thyroid bed confirmed three hypervascular thyroid nodules measuring 8, 27 and 16 mm. Thyroid hormone replacement was discontinued to facilitate a thyroid uptake scan; this will likely be followed by radioactive iodine therapy if she has ongoing biochemical subclinical thyrotoxicosis.
Graves' disease is an autoimmune condition characterised by the production of autoantibodies against the thyroid-stimulating hormone receptor: TSH-receptor antibodies (TRAb). TRAb stimulates target organs with the majority developing hyperthyroidism from stimulation of follicular cell production of thyroid hormone and about half developing thyroid associated orbitopathy. Total thyroidectomy removes target tissue for TRAb and controls hyperthyroidism. Surgical thyroid resection is usually followed by a reduction of TRAb levels in variable degrees; the degree of reduction remains controversial. The median TRAb value half-life has been estimated at 93.5 days after total thyroidectomy in patients without orbitopathy or smoking, such as our patient. This case therefore demonstrates that remnant thyroid cells after total thyroidectomy in Graves' disease can become stimulated under the mediation of TRAb causing recurrent thyrotoxicosis. Revision surgery would be challenging and therefore radioactive iodine necessary.

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Workshop D: Disorders of the adrenal gland

WD1

Cushing's Syndrome Secondary to Ectopic ACTH

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A 55 year old man was seen in an endocrinology clinic for possible diagnosis of Cushing's syndrome. He described a 6 months history of feeling lethargic and increased facial puffiness and abdominal fat. There Cushingoid signs on examination, namely; moon facies, thin skin and bruises with increased abdominal fat distribution. He was admitted to hospital following symptomatic hyperglycaemia and hypokalaemia (lowest reading 2.7 mmol/l). Initial laboratory results showed raised urinary free cortisol 18091 nmol/24 h, random cortisol 1281 nmol/l which was not suppressed by low or high dose Dexamethasone test along with raised ACTH level. Other test also showed pituitary insufficiency of hypogonadotrophic hypogonadism and hypothyroidism with low TSH levels. Radiographical images did not show evidence of pituitary adenoma but instead there was a nasal space tumour and bilateral bulky adrenal glands. Histology results confirmed the diagnosis of Cushing's syndrome secondary to Ectopic ACTH from an Olfactory Neuroblastoma. He was medical managed with Metyrapone and Ketoconazole prior to pituitary surgery. Complications of Cushing's such as diabetes was treated with Metformin and insulin while hypokalaemia with Spironolactone and oral potassium replacement. Following surgical resection of the tumour, all anti-Cushing's medications were stopped as he was no long producing excess cortisol. However, he continues to be on long-term Testosterone and Thyroid Hormone replacement.

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WD2

Metastatic Malignant Pheochromocytoma – Wildfire Spread of disease post resection

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Pheochromocytoma is a rare disorder of chromafin cells and can be incidentally found in 20% of adrenal masses. It can be malignant in upto 10% of the cases. Metastatic disease poses a great challenge and no definitive cure is yet established to treat these tumors.

We report a case of 54-year-old male patient with past medical history of anxiety who presented with acute left sided abdominal pain to the urology department at Norfolk and Norwich University Hospital with suspicion of renal colic. Computed Tomography Thorax and Abdomen with contrast Incidentally picked up 9 cm left adrenal lesion presumed to be hemorraghic with simple liver cysts and no evidence of malignancy. He was then referred to the endocrinology department for work up of the adrenal lesion. Clinical examination blood pressure was 142/87 and he had microspocic hematuria. Skin examination showed few benign intradermal naevi, skin tags, multiple neurofibromas and one café au lait Spot. There was no axillary freckling. Biochemical investigation revealed raised urine Normetadrealine 9.2 µmol/24 h (0.0–3.8) and plasma normetanephrine of 1606 pmol/l (120–1180) which raised suspicion of pheochromocytoma.

In consultation with Urologist Adrenalectomy was planned and optimization of Blood pressure was done with alpha and beta blockage. Pre Operative MIBG I123 whole body scan showed uptake of tracer in the left adrenal gland and the 4th segement of the liver. This was suggestive of Left sided pheochromocytoma with hepatic deposit which was then characterised with an MRI scan as solitary liver deposit measuring 1.7 cm.

Laparoscopic adrenalectomy was complicated by excessive haemorrhage due to large size of the tumor hence open laparotomy and nephrectomy was carried out. Patient remained hypotensive postoperatively and was started on replacement Hydrocortisone which was stopped after normal synacthen response. Histology confirmed immunoreactivity for CD56, Chromogranin A, Synaptophysin and S100 (weakly) and high proliferation fraction (Ki-67) of 10–20% in keeping with an aggressive pheochromocytoma. Genetic testing was still pending.

Patient was then referred to UCL for management of the solitary liver lesion. Urinary and plasma normetanephrine had normalised at 1 month interval, but he was unfortunately found to have widerspread Metastasis to the liver, lungs and bones on repeat CT scanning. It is presumed that due the aggressive nature of the tumor and intra-operative bleeding the malignant cells were seeded in the circulation and spread widely and rapidly. Although Surgical resection is rarely curative in Malignant pheohromcytomas, tumor debulking can reduce the toxic effects of catecholamines on the mycocardium and improve survival. Mostly treatment is palliative and without treatment the 5-year survival is generally less than 50%. This widespread metastasis in such short interval has not been reported previously in literature and the levels of catacholamines may not depict the true malignant potential of the tumor as in our case they were only modestly raised. We also suggest that in presence of metastatic disease with malignant pheochromocytoma adjuvant therapy is considered concomitantly and patients are counselled pre-operatively about poor prognosis.

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WD3

A young female patient with severe hypertension referred as Conn's syndrome

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This case highlights the importance of investigating for secondary causes of hypertension especially in young people. The patient was referred to exclude Conn's disease, this case outlines the limitations one may face when interpreting the results of subsequent tests. Miss K was 24 years old when she was initially admitted under the Nephrologists with headache, palpitations and significant hypertension with systolic blood pressure (BP) of 275.

Her echo and cardiac MRI ruled our coractation of aorta, renal function and renal Ultrasound normal. Her 24 h urinary metanphrines and cortisol are normal. CT renal angio-gram was reported initially in our local hospital as no evidence of renal artery stenosis and no adrenal abnormality.

She had multiple hospital admissions with gradually increasing regimen of anti-hypertensive medications: Spironolactone 50 mg OD, Ramipril 10 mg OD, Amlodipine 10 mg OD, Doxazosin 8 mg BD, Hydralazine 25 mg OD, Bisoprolol 5 mg OD and Indapamide 2.5 mg. Despite all the above medications her BP remained very high.

Renin: 21.2 nmol/l per h (0.3–3.9 nmol/L per h), Aldosterone: 1840 pmol/l (100–850 pmol/l). Renin:aldosterone ratio not suggestive of Conn's syndrome when test performed whilst taking Amlodipine and Ramipril. Results were consistent with secondary hyperaldosteronaemia.

At this point, with no obvious cause identified, she was referred for review by a specialist hypertension centre at Addenbrooke's, Cambridge. Following review she underwent a repeat CT Renal Angiogram, that confirmed the presence of a renal artery stenosis which was treated by angioplasty.

Currently she is very well and off any treatment.

She had commenced her on anti-hypertensive agents before requesting the renin:aldosterone ratio and other investigations. Her case highlights the difficulty in obtaining and interpreting accurate results when it may be dangerous to discontinue treatment in order to facilitate testing and the importance of considering these investigations before starting treatment if possible.

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WD4

Adrenal insufficiency- an incidental finding?

Ali Naqvi, Edouard Mills & Jeannie F Todd
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54 year old lady presented to the endocrine clinic. She was diagnosed with breast cancer and right ovarian tumour at the same time in August 2015. She had right lumpectomy of breast and Salpingo-Oophorectomy in November 2015. She was not feeling very well for the last few weeks. She complained of extreme lethargy and tiredness. She had blood test done on 15th November 2016 that showed prolactin level at 2849 nmmol/l with negative macroprolactin. Hence, she was referred to the endocrine clinic. Her prolactin level was normal in February 2016 at 170 nmol/l.

She has not had any galactorrhoea. She has been menopausal for the last four years. She has a family history of hypothyroidism. She did not have any steroid replacement.

Her weight was 53.70 kg, her blood pressure was 95/60 mmHg and no postural drop. Her vision was normal to confrontation. She did not have any Cushingoid features.

Her blood results came back on the same evening and showed a very low cortisol level, less than 20 nmol/l. Her prolactin level was normal at 426 milliunit/l, TSH 0.5 milliunit/l, T_4 11.6 pmol/l, IGF-1 25.1 nmol/l, Na 140 mmol/l, K 4.6 mmol/l. She had Short Synacthen test next day and it showed very low base line cortisol $<$ 20 nmmol/l, after Synacthen, cortisol was 52 and 72 nmol/l at 30 min and 60 min respectively. Her ACTH fluctuates between $<$5 and 35.0 ng/l. We arranged long synacthen and results are following.

Time	Cortisol		
T = 0min	68	**T = +2h**	103
T = +30min	87	**T = +4h**	108
T = +60 min	87	**T = +8h**	146
		T = +24h	432

The delayed rise in cortisol, in keeping with secondary adrenal insufficiency but one of her ACTH came back as 35 ng/l. So currently she is on prednisolone 3 mg and fludrocortisone 25 µg and imaging for her pituitary and adrenals are requested. Her renin aldosterone ratio is pending.

The presentation will focus on the following questions:

1. What treatment should be offered to the patient on the basis of her result for short synacthen test and long synacthen test and ACTH? What is the diagnosis, is it primary or secondary adrenal insufficiency?

2. Are additional investigations required before starting on steroid replacement?
3. Is long synacthen test helpful in this case?

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WD5

A case of iatrogenic Cushing's due to drug interaction

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Case
A 29-year-old gentleman with HIV was self-administering anabolic steroids. The abrupt discontinuation of the exogenous anabolic steroids resulted in fatigue and abdominal pain. A short synacthen test confirmed adrenal insufficiency.
His past medical history comprised of HIV which was stable on anti-retroviral therapy. He was commenced on Hydrocortisone 10 mg in twice daily.
Over 1 year he developed clinical Cushings. Discontinuation of hydrocortisone resulted in symptomatic improvement. CT scan confirmed atrophic adrenals. ACTH was <5 ng/l whilst on Hydrocortisone. His hydrocortisone replacement therapy was reduced from 10 mg twice daily to 5 mg twice daily to improve iatrogenic Cushing.
A Hydrocortisone Day Curve was performed to evaluate endogenous production and assess exogenous replacement therapy of 5mg twice daily.

Time interval (min)	Serum cortisol (nmol/l)	Salivary cortisol (nmol/l)
0	<30	3.1
60	95	10.4
120	322	15.5
180	350	45.3
240	253	9.7
300	387	8.7
360	374	51.0

Discussion
This gentleman was in adrenal insufficiency at presentation and developed iatrogenic Cushing's on physiological replacement therapy of hydrocortisone 10 mg twice daily. A Hydrocortisone Day Curve following a dose reduction to 5 mg twice daily reveals no endogenous cortisol production and supra physiological levels lasting for 6 h. This is likely to be a result of his HIV therapy drugs inhibiting the metabolism of hydrocortisone.
Conclusion
Treatment of adrenal deficiency with Hydrocortisone in patients with HIV therapy needs to be cautiously monitored. HIV therapy currently in place and their potency of inhibition of steroid metabolism will help guide initiation and dosing of hydrocortisone.

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WD6

New onset Addison's disease in a patient with previously confirmed hyperaldosteronism

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Primary Aldosteronism (PA) is caused by autonomous aldosterone production from the adrenal cortex (due to hyperplasia, adenoma or rarely carcinoma) and diagnosis is confirmed by elevated plasma aldosterone level with suppressed renin activity and localized further by CT scan of the adrenal glands and selective adrenal venous sampling (AVS) if required. On the other hand, Addison disease (also known as primary adrenal insufficiency) which represents the other face of the coin is a rare condition mainly due to autoimmune adrenalitis in the UK, Diagnosis is flagged by combination of hyponatraemia with hyperkalaemia, further confirmation is usually by arranging a short synacthen test (SST), ACTH level, autoantibody screening and CT/MRI of the adrenal glands. We present a case of a 79-years-old lady who has been initially diagnosed with primary hyperaldosteronism in 1999 which was confirmed clinically and biochemically at that time and commenced on spironolactone to control her symptoms and recently in June 2016 presented with features of primary adrenal insufficiency (AI) that has been confirmed biochemically as well with failed SST, elevated ACTH level and positive adrenal autoantibodies with an absolutely normal MRI of the adrenal glands. Controversial data were published about the evolution of PA after prolonged treatment with mineralocorticoid antagonist causing normalization of aldosterone levels, however occurrence of Addison's disease in a previously confirmed hyperaldosteronism is a rare unusual condition and whether this is due to biological decline of mineralocorticoid secretion with age or due to specific drug treatment or combination of both deserve further exploration as if this is confirmed it may justify periodical diagnostic assessment of patients particularly when the clinical presentation is not severe.

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WD7

Cushing's disease – the potential pitfalls of adrenal autonomy

Nathalie Bolding, Rebecca Gorrigan, Shang Shaho & William Drake
St Bartholomew's Hospital, London, UK.

A 54-year-old female presented to her GP with a 9-year history of poorly controlled hypertension (requiring five drugs) and type 2 diabetes mellitus, associated with central weight gain, low mood and poor wound healing. On examination she had clinical evidence of glucocorticoid excess. Cushing's syndrome was confirmed on low dose dexamethasone suppression testing (2+0 cortisol 857 nmol/l, 2+48 cortisol 346 nmol/l). Cushing's day curve demonstrated loss of circadian rhythm, with a mean cortisol of 702 nmol/l. Serum ACTH was detectable at 45 ng/l and inferior petrosal sinus sampling confirmed pituitary-dependent Cushing's disease. Pituitary MRI imaging demonstrated a markedly enlarged, predominantly empty pituitary fossa, with a sliver of tissue on the left hand side, raising the possibility of a previous macroadenoma which had auto-infarcted. Adrenal imaging showed bilateral marked nodular hyperplasia. The relative values of the ACTH and cortisol levels was thought to indicate that the adrenals had developed a degree of semi-autonomy. Medical therapy was commenced with 8 hourly metyrapone 750, 750, 1000 mg. The patient has been referred for conventional fractionated external beam radiotherapy, in light of the fact that trans-sphenoidal surgery would be unlikely to be curative and highly likely to result in a CSF leak due to the anatomy. It is likely she will require unilateral or bilateral adrenal surgery in the future. This case demonstrates that long-term ACTH stimulation of the adrenal glands can result in significant adrenal hyperplasia with autonomy and highlights the potential value of adrenal imaging, even in cases of proven ACTH-dependent pituitary disease.

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Workshop E: Disorders of the Gonads

WE1

Complete androgen insensitivity syndrome and breastfeeding

Gemma Fraterrigo & William Drake
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A 37-year-old lady with complete androgen insensitivity syndrome (AIS, 46,XY) presented with the desire to breastfeed her first child during the pregnancy of a surrogate mother. Her past medical history included thyrotoxicosis initially treated with block-and-replace regime, followed by multiple relapses, and definitive treatment with radioactive iodine ablation. There was a family history of complete androgen insensitivity syndrome (two sister out of four) and one sister had been treated for a malignant seminoma. Our patient had undergone a bilateral gonadectomy and has been on oestrogen replacement treatment since the diagnosis of AIS was made. One month prior to the baby's delivery date, the patient's oestrogen replacement treatment was switched from oral to transdermal and the dose was doubled to simulate the latter stages of normal pregnancy. After detailed discussion and counselling, the patient was also started on treatment with domperidone, initially 10 mg three times daily to induce hyperprolactinaemia, and was advised on the use of a manual breast pump. The dose of domperidone was gradually increased to 20 mg three times daily. She was advised to remove the transdermal oestrogen patch as soon as the surrogate mother went into labour. Such treatment allowed the patient to breastfeed the child upon arrival, essential in the emotional and physical bonding between infant and mother. Though the patient cannot be the genetic mother nor experience firsthand the gestation and labour of the child, breastfeeding the child for a month after her birth was of utmost importance to her. Oestrogen replacement treatment after the baby's delivery was switched back to the normal dose and oral formulation. Prolactin level measured during breastfeeding was 1416 munits/l. After breastfeeding her baby for a month, treatment with domperidone was stopped. We describe the case of a phenotypically female patient with compete AIS who was able to breastfeed her child, obtained via IVF and surrogate mother, for a month after delivery, allowing the patient to physically and emotionally bond with the baby.

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WE2

A case of complex fertility management decision

Shang Shaho & William Martin Drake
St Bartholomew's Hospital, London, UK.

A 29-year-old gentleman known to the pediatric endocrinologist for non-mosaic Klinefelter's (XXY) syndrome. The diagnosis was made opportunistically when a karyotype was organized following his presentation at age of 4 years with delayed developmental milestone. He went through puberty spontaneously and was transitioned to adult endocrinologist in September 2005. At the time he did not have symptom of hypogonadism but did have elevated gonadotropins in keeping with the diagnosis. The following few years he was diagnosed with Ulcerative colitis and dysfibrinogenaemia causing recurrent thromboembolic events. In 2014 was referred back to the endocrinologist at the time his main issues were a desire for increased muscle bulk and his attention was turning to fertility as he was in a stable relationship. Given the fact he requires indefinite anticoagulation pursuing fertility options by mean of testicular micro dissection was not straightforward. He was referred to another unit for consideration of other treatment options and exploration of the psychological aspects of infertility. Fine needle aspiration to the testis (testicular mapping) including its risk and benefit were discussed in detail with the patient. His female partner has a child of her own; this inevitably implicates any future NHS funding. This case highlights the complexity of fertility management options in a patient with Klinefelter.

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WE3

Clomiphene-induced pregnancies following secondary infertility and amenorrhoea, with puerperal alactogenesis

Bernard Freudenthal, Sabina Russell & Chris Baynes
Chase Farm Hospital, Royal Free London NHS Foundation Trust, London, UK.

History

A 38-year-old Afghan lady was referred for investigation of secondary amenorrhoea. She had had no menstrual periods since her first spontaneous pregnancy, except when recently taking a combined oral contraceptive pill (COCP). When living abroad, she conceived on three further occasions with clomiphene induction, resulting in two live births and one miscarriage. In addition, it was noted that she had never been able to breast-feed any of her children. She did not have a prior history or current features of hyperandrogenism or polycystic ovarian syndrome, and was of normal BMI.

Investigations

Pituitary profile tests while taking a COCP showed low prolactin of 24 mIU/l, but were otherwise unremarkable with cortisol 436 nmol/l, FSH 6.2 IU/l, LH 3.0 IU/l, free T4 10.7 pmol/l and TSH 2.82 mIU/l. When taken off the COCP for 3 months, serum oestradiol was 51 pmol/l and gonadotrophins were similar with FSH 6.3 and LH 4.4. DEXA scan showed femoral neck T-score −1.9 and lumbar spine T-score −1.4. Pelvic ultrasound, also 3 months after stopping the pill, showed normal appearance of the ovaries with right ovary 5.5 ml and left ovary 1.9 ml and endometrial thickness of 2.1 mm. A progesterone challenge test was performed prior to considering requesting pituitary imaging.

Discussion

It is challenging to provide a unifying diagnosis for the anovulation together with the alactogenesis and hypoprolactinaemia, given that FSH and LH are detectable within normal ranges. The abrupt absence of menstrual periods following first pregnancy with no usual features of ovarian failure, generalised hypopituitarism or previous polycystic ovarian syndrome is unusual. The history of successful conception by clomiphene alters the likely differential diagnosis, and the case should prompt informative discussion.

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WE4

Amenorrhoea in disorder of sexual development

Rakshit Kumar, Layla Thurston & Andrew Rodin
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We present here a case of diagnostic dilemma in a 34-year female presenting with Amenorrhoea. She was recently married and saw GP for infertility and history of hot flushes, melasma and migraine. Clinic history revealed primary amenorrhoea diagnosed in Brazil at age 15. Patient was told she was born without ovaries and was started on Premarin (oetrogen). She had normal female secondary sexual charcteristics, functional female genitalia and growth. On examination, she had a normal female genitalia and body habitus with height of 1.81 m. Investigations showed FSH:42 (H), LH 20 (H), Oestradiol 157, testosterone 1.1, Free testosterone index 0.7, SHBG 150 (H) and normal pituitary profile. Genetic studies revealed Male genotype 46,XY. US and MRI pelvis confirmed rudimentary uterus and right atrophic ovarian tissue. Differential diagnosis of this presentation of Disorder of Sexual Development (DSD) included Complete Androgen Insenstivity Syndrome (CAIS), Mayer-Rokitansky-Kuster-Hauser Syndrome (MRKH) or Swyer Syndrome. Normal female range testosterone ruled out CAIS and genotype of 46,XY confirmed a rare diagnosis of Swyer Syndrome.

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Workshop F: Disorders of the Parathyroid Glands, Calcium Metabolism and Bone

WF1

A case of hypercalcaemia in Hyperparathyroidism-jaw tumour syndrome

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A 52-year-old male patient first presented in 2007 with symptoms of polyuria, polydipsia and muscle pains. Initial investigations revealed a corrected Ca^{2+}: 3.45 mmol/l and PTH: 168 pmol/l. He was also vitamin D depleted so a diagnosis of severe primary hyperparathyroidism on a background of vitamin D deficiency was made and further investigations were requested to confirm the diagnosis. Unfortunately, the patient failed to attend all subsequent follow up appointments. He presented again in 2010, this time with a 1-year history of 25 kg weight loss, constipation, dysphagia

to solids and nocturia. On examination, he was cachectic and pale with a firm, solitary nodule in the left anterior triangle and small cervical and inguinal lymph nodes. Biochemical investigations revealed a corrected Ca^{2+}: 3.40 mmol/l, PTH > 263 pmol/l, ALP: 1,075 IU/l and Cr: 136 µmol/l. Ultrasound of the kidneys demonstrated increased echogenicity of the parenchyma bilaterally consistent with chronic kidney disease. X-ray of hands showed cortical re-absorption consistent with hyper parathyroid changes. He was admitted to the endocrinology ward to pursue further investigations urgently including a neck ultrasound with FNA, DEXA and MIBI scan. These investigations confirmed the diagnosis of parathyroid carcinoma with jaw tumour syndrome. He underwent Para thyroidectomy. He also received external beam radiotherapy post operatively due to incomplete surgical excision. He remained clinically and biochemically stable until 2014 when he re-presented with symptomatic hypercalcaemia and a high PTH of 144 pmol/l and was readmitted for bisphosphonate infusions and denosumab therapy. He had developed back pain and an MRI spine showed a sclerotic lesion on T8, which was biopsied only to confirm a parathyroid carcinoma metastases. After discussion at the thyroid MDT he was referred for urgent laminectomy under the neurosurgical team in addition to radiotherapy. There were no complications postoperatively and his corrected calcium after surgery has remained normal at 2.41 mmol/l with PTH: 9.6 pmol/l, which remains high due to secondary hyperparathyroidism from chronic kidney disease. The patient was referred for genetic testing and he was found to have a CDC73 deletion so his family members have been referred for further genetic screening and clinical evaluation.

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WF2

Giant parathyroid cyst – a rare cause of severe hypercalcaemia

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Parathyroid cysts (PCs) are rare lesions usually found during exploration of neck and mediastinal masses. Ten percent of PCs can be functional and release parathyroid hormones causing hypercalcemia. Most PCs are small but lesions greater than 1 cm can exert pressure effects on adjacent structures.. Diagnosis can be challenging as PCs can mimic thyroid masses with no focal tracer uptake during most functional imaging modalities. Fine needle aspiration (FNA) can help if coupled with intracystic parathyroid hormone (PTH) measurement. Functional and large cysts require surgical excision. We report a case of a 61-year-old male with incidental finding of severe hypercalcemia (corrected calcium 3.96 mmol/l) with a raised PTH (125.8 pmol/l; normal range 1.6–6.9 pmol/l) level during routine biochemical investigations. He had a large underlying mediastinal mass causing local compression, with inconclusive imaging for preoperative localisation of a suspected parathyroid adenoma. During surgical exploration (via a midline sternotomy), he was found to have a PC originating from the inferior pole of the right lobe of the thyroid gland. He underwent a right hemithyroidectomy with cystectomy resulting in significant reduction in intraoperative PTH levels. Histology was positive for a benign parathyroid adenoma with evidence of thyroid tissue surrounding the cyst. Postoperatively patient was nomocalcaemc with a PTH level in range and normal thyroid function tests. Surgery was complicated by recurrent laryngeal nerve palsy. In essence PCs although rare, should be considered in the differential diagnosis of neck masses warranting early surgical intervention to restore calcium homeostasis rapidly.

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WF3

Hypercalcaemia with inappropriate parathyroid hormone (PTH) levels

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A 74-year-old with a history of type 2 diabetes, chronic kidney disease, vitamin D deficiency and bronchiectasis was referred to the medical team with symptomatic hypercalcaemia with raised adjusted calcium of 3.80 mmol/l. Investigations also showed raised creatinine and vitamin D levels; normal parathyroid hormone (PTH) and angiotensin-converting enzyme (ACE). Myeloma screen was negative. A diagnosis of primary hyperparathyroidism was made in view of elevated calcium and normal PTH levels. On this admission, vitamin D supplements were stopped and she was treated with intravenous fluids and pamidronate. In the following weeks, CT and bone scan did not show any evidence of malignancy and there was no increased uptake on sistamibi scan. PTH-related peptide levels were not detected. Hypercalcaemia persisted despite cinacalcet, IV pamidronate and post-parathyroidectomy of all four glands. She was treated empirically with dexamethasone in view of malignancy being a differential diagnosis. PET-CT showed extensive lymphadenopathy of supraclavicular, mediastinal and hilar nodes. A biopsy was undertaken via endoscopic bronchial ultrasound (EBUS) revealed lymphocytes, a single epitheloid and giant cell granuloma. This confirms a diagnosis sarcoidosis with secondary hypercalcaemia.

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WF4

Primary hyperparathyroidism due to parathyroid carcinoma

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A previously well 50-year-old female was admitted to the acute medical unit with a 4-month history of fatigue, anorexia, polyuria, polydipsia and arthralgia. She was found to be hypercalcaemic with a corrected calcium of 3.84 mmol/l and, unexpectedly, an extremely elevated parathyroid hormone reported at > 200 pmol/l. Other results included an elevated alkaline phosphatase, hypophosphataemia and normal renal function. Treatment with intravenous 0.9% sodium chloride and pamidronate resulted in amelioration of the patient's symptoms. Her calcium improved only marginally, and she required ongoing intravenous rehydration to maintain a calcium level below 3 mmol/l. Ultrasonography of the neck and sestamibi scanning showed a right superior parathyroid mass. She was referred to an endocrine surgeon who went on to perform a parathyroidectomy and partial thyroidectomy. The clinical suspicion of parathyroid carcinoma was confirmed histopathologically. The patient's hypercalcaemia resolved immediately and PTH returned to normal. She was subsequently discharged on oral vitamin D supplementation. Approximately 2 weeks following discharge, the patient began experiencing widespread muscle cramps and paraesthesia. Corrected calcium was 1.78 mmol/l and PTH was elevated at 40 pmol/l indicative of secondary hyperparathyroidism and 'hungry bone syndrome'. She was treated with intravenous calcium infusion followed by oral calcium supplementation and high-dose alfacalcidol. This case reminds us that primary hyperparathyroidism is not always a benign condition and can rarely be due to parathyroid carcinoma. It also highlights the challenges of managing hypercalcaemia when the PTH is substantially elevated, the importance of hungry bone syndrome, and the necessity of liaison with our surgical colleagues in such cases.

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WF5

A case of non-parathyroid hormone-mediated hypercalcaemia

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We report a 61-year-old female with a history of bronchiectasis, primary Sjogren's syndrome and osteoporosis. She had taken oral glucocorticoids initiated by the rheumatologists for 10 years, which were stopped 2 years ago. She was referred to the Endocrine Clinic with a short history of polyuria and polydipsia. Biochemistry confirmed new hypercalcaemia and an acute kidney injury: corrected calcium 3.14 mmol/l, phosphate 1.13 mmol/l and 25-hydroxyvitamin D 66.4 nmol/l. At the time of referral, she was taking Adcal D3 1 tablet twice daily. After being encouraged to consume 2.5 l of fluid daily, her corrected calcium improved 2.66 mmol/l, phosphate 0.79 mmol/l, creatinine 128 umol/l and PTH low-end of normal reference range 2.2 pmol/l. Simultaneously, she was being seen by the respiratory physicians due to increased breathlessness, cough and fatigue. CT chest demonstrated a new increase in mediastinal lymph nodes and the development of parenchymal changes suggestive of sarcoidosis. Serum ACE was raised at 79 IU/L. She is currently awaiting an endobronchial ultrasound to facilitate a histological diagnosis of pulmonary sarcoidosis. Non-parathyroid hormone-mediated hypercalcaemia is a prevalent complication of sarcoidosis in 10–20% of patients, meaning it should be considered in all patients presenting with hypercalcaemia with no apparent cause. It is due to uncontrolled synthesis of 1,25-dihydroxyvitamin D3 by macrophages and leads to increased intestinal absorption and increased bone resorption of calcium. Treatment of

hypercalcaemia in sarcoidosis depends on the degree of hypercalcaemia and its persistence. General advice includes avoiding excessive sunlight, avoiding foods which are rich in vitamin D and consuming 2.5 l of fluid daily. For those patients who experience calcium levels >3 mmol/l or severe symptoms, oral glucocorticoids can be used, which inhibits enzymatic conversion of cholecalciferol to calcitriol via 1-alpha hydroxylase in macrophages. Furthermore, patients with sarcoidosis who receive vitamin D supplements are at increased risk of developing hypercalcaemia and therefore renal impairment.

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WF6

Primary hyperparathyroidism – that's easy for you to say

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Mr JP, a 64-year-old gentleman with a background of type 2 diabetes mellitius, ischaemic heart disease and hypertension was referred due to hypercalcaemia. He had a long history of hypercalcaemia, 9 years according to the biochemistry records and his adjusted calcium was 2.9 mmol/l on presentation. His parathyroid hormone level was 8.1 pmol/l. JP had clear symptoms of hypercalcaemia – polydipsia, polyuria, problems with concentration, fatigue, headaches and generalised aches and pains. Due to his high calcium level and PTH level he was diagnosed with primary hyperparathyroidism and due to the calcium level and his symptoms he was sent for localisation studies as work up for potential surgery. Sestamibi and ultrasound scans of his neck unfortunately did not reveal a clear site of a parathyroid adenoma, however after discussion with the surgeon, JP decided to proceed with a parathyroid exploration with a view to removing an adenoma. In the meantime, he was referred for an MRI scan of his neck and mediastinum before the procedure. MRI was unhelpful and he went for surgery and had three of four parathyroid glands removed (leaving the left upper behind) without and surgical complications. PTH and calcium levels fell after the surgery but not back into the reference ranges. JP was feeling better after the surgery although he still had some residual symptoms. The plan at this stage was to review again in 6 months and if the biochemistry remained deranged then to re-image with a view to removing the final gland. When see again in clinic in 6 months (9 months after surgery) JP was feeling lethargic and had headaches however the thirst and aches and pains had remained resolved. His adjusted calcium level was 2.96 mmol/l however his parathyroid hormone level was normal at 5.0 pmol/l. A 24-h urine collection for creatinine/calcium ratio was arranged in order to exclude concurrent familial hypocalcuric hypercalcaemia. He was advised to drink less tea (was drinking 20 cups/day) due to possible diuretic effect raising his calcium. The urine collection was normal. At the time of writing he is awaiting a repeat calcium level.

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WF7

Familial Hypocalciuric Hypercalacaemia

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Sixty six year old gentleman was referred to clinic with hypercalcaemia. He suffered with low mood, "funny turns", nocturia and loose stools with urgency to open bowels. He appeared tanned but no increase pigmentation in skin folds. There was no palpable lymphadenopathy or neck lump. The calcium levels were around three with low normal PTH of 3.7 (2.8 on repeat). He had not developed any complications like osteoporosis, fracture or renal stones secondary to hypercalcaemia. He had extensive investigations for his symptoms and hypercalcaemia.

Investigations- Calcium excretion index 25.9(<22 for FHH) and 24 h urine calcium was mid normal range at 6.23(repeat 5.32). Full blood count, renal function and liver function were normal with slightly raised plasma viscosity of 1.99. The myeloma screen was negative. Vitamin D and TSH was in normal range. Chest X-ray was showed no mass or enlarged mediastinal lymph nodes. Ultrasound and MIBI scan did not identify any parathyroid adenoma.

Other - 9 AM cortisol was 485. Faecal elastase and tissue transglutaminase were normal. No abnormality identified in colonoscopy. 24 h tape showed sinus rhythm. Urinary catecholamines were normal. CT scan of head, neck, chest, abdomen and pelvis did not reveal any cause for hypercalcaemia.

Since no conclusive cause was identified for his hypercalcaemia, genetic test were done to investigate for Familial Hypocalciuric Hypercalcaemia. The results confirmed the diagnosis and the patient was informed. It was explained to him that it had no implications for his long term health and does not require investigations in future.

This is an interesting case as urine calcium was NOT low which is not typical for Familial Hypocalciuric Hypercalcaemia; hence genetic test was done. It also a good case for learning as it highlights the investigations that need to be done to rule out other conditions which cause hypercalcaemia.

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WF8

A case of transient hypercalcemia

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We are presenting a common case of diagnostic dilemma with hypercalcemia.

This 66 year female patient was referred to Endocrine clinic with hypercalcemia. She was asymptomatic with blood tests showing C.Calcium of 2.73 mmol/l. This was followed by investigations to rule out primary or tertiary hyperparathyroidism.

Blood tests revealed high PTH of 12.2 pmol/l with low 25OH Vitamin D of 37 nmol/l, suggesting a diagnosis of tertiary hyperparathyroidism. Patient was started on Vitamin D replacement. Repeat blood tests after 6 months showed normal C.Calcium of 2.46 mmol/l, normal 25OH Vitamin D level of 85 nmol/l but persisting high levels of PTH at 11.5 pmol/l.

At this stage we planned to repeat bone profile in 3 months. Next set of bloods revealed normal Calcium, 25OH Vitamin D of 95 nmol/l and PTH of 10.5 pmol/l. Differentials of Primary Hyperparathyroidism vs Familial hypocalciuric hypercalcemia (FHH) were considered. Urine Calcium/Creatinine ratio was low 0.14 mol/mol suggesting FHH. This was followed by repeat urine calcium/creatinine ratio and 24 h urine calcium, both tests came back as normal, confirming primary hyperparathyroidism. Patient remained asymptomatic and eucalcemic, hence further investigations were deferred.

This was a good learning case with a common presentation in Endocrine clinics.

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WF9

Hypercalciuric hypocalcaemia – potential pitfalls and a novel treatment option

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The calcium-sensing receptor (CaSR) is a G-protein coupled receptor expressed in the parathyroid gland and kidneys. Loss of function mutations of the CaSR result in familial hypocalciuric hypercalcaemia (FHH). Rarer, gain of function mutations of the CaSR result in hypercalciuric hypocalcaemia and are inherited in an autosomal dominant pattern. The phenotype varies from asymptomatic individuals, to profound hypocalcaemia. We present a severely affected individual whose case highlights the potential pitfalls in treatment of hypercalciuric hypocalcaemia and a novel therapeutic approach with a continuous parathyroid hormone (PTH) infusion. A 16 year old, Caucasian female was referred to our unit with severe hypocalcaemia and frequent fits. She was born at 31 weeks gestation and aged 2 days old was noted to be hypocalcaemic, hypomagnesaemic and hypoparathyroid. The patient's mother had hypocalcaemia since childhood and an initial diagnosis of familial hypoparathyroidism was made. Genetic analysis aged 4 years identified a heterozygous missense CaSR mutation and she was diagnosed with hypercalciuric hypocalcaemia. Her medication included sandocal, magnesium, alfacalcidol and bendrofluazide. Biochemistry: Corr Ca++1.4–2.9 mmol/l, creatinine 101 μmol/l. Renal ultrasound:early nephrocalcinosis. A trial of subcutaneous PTH 1-34 20 μg bd and cessation of alfacalcidol/bendrofluazide, resulted in improved hypercalciuria, but persistent hypocalcaemic fits. She was thus trialled on a continuous PTH infusion, via an insulin pump (omnipod, unlicensed use) at 1.3 μg/h (concentration 125 μg/ml) resulting in a stable serum calcium, with no fits for 1 year. She was converted to a Medtronic pump due to supply issues and developed hypercalcaemia, requiring a reduction in PTH to 0.735 μg/h (concentration 35 μg/ml). This case demonstrates the potential severity of hypercalciuric hypocalcaemia and the very difficult balance between avoiding hypocalcaemic

seizures and hypercalciuria-associated nephrocalcinosis. It highlights the importance of avoiding vitamin D in this condition, which worsens hypercalciuria and nephrocalcinosis. Our patient has impaired renal function and her mother has end stage renal failure. This case demonstrates the dramatic clinical improvement and smaller PTH doses that can be achieved with a continuous PTH infusion. It also shows the potential variability in PTH delivery between different insulin pumps.

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WF10

Severe hypercalcaemia and osteoporosis in a patient with primary hyperparathyroidism

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A 60-year-old Caucasian lady was referred to the metabolic bone clinic for assessment of osteoporosis. Her risk factors for osteoporosis included gender, postmenopausal status, childhood immobility due to illness, previous severe vitamin D deficiency, COPD, as well as an extensive smoking and alcohol history. She had multiple previous fragility fractures involving her ribs and both radii. A DEXA scan revealed lumbar osteoporosis (T -4.0) and femoral osteopenia (T -2.0). Thoracolumbar X-rays revealed vertebral loss of height at L3. On examination her BMI was 22.8 kg/m^2. Systemic examination was unremarkable with no endocrine cause for her osteoporosis revealed.

Following the clinic appointment, routine blood tests showed adjusted calcium 4.39 mmol/l (2.2–2.6), phosphate 1.00 mmol/l (0.8–1.5), PTH 59.7 pmol/l (1.1–6.8), vitamin D 27.7 nmol/l (70–150), creatinine 85 umol/l, and alkaline phosphatase 120 IU/l (30–130). Serum protein electrophoresis and thyroid function were normal.

Although she was asymptomatic, she was admitted for further investigations and management. ECG revealed a prolonged PR but normal QT interval. She commenced intravenous rehydration and also required pamidronate and cinacalcet to control her calcium levels. Neck ultrasound revealed a probable left superior parathyroid adenoma measuring 11×7×9 mm and she was referred for an urgent neck exploration and parathyroidectomy. A CT thorax, abdomen and pelvis showed a chronic ill-defined 18 mm right hilar mass. There were concerns that this could represent a granulomatous disease, and a trial of prednisolone was commenced pending biopsy. Histology showed just reactive tissue and steroids were then stopped. A few weeks following her admission, she underwent a left inferior parathyroidectomy with remaining glands reviewed as normal. There was no soft tissue invasion or lymphadenopathy. Histology showed hypercellular parathyroid tissue with no definite features of malignancy.

Post-surgery, adjusted calcium was 2.39 mmol/l and PTH 6 pmol/l and she awaits a further DEXA scan to assess for improvement in her severe osteoporosis.

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Workshop H: Miscellaneous endocrine and metabolic disorders

WH1

A case of Von Hippel-Lindau, diagnosed following oral contraceptive pill health check

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Case

A 35 year old lady was found to be hypertensive following a blood pressure check in view of her oral contraceptive pill use. Because of her young age she was referred to the Hypertension Clinic. She underwent a renal ultrasound as part of the screening for end organ damage in view of her drug-resistant hypertension. The ultrasound- and subsequent CT imaging- revealed a 2.8 cm left adrenal mass measuring 44 Hounsfield Units. She denied any palpitations or headaches but did report to suffer from intermittent sweats and flushes, almost on a daily base. In addition she described an occasional sense of impending doom. Three 24 h urine collections for metadrenalines showed consistently raised normetadrenaline levels. MIBG scan showed that the mass was MIBG avid. Her blood pressure normalized on Phenoxybenzamine and she underwent an uncomplicated laparascopic left adrenalectomy. The histology of the tumour was in keeping with a pheochromocytoma. Post-surgery her bloodpressure has remained normal without medication and she is no longer experiencing symptoms of flushing. Repeat 24 h urine collections for metadrenalines have been negative. Genetic screening, requested prior to surgery, revealed that she carries a mutation in the Von Hippel-Lindau (VHL) gene. Up until now, she has not been diagnosed with other manifestations of VHL; Ophtalmology review was negative for retinal haemangioblastoma and MRI investigations were negative for cerebellar- and myelum haemangioblastoma, endolymphatic sac tumours, renal cell carcinoma, pancreatic cysts and pancreatic neuro-endocrine tumours. Her family is currently undergoing genetic screening.

Discussion

i) The clinical diagnosis of pheochromocytoma can be difficult. In our case hypertension in a relatively young person led to renal imaging, which prompted the analysis of an adrenal mass. ii) Approximately 30% of patients diagnosed with a pheochromocytoma are found to have an underlying genetic disorder, such as VHL, MEN2 and neurofibromatosis type 1. Early diagnosis of inheritable tumour syndromes and surveillance for additional tumour manifestations are key, both in the presenting patient and their extended family. iii) VHL patients might benefit from a combined Endocrinology-Genetics MDT for their lifelong follow-up.

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WH2

An unusual presentation of multiple endocrine neoplasia 1 (MEN1)

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Case history

A 36 year-old man was referred to our department for further evaluation of a large adrenal and multiple liver mass lesions. These had been found on an abdominal ultrasound requested for a 3 week history of mild, episodic abdominal pain. He was asymptomatic but described mild night sweats for over 10 years.

Investigations, results and treatment

Hyperparathyroidism was evident, with a serum corrected calcium 3.04 mmol/l (2.15–2.65), PTH 16 pmol/l (1.6–6.9). Ultrasound of the neck showed three enlarged parathyroid glands. Two 24 h urine collections showed raised normetadrenaline levels >70 000 nmol/l (<4400) and raised 3-methoxytyramine levels >3200 nmol/l (<2500). Plasma glucagon was 65 pmol/l (<50). Cross sectional and functional imaging revealed 8.6×6.3 cm inhomogenous adrenal mass with avidity to FDG and MIBG, 2.2×1.7 cm pancreatic tail mass with avidity to FDG alone and 6.3×3.5 cm extracapsular low density mass antero-lateral to the right lobe of the liver with no demonstrated metabolic activity. Genetic testing identified a previously described mutation in intron 4 of the MEN1 gene. He underwent a right adrenalectomy, 3 ½ gland parathyroidectomy and thymectomy and distal pancreatectomy in separate operations at a different institution. Histology revealed a macroscopically excised phaeochromocytoma which demonstrated areas with loss of heterozygosity, fully excised pancreatic 20 mm tumour positive for CGA, synaptophysin and CK19 and biopsy of the perihepatic mass revealed benign cartilaginous tissue. He remains well with no biochemical or radiological evidence of disease recurrence.

Conclusions and discussion points

This case describes an unusual de novo presentation of multiple endocrine neoplasia type 1 (MEN1). Phaeochromocytomas have been reported in MEN1 but are very uncommon. The management dilemma of this case relates to the approach to surgical strategy. Should you monitor the pancreatic tail mass, attempt resection at the time of phaeochromocytoma resection or remove it separately? Does the histology inform our monitoring for the future?

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WH3

Von Hippel–Lindau disease and pre-implantation genetic testing for *in-vitro* fertilisation

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Case history

Von Hippel–Lindau disease (VHL) was confirmed in this 36 year old gentleman when he was 13. His father's diagnosis of metastatic renal cell carcinoma (RCC)

and paragangliomas prompted genetic testing and revealed mutation c.499C>T p. (Arg167Trp) which is associated with type 2B VHL in this patient and his sister. Shortly after diagnosis, bilateral phaeochromocytomas were resected with a bilateral adrenalectomy. At age 17, the patient developed tinnitus, deafness to high-pitches and unsteadiness when imaging confirmed an endolymphatic sac tumour on the left which was surgically removed. At age 27, surveillance imaging revealed a C1 spinal haemangioblastoma which was surgically resected. At age 35, he and his partner underwent pre-implantation genetic testing prior to undergoing successful *in-vitro fertilisation* and subsequent delivery.

Investigations

Routine surveillance: *Central Nerves System*: Magnetic resonance imaging (MRI) of the brain and spine confirms the absence of a right side endolymphatic sac and no residual disease of the left, a small cerebellar haemangioblastoma, which will be monitored, and expected abnormal architecture at the site of his resected haemangioblastoma at C1. *Ophthalmic*: Unremarkable. *Abdomen (non-renal):* A remnant of adrenal tissue remains but the size is stable. Two small lesions, consistent with islet cell tumours, were seen in the pancreas, one in the head, which remains stable in size, and one in the ucinate process which is no longer visible. *Abdomen (renal):* Simple cysts are seen in the kidneys but require no intervention, eGFR >90 ml/min. *Biochemistry:* Normal urinary metanephrines.

Conclusion and points for discussion

This case describes the clinical course of type 2 VHL and illustrates the requisite routine imaging and biochemical surveillance. Of importance is the monitoring of renal cysts due to the high risk of RCC. Points for discussion include the contemporary genetic nomenclature and relation to the clinical presentations, emerging treatment for haemangioblastomas; surgery vs radiotherapy, and fertility considerations with pre-implantation genetic testing and IVF considerations for patients of reproductive age.

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WH4

The multiple tumours of MEN1 and some interesting chest imaging

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A 59 year old lady attended endocrinology clinic for her annual review. She had presented at the age of 28 years with symptomatic hypoglycaemia. She was diagnosed with an insulinoma and underwent resection of the tail of the pancreas. She was concurrently found to be hypercalcemic and underwent two gland parathyroidectomy. Genetic testing confirmed a heterozygous frameshift mutation in exon 3 of the MEN1 gene (c.628_631 del.) and a diagnosis of multiple endocrine neoplasia type 1 was made. Over the past three decades she has been diagnosed with two malignant melanomas and a breast fibroadenoma, which have been associated with MEN1. She also has gastric varices. She has four children, two of whom have tested positive for MEN1. At her latest review pancreatic MR imaging demonstrated two stable islet cell tumours. Chromogranin A had risen from 20 to 86 pmol/l over 3 years, associated with a rise in gastrin from 287 to 2354 pmol/l in 1 year. The patient's symptoms were well controlled on omeprazole 40 mg bd. The patient remained persistently hypercalcaemic (Corr Ca++ 2.9 mmol/l, PTH 8.1 pmol/l) and was asymptomatic. She had no evidence of renal calculi on ultrasound imaging and DEXA scan showed osteopaenia of the femoral neck and lumbar spine. She drinks a minimum of 2.5 l of water per day and takes vitamin D supplements. Pituitary MRI and biochemistry were normal. As part of the new screening guidelines the patient underwent chest imaging which showed progressive nodular CT change, associated with modest uptake on FDG-PET and reduced transfer factor (80% predicted). This is thought to represent diffuse idiopathic pulmonary neuroendocrine cell hyperplasia (DIPNECH). Biopsy would only be possible via a VATS procedure and as the patient has no respiratory symptoms she will be monitored with serial CT scans. She has been referred for a gallium dotatate PET. This case demonstrates the multiple endocrine and other tumours associated with the MEN1 syndrome. It also demonstrates the radiological appearances of DIPNECH, a rare condition considered to be a precursor of peripheral carcinoid tumours.

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WH5

A complex case of Von Hippel Lindau syndrome: RCC vs NET metastases?

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Case history

A 45 year old lady with Von Hippel Lindau syndrome with a complex past medical history presented with symptoms of cauda equina compression. Past medical history includes cerebellar hemangioblastomas, bilateral retinal angiomas, right sided renal cell carcinoma and renal carcinoid tumour treated with partial and then completion nephrectomy, bilateral phaeochromocytoma treated with bilateral adrenalectomy, metastatic neuroendocrine tumour treated with Whipple's procedure, radiofrequency ablation and embolization of liver metastases and temozolamide, and secondary diabetes mellitus.

Investigations

Whole spine MRI, Octreotide scan, NM bone whole body with SPECT, brain CT, L femur X-ray.

Results and treatment

Octreotide scan was negative for octreotide positive disease. Whole spine MRI revealed a new large expansile and destructive bony vertebral mass in L2 region causing tight cauda equina compression, highly vascularized in keeping with metastatic disease, and a previously noted small non compressive lesion at L5. This L2 lesion was initially thought to be a renal cell carcinoma metastasis. Patient was initially treated with dexamethasone and then neurosurgically for debulking of L2 lesion and L1-3 fixation. Histology revealed metastatic neuroendocrine carcinoma. During the post-operative period while mobilising the patient had a pathological right humeral shaft fracture through lytic lesion treated with right humeral intramedullary nail fixation. NM Bone whole body with SPECT revealed focal areas of increased uptake in anterior skull, posterior skull, midshaft of right humerus, and midshaft of left femur in keeping with bone metastases. Brain CT showed a destructive lesion of left parietal/occipital bones with well-defined surrounding soft tissue compartment measuring 3.7×3.4 cm, confined to extra-axial compartment and causing inward bowing of dura with local mass effect in parietal lobe. Similar 1×1.5 cm destructive lesion in right frontal lobe breaching only inner table of calvarium was also shown. These lesions seem to be new metastatic deposits. Patient has no central nervous system or cerebellar symptoms. In view of asymptomatic skull bone metastases and previous radiotherapy to the posterior fossa decision was made against radiotherapy to the skull/brain. Patient was treated with bisphosphonate infusion and received radiotherapy to L1-3, left femur and right humerus, followed by systemic treatment with Temozolamide.

Conclusions and points for discussion

We describe a complex case of VHL syndrome: back pain leading to diagnosis of L2 lesion initially thought to be a metastasis of RCC that resulted to be a NET metastasis. The post neurosurgical period was complicated by a pathological right humerus fracture, which led to the finding of multiple bone metastases and required nail fixation, bisphosphonate infusion, radiotherapy and systemic treatment with Temozolamide. Ongoing management questions include whether gallium dotate scan might be beneficial for consideration of treatment with lanreotide and whether investigation is necessary to clarify the origin of the bone metastases.

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Additional Cases

CB1

Primary hyperparathyroidism in a patient with thyroid hormone resistance

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Introduction
Thyroid hormone resistance due to a TRα mutation is rare and only recently described. We report a case of primary hyperparathyroidism in an adult patient with a known TRα mutation.
Case presentation
A 60 year old female was referred with hypercalcaemia and hyperparathyroidism that persisted despite correction of vitamin D deficiency. Parathyroid hormone levels continued to be elevated or inappropriately normal with hyper- or normocalcaemia. She had a normal range urinary calcium excretion and normal renal ultrasound. DEXA scan showed osteoporosis. Sestamibi scan failed to localise a parathyroid adenoma. 'Primary hypothyroidism' was identified at a young age (3 years) and she received continuous thyroid hormone replacement. As both of her children developed early life hypothyroidism; genetic assessment was performed leading to diagnosis of thyroid hormone resistance syndrome due to thyroid hormone receptor alpha mutation (heterozygous A263V mutation in the THRA gene). Her condition presenting as selective thyroid hormone resistance and hypothyroidism, she was continued on L-T_4. She declined surgical exploration without prior localization of the parathyroid lesion, and as she does not have symptoms of hypercalcaemia she remains under surveillance and is being investigated for genetic causes of hypercalcamia (including calcium sensing receptor CaSR gene mutation) and possible molecular pathogenesis that links between thyroid hormone resistance and hypercalacaemia.
Conclusion
While thyroid hormone receptor alpha mutation is a rare genetic mutation; the combination of primary hyperparathyroidism and thyroid hormone resistance is very rare and an underlying genetic pathogenesis linking the two is yet to be determined.
Keywords: Thyroid hormone resistance, TRα, hypercalcaemia, hyperparathyroidism.

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CB2

Cushing's disease in 19 years old student

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A 19 years old student from Bahrain,she has Diabetes mellitus Type II on metformin tablet twice daily for two and half years, she has hypertension uncontrolled on Coveram 5/5 perindopril/amlodipine increased twice daily but still uncontrolled. She has presented with subclinical hypothyroidism TSH is high above 5 and normal T_4. Clinically she has mild abdominal obesity, acne, and mood changes.
Investigations
FBS 7.3 urea 2.9 Cortisol 769.3 at 0800 h. Short Dexamethasone suppression test failed to keep cortisol level normal, while high dose 48 hours dexamethasone suppression test reverted to normal. ACTH 53 pg/ml normal is below 46. MRI done normal. It was mistake they did MRI brain only although request was clear. MRI pituitary Micro adenoma. So referred to neurosurgery (surgery microscopic) done. After surgery patient blood pressure was well controlled, she stopped anti Hypertension and diabetic medications. She was temporally on cortef tablet 10-5-5. Following sequence in endocrine treatment is very successful in treating patients.
Discussion
Dose this patient needs further tests or what was done is enough. Is this situation every time or there is challenges in treatment.

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CB3

Severe hypercalcaemia associated with inflammatory diarrhoea in a young male

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Introduction
Severe hypercalcaemia is a medical emergency which can result in severe cardiovascular and neurological dysfunction. Pathophysiological mechanisms of hypercalcaemia include increased osteoclastic activity, osteolytic bone metastasis, extra-renal activation/production of 1,25 hydroxyvitamin D and decreased urinary calcium excretion. Understanding these mechanisms is important to guide the diagnostic process and the long-term management of hypercalcaemia.
Case report
We present a 26 years old Afro-Caribbean man who presented with 8 months history of diarrhoea, 10 kg weight loss, abdominal pain and arthralgia; associated with a recent history of dry cough and breathlessness. He was not on any long term medicines. Apart from bilateral episcleritis, clinical examination was not of note. Blood tests showed a corrected serum calcium of 4.18 mmol/l, serum phosphate of 1.73 mmol/l and acute kidney injury. Parathyroid hormone was fully suppressed with normal serum angiotensin converting enzyme level and normal vitamin D. Parathyroid hormone related peptide and 1,25 hydroxyvitamin D were undetectable. Serum immunoglobulins were normal and antinuclear antibodies were negative. Computed tomography scan of chest and abdomen demonstrated features of organizing pneumonia (not typical of sarcoidosis) and oedematous proximal small bowel. Upper gastrointestinal endoscopy and colonoscopy showed diffuse patchy inflammation of the colon and duodenum. Capsule endoscopy showed ulceration and blunted villi with inflammation in the distal small bowel and terminal ileum. Colonic and duodenal biopsies demonstrated diffuse chronic inflammation. No granulomata were seen. Cultures for bacteriology and tuberculosis were negative. Renal biopsy showed tubular calcification in keeping with hypercalcaemia, no significant inflammation or granulomata. He was treated with prednisolone 20 mg tds and intravenous normal saline hydration. His corrected calcium improved to 3.08 mmol/l within 10 days with normalization of his kidney function. Hydroxychloroquine 200 mg od was added with subsequent normalization of corrected calcium to 2.38 mmol/l within 7 days. He was discharged on hydroxychloroquine 200 mg once daily and prednisolone 15 mg once daily, which was then reduced to 10 mg once daily. Video assisted thoracoscopic lung biopsy and push enteroscopy for small bowel biopsy were planned as outpatient. Unfortunately, the patient declined any further investigations as he remained completely asymptomatic. Although the final diagnosis has not been reached yet, the impressive response to glucocorticoids and hydroxychloroquine is suggestive of a macrophage dependant inflammatory process responsible for the severe hypercalcaemia, with low grade gastrointestinal lymphoma remaining a possible cause.

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CB4

A calcium conundrum

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Mr RP is a 69 year old gentleman who presented to A&E following a fall. He described several weeks of lethargy, ataxia, confusion and polydipsia. He had a past medical history of hypertension and COPD, but was otherwise fit and well. On admission bloods revealed an adjusted calcium of 5.25, with a PTH of 2.4. He also had a significant AKI with an eGFR of 22. After initial management with IV fluids, a cautious dose of pamidronate was given. Basic investigations did not identify a cause for the hypercalcaemia. Bendroflumethiazide was stopped, but it was not felt that this was the sole cause. Over the next 10 days, his symptoms resolved and he was discharged with an adjusted calcium of 2.83 and eGFR 35. Over the forthcoming months he had extensive investigations into the cause of the hypercalcaemia. Malignancy was thought to be most likely given the low PTH, despite no constitutional symptoms. Bloods revealed PSA 1.7 and ALP 109, and no evidence of Multiple Myeloma. CT-chest/abdomen/pelvis and bone scan were normal. Recurrent PTH levels were low but detectable. In light of a PTH of 2.4, despite a calcium above 5, it was felt prudent to investigate further for primary hyper-parathyroidism. Both US and Sestmibi scan of the parathyroids did not reveal an adenoma. The only abnormality of note on his bloods was a raised ACE at 84. Following discussion with the respiratory team and the radiologists, it was felt that a diagnosis of Sarcoid was a possibility, however there was little evidence to support this diagnosis and no lesion to biopsy. PTHrP result is pending. Regular monitoring of the calcium was performed, which varied dramatically on a week-to-week basis. On two occasions over the last couple of months, the calcium was found to be greater than 3, but without intervention dropped to the normal range within 1 week.
This case posed a number of difficulties for us as physicians.

i) What further investigations could help make a diagnosis?
ii) Are there any cyclical causes of hypercalcaemia?
iii) How frequently do we need to monitor this gentleman's calcium level?

iv) At what calcium shall we treat this gentleman given the drastic variation in results?
v) How do we manage expectations in our patients when a diagnosis is not found?

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CB5

Complex combinations of late effects

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Case history
This 32 year old lady developed anterior and posterior pituitary failure following chemotherapy, radiotherapy and bone marrow transplant for acute lymphoblastic leukaeamia in childhood and subsequent CNS recurrence. During her teens, she required GH replacement with which she was poorly compliant. She had a mastectomy and currently takes hormonal treatment for oestrogen receptor positive T2N1M1 breast cancer, which is possibly a consequence of her total body irradiation. Complications of her pituitary dysfunction have been adrenal insufficiency during acute illness and fluid balance problems during a trip to the tropics. Additionally, she has developed bilateral posterior subcapsular ocular lens opacities, mixed dyslipidaemia and classical migraines.
Investigations
Latest blood tests; TSH <0.01 munit/l, FT_4 20.00 pmol/l, 25-hydroxyvitamin D 35 nmol/l, cortisol day curve confirmed adequate replacement, ACTH <5 ng/l, FSH 1.3 unit/l, LH 0.7 unit/l, oestradiol <19 pmol/l, prolactin 249 munit/l, SHBG 134 nmol/l. A brain MRI scan in 2005 was unremarkable. DEXA scan showed a T score of -2.2.
Current management
Pituitary replacement: hydrocortisone 10/5/2.5 mg, L-T_4 75 µg OD, desmopressin 10 µg BD intranasal. *Bone health:* Calcium supplementation and alendronic acid 70 mg once per week. *Breast cancer management:* Letrozole OD and the patient is considering prophylactic bilateral mastectomy and breast reconstruction. She was also previously also taking GH 0.9 mg/1.0 mg on alternate days, ethinlyoestradiol 2 µg OD and norethieterone 5 mg OD on days 1–14 of menstrual cycle; stopped on account of the breast cancer.
Conclusion and points for discussion
This is a case of an adult with anterior and posterior hypopituitarism because of childhood lymphoblastic leukaemia and its treatment. It illustrates the complexity of late effects of cancer treatments and important interactions of disease management. Points for discussion include the peri-operative management of patients with pituitary failure, management of symptoms of ovarian failure in the presence of a co-existing oestrogen-receptor positive tumours and the use of exogenous GH in patients with pituitary failure and concomitant malignancy.

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CB6

Complex thyroid cancer with likely underlying TSHoma – a medical challenge

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A 22-year-old Caucasian female was referred to thyroid specialist clinic due to difficulties fully suppressing her thyroid stimulating hormone (TSH) despite high dose combination therapy of triiodothyronine (T3) and thyroxine (T4) treatment. She had a well-differentiated thyroid -carcinoma of left thyroid lobe treated with left lobectomy. Several other small primary thyroid carcinoma lesions were detected on further examination of the histology which lead to completion thyroidectomy and radioiodine treatment.
On assessment, she denied any symptoms of dysthyroidism. Her great-grandmother had goitre; otherwise there is no other evidence of thyroid disease in the family. Clinically, she was euthyroid with no evidence of recurrence or lymphadenopathy. Anterior pituitary function tests were all within normal range and MRI scan of pituitary gland indicated a 30-mm macroadenoma. Thyroid function tests showed free T4 of 29.8 pmol/l (10–22 pmol/l), free T3 of 10.7 pmol/l (3.1–6.8 pmol/l) and a TSH of 0.06 mU/l (0.3–4.5 mmol/l). Her thyroglobulin level was undetectable. Following evaluation, she was diagnosed with biochemical hyperthyroidism and likely pituitary incidentaloma and was advised to discontinue treatment with T3 to prevent thyrotoxic effects.
On follow-up, patient was pregnant; otherwise clinically unremarkable. However, her pituitary mass had grown and was abutting the optic chiasm. Thyroglobulin remained undetectable. The tumour growth was attributed to pregnancy changes and patient was managed in joint obstetric and endocrine clinic. Post-partum scans showed macroadenoma with no change in size. Thyroglobulin remained undetectable and alpha sub-unit was low. However, TSH remained at detectable range. Patient did not wish to undergo possible curative surgery as it may affect her chance of future pregnancies. Following multi-disciplinary team discussion, somatostatin analogue was trialled. TSH level supressed to undetectable range with somatostatin; however, tumour size did not change. Currently, patient is managed on a combination of T4 and somatostatin analogue under close follow-up.

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CB7

Amiodarone induced thyrotoxicosis? type2

Amutha Krishnan & Emran Ghaffar Khan
Nobles Hospital, Douglas, Isle of Man, UK.

Case history
62 Year old male known to have inflammatory bowel disease, ischemic cardiomyopathy, ICD implant for sustained ventricular tachycardia was referred to endocrine clinic by the GP for thyrotoxicosis which was found on lab workup for worsening diarrhoea and tiredness.
Drug history
Amiodarone 200 mg od since 2012, Bisoprolol, Eplerenone, Atorvastain and Pentasa.
Investigations
FT4 48.6 pmol/l (6.5–17.0) TSH0.03 (0.35–4.94) TPO antibodies negative. USG thyroid showed a normal gland with normal vascular flow.
Results and treatment
Diagnosed as amiodarone induced thyrotoxicosis (AIT) and initially was commenced on carbimazole 40 mg and prednisolone 40 mg was added after 4 weeks as there were no improvement in his thyroid function test results. His carbimazole was stopped by his GP 2 weeks after as the patient developed severe rash. As TFTs improved by then we continued him on tapering dose of prednisolone.He was advised to continue amiodarone by the cardiologist in view of his VT and cardiomyopathy. His colitis flared up when prednisolone dose was tapered down to 10 mg, Hence GP increased prednisolone to 20 mg though his FT4 14.2 and TSH 1.9 were in normal ranges then. He was commenced on azathioprine by the gastroenterologist and his prednisolone gradually withdrawn as the patient developed steroid induced diabetes. He is asymptomatic with latest FT413.5 and TSH12.6

	16.10.15	17.11.15	07.12.15	01.02.16	22.09.16	16.11.16
T4	48.6	42.9	23	14.2	14.4	13.5
TSH	0.03	0.03	0.07	1.9	11.03	12.6

Conclusions and points for discussion

i) Diagnostic and therapeutic challenges in AIT
ii) Amiodarone-whether to stop or not?

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CB8

Androgen deprivation in testicular cancer by way of macroprolactinoma – balancing the two pathologies

James Pittaway, Jonathan Shamash & William Drake
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Case history
A 72 year old gentleman from Zimbabwe presented to clinic with MRI pituitary findings of $1.5\times1.4\times1$ cm pituitary macroadenoma. This had been discovered incidentally during outpatient investigation for severe headaches refractory to standard analgesia at another hospital. The mass was in contact with but not compressing the optic chiasm. He did not describe any visual loss. He had an

unintentional weight loss of 10 kg in the last 2 months. He described decreased libido for the last 3 years and no erections including the mornings. He has no other past medical history other than glaucoma. Examination of the testes revealed small testes bilaterally approximately 6–8 ml in volume. Visual fields were minimally decreased temporally to confrontation with red pin.
Investigations, results and treatment
Pituitary profile revealed a raised prolactin of 19 204 mU/l (0–496) after PEG precipitate. The rest of his anterior pituitary function was within normal limits. Testosterone was suppressed at 1.6 nmol/l (9–27). PSA was raised at 4426 mcg/l (0–4.4) and liver function revealed a raised ALP of 758 U/l (30–130). On the basis of these blood tests he was diagnosed with a macroprolactinoma and metastatic prostate cancer and referral to oncology was made.
Whole body CT and bone scans revealed widespread bony disease including a burden of disease in the skull. He was started on docetaxel and also cabergoline. His headaches improved but after his PSA dropped but after some time his testosterone level started to rise. Cabergoline was stopped and he remains headache free. He has not had any visual disturbance and MRI pituitary reveals no compromise of the optic chiasm. His prostate cancer disease burden is currently stable on enzalutamide 18 months on from diagnosis.
Conclusion and points for discussion
This case describes a gentleman with metastatic prostate cancer who is biochemically castrate secondary to a macroprolactinoma. The management dilemma of this case is balancing the treatment of each as they are interrelated and how best this should be done.

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CB9

PTH independent hypercalcaemia – diagnosis dilemma

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Summary
A case of intermittent hypercalcemia with a suppressed parathyroid hormone (PTH) for nearly 3 years. It was found that high calcium specifically occurred in summer and each one lasted for about a month. Once calcium normalised the renal function also improved. Exact aetiology has not been found even after extensive investigations.
Case presentation
A 67-year-old gentleman first presented to the endocrine clinic in early 2014 with Hypercalcemia. Tests revealed hypercalcemia (adjusted calcium 2.76 mmol/l) with supressed PTH (0.7 pmol/l), and low vitamin D of 44 nmol/l. The working diagnosis was hypercalcemia with suppressed PTH. Bendroflumethazide was stopped and no other precipitants found. He also had mild anaemia and mild renal impairment (previously normal renal function). Calcium normalised with hydration.
Subsequent investigations showed negative myeloma screen. CT CAP in 02/2014 showed bilateral multiple areas of ground glass opacification. Chest physicians diagnosed it as possible sarcoidosis, however appearances resolved on follow up CT done on 08/2014.
Since early 2014 he had few more admissions in Aug 2014, Jul 2015 and Jul 2016; notably all these admissions were in summer and he presented with hypercalcaemia with suppressed PTH and acute kidney injury. In between these admissions, his calcium remained normal. In July 2016, he developed Complete heart block which has not resolved with correction of calcium and a pacemaker was inserted. Repeat CT CAP in 08/2016 did not show any features of malignancy or reveal any lung parenchymal disease or any features of sarcoidosis. More extensive investigations were performed including PTHrP, 1,25 vitamin D and serum ACE, all of which came back normal. He had recent admission in November 2016 with similar presentation. This time it was precipitated by a small dose of Cholecalciferol. It returned to normal with adequate hydration and iv bisphosphonate.
Questions to the panel

i) The cause of Hypercalcemia, is it cyclical hypercalcemia?
ii) Are there any other investigations that need to be done?
iii) Is there any genetic cause?
iv) Is it sarcoidosis which is intermittently active? If it is the case, how do we diagnose?

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CB10

A challenging case of dual endocrine pathology

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A 39-year-old man presented with thyrotoxicosis and was diagnosed with Graves' disease. Despite high doses of anti-thyroid medication for 18 months, he remained biochemically and clinically hyperthyroid. Therefore, a thyroidectomy was planned. Four days before surgery, he developed double vision and was referred for urgent Neurosurgical review at our centre. On further questioning, he reported a 12-month history of lethargy and low libido. On examination, he had right 6th cranial nerve palsy and a partial right ptosis. A pituitary MRI showed a large suprasellar lesion with right cavernous sinus involvement. Biochemistry showed prolactin 37 384 mU/l (macroprolactin negative), testosterone 1.6 nmol/l, LH 1.5 IU/l, FSH 1.7 IU/l, T4 16.4 nmol/l, T3 7.2 nmol/l, TSH <0.01 mU/l, cortisol 53 nmol/l and IGF-1 23.2 nmol/l (13–50 nmol/l). His case was discussed at the ICHNT Pituitary MDT meeting. His thyroidectomy was cancelled and he commenced cabergoline 0.5 mg/week and hydrocortisone replacement. Serum prolactin fell quickly to 5456 mU/l after one dose of cabergoline 0.5 mg. Both the right-sided ptosis and 6th nerve palsy resolved.
With regards to his thyroid dysfunction, TSH receptor antibody level was raised at 2.1 u/ml (ULN 0.3) with a persistently elevated fT3 and undetectable TSH. He elected for radioactive iodine treatment rather than thyroidectomy as a definitive cure for his Graves' disease. Nine weeks following radioactive iodine, he commenced thyroxine replacement (fT4 9.3 nmol/l).
A subsequent pituitary MRI showed a significant reduction in the size of the prolactinoma, albeit with persistent right parasellar extension to the cavernous sinus. Prolactin reached a nadir of 437 mU/l. However, he demonstrated low mood, with aggression and anger at out-patient review. After multiple discussions, he proceeded with a trial without cabergoline. Nine months later, his prolactin is static at 2494 mU/l and his mood improved. An interval pituitary MRI is being scheduled.
Questions for discussion
What are the options for this gentleman if his prolactin rises without dopamine agonists? Will surgery alone be sufficient in view of probable cavernous sinus involvement?

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CB11

ACTH-dependent Cushings and secondary amenorrhoea: where is the source and are they linked?

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A 37-year-old female referred by gynaecologist with elevated serum testosterone found on evaluation for amenorrhoea. She has had oligomenorrhoea for 5 years with induced bleed on Logynon and no change on stopping. Episodes of feeling hot and sweaty and going red in face. On examination: weight 68 kg, BMI 25 kg/m^2, euthyroid, euadrenal, no hirsutism. BP: 116/86 (lying) & 110/90 (standing for 2 min). Available blood results: Estradiol- 43–91; LH- <0.1–6.9 and FSH- 0.3–15.4; S Testosterone- 6.6 and 3.5 and 4.1 nmol/l; DHEAS- 15.9 umol/l. Others- prolactin, TSH, Vit B12, folate, HbA1c- all normal. Elevated DHEAS suggesting adrenal source. Available CT abdomen (noncontrast)- adrenals normal. TA and TV USS- ovaries small without follicles. Suspected possible premature ovarian failure! But LH and FSH not suggesting of and source of Testosterone and DHEAS still unclear. Evaluation: FBC- normal, Corr Ca^{2+} 2.38 mmol/l, PO_4- 0.70 mmol/l, T Chol.- 5.4 mmol/l, HDL- 1.6 mmol/l, Vit D- 84.3 nmol/l; HbA1c- 40 mmol/mol, Prolactin- 152 mU/l, TSH- 0.63 mU/L, S Cortisol- 1144 nmol/l (09:45am), Testosterone- 2.4 nmol/l, E2- 77 pmol/l, FSH- 3.1 and LH 0.1 u/l, 17OHP- 2.1 nmol/l (1.9–6.5). Tests suggesting cortisol excess and normalised testosterone. DHEAS- 15.6 umol/l (1.7–9.2). Further evaluation: 24-h urine cortisol excretion- 1127 and 368 nmol/24 h (100–379); ODST (1 mg) S Cortisol – 1317 nmol/l. LDDST (0.5 mgx8) S Cortisol- basal 1251 nmol/l and 48 h 459 nmol/l; ACTH (with basal sample)- 54 nmol/l. Tests suggests ACTH-dependent Cushings possible pituitary source. Review: bruising easily, now round face, with slight flushed appearance, small base of neck hump. Further evaluation: HDDST (2 mgx8); basal 1332 and 48 h- 1507 nmol/l; MRI pituitary- normal. Suggesting possible ectopic source of ACTH. BMD: osteoporosis in spine and osteopenia in hips. Further review: very flushed, more cushingoid in her facial appearance, weight stable, BP- 130/84. Contrast CT (neck to pelvis)- poorly

enhancing rounded lesion in liver with nonspecific appearance, nil else. Where is source of ACTH and how to explain various hormonal abnormalities?

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CB12

Investigating menstrual disturbance: a series of unfortunate events

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A 20-year-old lady presented to her GP with menstrual irregularity and worsening right-sided headaches with associated vomiting. Blood tests showed a mildly raised testosterone level and a markedly raised prolactin level, approximately 80% of which was macroprolactin (normal monomeric prolactin level). She was subsequently referred by her GP for an MRI Pituitary, which was reported as showing a 6-mm hypoenhancing lesion. Medical history includes migraines and hayfever; she was on no regular medications. She reported persistent menstrual disturbance, but no other symptoms, when seen in endocrine OP some 9 months later. There was no clinical evidence of hirsutism. Repeat blood tests confirmed normal monomeric prolactin in the context of macroprolactinaemia; as well as biochemical hyperandrogenism. Review of her MRI scan in our local MDT discounted the possibility of a pituitary lesion. Pelvic sonography confirmed the presence of multiple ovarian cysts. This case illustrates a series of unfortunate mishaps that can lead to over-investigation and misdiagnosis of menstrual disturbance.

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CB13

Primary aldosteronism – management can be challenging and complex

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Africo Caribbean gentleman was referred to our endocrine department for management of secondary hypertension. He initially presented to his doctor with headache and he was found to be hypertensive with systolic BP of 200 mmHg and hypokalaemia. A renal MRI showed a right adrenal mass, 2.9×2.4 cm, and echocardiogram showed moderate left ventricular hypertrophy and diastolic dysfunction. He had normal 24 h excretion of metanephrines and free cortisol. An aldosterone renin ratio, after stopping interfering drugs, was 2320, consistent with Conn's syndrome. He also had a saline suppression test, which showed baseline aldosterone to renin ratio of 5700 and post saline infusion, the aldosterone remained elevated at 250 pmol/l. Subsequently he had adrenal venous sampling (AVS) that showed right adrenal aldosterone-to-cortisol (AC) ratio at 3.4, a left adrenal ratio of 8.3 and an IVC ratio of 3.3. The lateralisation index was left dominant at 2.44 (8.3/3.4), contralateral suppression index was 1.03 (3.4/3.3). This was interpreted as being suggestive of bilateral disease. He had repeat MRI Adrenal and comparison was made with previous MRI. The 2.5 cm right adrenal lesion was unchanged in size and appearances and the left adrenal gland appeared normal. He had an FDG-PET scan that showed right adrenal lesion, demonstrated low level FDG uptake. The level of metabolic activity in the left adrenal is within the normal limits. Currently he is on Spironolactone 75 mg OD, Valsartan 160 mg and Amlodipine 10 mg. He feels 'washed out and weak', and would like to consider surgical options for the treatment of his Primary hyperaldosteronism. The presentation will focus on the following questions:
(1) Does this gentleman require adrenal surgery, and if so, what would be the recommended procedure?
(2) Are additional investigations required before surgery can be recommended?
(3) Are alternative medical treatments required for the hypertension?

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