Hammersmith Abstracts

11th Hammersmith Multidisciplinary Endocrine Symposium 2016

Hammersmith Hospital 11th Multidisciplinary Endocrine Symposium

Friday 9th December 2016

Wolfson Education Centre, Hammersmith Hospital

8.30am Registration and Coffee

8.50am Welcome and Introduction: Mr. Fausto Palazzo, Prof Karim Meeran and Prof. Waljit Dhillo

Session 1: Endocrine Failure (James Ahlquist)

9.00am Hypothyroidism: The benefits of T3 supplements – truth or myth
Prof Graham Williams (Imperial College)

9.30am Adrenal insufficiency: Back to the future
Prof Karim Meeran (Imperial College)

10.00am Clinical case: The Adrenal Full House
Dr Amir Sam (Imperial College)

10.15am Endocrine challenges pancreatic failure/ total pancreatectomy
Dr Bernard Khoo (Royal Free Hospital)

10.45am Coffee Break

Session 2: More Endocrine Failure

11.10am Hypoparathyroidism: Long term consequences and novel treatments
Prof Bill Fraser (Norwich)

11.45am Clinical case OC1 (W008): An unusual case of Autoimmune Hypoparathyroidism: presenting as Congestive cardiac failure and Myocardial Infarction.

12.00 noon Clinical case OC2 (W009) Challenges in concurrent liver and pancreatic insufficiency: glucose control on a tight rope

12.15pm Lunch & Poster session

Session 3: Hyperparathyroidism (Chair Fausto Palazzo)

1.00pm Hyperparathyroidism in pregnancy & miscarriage
Dr Marcus Martineau (West Middlesex University Hospital)

1.30pm Lithium induced HPT
Dr Jeremy Cox (St Mary's Hospital)

2.00pm Clinical Case OC3 (W016) Hypercalcaemia in a patient on Lithium therapy

2.15pm Coffee break

Session 4: Hyperparathyroidism (Waljit Dhillo)

2.45pm Genetic testing in work up of hypercalcaemia (FHH/MEN1&@)
Prof Neil Gittoes

3.15pm Endocrine surgery: Technique versus technology
Mr David Scott Coombes (Cardiff)

3.45pm Clinical case OC4 (W013) : Large Undescended Inferior Parathyroid Adenoma Masquerading as part of Retropharyngeal Large Multinodular Goitre.

4.00pm Clinical case OC5 (W014): Thoracic Surgery for Persistent Hyperparathyroidism due to Ectopically Located Parathyroid Tissue

4.15pm Feedback and Close

W001	A rare occurrence of adrenal leiomyosarcoma
W002	Thyrotoxic Cardiomyopathy - A reversible entity
W003	Drug-Induced Inappropriate Antidiuretic-Hormone Activity and Hypokalemia induced Diabetes insipidus- safe use of 1.8% hypertonic saline in acute severe symptomatic hyponatremia
W004	Missing Periods – the Case of a Pituitary Carcinoma
W005	Identification of rs6161, previously designated a benign variant of CYP11A1, as a recurrent, pathogenic splicing mutation
W006	Suppression of parathyroid hormone in two patients with severe hypomagnesaemia
W007	Large Undescended Inferior Parathyroid Adenoma Masquerading as part of Retropharyngeal Large Multinodular Goitre
W008 OC1	An unusual case of Autoimmune Hypoparathyroidism: presenting as Congestive cardiac failure and Myocardial Infacrction
W009 OC2	Challenges in concurrent liver and pancreatic insufficiency: glucose control on a tight rope
W010	Use of a treatment pathway in Primary Hyperparathyroidism to ensure consistent care and appropriate use of Cinacalcet in patients unable to have operative intervention
W011	Beer Potamania- A reversible cause of symptomatic severe hyponatremia
W012	Breast cancer metastases to the thyroid gland – An uncommon sentinel for diffuse metastatic disease
W013 OC4	Large Undescended Inferior Parathyroid Adenoma Masquerading as part of Retropharyngeal Large Multinodular Goitre
W014 OC5	Thoracic Surgery for Persistent Hyperparathyroidism due to Ectopically Located Parathyroid Tissue
W015	Something to chew on: a rare cause of hypercalcaemia

W016 OC3	Hypercalcaemia in a patient on Lithium therapy
W017	IgG4 related hypophysitis-a diagnostic challenge
W018	Neuroendocrine tumour diagnosed in pregnancy, the challenges of complex disease
W019	The long search for an occult ectopic ACTH-producing tumour
W020	Case Report: The Curious Case of Misleading Cortisol
W021	Palpitations and the Pituitary – the Case of Assay Interference

W001

A rare occurrence of adrenal leiomyosarcoma

Aditi Sharma, Elisa Lewington-Gower, Ana Pokrajac

Leiomyosarcomas are rare tumours that can arise from smooth muscle cells anywhere in the body; common sites include uterus, GI tract and retroperitoneum. We report an unusual case of a patient with an adrenal incidentaloma confirmed as a leiomyosarcoma on histology.

A 61-year-old Caucasian female was investigated for chronic abdominal pain. CT scan of the abdomen and pelvis did not show any pathology other than an incidental 2.3cm right adrenal nodule. She underwent CT and MRI of the adrenals to further characterise the lesion. These were reported as 'indeterminate' but likely a benign adrenal incidentaloma. Patient was then referred to our Endocrinology department whereby further testing confirmed a non-secretory tumour. A follow up CT adrenal scan at 6 months showed an increase in the size of the tumour to 3cm, of heterogeneous density with delayed washout of contrast with a low-enhancing centre, requiring an urgent referral for surgery. Although the pre-operative investigations suggested an adrenal tumour, at surgery the tumour was even larger and found to be invading the IVC and was clearly malignant. A planned laparoscopic procedure was changed to an open procedure and a grade 2 right per-adrenal leiomyosarcoma was resected with resection of the lateral wall of the IVC. Interestingly, the attached adrenal gland was normal. Patient is currently followed up in a dedicated leiomyosarcoma centre.

This case highlights the importance of appropriate radiological assessment in adrenal incidentalomas by experienced adrenal radiologists. Monitoring of patients with suspicious looking lesions even if not meeting the initial criteria for surgery should be rigorous and discussed in a dedicated multi-disciplinary team

W002

Thyrotoxic Cardiomyopathy - A reversible entity

S Qureshi, S G Wijetilleka, K Yoganathan, A Stevenson, S Wright, S Ladbrokes, R Kaushal

Graves thyrotoxicosis rarely causes heart failure in healthy patients. It is a presentation in only 6% of thyrotoxicosis patients and conventional treatment for hyperthyroidism normally reverses these cardiovascular manifestations.

We present a patient who presented with 3 week history of progressive breathlessness, palpitations and sleeping in chair. She had a past history of being seen in the endocrine clinic 8 weeks prior to the presentation with a relapsed Graves disease treated with small dosage of Carbimazole. She had a past medical history of subtotal thyroidectomy with subsequent hypothyroidism in 1967. She remained asymptomatic until her current presentation with no active treatment. Her thyroid ultrasound thyroid showed multi-nodular goitre and a fine needle aspiration was not advised.

Her examination revealed various features of congestive cardiac failure, moderate goitre and thyroid eye disease. Her echocardiogram showed an ejection fraction of 20-25% on admission and akinetic cardiac wall abnormality (figure 1). Her previous echocardiogram 7 years early was within normal parameters. Her biochemical tests on admission were T4 38 p mol TSH <0.01 m IU/L and TSH antibodies were positive. She was initiated on Carbimazole 20 mg and Propanolol 40 mg three times and she showed dramatic improvement in her congestive cardiac failure. She was subsequently discharged from hospital on anti-thyroid therapy. She was scheduled for a follow up echocardiogram in 4-6 weeks and we hope to find that her repeat echocardiogram will improve with biochemical improvement in her thyroid function tests.

The clinical manifestations of thyrotoxic cardiomyopathy are secondary to an unknown aetiology. An assessment of thyroid hormone status in all patients with congestive cardiac failure may permit identification of a potentially reversible cause in this patient group.

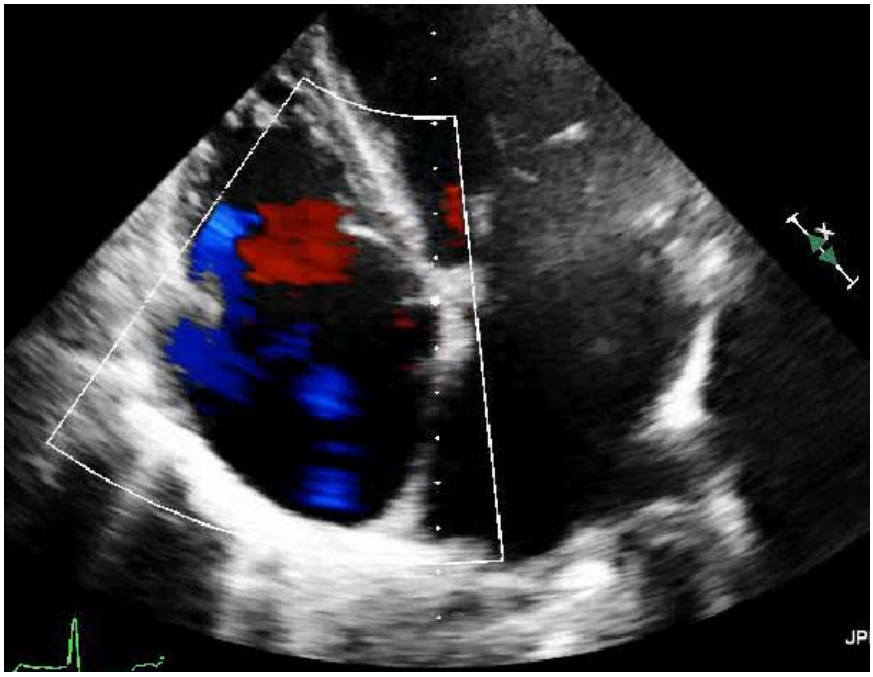

Figure 1. Echocardiogram

W003

Drug-Induced Inappropriate Antidiuretic-Hormone Activity and Hypokalemia induced Diabetes insipidus- safe use of 1.8% hypertonic saline in acute severe symptomatic hyponatremia

S Qureshi, S G Wijetilleka, K Yoganathan, A Stevenson, S Wright, S Ladbrokes, R Kaushal

Hyponatremia can be a manifestation of physiological processes, systemic disease processes or drugs. It is important to establish the cause early as it is potentially remediable and reversible. The symptoms of hyponatremia secondary to SIADH are attributable to the severity of hyponatremia and rapidity of syndrome development.

We present a challenging case of a 55 year old lady who re-presented to our emergency department with acute confusion, problem with her balance and slurred speech with a serum Na of **98 m mol** after being discharged with an earlier presentation with **Na 120 m mol**. She had a past medical history of treatment with anti-depressants i.e. Quetiapine, Duloxetine, Vortioxetine etc. It was agreed for her anti-depressants to be stopped due to various side effects but she chose to take her previously prescribed therapy without advice from her GP. Her blood sugar on admission was normal and there were no signs of renal/cardiac failure. Her anterior pituitary screen was unremarkable.

On examination there was no neurological lesion and her CT brain was normal. She was clinically euvolaemic but her GCS was 4 on admission.

She was initially managed with 3% hypertonic saline in HDU but remained polyuric after discharge from HDU with confirmed biochemical hypokalemia. It was agreed for her polyuria to be treated with Desmopressin until it was reversed and then treated her euvolaemic hyponatremia with ward based care with 1.8% hypertonic saline. Her MRI Pituitary revealed micro-prolactinoma with no active lesion in posterior pituitary. Her serum Na normalised after strict fluid restriction and use of hypertonic saline on the ward. She was discharged with follow up in the local ambulatory care unit.

Learning Points:

- Patients presenting with severe symptomatic hypontraemia should have routine investigations but a careful history of previous medications i.e. Anti-psychotics etc. shall be excluded.
- Acute symptomatic hypontraemia is managed conventionally with fluid restriction on medical wards which is challenging but use of 1.8% hypertonic saline to reverse acute hyponatraemic state is not common practice yet.
- Our case highlights the importance and safe use of 1.8% hypertonic saline in acute life threatening symptomatic hyponatremia for continued ward based care with excellent results.

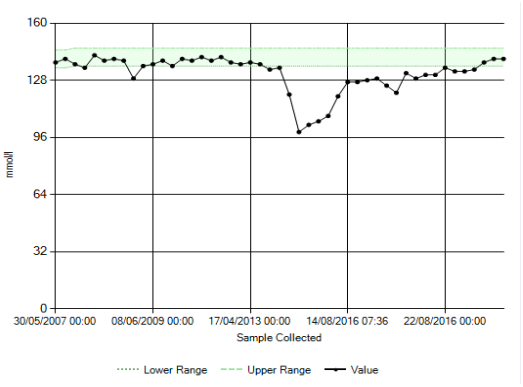

Figure 1- Serum Na and response to Tx

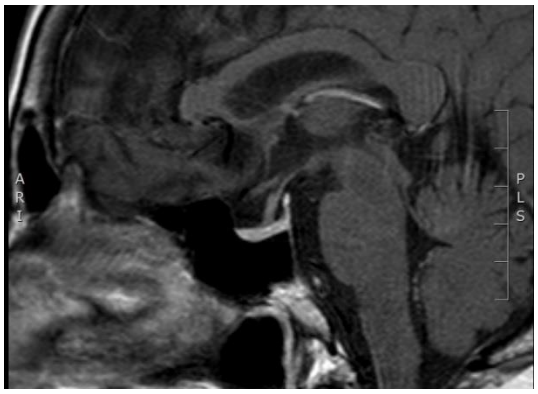

Figure 2- Microprolactinoma

W004

Missing Periods – the Case of a Pituitary Carcinoma

S G Wijetilleka, S Qureshi, R Kaushal

We would like to present the rare case of pituitary carcinoma. We reviewed a 21 year old female with normal development and BMI and 18 months of secondary amenorrhoea after stopping the oral contraceptive pill. She experienced occasional headaches but had no other symptoms and signs of pituitary disease.

- Post-clinic bloods revealed 9am morning cortisol – 123, oestradiol 103, FSH 1.3, LH 0.4, TSH 2.17, T4 - 5.8. IgF-1 and Prolactin in the normal range. Her HCG was negative, her vitamin D – 25.6 and her FBC and U&E were normal.

- We advised replacement with Hydrocortisone 10mg at 8am, 5mg at noon, 5mg at 1800 and Levothyroxine 50mcg od. Microgynon 30 if periods do not return after commencing levothyroxine. We also advised Vitamin D replacement (cholecalciferol).

Pituitary mass seen on MRI Sept 2015 - 2.8 cm likely pituitary macroadenoma with compression of the optic chiasm superiorly. Images reviewed in pituitary MDT at St George's Hospital. Patient's mass akin to a craniopharyngioma upon review of radiology. Patient seen at St George's on 28/10/15; she elected for transphenoidal surgery which took place in January 2016. The histology showed a pituitary carcinoma and our patient went on to have pituitary radiotherapy.

At West Middlesex Hospital, we ordered a pre-operative ECHO (family history of HOCM) and monitored the patient for signs of diabetes insipidus. Her U&E at present is within the normal range, with a serum osmolality of 290 and a urine osmolality of 76. She is currently euvolaemic and on hormone replacements.

W005

Identification of rs6161, previously designated a benign variant of *CYP11A1*, as a recurrent, pathogenic splicing mutation.

A Maharaj*, F Buonocore*, E Meimaridou, T D Cheetham, C Brain, E Gray, J Suntharalingham, N Striglioni, H Spoudeas, M Donaldson, J C Achermann*, L A Metherell*

Background: The genetic variant chr15:74635368 in *CYP11A1* (SNP ID rs6161 c.940G>A) has a minor allele frequency of 0.0024 (ExAC); At protein level (p.E314K) it is predicted to be benign with heterologous expression of the mutant protein showing no functional difference from wild-type. *CYP11A1* encodes the P450 side chain cleavage enzyme which initiates the steroidogenic cascade by conversion of cholesterol to pregnenolone. Severe deficiency of this enzyme is characterised by disordered sexual differentiation in addition to adrenal and gonadal insufficiency but partial loss-of-function mutations can present with a milder phenotype.

Results: In ten patients, with variable presentations, sequencing and segregation analysis revealed the presence of the rs6161 variant in exon 5 of *CYP11A1* in compound heterozygosity with deleterious mutations on the other allele. This rs6161 variant is enriched in our patient cohort with an allele frequency = 0.015 ($p<0.01$). We hypothesized that it affected the gene at the RNA level, perhaps altering splicing, or that it represented a marker for another intronic/regulatory change within the gene. An *in vitro* splicing assay, utilising minigene constructs incorporating wild-type or variant exon 5 sequences, revealed markedly reduced splicing of variant exon 5.

Conclusion: Previously considered a benign variant, rs6161 occurs more frequently than expected in patients, alters splicing and is pathogenic when in combination with a disruptive change on the other allele. It represents a partially inactivating mutation and can lead to mild phenotypic presentation.

Impact: With the advent of whole exome and genome sequencing the necessity of assessing whether a sequence variant is deleterious or not becomes key. *In silico* prediction tools may not be adequate to assign causality and hence analysis of exonic and non-exonic variants will need to be performed *in vitro* not only considering the change at protein but also at nucleic acid level.

N.B. Local Ethical Committee approval obtained.

W006

Suppression of parathyroid hormone in two patients with severe hypomagnesaemia

E Mills, S Narayanaswamy, A Naqvi, J Todd & F Wernig, Hammersmith Hospital, Imperial College Healthcare NHS Trust, London

Hypocalcaemia can be associated with coexisting hypomagnesaemia, which induces hypoparathyroidism as well as resistance to parathyroid hormone (PTH). Here, we present two patients with severe hypomagnesaemia and associated hypocalcaemia.

Patient A was an 88-year old woman who was admitted via the Emergency Department with general malaise, twitching and tremors following a short illness with diarrhoea. She had a medical history of diastolic heart failure and was taking furosemide, perindopril and lansoprazole. She was found to have severe hypocalcaemia 1.51mmol/L with inappropriately normal PTH 5.7pmol/L. Magnesium was undetectable <0.25mmol/L and 25-hydroxyvitamin D 102.2nmol/L. Patient B was a 61-year old woman who was referred to the Endocrinology Clinic with tingling, numbness and leg cramps. She had a jejunal bypass 40-years previously and had experienced chronic diarrhoea since. Due to severe reflux, she was taking both lansoprazole and omeprazole. She was found to have severe hypocalcaemia 1.64mmol/L, inappropriately normal PTH 6.5pmol/L, undetectable magnesium <0.25mmol/L and 25-hydroxyvitamin D deficiency 14nmol/L.

In both patient's, after the administration of intravenous magnesium, there was an immediate and a significant increase in the levels of circulating PTH; in Patient A PTH rose to 26.9pmol/L and in Patient B to 23.8pmol/L. In both cases, this led to a concomitant improvement in hypocalcaemia and resulted in normocalcaemia. Patient B also received intramuscular vitamin D replacement. Patient A was discharged from hospital with oral calcium supplements and has remained normocalcaemic. Her PPI was discontinued. However, in Patient B, despite oral calcium supplements, calcium and magnesium gradually fell in the weeks following the magnesium infusion despite stopping both lansoprazole and omeprazole. This was felt to be due to on-going diarrhoea, which would be exacerbated by oral magnesium supplements and she has been referred to the Gastroenterologists.

Like calcium, magnesium plays a crucial role in the regulation of PTH secretion. These cases demonstrate the blunted PTH secretion in patients with severe hypomagnesaemia. Profound hypomagnesaemia decreases the release of PTH and induces skeletal resistance to PTH which can result in severe hypocalcaemia. In both cases, hypomagnesaemia will have been driven by diarrhoea along with long-term proton pump inhibitor use, which is known to reduce intestinal magnesium absorption.

W007

Large Undescended Inferior Parathyroid Adenoma Masquerading as part of Retropharyngeal Large Multinodular Goitre

A Plonczak, A N Di Marco, F F Palazzo, Department of Thyroid & Endocrine Surgery, Hammersmith Hospital, Imperial College Healthcare NHS Trust

The Case: A 71 year old female with asymptomatic biochemically proven primary hyperparathyroidism and compressive symptoms from a very large toxic multinodular goitre is presented. Examination revealed a very large goitre with distension of the external jugular veins suggestive of thoracic inlet compression. Localisation studies in the form of ultrasound neck and SestaMIBI failed to identify a parathyroid adenoma. CT of the neck and upper thorax noted the left thyroid lobe to be larger with a significant retropharyngeal component, extrathoracic tracheal compromise and minor retrosternal extension.

A combined total thyroidectomy and parathyroidectomy via a cervical approach was performed. The right thyroid lobe was mobilised first, during which an enlarged right superior parathyroid gland was identified. The left lobe of the thyroid was then mobilised and a classically positioned but abnormally large left superior parathyroid gland was found and safely removed. Subsequently, during mobilisation of the highly developed superior pole of the left thyroid lobe, a separate retropharyngeal structure, superomedial to the lobe, was encountered and mobilised. This structure, measuring up to 63mm, appeared to be separate from the thyroid and was recognised as a very large adenoma of what we interpret as a non-descended left inferior parathyroid gland.

Post operative recovery was uneventful, including normalisation of biochemistry. Histology showed a multinodular goitre with an incidentally found 0.2mm papillary thyroid carcinoma and hyperplasia of all three parathyroid glands, including the very large (21g) non-descended left inferior gland.

Discussion: The largest adenoma in this case was many hundreds of times the size of a non-pathological gland. Giant parathyroid adenomata are defined as weighing ≥35g and are not necessarily functional[1]. The location of this gland was characteristic of a non descended inferior gland. These occur in <1% of cases. Coexistent thyroid and parathyroid pathology is not unusual, with rates of synchronous parathyroid and thyroid surgery in patients with PHPT reported in up to 29%[2]. Despite this, we believe this case to be unique in the published literature given the huge goitre, unusual nature of the parathyroid disease and coincidentally found microcarcinoma. However, the primary value of this case lies in the illustration of the difficulties of parathyroid localisation in the presence of a large goitre.

References:

1. Spanheimer PM et al. Do giant parathyroid adenomas represent a distinct clinical entity? Surg (United States). 2013;154(4):714–9.
2. Ryan S et al. Co-existent thyroid disease in patients treated for primary hyperparathyroidism: implications for clinical management. Eur Arch Otorhinolaryngol [Internet]. 2015;272(2):419–23.

W008

An unusual case of Autoimmune Hypoparathyroidism: presenting as Congestive cardiac failure and Myocardial Infacrction

Neelam Khalid, Hadleigh Cuthbert, Adie Viljoen, Peter Winocour, Sagen Zac-Varghese, Kanchana Rajaguru, Lister Hospital, Stevenage

66 year old lady was admitted to the A&E department with shortness of breath and chest tightness. She had a background of hypertension and migraine. There was no significant family history and no past medical history, in particular no previous neck irradiation or surgery. Her admission blood tests revealed low corrected calcium of 1.35 mmol/L. She denied any symptoms of hypocalcemia. Clinical examination was normal, there was no paraethesia, tetany or other neurological findings and she had a negative Trousseau and Chvostek's sign. The lack of signs and symptoms pointed to a chronic cause for the hypocalcaemia. She was of normal height and had normal bone development. She had 2 children and had no problems during pregnancy. Her children did not have any known medical conditions and were of normal height and had developed normally. Her PTH was undetectable at a level of <0.0 pmol/L (repeated three times and discussed with the biochemistry department to exclude assay interference). 25-HydroxyVitamin D3 was sufficient at 63.6 nmol/L.

Electrolytes showed marginally raised phosphate 2.52 mmol/L, normal Magnesium 0.82mmol/L and normal renal function. Her thyroid function tests and serum cortisol were normal. She was commenced on Calcium infusion, 10% calcium gluconate at 50ml/hr and this was titrated according to response. She remained on Calcium infusion for 72 hrs. Following this her adjusted calcium was 2.20 mmol/L. She was commenced on oral Calcium carbonate 1g BD and 0.5 mcg Alpha calcidol daily to maintain her calcium levels. Whole body CT was performed and it ruled out any infiltrative/malignant process.

Her initial ECG showed QT_C prolongation 487 and ST depression in V4-V6. She had a raised troponin and was treated for acute coronary syndrome. An echo showed severe left ventricular systolic dysfunction. An angiogram was performed which showed severe multivessel atherosclerosis and she was referred for a CABG.

Learning point: This was a rare case of likely Autoimmune Hypoparathyroidism, presenting following an acute coronary syndrome. Autoimmune Hypoparathyroidism can occur as two main etiologies, either as an isolated enzyme deficiency of PTH or in connection with other endocrine enzyme deficiencies. In cases of hypoparathyridism, where there is no history of thyroid or neck surgery, it is also important to rule out infiltrative process.

W009

Challenges in concurrent liver and pancreatic insufficiency: glucose control on a tight rope

Andrianni Karra[1], Pei Chia Eng[1], Maria Phylactou[1], Manninder Gill[2], Anna Pullen[3], Lucy Fermor[1], Sanjeev Mehta[1], Nick Oliver[4], Kevin Baynes[1]
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[2]Department of dietetics, Ealing hospital
[3]Department of clinical psychology, Ealing hospital
[4]Department of diabetes and endocrinology, Charing Cross Hospital

Introduction: Control of plasma glucose concentration requires flawless cooperation between the pancreas, liver and the intestine. In the event of diminished pancreatic function, impaired incretin secretion and loss of islet cell insulin and glucagon secretion contribute to brittle diabetes characterized by marked glycaemic lability.

Case and Investigations: We present a case of a 29-year old man who was diagnosed with pancreatic diabetes resulting from chronic alcohol misuse. He had significant weight loss and had extensive investigations for low body mass index. His computed tomography scans of his chest; abdomen and pelvis revealed gross chronic pancreatitis but did not suggest any evidence of malignancy. His tumour markers – CEA and CA19-9 were elevated at 6.6 and 251 suggesting chronic pancreatitis rather than malignancy. Further investigations including an oesophagoduodenoscopy and biopsy excluded gastrointestinal ulcer and malignancy. Hepatitis screen, liver autoantibodies, coeliac screen, alpha -1 antitrypsin, ceruloplasmin, copper and HIV serology have been negative. Despite being treated with a basal bolus regimen consisting of small doses of meal time novorapid and small doses of long acting basal insulin, his capillary blood glucose control had remained erratic ranging from 1 mmol/L to 47 mmol/L. Managing his hypoglycaemic episodes was challenging as these episodes were poorly responsive to glucagon therapy and could only be managed with intravenous dextrose. As he had repeated hypoglycaemic episodes, he developed impaired hypoglycaemic awareness and this eventually led to a severe hypoglycaemic event after which he sustained a basal ganglia infarction. Investigations for hypoglycaemia excluded endogenous insulin secretion as his paired plasma C-peptide, plasma insulin were appropriately suppressed at plasma glucose of 2mmol/L. IGF2 levels were within normal limits and urine sulphonylurea screen was negative. An early morning serum cortisol level is adequate at 819nmol/L.

Discussion: Control of hyperglycaemia and preventing hypoglycaemia remains the primary treatment target in pancreatic diabetes. There are no guidelines regarding the management of such patients. Abstaining from alcohol and smoking cessation reduce toxic insults to the pancreas. Pancreatic enzyme replacement aids fat digestion and provides incretin to maintain glucose tolerance during meal ingestion. Since the endocrine defect is insulin deficiency, most experts recommend multi dose basal bolus regimen including carbohydrate counting for flexible prandial coverage, with consideration of continuous subcutaneous insulin infusion or pump therapy. As the degree of beta cell impairment is seldom absolute, insulin dosing for such patients is challenging as the continued loss of glucagon secretion and replacement

insulin doses may unpredictably predisposes to hypoglycemia. We would like to highlight the complexity in the management of this patient. Our patient has been referred to tertiary center and is being worked up for liver and pancreatic transplant.

W010

Use of a treatment pathway in Primary Hyperparathyroidism to ensure consistent care and appropriate use of Cinacalcet in patients unable to have operative intervention

Mr Joshua Hamby, Mrs Vanessa Hamby (Medical Students- American University of the Caribbean), Dr Steven McCann (Consultant Chemical Pathologist), Dr Richard C. Bell (Consultant Physician) Stepping Hill Hospital- Stockport NHS Foundation Trust

Objectives: To validate the use of a Primary Hyperparathyroidism treatment pathway and shared care management plan used in a secondary care setting.

Methods: We compared the biochemical laboratory data of patients presenting in 2012 and 2015 with an adjusted calcium >2.85mmol/L. These data were reviewed to identify cases of hyperparathyroidism, utilising biochemical and electronic patient record data.

Results: In a population area of around 350,000 we perform approximately 75,000 calcium tests per year. In 2012 and 2015 using an adjusted calcium threshold of >2.85 we identified 270 and 226 unique patients with significant hypercalcaemia. Of these 87 and 67 were determined to have primary hyperparathyroidism. When comparing treatment with parathyroidectomy or cinacalcet we identified a younger cohort treated surgically average age 62 and 65 (2012-2015) as opposed to average age 80 and 83 years (2012-2015) treated with cinacalcet.

Using the presented treatment pathway which biases towards use of cinacalcet if risk factors for admission are present such as falls risk or history of dementia/ delirium. We show consistent use of cinacalcet and surgery (19 cinacalcet and 19 surgery in 2012 vs 10 cinacalcet 21 surgery in 2015).

Conclusions: Although cinacalcet use in routine hyperparathyroidism is relatively new and therefore a growth treatment area. We identify a pathway of treatment that prevents its overuse as a first line treatment. When comparing parathyroidectomy vs cinacalcet treatment we found each treatment use was geared to specific age cohorts which remained consistent in 2012 and 2015.

W011

Beer Potamania- A reversible cause of symptomatic severe hyponatremia

A Stevenson, SA Qureshi, S.G.Wijetilleka, K. Yoganathan, S. Wright, S. Ladbrookes, R.Kaushal

We present a 65 year old male brought in following a fall at home. He had a history of T2DM, COPD and congestive cardiac failure. His drug history included, amongst others, irbesartan and furosemide, although his compliance with medications was low. His plasma sodium on admission was **104 mmol/L** (136mmol-146mmol). He had normal potassium of 5.1 mmol/L (3.5-5.1mmol/L) and his renal function was normal. On examination his GCS was 13/15 and he was clinically hypervolaemic. He had was anaemic with a haemoglobin of 122 (NR 132-171) and an MCV of 110 (NR 80-100) with normal liver function tests. He had a history of alcohol abuse consuming around 20 units/day for the past 20 years. His serum osmolality was 232mosm/kg (275-295mosm/kg) with a urine osmolality of 488mosom/kg (50-1200mosm/kg) and a urinary sodium of 13mmol. His random cortisol was 635nmol/L (185-625nmol/L), he had a normal thyroid function (TSH 2.73, free T4 = 12.3pM) and a normal lipid profile (triglycerides 0.87). Chest X-ray revealed an enlarged heart with upper lobe dilation but no obvious consolidation.

The initial impression was this was hyponatraemia secondary to CCF and was initially with fluid restriction & furosemide was initiated. It was decided that given his comorbidities he was a ward-based ceiling of care only. His sodium failed to improve on this regime and in 48 hours his GCS had dropped to 6/15. He also developed a respiratory acidosis, thought to be due to fluid overload, necessitating NIV. Treatment was then commenced with careful infusion of 1.8% hypertonic saline, checking sodium levels every 6 hours. His sodium gradually increased from 106mmol/L to 128mmol/L over 72 hours. With this his GCS improved, along with his respiratory effort, he was able to be taken off NIV and has been medically stable ever since.

Learning Points:

1. Chronic alcoholic patients are commonly found to be hyponatraemia. In this case one contributory factor was certainly fluid overload secondary to alcoholic cardiomyopathy.
2. In beer potomania, chronic consumption of beer, which is a hypotonic alcoholic solution, combined with protein malnutrition, can lead to profound hyponatraemia.
3. This case illustrates how the careful administration of additional solute in the form of 1.8% hypertonic saline can provide gentle correction in serum sodium leading to the patients neurological recovery, but minimizing the risk of overcorrection and central pontine myelinolysis.

W012

Breast cancer metastases to the thyroid gland – An uncommon sentinel for diffuse metastatic disease

A M Plonczak, A N Di Marco, R Dina, D Gujral, F F Palazzo, Department of Thyroid & Endocrine Surgery, Hammersmith Hospital, Imperial College Healthcare NHS Trust

Aim: To illustrate a rare case of breast metastases to the thyroid gland, present a literature review and discuss its clinical significance

Background: Metastases to the thyroid are rare. The most common cancer to metastasize to the thyroid is the kidney, followed by gastrointestinal tract tumours, lung, skin and only rarely the breast. Outcomes in malignancies metastatic to the thyroid overall are poor. There are no prospective studies addressing the role of surgery in metastatic disease of the thyroid. Isolated thyroidectomy has been proposed as a local disease control option to palliate and prevent the potential morbidity of tumour extension related to the airway.

Case description: A 62 year old female was diagnosed with bilateral carcinoma of the breast in 2004, for which she underwent bilateral mastectomy. The pathology revealed multifocal disease on the right: grade (G)1+ G2 IDC 0/20 lymph nodes (LN) and G1 IDC + DCIS N1 2/18 LN left-sided disease. Surgery was followed by adjuvant chemotherapy and regional radiotherapy. The disease was under control on predominantly hormonal therapy until 2016, when the patient developed cervical lymphadenopathy. The fine needle aspiration (FNA) cytology of the thyroid was reported as papillary thyroid cancer; however, the biopsy of the left lateral nodal disease was more suggestive of breast malignancy. The patient underwent a total thyroidectomy and a clearance of the central compartment lymph nodes. The histopathological analysis was consistent with metastatic breast cancer, with no evidence of a primary thyroid malignancy.

Conclusion & Clinical Significance: In conclusion, a past history of a malignancy elsewhere should raise the index of suspicion of metastatic disease in patients presenting with a thyroid lump with or without cervical lymphadenopathy. If confirmed on FNAB it is usually a poor prognostic sign and indicative of disease beyond cure. The empirical thyroidectomy can be considered in select patients for local disease control.

W013

Large Undescended Inferior Parathyroid Adenoma Masquerading as part of Retropharyngeal Large Multinodular Goitre

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The Case: A 71 year old female with asymptomatic biochemically proven primary hyperparathyroidism and compressive symptoms from a very large toxic multinodular goitre is presented. Examination revealed a very large goitre with distension of the external jugular veins suggestive of thoracic inlet compression. Localisation studies in the form of ultrasound neck and SestaMIBI failed to identify a parathyroid adenoma. CT of the neck and upper thorax noted the left thyroid lobe to be larger with a significant retropharyngeal component, extrathoracic tracheal compromise and minor retrosternal extension.

A combined total thyroidectomy and parathyroidectomy via a cervical approach was performed. The right thyroid lobe was mobilised first, during which an enlarged right superior parathyroid gland was identified. The left lobe of the thyroid was then mobilised and a classically positioned but abnormally large left superior parathyroid gland was found and safely removed. Subsequently, during mobilisation of the highly developed superior pole of the left thyroid lobe, a separate retropharyngeal structure, superomedial to the lobe, was encountered and mobilised. This structure, measuring up to 63mm, appeared to be separate from the thyroid and was recognised as a very large adenoma of what we interpret as a non-descended left inferior parathyroid gland.

Post operative recovery was uneventful, including normalisation of biochemistry. Histology showed a multinodular goitre with an incidentally found 0.2mm papillary thyroid carcinoma and hyperplasia of all three parathyroid glands, including the very large (21g) non-descended left inferior gland.

Discussion: The largest adenoma in this case was many hundreds of times the size of a non-pathological gland. Giant parathyroid adenomata are defined as weighing ≥35g and are not necessarily functional[1]. The location of this gland was characteristic of a non descended inferior gland. These occur in <1% of cases. Coexistent thyroid and parathyroid pathology is not unusual, with rates of synchronous parathyroid and thyroid surgery in patients with PHPT reported in up to 29%[2]. Despite this, we believe this case to be unique in the published literature given the huge goitre, unusual nature of the parathyroid disease and coincidentally found microcarcinoma. However, the primary value of this case lies in the illustration of the difficulties of parathyroid localisation in the presence of a large goitre.

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W014

Thoracic Surgery for Persistent Hyperparathyroidism due to Ectopically Located Parathyroid Tissue

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Failure to cure hyperparathyroidism (HPT) at the initial operation has an estimated incidence in the UK of 5.2%, rising to 15.4% nationally for re-operations.[1] Ectopic glands including thoracic parathyroids are amongst the less common causes of failure to cure at first time surgery.[2] The successful management of persistent and recurrent HPT requires close collaboration between the endocrinologist, radiologist and surgeon and with this, cure rates close to those for primary surgery may be achieved.[3] However, as illustrated by this case series, ectopic thoracic disease presents particular challenges. 8 patients are presented who underwent re-operative parathyroid surgery for ectopic parathyroid glands in the thorax. One patient had renal HPT and all others, primary HPT with one MEN-related and the other 7 sporadic. Median age at reoperation was 43 years (range 18-65). The number of previous unsuccessful procedures was 1 in 6 cases, 2 in 1 case and 3 in 1 patient. The strategy in all cases was to (1)reconfirm the diagnosis with repetition of the biochemistry and confirm existence of an indication to intervene, (2)establish the details of previous intervention (based on operating notes and histology) (3)perform further selected imaging and (4)plan surgery with appropriate cardiothoracic surgeon back up where required. The ectopically located gland was identified on NM imaging in 3 cases, 1 with concordant USS and 2 with concordant CT/MRI and 5 patients had diagnostic venous sampling, 3 with concordant CT/MRI. The surgical approach was via median sternotomy in 4 cases, manubrial split in 2 and thoracoscopy in 2. Intraoperative PTH was used in all patients bar one, whose operation predated the availability of this technology. Intermittent monitoring of the recurrent laryngeal nerve was used selectively (in 4 cases). A single abnormal gland was removed in all cases except one in whom a full mediastinal clearance was performed (based on venous sampling) with no parathyroid tissue detected histologically. In this patient the calcium normalized, as did PTH initially, before rising to 8.8pmol/l by 4 months (in the context of a corrected calcium of 2.48mmol/l and vitamin D of 47.8nmol/l). Histology confirmed abnormal parathyroid tissue in all but the case above. A further equivocal result was seen in the renal HPT case, where the PTH decreased by over 50% intraoperatively but remained elevated at long term follow up with eucalcaemia. 6 patients had definite biochemical cure, although 2 had not yet reached 6 month follow-up and 2 were rendered predictably or intentionally hypoparathyroid (3 gland excision in one patient and subtotal excision for MEN in the other at previous surgery). This small series illustrates the challenges in re-interventions for persistent HPT due to thoracic disease which include: (1) the difficulty in achieving accurate pre-operative anatomical localization, (2) the intraoperative localization of a small gland in a large fat filled cavity and (3) the decision to induce hypoparathyroidism as the only way to achieve cure in some cases – something that the arrival of recombinant PTH may avert.

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W015

Something to chew on: a rare cause of hypercalcaemia

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A 71 year old gentleman with a background of ameloblastoma of the mandible, primary hypothyroidism, bipolar affective disorder and chronic kidney disease was referred from the psychiatry team with a corrected calcium of 3.09 mmol/L (normal range 2.20-2.60). He reported a three week history of constipation and loss of appetite along with unintentional weight loss over the preceding few months. He was on Levothyroxine 100micrograms daily and had previously been treated with Lithium carbonate for his bipolar affective disorder, which was stopped many years prior to admission. He was diagnosed with mandibular ameloblastoma in 2009 following a left sided mandibular biopsy but declined surgery at the time.

Further investigation of his hypercalcaemia revealed an appropriately suppressed level of Parathyroid hormone (PTH) at 0.9 pmol/L (normal range 1.6-6.9) and slightly low Vitamin D of 44nmol/L (51-163). He was biochemically euthyroid with a TSH of 2 mIU/L (normal range 0.27-4.20) and free T4 17.26 pmol/L (normal range 12-22). His 9 am Cortisol was satisfactory at 406 nmol/L and his serum angiotensin converting enzyme was also normal at 27 U/L (normal range 8-52). There was no evidence of myeloma as evidenced by normal serum immunoglobulins and urine electrophoresis. The result of his serum PTH-related peptide level is awaited.

A scan of the patient's chest, abdomen and pelvis revealed the already known lesion on the left mandible, mediastinal lymph nodes, adenomatous adrenals and small calculi within the bladder; but no evidence of a solid malignancy. Subsequent imaging of the neck further characterised the left mandibular lesion as partly solid and partly cystic, measuring 7.2cm by 6 by 4.6 cm, with evidence of mass effect. In addition there was a 1.5cm lytic lesion on the right mandible, associated with the root of the molar. In light of the above the case was discussed at the Head and Neck MDT at the Royal Marsden Hospital who advised intervention with a left hemimandibulectomy and simultaneous encapsulation of the right lytic lesion, which was likely to be a radicular cyst of the lower right sixth tooth.

Ameloblastoma is a very rare, benign tumour that develops most often in the jaw near the molars. It can be very aggressive, growing into the jawbone and causing swelling and pain. Malignant transformation is rare as are metastases, but they do occur, and both may be associated with hypercalcaemia. Here we present a case of hypercalcaemia in a gentleman with known ameloblastoma of the mandible, in whom all the common causes of hypercalcaemia have been excluded, raising the possibility that the new hypercalcaemia signifies malignant transformation of his previously benign ameloblastoma.

W016

Hypercalcaemia in a patient on Lithium therapy

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Case history: We present an interesting case of 64 years old lady who was recently referred by GP to our endocrine clinic for management of hypercalcemia. She has past medical history of Asperger's syndrome, learning difficulties and bipolar disorder. She has no family history of hypercalcemia or nephrocalcinois. She has been on Lithium since 2011. She is asymptomatic apart from vague abdominal pain and constipation for which she has seen a gastroenterologist.

Hypercalcemia workup revealed corrected calcium ranging between 2.73-2.88mmol/L (normal-2.1-2.6) ,PTH-9.3 pmol/L(normal 1.5-7.6), VitD-49nmol/L(normal 75-200), ALP-69 IU/L(normal 30-130) , eGFR-55 ml/min(normal>60). Patient could not manage 24 hour urine collection due to learning difficulties. She never had bone profile tested predating Lithium start.

CT whole body arranged by gastroenterologist excluded malignancy, sarcoid or kidney stones. US neck however has revealed left-sided parathyroid adenoma. DEXA scan is pending. She is on Vitamin D replacement now.

The possible aetiology of hypercalcaemia in our case is thought to be PHPT or Lithium induced.

Discussion: The prevalence of LAH is estimated to range from 4.3-6.7% and is higher than overall prevalence of HPT. It is still unclear whether lithium initiates HPT or promotes an underlying subclinical state of HPT. It was shown that lithium causes a shift in the inhibitory set point for PTH secretion to a higher serum calcium concentration. It is believed to either unmask or accelerate previously unnoticed hyperparathyroidism. We review the available literature and evidence to understand the pathophysiology.Of this poorly understood endocrine disorder and also keen to learn from experiences in other centres re management.

Question to Panel: Should she procced with parathyroidectomy or managed conservatively?

References:

1) Lithium-associated hyperparathyroidism: report of four cases And review of the literature. Auryan,Szalat1, Haggi Mazeh2 and Herbert R Freund2
2) Lithium Associated Hyperparathyroidism: An Evidence Based Surgical Approach Umashankar K. Ballehaninna1,2, Steven M. Nguyen3, Ronald S. Chamberlain1,3,4

W017

IgG4 related hypophysitis-a diagnostic challenge

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Introduction: Isolated IgG4 related hypophysitis is a rare condition. Headache, visual field defects, pituitary hormones insufficiency are the main clinical features. There a few diagnostic challenges as MRI findings might mimic other conditions. Pituitary biopsy is the gold standard investigation.

Case Presentation: A 34 year old lady presented with headache, blurred vision. MRI brain revealed pituitary macroadenoma (19x15x11mm elevating the optic chiasm). She has been on Progesterone only pill for some years so didn't have regular periods. She had 2 children with no further plans of pregnancy. Biochemical workup revealed Prolactin 1076 milliunit/L, Cortisol 50 nmol/L, TSH-0.22 milliunit/L, FT4-4.4 pmol/L, LH <0.5IU/L, FSH-1.7 IU/L, Oestradiol <70 pmol/L. RA factor, complements normal, ANA, ANCA negative, IgG4 1.53g/L (<1.3), chest x-ray was normal. She was commenced on Hydrocortisone and Levothyroxine. Has complained of polyuria and polydipsia. Water deprivation test confirmed Cranial Diabetes Insipidus, she was commenced on Desmopressin.

She underwent Pituitary biopsy as per the recommendation of pituitary MDT. Histopathology of the specimen revealed initial diagnosis of Lymphocytic Hypophysitis. Further immunostaining revealed IgG4 related disease. She has been commenced on Prednisolone 60 mg OD with gradual down titration to a maintenance dose. Has been referred to a specialist with an interest in IgG4 related disease for the consideration of immunotherapy. Repeat MRI Pituitary has revealed reduction the size of the pituitary macroadenoma.

Conclusion: IgG4 related disease commonly presents with multisystem involvement. Literature review reveals only a few cases of isolated IgG4 hypophysitis. Misdiagnosis can be avoided by a careful and dedicated cytological analysis.

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Revisitation of autoimmune hypophysitis: knowledge and uncertainties on pathophysiological and clinical aspects. Pituitary 2016, Aug 8

W018

Neuroendocrine tumour diagnosed in pregnancy, the challenges of complex disease

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The appropriate diagnosis and treatment of neuroendocrine tumours often involves collaboration between specialists in multiple disciplines, using specific biochemical, histological and surgical methods. The 28 years old lady presented with epigastric abdominal pain at 16 weeks of pregnancy. She was found to have pancreatic head lesion of 4.5 cm. She had EUS, showed 4 cm solid lesion at head of pancreas adjacent to portal vein and SMV without obvious invasion, subsequently FNA was positive for chromogranin and synaptophysin, consistnet with low grade Neuroendocrine tumour (NET). The obstetrician were very keen for her to get to 30 to 32 weeks with steroid cover before delivery, therefore it was discussed in NET MDT to monitor her over next few weeks of her pregnancy. She had elective caesarean at 30 weeks of gestation. After delivery, CT scan showed a large tumour, which appeared to involve the SMV and octreotide scan showed localised disease only. She was admitted 2 weeks after delivery for proposed elective Whipple's procedure. The laparotomy revealed no evidence of metastases, but tumour extended posterior to the superior mesenteric artery. As artery was involved it was not possible to resect the tumour. The histology of lymph node showed an NET Ki-67 2 to 3 % with negative hormonal markers. She was referred for consideration of radiotherapy and chemotherapy. Her 4.5cm non functioning pancreatic neuroendocrine tumour reduced to 2.6 cm post pregnancy prior to chemotherapy but it was mainly due to reduction of cystic component. She did have a course of chemotherapy with 5-FU and Streptozocin but did not complete her cycles. She decided to have Nanoknife therapy to pancreatic neuroendocrine privately. Her pain improved after Nanoknife therapy. She was started on somatostatin analogue. It was decided against surgery in the MDT meeting because of possible complication associated with surgery and disease was stable on somatostatin analogue. The patient decided to have surgery at Heidelberg. She had Whipple's resection and then partial gastrectomy, because of gastric ischaemia, it was further complicated by ascites and chyle fistula. The histology showed complete resection of the tumour with clear margins, Ki 67 was 10 % but there was some lymphovascular invasion and 1/32 lymph node positive, T3N1V1pN1R0. She developed diarrhoea and weight loss after extensive surgery. She was also diagnosed with bile salt malabsoption. The repeat gallium DOTATATE PET CT scan did show no focal tracer activity but showed moderate ascites of disease. She currently has significant diarrhoea and difficult gaining weight.

The primary treatment goal for patients with neuroendocrine tumours is curative with symptoms control and limitation of progression of disease. Surgery is a possible curative approach but can lead to significant morbidities, because of tumour location. Chemotherapy and Somatostatin analogue can also limit the progress of disease when tumour is inoperable and disease remains stable but it needs multi disciplinary team input and regular imaging to see progress of disease.

W019

The long search for an occult ectopic ACTH-producing tumour

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Ectopic adrenocorticotropic hormone (ACTH) production accounts for 10-20% of all endogeneous Cushing's syndrome. The commonest tumour types are those with neuroendocrine features, such as carcinoid tumours; bronchial carcinoid accounts for 10%. Such cases are challenging and may require repeat investigations over many years. The ideal treatment is curative surgery of the underlying tumour. In difficult cases, such as non-operable or non-localised tumours, bilateral adrenalectomy with glucocorticoid and mineralocorticoid replacement 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. At 16 years post adrenalectomy, two right lung nodules became apparent on subsequent surveillance imaging. One nodule was identified in the upper part of the right lower lobe and another central nodule close to the bronchus. 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. 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 bronchial typical carcinoids measuring 24mm and 13mm 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. Since surgery, her tan significantly decreased. ACTH fell from a preoperative level of 1465ng/L to 12ng/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. These tumours are best managed in a multidisciplinary setting involving the endocrinologists, endocrine surgeon, radiologist and pathologist.

W020

Case Report: The Curious Case of Misleading Cortisol

Luke Flower, Sogha Khawari, Imperial College Healthcare NHS Trust

A 73 year old female presented to our endocrinology ward having been admitted with hyperglycaemia and ketosis, discovered when attending hospital for a PET scan to investigate a right-sided adrenal mass.

A Short Synacthen Test was performed prior to this admission, following initial discovery of adrenal mass, demonstrating a consistently raised cortisol. This, in combination with her poorly controlled diabetes, was suggestive of a cushingoid picture.

On admission to our hospital, she was found to have a raised cortisol of 667. Her past medical history included a known right adrenal mass with IVC thrombus, osteoporosis and previous rectal prolapse repair.

During this admission, a large mass was found on CXR in the mid-zone of her left lung. After CT and lung MDT, it was concluded that the mass was likely a primary lung cancer in the lower left lobe and the right adrenal abnormality represents a metastatic deposit. It was thought that a lung malignancy may have been producing ectopic ACTH and resulting in a cushingoid picture. ACTH immunohistochemistry was performed on the lung mass biopsy, however this was negative.

A low dose dexamethasone suppression test was performed to help confirm a diagnosis of Cushing's syndrome. The results were: initial ACTH 59.8 and cortisol 537, at 24 hours ACTH 8.0 and cortisol 75, at 48 hours ACTH 6.3 and cortisol 56. This demonstrated a suppressed cortisol, in keeping with a normal physiological response - making Cushing's syndrome unlikely.

In conclusion, following an initial raised morning cortisol and an adrenal mass there was a high suspicion of Cushing's syndrome. Following more specific investigations it was demonstrated that this cortisol may have been a misleading result and this diagnosis was in fact unlikely. In retrospect the raised cortisol may have been stress related due to illness, multiple recent hospital admissions and her new diagnoses. This highlights the clinical importance of utilising specific investigations and keeping an open diagnostic mind.

W021

Palpitations and the Pituitary – the Case of Assay Interference

S G Wijetilleka, S Qureshi, R Kaushal

We would like to present a pituitary incidentoloma which was initially thought to be a TSH-oma. We reviewed a 53 year old Indian male with no past medical history who presented to his GP with palpitations in December 2015. He reported weight loss and six loose stools per day. He attended endocrine clinic in Jan 2016.

- Post-clinic bloods revealed TSH of 2.13, T4 of 21.3, T3 – 5.4, TSH receptor antibody of 0.5, FBC and U&E were normal.
- USS thyroid revealed a small nodule in the left thyroid (3.7mm), otherwise normal left and right lobes. The patient was sent for an NM thyroid scan which was a normal study with no evidence of a toxic adenoma.
- He was started on carbimazole 20mg od and propranolol 10mg tds prn.

He returned to endocrine clinic in April 2016 and was clinically euthyroid – TSH was 4.89, T4 was 27.5 and T3 was 5.7. His FBC, UE and LFTs were normal. 9am cortisol was 143, FSH 7.4, LH 4.5, PRL 114, IGF1 34,testosterone 8.7 and his SHBG was 31.6 (FAI 27.5%).

We performed a short synacthen test which was within normal limits and invited this gentleman for a pituitary MRI in May 2016.

A pituitary mass was seen on MRI in May 2016; a 4mm cyst was visualised behind the infundibulum.

He was discussed in the pituitary MDT at Imperial College in July 2016. He was asked to attend Imperial for further bloods, namely thyroid function tests with different assays. Conditional to this he was invited for a TRH test and a T3 stimulation test and an alphasubunit radio was sent.

Repeating bloods at Imperial College led to his TSH, T4 and T3 being within normal limits. As our patient was clinically euthyroid now and remains completely asymptomatic on further clinical review.

Save the date for 12th Hammersmith Multidisciplinary Endocrine Symposium:

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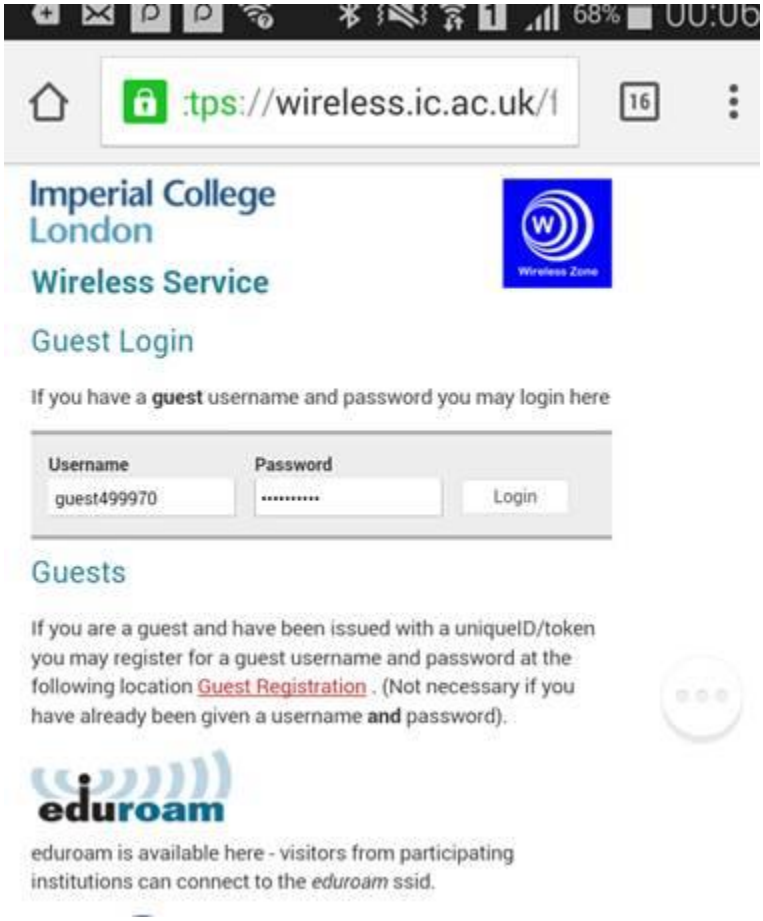

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