Hammersmith Abstracts

14th Hammersmith Multidisciplinary Endocrine Symposium 2019

Friday 6th December 2019

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Hammersmith Hospital 14th Multidisciplinary Endocrine Symposium
Provisional programme Fri 6th Dec 2019

Wolfson Conference Centre, Hammersmith Hospital, London

CPD approved for 6h code 126823

8.30am Registration & Coffee

8.55am Welcome and Introduction
(Fausto Palazzo, Karim Meeran & Waljit Dhillo)

Session 1: Hyperaldosteronism (Chair James Ahlquist)

9.00am Hyperaldosteronism: making the right diagnosis
(and getting the correct side)
Prof Mark Gurnell (Cambridge University)

9.30am Adrenal vein sampling for Conn's syndrome – is it necessary?
Prof Karim Meeran (Imperial College)

10.00am *International Guest Lecture 1*
Conn's Syndrome: Are we operating on enough patients & are we doing it correctly?
Prof Menno Vriens, Utrecht, Holland.

10.30pm Clinical Case 1 (Z032) A Surgical Treatment for Cardiomyopathy.
Anneke Graf, UCLH Foundation Trust.

10.50am Coffee Break

Session 2: Multidisciplinary Endocrine Surgery (Chair Prof Tricia Tan)

11.20am Endocrine tumours in the chest and how to remove them.
(including thoracoscopic PTX and ectopic ACTH removal).
Mr John Anderson, Hammersmith Hospital.

11.50am Parathyroid carcinoma
Mr Fausto Palazzo, Hammersmith Hospital

12.20pm Clinical case 2: (Z011) A rare case of multi-organ failure due to acute Parathyroid crisis, **Beenish Inayat**, Great Western Hospital NHS Foundation Trust, Swindon.

12.40pm Clinical Case 3: (Z025) A Case Report of Concomitant Diagnosis of Multiple Myeloma and Primary Hyperparathyroidism.
Eithar Deyab, North Middlesex Hospital.

12.50pm Lunch & Poster session

12.50pm Lunch & Poster session

Session 3: <u>Phaeochromocytoma</u> (chair Miss Aimee di Marco)

1.50pm Why and how to block phaeochromytoma patients for surgery
Dr Hilary Bridge (Oxford)

2.10pm *International Guest Lecture 2*
Breaking news in Phaeochromocytoma surgery!
Prof Martin Walz, (Essen Germany)

2.50pm Coffee break

Session 4: Primary Hyperparathyroidism (Chair Dr Emma Hatfield)

3.10pm The NICE guidelines for pHPT: what should we be doing?
Prof Neil Gittoes (Birmingham)

3.40pm Imaging in first time parathyroidectomy: Help or hindrance?
Mr David Scott-Coombes (Cardiff)

4.00pm New frontiers in imaging in re-operative parathyroidectomy
Tara Barwick (Charing Cross Hospital)

4.20pm Clinical cases **(Chair James Ahlquist)**

4.20pm Clinical Case 4: Z029: A case of possible non-secretory metastatic phaeochromocytoma
Zeenat Banu, Royal Free Hospital.

4.35pm Clinical Case 5: Z012: Bilateral adrenal incidentalomas, unilateral Conn's and subclinical Cushing's syndromes
Tessa Glyn, Natasha Thorogood, University Hospital Bristol

5.00pm Close and Feedback

Z001	Phaeochromocytoma/paragangliomas in patients with Neurofibromatosis-1: time to re-visit the clinical practice guidelines?
Z002	Lithium induced endocrinopathy; a reversible entity!
Z003	Idiopathic Isolated ACTH deficiency- How much do we know?
Z004	Constrictive pericarditis in Primary Hyperparathyroidism: An unusual presentation
Z005	Inpatient pheo crisis in neurofibromatosis type1 (NF 1) "Triggers and management", role of magnesium sulphate in management of pheo crisis
Z006	I would love to remove my head = pituitary apoplexy
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Z008	Regional audit into the indeterminate thy3f cytology thyroid nodules and their resulting histological malignancy rate at Luton & Dunstable Hospital and Lister Hospital
Z009	A giant cystic pheochromocytoma presenting with myocardial infraction - a case report
Z010	Case Report: A Striking presentation of a Rare Invasive and Aggressive co-secreting Growth Hormone and ACTH Pituitary Macroadenoma
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Z012	Bilateral adrenal incidentalomas, unilateral Conn’s and subclinical Cushing’s syndromes
Z013	Immune-Mediated Endocrinopathies
Z014	Lessons from prednisolone day curves in adrenal insufficiency: Can we tailor anti-inflammatory steroid treatment based on serum prednisolone levels?
Z015	Pre-operative diagnosis of Parathyroid Cancer: a single centre experience

Z016	A case of refractory hypercalcemia. Elusive diagnosis and difficult management
Z017	The use of computed Tomography as a first-line imaging modality in patients with primary Hyperparathyroidism
Z018	Identification and Management of Familial Hypocalciuric Hypercalcemia Type 3 in a District General Hospital
Z019	Does α and β blockade in patients undergoing adrenalectomy for phaeochromocytoma increase postoperative morbidity? - An Institutional analysis
Z020	Case of Grave's ophthalmopathy complicated by ocular myasthenia gravis
Z021	Genetic testing in patients with primary hyperparathyroidism: when is screening worthwhile?
Z022	Myxoedema coma with severe hypoxia
Z023	Persistent hypertension post adrenal resection in a patient with surgically managed Conn's Syndrome
Z024	Paraganglioma Crisis: the challenges of a silent neuroendocrine neoplasm
Z025	A Case Report of Concomitant Diagnosis of Multiple Myeloma and Primary Hyperparathyroidism
Z026	Phaeochromocytoma presenting as an acute coronary syndrome
Z027	An Addisonian Crisis following recent Diagnosis and Treatment for Tuberculosis
Z028	Synchronous papillary (pT1a(m)N0) and medullary (pTxN1b) thyroid cancer – a diagnostic pitfall
Z029	Case of possible non-secretory metastatic Phaeochromocytoma
Z030	An unusual case of SIADH in patient with HTLV-1 positive infection

Z031	Investigating hypokalaemia – don’t be Conn’d
Z032	A Surgical Treatment for Cardiomyopathy
Z033	Graves thyrotoxicosis presenting late in pregnancy: Additional challenges, considerations and multidisciplinary teammates
Z034	Development of primary hyperparathyroidism after cardiothoracic surgery: a case of ectopic mediastinal parathyroid adenoma
Z035	Management strategies for TSH secreting pituitary adenomas (TSHomas)
Z036	The “Jekyll and Hyde” of Cabergoline therapy
Z037	
Z038	

Z001

Phaeochromocytoma/paragangliomas in patients with Neurofibromatosis-1: time to re-visit the clinical practice guidelines?

A Al-Sharefi[1], U Javaid[1], P Perros[1], J Ealing[2,3], P Truran[4], S Nag[5], S Kamaruddin[6], K Abouglila[6], F Cains[3], L Lewis[3], R A James[1]

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Introduction: Patients with neurofibromatosis type 1 (NF1) are at risk of developing phaeochromocytomas/paragangliomas (PHAEO/PG). Unlike in other familial PHAEO/PG syndromes, there are no published guidelines regarding screening in asymptomatic or normotensive patients with NF1. This strategy may be associated with preventable morbidities in those patients who ultimately present with symptomatic PHAEO/PG.

Objective: To describe the mode of presentation and the incidence of adverse clinical outcomes attributed to PHAEO/PG in NF1.

Methods: A retrospective study was performed in a tertiary referral centre in Northern England in collaboration with the national complex NF1 centre. Hospital records and databases between 1998–2018 were searched.

Results: Twenty-seven patients with NF1 and PHAEO/PG were identified. In all but one, PHAEO/PG was diagnosed after NF1. The median age at the time of diagnosis of PHAEO/PG was 43 years (range 22–65) and 21/27 (78%) were females. The diagnosis was mostly incidental in 13/27 (48%) while classical PHAEO/PG symptoms were found in 15/27 (56%), and hypertension was found in 14/27 (52%) of NF1 patients prior to PHAEO/PG diagnosis. No patient had undergone biochemical screening for PHAEO/PG. Metastatic disease was evident in 2/27 patients, 8 suffered potentially avoidable complications attributed to PHAEO/PG (including two deaths).

Conclusion: The course of PHAEO/PG in NF1 is associated with an unpredictable presentation and potentially avoidable adverse outcomes. We recommend that routine biochemical screening for PHAEO/PG should be part of the care package offered to all patients with NF1 by regular measurements of plasma free or urinary fractionated metanephrines starting from early adolescence and repeated every 3 years.

Z002

Lithium induced endocrinopathy; a reversible entity!

A Ali, M Modi, S Qureshi, West Middlesex University Hospital, London, United Kingdom

Abstract:
Lithium, commonly used as a mood stabiliser in bipolar affective disorder, is associated with an increased risk of hyperparathyroidism and nephrogenic diabetes insipidus. The consequent hypercalcaemia and severe dehydration can prove to be life threatening. Prompt identification and management is therefore essential in limiting the adverse effects of these endocrinopathies, particularly in the context of frail and co-morbid patients.

Case: An 85-year-old lady presented to West Middlesex Hospital with worsening confusion and polyuria. She had a known history of bipolar disorder treated with lithium for the past 9 years, and recent hospital admissions for treatment of a severe community acquired pneumonia and bilateral pulmonary emboli. She was found to be hypercalcaemic (2.90 mmol/L) and clinically dehydrated. Initial screening blood tests revealed hyperparathyroidism, with a parathyroid hormone level of 12 pmol/L. Despite intravenous rehydration and improvement in her calcium levels, she developed worsening hypernatremia, hypotension and declining renal function. The hypernatremia was likely exacerbated iatrogenically by intravenous sodium chloride administration. Upon catheterisation, it was noted that she was passing large volumes of urine, with a significant negative fluid balance despite intravenous fluid therapy. The suspicion of nephrogenic diabetes insipidus was raised given long-term use of lithium, with raised serum osmolality of 350 mmol/L and relatively dilute urine with osmolality of 275 mmol/L. Following careful liaison with the psychiatry team, lithium therapy was gradually weaned down and stopped, with close monitoring of the patient's mental state and trial of an alternate mood stabiliser. Her urine output gradually fell, her calcium levels normalised, and her confusion improved significantly. No further investigation of her hyperparathyroidism was deemed appropriate given her multiple co-morbidities.

Discussion: This case highlights that prompt identification and management of lithium induced hyperparathyroidism and nephrogenic diabetes insipidus can prevent clinical deterioration and death from severe dehydration. A high index of suspicion needs to be had to detect these endocrinopathies in patients on long term lithium therapy. There is understandable concern regarding withdrawal of mood stabilising medication due to risk of relapse in mental health, but by working closely with the psychiatry team, this is a sensible management option.

Z003

Idiopathic Isolated ACTH deficiency - How much do we know?

Y Chok, M Martineau, West Middlesex University Hospital.

Introduction: Idiopathic isolated ACTH deficiency (IIAD) is a rare cause of adrenal insufficiency, which was first described by Steinberg in 1954. 300 cases have been described in the literature, however due to the lack of uniformity in the diagnostic criteria, the prevalence of IIAD is difficult to be established. Here we describe a patient with IIAD after an acute illness. He has been followed up for 4 years.

Case presentation: A 42 years old flight attendant who had no known medical illness was first hospitalised in Kuala Lumpur for gastroenteritis with pre-renal impairment and hyponatremia. He presented with headache, nausea, vomiting, abdominal pain and diarrhoea. In Singapore, a diagnosis of Legionella pneumonia was made. A short synacthen test performed for investigation of hyponatremia (Na 123 mmol/L) showed inadequate response (0 min: 11nmol/L, 30 mins: 56nmol/L and 60 mins: 71nmol/L) and patient was commenced on prednisolone. Thyroid function test was normal, HIV antigen and antibody test were negative. Tests for serum ACTH, FSH, LH, testosterone, IGF-1, and prolactin were not performed during the acute event.

We first met him 2 months after his acute illness. Prior to this incident he had been fit and well with stable weight, absence of excessive lethargy or increased skin pigmentation. A repeated Short Synacthen test 6 months after acute illness showed ACTH 5.6ng/L, baseline cortisol at 0 min is 10 mmol/L, cortisol at 30 mins is 27 mmol/L and cortisol at 60 mins is 28 mmol/L. The rest of the pituitary hormones was normal at 6 months post-acute illness. MRI of the pituitary 7 months post-acute event was normal. Aldosterone and plasma renin aldosterone ratio are normal. These results were consistent with the diagnosis of isolated ACTH deficiency.

We followed the patient up with 6 monthly clinic appointments, regular biochemistry tests and thyroid function tests. He remained fit and well on Prednisolone 3mg once a day. During a recent clinic visit, 4 years after patient’s acute illness, pituitary hormone function tests and short synacthen test were repeated. Apart from ACTH, pituitary hormone function tests were normal. SST revealed inadequate response at both 30 and 60 minutes, further confirming the diagnosis of Isolated ACTH deficiency.

Discussion: In adults, isolated ACTH deficiency (IAD) has been described after discontinuation of glucocorticoid therapy, lymphocytic hypophysitis, traumatic brain injury, hypothyroidism, and post radiotherapy. Cases of Nivolumab induced IAD has been increasingly reported with the increase usage of checkpoint inhibitors over the last few years. IIAD is a diagnosis of exclusion.

The diagnosis of IIAD can be easily missed as it often presents insidiously. Hyponatremia can frequently be the only abnormality on biochemistry testing, and they are usually euvolaemic. Case series have revealed a close association of IIAD and autoimmune diseases, in particular with autoimmune hypothyroidism. Other

autoimmune diseases associated with IIAD have also been reported, e.g. Graves disease, Crohn's disease and Type 1 Diabetes. The high prevalence of autoimmune diseases in IIAD would suggest that at least some of the IIAD cases might be autoimmune in origin.

The natural history of IIAD is not well elucidated. 2 cases of complete recovery of hypothalamic pituitary adrenal axis in IIAD has been described in the literature. Initial IIAD can also progress to more extensive hypopituitarism. Consequently, it is advisable to retest pituitary hormones to ensure the integrity of all pituitary axes and also to reassess the need to continue glucocorticoid.

With regards to corticotrophin stimulation test, conflicting reports regarding its response has been described in the medical literature. This might suggest that IIAD is a heterogeneous disorder which can be caused by either pituitary ACTH deficiency or a hypothalamic Corticotrophin-releasing hormone deficiency.

Z004

Constrictive pericarditis in Primary Hyperparathyroidism: An unusual presentation

A Iqbal, B Inayat, N Haya, The Great Western Hospital NHS Trust Foundation Swindon

Abstract: The classic symptoms of primary hyperparathyroidism have evolved considerably and ranges from no symptoms to minimal symptoms. We present a case of 81 years old man who presented with signs & symptoms of heart failure (pedal oedema, shortness of breath). Examination revealed elevated JVP, pulses paradoxus, pericardial knock, decreased breath sounds and pedal oedema. Chest x-ray showed right pleural effusion & pericardial calcification. Laboratory investigations showed hypercalcemia with low serum phosphate level and raised PTH level. CT scan of the neck, parathyroid sestamibi scan findings were consistent with parathyroid adenoma. Echocardiography, CT thorax showed constrictive pericarditis due to pericardial calcification and bilateral pleural effusion. Patient was treated with pamidronate for hypercalcemia and diuretics for heart failure. On his second presentation with high calcium level, pamidronate, cinacalcet and alendronic acid were given. On third admission with heart failure, patient was treated with diuretics and had upper normal calcium.

Hyperparathyroidism is associated with multiple clinical and biochemical abnormalities, but its role in heart disease is not clear. There is strong evidence that these patients have increased risk of death due to cardiovascular disease. Hyperparathyroidism can cause hypertension, left ventricular hypertrophy, coronary artery disease, valvular calcification, myocardial calcific deposits and arrhythmias. Although risk of constrictive pericarditis in end stage renal disease induced hypercalcemia is known in few case reports. We present a case of patient who developed constrictive pericarditis secondary to primary hyperparathyroidism, as we feel it is important to highlight the possibility of constrictive pericarditis in these cases.

All other causes of constrictive pericarditis were considered, detailed history for various causes, clinical signs and number of tests were done to exclude other causes.

This patient with complex presentation was managed by multidisciplinary team including endocrinologist, cardiologist and ENT surgeon. His symptoms of heart failure improved with medical therapy. Multidisciplinary cardiology meeting decided that he will not be a candidate for pericardectomy or pericardial stripping. After multidisciplinary meeting with ENT and Endocrine surgeons, it was decided that in a view of his constrictive pericarditis patient will not be a good candidate for surgical intervention.

Z005

Inpatient phaeo crisis in neurofibromatosis type1 (NF 1) "Triggers and management", role of magnesium sulphate in management of phaeo crisis

R Eltayeb, S Bitat, B Khoo, E Karra, S Ali, Royal Free Hospital

Background: Phaeochromocytomas are catecholamine-secreting tumours arising from chromaffin cells in the adrenal medulla. Phaeochromocytoma crisis (PCC) is an endocrine emergency associated with significant mortality estimated at ~15%. We describe herein a case where multidisciplinary team management was crucial in safely carrying the patient through a phaeo crisis and highlight precipitating factors and the role of magnesium in the treatment of phaeo crisis.

Case Presentation: A 63-year-old female with neurofibromatosis type 1 (NF-1), and previous renal artery stenosis bypass surgery, was admitted via A&E with increasingly frequent episodes of sweating, headache, dyspnoea and palpitations. Troponin was raised at 72 ng/l, progressively rising to 364 ng/l. CT pulmonary angiogram revealed a 6 cm left sided adrenal mass with cystic features. The possibility of phaeochromocytoma was raised and later confirmed by elevated metadrenaline at 5834pmol/L and normetadrenaline at 4198pmol/L). Doxazosin was commenced for alpha blockade with a view to introducing beta blockade at a later point. Subsequently she developed a narrow complex tachycardia treated with amiodarone, and chest pain treated with morphine and metoclopramide. She had a coronary angiogram which revealed unobstructed coronary arteries. Post angiogram she developed a broad complex tachycardia (recurrent VT) and uncontrolled hypertension at 254/112mmHg. She was admitted to ITU, and treated with magnesium sulphate infusion, phenoxybenzamine and verapamil. With magnesium sulphate, her condition stabilized, and BP controlled. She was discharged on phenoxybenzamine and verapamil. She underwent elective left adrenalectomy, nephrectomy, distal pancreatectomy and splenectomy. Histopathology revealed appearances of an adrenal medullary haemorrhagic pseudocyst, arising within a phaeochromocytoma. The lesion was completely excised with no involvement of the kidney or spleen and 0/3 lymph nodes, PASS score1.

Discussion: Phaeochromocytoma was considered early and blockade commenced.

1) Nevertheless, patient had a crisis. Likely precipitants were:
    a) CTPA- IV, albeit non-iodinated contrast is considered safe.
    b) Possibly contributed by intra-arterial contrast during the coronary angiogram.
    c) Metoclopramide use may have had a contributory role, which can worsen the crisis.

2) Magnesium is useful in the management of severe refractory hypertensive emergencies. It acts by:
    - Direct vasodilating action on arterioles.
    - Inhibition of catecholamine release.
    - Reduction of Alpha receptor sensitivity.

Z006

I would love to remove my head = pituitary apoplexy

R Eltayeb, N Kaimal, Royal Free Hospital.

Abstract: Pituitary apoplexy is a medical emergency and rapid replacement with hydrocortisone may be lifesaving. Apoplexy is often the first manifestation of an underlying pituitary adenoma. We report a case of apoplexy in a patient with an undiagnosed pituitary adenoma who presented with sudden onset headache and subtle neurology in the form of minor left ptosis.

A 64-year-old male with a background of hypertension, asthma-COPD overlap syndrome and bronchiectasis presented to A&E with acute sudden onset headache, vomiting and dizziness. He denied any visual symptoms. Examination revealed very mild left sided ptosis with no other neurological deficit. Urgent non-contrast CT head showed no acute haemorrhage or infarction.

Due to the persistent symptoms and unexplained partial ptosis, an out of hours CT angiogram was requested. This showed a 4 x 5 mm aneurysm of the distal MCA, enlargement of the pituitary fossa and a possible pituitary mass. Urgent ophthalmology review confirmed left partial ptosis and bitemporal hemianopia with possible left ischemic optic neuropathy. Hormonal profile showed random cortisol of 144 nmol/l (whilst on prednisolone 30 mg/d for COPD), normal thyroid function tests and prolactin. Testosterone was low at 5.9nmol/l. Pituitary MRI with contrast done on the following day showed an enlarged pituitary measuring 2.1 X 2.2cm.

Heterogeneous signal was noted on T1 weighted images with central low signal but peripheral high signal suggestive of blood products, in addition to the MCA aneurysm. He was transferred to the local neurosurgical centre and underwent transsphenoidal pituitary surgery. Postoperative visual field assessment showed full recovery. He was started on hydrocortisone replacement. The MCA aneurysm is being managed conservatively.

Learning points:

1. Pituitary apoplexy often occurs in undiagnosed pituitary tumours
2. Subtle neurology in association with other symptoms should trigger further evaluation
3. Dual pathology can co-exist, such as aneurysm and apoplexy
4. Multidisciplinary approach is crucial.

Z007

Case report: Multi-factorial hyponatraemia in hypopituitarism

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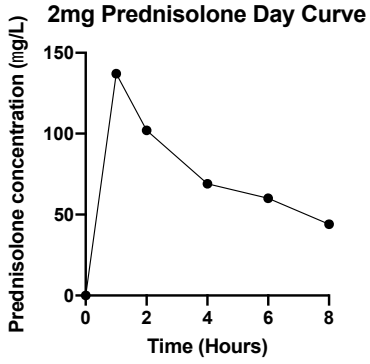
Introduction: Glucocorticoid replacement therapy is essential in treating patients with adrenal insufficiency. Administering the right dose and reproducing the pulsatile release of cortisol is still a major challenge. Hyponatraemia can occur after periods of hypocortisolism, but the mechanism is poorly understood. We present the case of a patient with known pituitary failure following transsphenoidal surgery and pituitary radiotherapy in 2012 for a craniopharyngioma, who developed hyponatraemia following a reduction in her prednisolone dose.

Report of the case: A 74-year-old female attended follow up pituitary clinic in July 2019. She reported tiredness spanning the past month, with a persistent cough, confusion (reported later by her husband) and intermittent vomiting episodes following reduction of her prednisolone to 1mg daily (8-hour level: 40 µg/L) from a previously stable 2.5mg daily. Routine bloods revealed she was hyponatraemic with a sodium of 114 mmol/L and slightly hyperkalaemic (potassium 5.5 mmol/L). Her previous serum sodium was ~131 mmol/L and baseline creatinine of ~130 µmol/L with normokalaemia (from 2017). She was called back and admitted to the hospital.

Her biochemistry on admission was: Sodium: 114 mmol/L; Potassium: 5.2 mmol/L; Creatinine: 101 µmol/L; eGFR: 47 ml/min; Serum osmolality: 240 mosm/L; Urine osmolality: 310 mosm/L; Urine sodium: 74 mmol/L
Urine potassium: 40.0 mmol/L; TSH: 0.72 mU/L Free T4: 11 pmol/L.

The patient's additional past medical history includes type 2 diabetes (HbA1c- 71 mmol/mol in January 2019), hypercholesterolemia, asthma, right nephrectomy for renal cell carcinoma (2010). Her growth hormone, gonadotrophin, and glucocorticoid axes were deficient, with only the latter being treated with replacement. In addition to antihypertensives and inhalers for asthma and atorvastatin, her medication included gliclazide 40mg BD, linagliptin 5mg OD, metformin 500mg BD and importantly, prednisolone 1mg OD. Her prednisolone day curve on 2mg is plotted below:



On examination, the patient was noted to be dehydrated and hypovolaemic as a result of the vomiting. She was given IV normal saline slowly over 12 hours in addition to 100mg hydrocortisone QDS. Her blood sodium dramatically improved overnight from 116 mmol/L to 125 mmol/L. Urine MC&S grew E. coli sensitive to ciprofloxacin which likely contributed to her decompensation. The response to saline confirms that she was partly dehydrated.

Despite treatment with antibiotics, careful rehydration and IV glucocorticoids, the patient's sodium later came down to 122 mmol/L, one week after admission, suggesting that in addition to the dehydration, she also had partial SIADH. She required further fluid restriction to 1 L/day, before the sodium returned to 128 mmol/L and the patient was discharged on prednisolone 3mg OD as glucocorticoid replacement.

This patient had acute adrenal insufficiency, precipitated by a UTI and reduction of glucocorticoid dosing, complicated by vomiting and a background of probable SIADH. It is possible that the apparent SIADH occurred because of the lack of glucocorticoid affecting free water clearance.

Learning points:

1. Reducing a patient's long-term steroid exposure is important but must be done carefully to avoid acute illness.
2. Extra care must be taken when the baseline sodium is already low or there are pathologies affecting salt-water homeostasis, such as SIADH or diabetes insipidus.
3. The diagnosis of SIADH can only be made in patients who have adequate levels of thyroxine and glucocorticoids, as apparent SIADH occurs when either of these are insufficient.

Z008

Regional audit into the indeterminate thy3f cytology thyroid nodules and their resulting histological malignancy rate at Luton & Dunstable Hospital and Lister Hospital

O Kenyon, A Shaladi, M Ally, V Blackabey, E Schechter, R Bance, D Ravichandran, P Loizou, G Mochloulis, P Kothari, Luton & Dunstable Hospital and Lister Hospital

Abstract: Fine needle aspiration cytology (FNAC) is a valuable investigation for thyroid nodules[1]. Thy3f grading is used when a follicular neoplasm is suspected however benign and malignant histology cannot be distinguished by cytology alone[2]. We accessed all thyroid related cytology in Luton & Dunstable Hospital between October 2017 and January 2019, yielding 29 patients with Thy3f classifications of which final histology was available for 19 (66%). In Luton & Dunstable 15 patients had US grading available, comprising one U2 (benign), 13 (87%) were U3 (indeterminate), and one U4 (suspicious).

In Lister Hospital we identified all patients discussed at MDT with the term Thy3F From June 2014 to June 2017, yielding 46 patients, of which 41 patients had final histology (89%). All patients had US grading available: 6 were graded U2/U3, 32 (70%) were U3, 3 were U3/4, 4 were U4 and 1 were U4/5.

The histology ranged from follicular adenoma (with and without oncocytic changes); hypoplastic nodule with focus of micropapillary carcinoma; follicular carcinoma; papillary carcinoma; to a plasmacytoma (corresponding to the U2 grading).
We found a malignancy rate of 53% (10/19) in Luton & Dunstable Hospital and 54% (22/41) in Lister Hospital averaging 53.3%. This falls high within the quoted malignancy rates in the literature (18-54%)[3] which may be due to the patient demographics and we plan to expand the time scale to capture more patient data.

[1] Bajaj, T., De M. & Thompson, A. (2006) Fine needle aspiration cytology in diagnosis and management of thyroid disease. *Journal of Laryngology and Otology*, **120**, 467-469
[1] Perros, P. *et al* (2014) British Thyroid Association Guidelines for the Management of Thyroid Cancer. *Clinical Endocrinology*, **81** (Suppl. 1), 1-122
[1] Alexander, V., Rudd, J., Walker, D. *et al* (2018) Thy 3F and 3a malignancy rate, a multisite regional retrospective case series. *Annals of the Royal College of Surgeons of England,* **100**, 545-550

Z009

A giant cystic pheochromocytoma presenting with myocardial infraction - a case report

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Introduction: Cystic pheochromocytoma is a rare neuroendocrine tumour, usually asymptomatic at presentation making pre-operative diagnosis difficult. We report a case of giant pheochromocytoma which appeared as a large cystic adrenal lesion acutely presenting with a ST elevation myocardial infarction.

Case Description: 42 years old female presented with Electrocardiographic evidence of acute anterolateral ST elevation myocardial infarction with a normal coronary Angiogram. She had a history suggestive of adrenergic spells and poorly controlled diabetes, hypertension for 5 years. 24 hour total urinary Metanephrines was 45.7mg/24 hours (<1mg/24 hours) and contrast enhanced CT abdomen revealed a purely cystic right suprarenal lesion with enhancing thin wall, measuring 8cm x 8.5cm x 7.5 cm. Whole body Magnetic Resonance Imaging (MRI) showed the same mass, 6.3x 6.2x7.1cm as hypointense in T1W images with a hyperintense rim and heterogeneous hyperintensity. She underwent surgery following optimal pre-operative preparation and macroscopically the lesion was circumscribed with a size of 10.5x 90x 7.5cm filled with homogenous brownish material, falling in the category of giant cystic pheochromocytoma. Histology and Immunohistochemical analysis showing strong positivity for Chromogranin and synaptophysin confirmed the diagnosis of pheochromocytoma with a Ki 67 index of 6.5%. Imaging was negative for distant metastasis.

Conclusion: Phaeochromocytoma is heterogenous in clinical presentation as well as in radiological appearance. Therefor high degree of suspicion and use of highly sensitive investigations in ruling in or out of diagnosis are crucial.

Z010

Case Report: A Striking presentation of a Rare Invasive and Aggressive co-secreting Growth Hormone and ACTH Pituitary Macroadenoma

R Banatwalla, V Horden, D Russell-Jones, Royal Surrey County Hospital

Introduction: Highly invasive Pituitary tumours are rare and represent difficult management problems. Co-secreting tumours are rare, and we report a case exhibiting both features.

Case: 47-year-old gentleman, working as a flight attendant became aware that his peripheral vision was not normal in 2006. The formal visual fields testing reported bitemporal hemianopia and MRI pituitary scan reported a large pituitary mass extending upwards and stretching the optic chiasm. The only symptom he had slight lack of libido. There were no clinical and biochemical signs suggestive of any endocrine syndrome at presentation. He underwent an uneventful Transsphenoidal Hypophysectomy in 2006. The histology was unusual and revealed co-secretion of both ACTH and GH with two separate populations of secretary granules within each cell on electron microscopy[1]. Post-operatively he did make good recovery, but he required hydrocortisone replacement. Serial imaging revealed a steady growth of his pituitary tumour leading to further surgeries in 2007 and 2014, which was complicated by a post -operative CSF leak. He underwent stereotactic radiotherapy in 2009 and 2016. In 2018, he developed florid Cushings confirmed on biochemical testing. Medical treatment was started, and bilateral adrenalectomy was performed with resolution of the Cushingoid state. His pituitary tumour continued to grow with symptoms of facial numbness and headache presumably due to cavernous sinus invasion.

Treatment options are now limited. Should he be commenced on Temozolomide or Octreotide or should undergo stereotactic radiotherapy be considered for the pituitary tumour invading the skull base (Pituitary bed has received maximum allowable radiation dose).

Discussion: This unusual tumour has demonstrated to co-secrete GH and ACTH that initially presented as a silent adenoma has proven to be resistant to treatment and transformed into a highly metabolically reactive tumour**.**

Reference:

1. Puslakar et al. A case of Silent ACTH/ GH adenoma. Endocrine abstracts (2008)**15**P216.

Z011 and OC002

A rare case of multi-organ failure due to acute Parathyroid crisis

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Abstract: Parathyroid crisis also described as a parathyroid storm or parathyrotoxicosis, is a rare and life-threatening complication of primary hyperparathyroidism. Parathyroid adenoma and hyperplasia are common causes of primary hyperparathyroidism. Parathyroid cancer can rarely cause primary hyperparathyroidism (0.5%-4%). Atypical parathyroid adenomas, a sub-set of parathyroid neoplasms, with uncertain malignant potential, are also found to be associated with acute and sever hypercalcemia.

Hypercalcemia is severe, usually > 3.5mmol/L with associated signs and symptoms of multi organ dysfunction, including metabolic encephalopathy, renal insufficiency, gastrointestinal symptoms and cardiac dysfunction. Parathyroid crisis requires prompt recognition, immediate medical management of hypercalcemia followed by urgent surgery. Initial management includes aggressive fluid resuscitation, cardiac monitoring and administration of intravenous bisphosphonates.

We present a case of 58 years old man with background of mild learning difficulties, COPD, Ischemic heart disease, depression and high BMI. His medications include Amitriptyline, Aspirin, Docusate, GTN, ISMN, Lisinopril, Mirtazapine, Risperidone, Senna, Simvastatin and Topiramate. He presented to ED with acute confusion, recurrent falls, slurred speech and increased urinary frequency. Systemic examination was unremarkable. CT head and subsequent MRI head ruled out stroke. Laboratory investigations revealed new hypercalcemia of 4.64mmol/L (2.12-2.62), PTH 267pmol/L (1.8-6.8), AKI stage 2, vitamin D 19nmol/L, and Myeloma screen negative.

Parathyroid ultrasound showed a right lower gland adenoma. SPECT CT, was not possible due to high BMI. CT Chest abdomen pelvis showed some coronary artery calcification and 8mm stone in left kidney.

His clinical presentation was thought to be due to acute severe hypercalcemia secondary to aggressive parathyroid cancer. He was placed under close monitoring, and resuscitated with intravenous fluids, loop diuretics, cinacalcet and bisphosphonate.

His clinical and metabolic derangement improved and serum calcium improved to normal level, 2.46mmol/L.

Due to high surgical risk, he was transferred to tertiary centre for parathyroidectomy. A very experienced endocrine surgeon described his surgery challenging due to co-morbidities, and surgical tissue adherent to surrounding structures. Histology confirmed features suggestive of atypical parathyroid adenoma with, occasional tumour cells, but low proliferation activity(<2%). Study of Immunohistochemical markers and epigenetics is underway.

Z012 and OC005

Bilateral adrenal incidentalomas, unilateral Conn's and subclinical Cushing's syndromes

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Abstract: We present the case of a 60-year-old woman with bilateral adrenal incidentalomas and no stigmata of endocrinopathy. Biochemical investigations were consistent with autonomous cortisol secretion: ODST 593nmol/L; ACTH 1ng/L; two 24hr UFC collections 414 and 518nmol/24hrs (<120nmol/24hrs). Cortisol after LDDST was 587nmol/L. Final cortisol following Yanovski test was 377nmol/L (<38nmol/L). Two urinary collections for catecholamines were normal, as was initial ARR. CT abdomen showed two lesions on the left (2.6cm and 1.9cm) and one on the right (1.9 cm), unchanged from 2012. Her co-morbidities included HTN requiring three agents and T2DM. Her BMI was 23 kg/m2. Her DEXA was normal.

She was referred for AVS and metomidate PET scan for lateralisation of excess cortisol production. An active nodule with increased uptake of C-metomidate in the upper part of the isthmus of the left adrenal gland was seen. There was no gradient for cortisol production, but a clear aldosterone gradient from the left adrenal gland (8,000 mmol/L) compared to the right (194 mmol/L) was observed. Genetic testing for ACTH independent macronodular adrenal hyperplasia was negative. Metyrapone was commenced, normalising her cortisol secretion.

Left adrenalectomy was performed in June 2019. Histology revealed three adrenocortical adenomas with Weiss score 2. Postoperative morning cortisol was 507nmol/L. ODST one month later revealed unsuppressed cortisol at 547nmol/L. Her postoperative plasma renin activity was normal. Her BP was 138/86 on lisinopril, amlodipine and doxazosin and HBA1c 52mmol/mol on sitagliptin.

This case highlights the diagnostic challenges arising in a patient with functional bilateral adrenal nodules. It demonstrates the current difficulties in lateralising excess cortisol secretion, and how, whilst attempting to do this, co-existing hyperaldosteronism was picked up.

The post-operative results suggest that although the primary hyperaldosteronism has been cured by the left adrenalectomy, autonomous cortisol secretion continues. Despite not being clinically Cushingoid, she has co-morbidities, which may well be a consequence of this. She is now faced with the decision of whether to have a right adrenalectomy, rendering her hypoadrenal, or life-long medical management of hypercortisolaemia.

Z013

Immune-Mediated Endocrinopathies

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Abstract: Immune checkpoint inhibitors are being increasingly used successfully for cancer treatment. However, this has led to the emergence of endocrine-related adverse events. Clinicians' awareness of these endocrine side effects is key and a Multidisciplinary Team Approach is recommended. I am presenting 3 cases of Immune-related endocrinopathies, managed with the Oncology team at Charing Cross Hospital.

Case 1: A 75-year old gentleman was commenced on palliative Pembrolizumab for lung adenocarcinoma. He developed intermittent palpitations and thyroid blood tests showed thyrotoxicosis with TSH < 0.01milliunit/L, T4 36.8 and T3 10.2 pmol/L. He had no thyroid eye disease and no goitre on examination and his TSH receptor antibody was mildly positive at 0.7 unit/L (NR: <0.4). It was felt this is in keeping with Pembrolizumab-induced thyroiditis and the patient was monitored closely and symptomatically managed on beta-blockers. After 2 months, he developed overt hypothyroidism with TSH 35.66 milliunit/Land undetectable T4. He is currently on levothyroxine replacement.

Case 2: A 78-year old gentleman was on a course of adjuvant Pembrolizumab for melanoma. He was admitted to hospital with nausea and postural hypotension. Early morning and random cortisol levels were undetectable with associated ACTH of 15.4 ng/L. The rest of his pituitary screen was normal and a Short-Synacthen Test (SST) showed an inadequate adrenal response. We felt this was a case of Pembrolizumab-induced adrenalitis. The patient was started on a replacement dose of Prednisolone 5mg and fludrocortisone 50 mcg. He was counselled about sick day rules and given a steroid card.

Case 3: A 72-year-old gentleman was receiving Nivolumab and Ipilimunab for metastatic renal cell carcinoma. He presented to hospital with a collapse and diarrhoea. Early morning cortisol were low at 90 and 102 nmol/L with undetectable ACTH. The rest of his pituitary screen revealed secondary hypothyroidism and secondary hypogonadism. A SST was performed which showed an increment of over 150 nmol/L and 250 nmol/L of cortisol at 30 and 60-minute respectively. We felt this was a case of immune-related hypophysitis and the SST was misleading due to normal/intact adrenal function. The patient was started on prednisolone for colitis and maintained on a replacement dose of 5 mg. A pituitary MRI showed no pituitary lesion.

Z014

Lessons from prednisolone day curves in adrenal insufficiency: Can we tailor anti-inflammatory steroid treatment based on serum prednisolone levels?

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Background: The recent BTS/SIGN 2019 guidelines for the diagnosis and management of asthma continue to advocate a step-wise approach to treatment. In patients with difficult asthma, that cannot be managed by maximal inhaler therapy and leukotriene receptor antagonists, oral glucocorticoid therapy with prednisolone may be necessary.

With greater past-year and lifetime cumulative exposure to glucocorticoids, individuals face an increasing risk of mortality and risk of developing iatrogenic adrenal insufficiency or Cushing's syndrome. As such, respiratory physicians managing difficult asthma will use the lowest possible dose of oral prednisolone to manage the condition and will consider biologics (anti-IgE antibody or anti-IL5 antibody treatment), if prolonged or frequent courses are required.

Case history: Patient (A), a 49-year-old female patient with difficult asthma is under the care of the respiratory physicians. Despite maximal treatment, her asthma remains poorly controlled... A dose of prednisolone of 20mg was insufficient to suppress markers of type-2 inflammation or control symptoms. Currently, she is only responding to 40mg Depo-Medrone (methyl-prednisolone), administered IM fortnightly.

In order to investigate why 20mg of prednisolone is insufficient, but 40mg of Depo-Medrone is effective, a prednisolone day curve was undertaken.

Results: Patient (A)'s prednisolone day curve data is available below. The maximum concentration (Cmax) achieved on 20mg prednisolone was 222 µg/L and the area-under-the-curve (AUC) was 1152 µg.h/L. The terminal half-life was 3.85h.

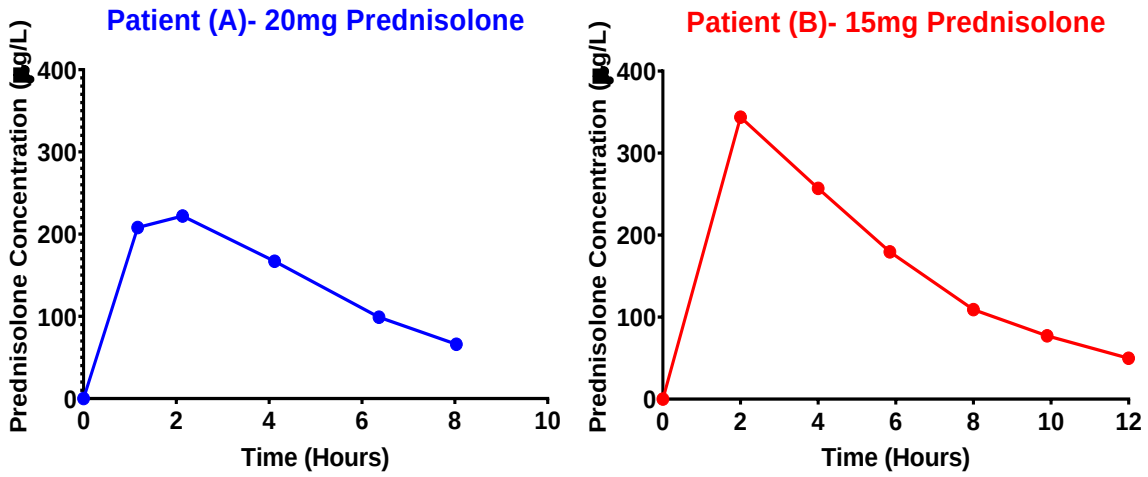
This was compared to data held on Patient (B), who had a historic day curve on 15mg prednisolone (day curve profile below). The Cmax for this patient was: 344 µg/L, with an AUC of 1969 µg.h/L. The terminal half-life was 3.38h.

Further historic data from 6 patients (with a total of 10 prednisolone day curves) receiving a mean prednisolone dose of 7.9mg (SD-4.4mg), showed a statistically significant positive correlation between dose and maximum serum concentration with coefficient r=0.788 (p=0.007) and also between dose and AUC with r=0.918 (p<0.001). Thus, both Cmax and AUC increased in a dose-dependent pattern. Mean terminal half-life of prednisolone was 3.73h (SD=0.64). Mean Cmax and AUC was 279.8 µg/L (SD- 98.7 µg/L) and 1430.1 µg.h/L (SD-542.7 µg.h/L) respectively. We now use 8-hour levels to guide glucocorticoid replacement therapy, and the target 8-

hour level for those on 2mg to 5mg is 15 µg/L-25 µg/L. In the historic patients, the mean 8-hour level was 82.5 µg/L (SD- 35.3 µg/L), and the 8-hour level for Patient B was 109.2 µg/L while the 8-hour level for Patient A on 20mg was only 66.0 µg/L.

Conclusions: The terminal prednisolone half-life in Patient (A) is comparable to Patient (B) and the pooled patient data. This indicates normal prednisolone elimination in Patient (A). There is however, a lower Cmax and AUC when compared to patients taking lower doses of prednisolone, suggesting increased first pass metabolism of the tablets. Given that the patient responded to IM glucocorticoids, it is likely that the patient has an altered first pass metabolism, and that there is increased liver cytochrome activity which is bypassed by parenteral steroid administration. The respiratory team were advised to review the patient's other medication, with a view to rationalising any liver enzyme inducers.

This case demonstrates the utility of prednisolone day curves in patients using anti-inflammatory doses of prednisolone. In this case, there was scope to escalate the patient's prednisolone dose without concern of unnecessarily increasing steroid exposure. A better profile of the patient could have been generated with quantification of cortisol binding globulin, and urinary steroids. In type-2-high asthma, steroid doses should be titrated to anti-inflammatory markers and biologic therapies used for patients on ≥ 5mg prednisolone / day. This work shows that, with more data, greater confidence about prednisolone dosing can be obtained by investigating absorption, in those patients where there is disparity between the clinical effects of oral and injectable steroids.



Z015

Pre-operative diagnosis of Parathyroid Cancer: a single centre experience

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Background: Parathyroid Cancer is a rare condition (<3% of patients diagnosed with Primary Hyperparathyroidism). Under staging and under treatment of parathyroid cancer can have disastrous consequences for patients. The risk of local recurrence, distal metastases and death is estimated as double of those initially diagnosed and treated appropriately.

Objective: This study shows the experience of a single centre in London with the objective of demonstrating and highlighting the importance of preoperative diagnosis, staging and initial treatment of this rare and aggressive disease, by showing the difference and improved outcomes for patients appropriately staged and treated. It also highlights the importance of simple recognition tools such as the Schulte's 3 + 3 rule for suspicion of Parathyroid Cancer.

Design and methods: Retrospective single centre review. The data consists of patients whom underwent oncological Parathyroidectomy between 2005 and 2018 at King's College Hospital. SPSS software was used for analysis

Results: 28 patients were included in the study: 8 patients underwent local excision and 20 underwent oncological resection (unilateral en bloc resection of thyroid lobe, both parathyroids and level VI lymphadenectomy). 3 patients who underwent local excision elsewhere had local recurrence and were referred to our centre for further treatment. One patient with pre-operative lung metastases, underwent oncological resection had also local recurrence and died after 12 years follow up. Twenty-four patients are free of disease. Average pre-operative Calcium was 3.01nmol/L, PTH 287ng/L and size 36mm.

Conclusion: Under staging and under treatment leading to inappropriate initial management showed a 12-fold increase in recurrence rates. The only observed recurrence in patients that underwent initial EB resection was related to advance staging due to distant metastasis. This highlights the importance of recognising pre-operative indicative factors described above for appropriate surgical planning, reducing risk of recurrence and death.

Z016

A case of refractory hypercalcemia. Elusive diagnosis and difficult management

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Para-neoplastic Hypercalcemia usually presents with markedly elevated serum calcium levels. Several potential mechanisms leading to the development of hypercalcemia of malignancy have been described, including parathyroid hormone-related peptide-mediated humoral hypercalcemia, osteolytic metastasis-related hypercalcemia, 1, 25 Vitamin D-mediated hypercalcemia, and parathyroid hormone mediated hypercalcemia. In addition to detailed history and physical examination, markers for above mentioned processes should be measured.

We present a case of refractory hypercalcemia in an 85 years old, Caucasian man, with background of myelodysplastic syndrome, pancytopenia, splenomegaly and renal impairment. He initially presented to acute admissions, through his GP, with new onset, severe hypercalcemia and worsening renal impairment. Physical examination was unremarkable. Medications included Colecalciferol, Ganfort, Paracetamol. Laboratory investigations showed Calcium 3.44mmol/L (2.12-2.62), PTH <0.6pmol/L (1.8-6.8), Vitamin D 39nmol/L. Myeloma screen was negative, B2 microglobulin was high. CT abdomen and pelvis showed enlarged spleen, T6 vertebral collapse. Bone marrow biopsy showed no evidence of myeloma and JAK2 mutation negative. Bone scan showed normal uptake. 1,25 dihydroxyvitamin D was 134pmol/L (55-139). Subsequent PET scan showed splenomegaly with increased uptake.

Above pattern of Lab results confirm a process of PTH independent hypercalcemia. He was resuscitated with intravenous fluids, renal dose pamidronate, and calcitonin. Regular Laboratory monitoring was arranged. Subsequently, zolendronate was given due to refractory hypercalcemia. Hematologist did not consider Myelodysplasia to be the likely cause of severe hypercalcemia. A possible presence of intra-splenic lymphoma has been raised.

There is continuous MDT involvement including Endocrinologist, Haematologist and radiologist. Tertiary centre-based haematology MDT suggested spleen biopsy or splenectomy if biopsy not possible. Interventional radiologist is concerned about increased risk of bleeding from spleen biopsy given age, frailty and co-morbidities. Patient declined spleen biopsy. MDT planned to start trial of empirical steroids, and to study blood film for flow cytometry.

This case clearly illustrates importance of MDT role in the management of this complex case where diagnosis still remain elusive and optimal management challenging.

Z017

The use of computed Tomography as a first-line imaging modality in patients with primary Hyperparathyroidism

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Introduction: Successful outcomes in primary hyperparathyroidism (PHPT) relies on accurate localisation of the culprit parathyroid glands. Concordant findings on the neck ultrasound (US) and Sestamibi (99mTc scintigraphy) are currently considered the 'gold' standard. However, Computed Tomography (CT) scan has also been used in the pre-operative planning of parathyroidectomy.

We report a 73-year-old lady, presented to her GP with fatigue and tiredness. Biochemically proven to have PHPT. CT parathyroid and US scans were performed for adenoma localisation and both scans revealed descended right superior parathyroid adenoma.

In our centre we conducted a study to assess the accuracy of CT scan in localising abnormal parathyroid glands in such patients.

Study Methods: This is a prospective study of 75 patients with PHPT who underwent neck US and CT scans as their first line imaging between January 2017 and January 2019. Sestamibi was reserved for patients with diagnostic uncertainty (n=7), although many patients (n=47) had already a Sestamibi scan performed at the referring centres.

During surgery, the identification and extirpation of the abnormal gland was followed by intra-operative PTH measurements. The modified Miami criterion was used to define intra- operative cure.

The postoperative values of serum calcium and PTH were recorded at specific times (i.e. direct post- operative [<24 hours of surgery], 3 months after surgery and at their final follow-up at 6 months).

Results: 77.3% of patients were female with median age of 57 years. The overall sensitivity [95%-CI] per diagnostic tool was 60% for US scan, 87% for CT scan, and 33% for Sestamibi (overall $p<0.05$). In 17 (22.7%) patients, CT was the only modality which correctly identified abnormal glands.

Eight patients (10.7%) in whom none of the imaging modalities was correct had bilateral neck exploration, in two of them (25.0%) the adenoma was in an ectopic location and five (62.5%) had multiglandular disease.

The combined sensitivities for multimodality imaging were also calculated. The standard combination of US and Sestamibi resulted in a sensitivity of 65%, this was statistically less than the sensitivity for CT alone ($p<0.001$). The combination of US and CT resulted in a sensitivity of 88%, this was not statistically significantly different from that of CT alone ($p>0.99$). Sestamibi was not helpful in patients with negative combined US and CT scans.

Discussion and conclusion: Several factors influenced imaging sensitivity in this study; the first factor was the weight and size of parathyroid adenoma. The sensitivity of CT for localizing abnormal parathyroid glands weighing <1.0g was 81%, significantly better than that of US (54%; $p=0.002$) or US & Sestamibi (62%; $p=0.04$). Combining the results of US & CT in patients with smaller glands did not lead to an increase in the sensitivity compared with CT alone ($p>0.99$).

The second important factor was ectopic parathyroid adenoma. The sensitivity for CT alone was 82% which was statistically better than US (50%; $p=0.039$) and combined US & Sestamibi (57%; $p=0.07$). However, combining US&CT led to increased accuracy of 86%; $p=0.016$).

The diagnosis of multiglandular disease seemed to be the most difficult, the sensitivity for US & Sestamibi was of 40% and the accuracy rate for combined US&CT was statistically similar at 50%.

The combination of US and CT was able to correctly identify the location of parathyroid adenoma in 88% of patients, with a relatively better diagnostic accuracy for smaller and ectopic adenomas.

This finding suggests that a change of current paradigm of performing US and Sestamibi is desirable and US & CT should be considered as a first-line imaging modality in patients with PHPT considered for surgery.

Z018

Identification and Management of Familial Hypocalciuric Hypercalcemia Type 3 in a District General Hospital

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We present a 29-year-old Caucasian lady, referred to Endocrinology with an asymptomatic, elevated adjusted calcium of 2.84 mmol/L.

Of interest, her mother had previously been investigated for apparent recurrent hyperparathyroidism and diagnosed with FHH type 3. Her mother had previously been discharged from the endocrine service.

Initial biochemistry, revealed a low urinary calcium creatinine clearance ratio of 0.002, an elevated PTH 10.92 pmol/ L, phosphate 0.97 mmol/L and ALP 81 U/L. Genetic testing confirmed a heterozygous AP2S1 mutation consistent with FHH type 3.

Familial hypocalciuric hypercalcaemia (FHH) is a rare, genetically heterogenous condition, characterised by raised serum calcium concentrations and low levels of urinary calcium excretion. The majority of FHH cases can be confirmed via genetic testing for a calcium sensing receptor (CASR) gene mutation. Loss of function mutations lead to FHH type 1 which is a benign condition. FHH3 is a much rarer genetic variant with normal CASR genes, accounting for 5-8 % of total FHH cases.

It is important to consider FHH in patients with hypercalcaemia and very low urinary calcium creatinine clearance ratio. However, differentiating FHH from primary hyperparathyroidism is in some cases difficult due to the similarity in clinical and biochemical features. Genetic confirmation of FHH may be required to avoid unnecessary surgical intervention. Specific genetic testing for CASR mutations will lead to a missed diagnosis of FHH3. Gene panel testing, including testing for AP2S1, is a more sensitive method to identify underlying genetic disorders.

People with FHH type 1 can be safely discharged from the endocrine service and need no further investigation or monitoring. FHH type 3, however, is not considered a benign condition and is associated with severe osteoporosis and some cognitive deficit. The patient and her mother will now be monitored life-long for symptoms of hypercalcaemia alongside investigation for osteoporosis including vitamin D and bone mineral density levels.

Z019

Does α and β blockade in patients undergoing adrenalectomy for phaeochromocytoma increase postoperative morbidity? – An Institutional analysis

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Introduction: Adrenalectomy for phaeochromocytoma (PCC) is associated with a higher rate of perioperative complications and even mortality. Historically the decreased mortality to under 3.0% has been attributed to the preoperative of α-blockade however this practice is now being challenged. The aim of our study is to present our institutional experience in preoperative alpha-blocking of PCC and its effect on postoperative morbidity.

Methods: This is a retrospective study from our institutional database. All patients undergoing adrenalectomy for PCC from September 2017 to October 2019 were included. All patients are routinely α and β blocked. Intraoperative hemodynamic instability (IHI) was assessed through number of Systolic blood pressure (SBP) episodes >160mmHg, episodes of SBP<80mmHg, use of vasoactive drugs and volume of intraoperative crystalloids. Postoperative morbidity and characteristics, such as hospital stay, complications BP and hemoglobin, were also evaluated. Results: A total of 36 patients were included (19 male, 17 female) with mean age 55(±16) years underwent adrenalectomy for PCC. Bodyweight ranged from 46 to 110kg (mean 77.5±15 kg). The mean maximum diameter of tumors was 44.5±27.8mm.

Pre-operatively the mean SBP was 147.11±29.9mmHg. Eighteen patients (50%) had no hypertensive episodes, 13 (36%) patients had 1-5 episodes of SBP>160mmHg, 4(11%) patients 6-10 episodes and 1(2%) patient a single hypertensive episode during the surgical procedure. Eight (22%) cases had no intraoperative hypotensive episodes, 21(58%) had 1-5 hypotensive (SBP<80mmHg) episodes, 6 (16%) showed 6-10 episodes, while only 1(2%) patient presented intraoperatively 15 episodes of SBP<80mmHg. Fourteen patients received vasoactive drugs during the operation. The mean volume of intraoperative crystalloids was 2.24±0.84l. The mean operative time was 114.2±59min.

Postoperatively, the mean SBP was 115.8±16.9mmHg. The mean hemoglobin on postoperative day 1 was 113.9±24.9g/l. No patient presented with arrhythmia during adrenalectomy, and none needed blood transfusion or ICU admission. Three cases presented complication regarding to their respiratory system, but no intervention was needed. No reoperation or readmission was required.

Conclusions: IHI remains numerically significant in PCC surgery even with α and β blockade. Omitting blockade would appear empirically questionable but only a randomised controlled trial of surgery with and without blockade will provide an answer to this new question.

Z020

Case of Grave's ophthalmopathy complicated by ocular myasthenia gravis

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Myasthenia Gravis is a rare autoimmune disease marked by muscular weakness without atrophy characterized by the presence of anti-acetylcholine receptor antibodies. Grave's disease is an autoimmune disorder affecting the thyroid gland resulting commonly in hyperthyroidism due to stimulation of the thyroid receptor antibody. Coexisting myasthenia gravis and Grave's disease is rare.

We present the case of a 58-year-old patient who presented with swelling, redness and irritation of both eyes and was diagnosed as having Grave's orbitopathy by the ophthalmologist and referred to the endocrine service. She had positive thyroid receptor antibodies and was commenced on carbimazole. Her initial thyroid function showed a T3 28.2, a T4 of 34.2 and a suppressed TSH. CT orbit (done as she could not tolerate an MRI) showed bilateral proptosis with symmetrical enlargement of the medial recti and asymmetrical enlargement of the right inferior and left superior recti, supportive of thyroid orbital disease. However, the presence of bilateral lid ptosis was found to be unusual and on probing she described worsening of ptosis as the day progressed and weakness of her arms and a slower gait in the last few weeks.

Her speech was also found to be slightly nasal. She was referred to neurology to investigate the possibility of coexistent myasthenia. She was subsequently admitted and received iv immunoglobulin and pyridostigmine with a clinical diagnosis of myasthenia gravis. Her acetylcholine receptor antibody subsequently came back as being strongly positive confirming myasthenia. At the end of 5 days, there was a marked improvement in her gait, upper arm weakness and degree of ptosis although her eye movements were still restricted. She is currently on pyridostimine, a block and replace regimen for thyroid disease, azathioprine and a weaning dose of oral steroids.

Conclusion: The occurrence of Grave's disease and myasthenia may indicate the patient has a genetic predisposition to autoimmune diseases. Muscle weakness may be associated with both conditions and can confuse the diagnosis. A high index of suspicion when the clinical picture does not explain all the symptoms and signs of the patient would help to clarify an alternative diagnosis.

Z021

Genetic testing in patients with primary hyperparathyroidism: when is screening worthwhile?

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Background: Primary hyperparathyroidism (pHPT) affects 1:1000 of the population but increases in prevalence with age, such that 2% of post-menopausal women have the condition. Approximately 95% of pHPT is sporadic and 5% related to familial inherited disorders, most commonly Multiple Endocrine Neoplasia type 1 syndrome (MEN1). Almost all MEN1 patients develop pHPT, and the disease is biochemically evident in over 90% by the age of 20 years. According to international guidelines, it is therefore recommended that genetic testing be performed in patients diagnosed with pHPT below the age of 30 years; however, the threshold age for testing remains controversial. The purpose of this study was to assess the use of genetic testing in early onset pHPT, to determine the yield of genetic abnormality and possibly the optimal age for genetic testing.

Methods: This was a retrospective observational study of a prospectively collected database of all patients 40 years and under, that underwent parathyroidectomy between February 2010 and February 2019. Electronic patient records were used to identify: age at operation; number of parathyroid glands removed; whether a referral for genetic testing was made and the results of any genetic tests. For patients referred for genetic testing, the genes most commonly tested for mutations included MEN1, MEN2, CASR, CDC73, CDKN1A, CDKN1B, CDKN2B and CDKN2C.

Results: An initial database search yielded a total number of 158 patients that fulfilled the above criteria, of which 70 patients were identified that had all required electronic notes available for analysis. Of these 70 patients, 48 had undergone genetic testing, and 15 were confirmed to have a gene mutation causing pHPT. A small proportion of patients who tested positive, were determined to have variations of uncertain significance. The age of patients who tested positive ranged from 19 to 40 years old, with a mean age of 30 years.

Conclusions: Our study demonstrates a high yield of causal genetic abnormalities in young patients undergoing parathyroidectomy. Genetic testing is therefore justified in younger patients diagnosed with pHPT, even up to the age of 40 years, since it may impact on surgical strategy and patient informed consent for surgery.

Z022

Myxoedema coma with severe hypoxia

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Myxoedema coma is a rare but treatable endocrine emergency however it must be considered as a differential diagnosis as symptoms overlap with other much more commonly occurring critical conditions.

The mortality rates may be as high as 30–60%, particularly with delayed treatment [1]

The cardiovascular manifestations in myxoedema coma tend to be especially severe and life threatening in comparison to other symptoms [2]. Early symptoms of the disease comprise bradycardia and low cardiac output due to decreased cardiac contractility.

Pericardial effusion may also be present in such cases due to increased vascular permeability [3].

Myxoedema coma can also be complicated by alveolar hypoventilation and respiratory failure. Alveolar hypoventilation occurs due to decreased ventilatory response to hypoxia and hypercapnia [4,5]. There is weakness of respiratory muscles as well as a decrease in the central drive [4,6]

Abstract: We report a case of a 28-year-old Polish woman, recently travelled to the UK, presented to the A&E with 3-week history of breathlessness and bilateral leg swelling. She had a history of Trisomy 21, amenorrhoea for 4 months and orthopnoea with periorbital oedema with conjunctival congestion. Saturation 70% on air, 96% on 15 liters of oxygen, heart rate 42, blood pressure 103/61, temperature 36.2.

Investigations and management: Echocardiogram showed 3.3cm pericardial effusion, low voltage ECG and tests showed TSH >100mIU/L (0.4 – 4.4) with FT4 0.05pmol/L (12-22). She initially had type 1 respiratory failure and required intubation and ventilation. Pericardial effusion was drained twice. She was treated with intravenous liothyronine and hydrocortisone along with levothyroxine via nasogastric tube. CT pulmonary angiogram showed bilateral lower zone collapse and complete collapse of the left upper lobe with widespread ground-glass consolidation throughout the remaining aerated lung with an element of congestive failure. Despite pericardial drainage, she later developed type 2 respiratory failure and despite intubation and ventilation required transfer for surgical tracheostomy and long wean from ventilation. She was discharged on oral levothyroxine and nocturnal CPAP.

Discussion: Pericardial effusion is described in literature as a complication of myxoedema. These usually are exudative with high cholesterol content, but can occasionally be transudative

.

Respiratory failure is well recognised in myxoedema coma with reduced ventilatory response to carbon dioxide, decreased diffusion capacity, respiratory muscle weakness and decreased breathing capacity. Abnormalities suggestive of fibrotic lung disease have been described. Ventilatory response usually improves after treatment. In our patient, this did not happen and it took over two months to wean her off. Ground glass consolidation and hypoxia requiring prolonged ventilation and tracheostomy is very rare.

References

1. Wall CR - Myxedema coma: diagnosis and treatment. American family physician. 2000;62:2485–90. [PubMed] [Google Scholar]
2. Chaudhari D, Gangadharan V, Forrest T - Heart failure presenting as myxedema coma: case report and review article. Tennessee medicine: journal of the Tennessee Medical Association. 2014;107:39–41. [PubMed] [Google Scholar]
3. Klein I, Danzi S - Thyroid disease and the heart. Circulation. 2007;116(15):1725–35. [PubMed] [Google Scholar]
4. Wilson WR, Bedell GN - The pulmonary abnormalities in myxedema. The Journal of clinical investigation. 1960;39:42–55. [PMC free article] [PubMed] [Google Scholar]
5. Zwillich CW, Pierson DJ, Hofeldt FD, et al. - Ventilatory control in myxedema and hypothyroidism. The New England journal of medicine. 1975;292:662–5. [PubMed] [Google Scholar]
6. Laroche CM, Cairns T, Moxham J, et al. - Hypothyroidism presenting with respiratory muscle weakness. The American review of respiratory disease. 1988;138(2):472–4. [PubMed] [Google Scholar]

Z023

Persistent hypertension post adrenal resection in a patient with surgically managed Conn's Syndrome

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We present the case of a 49-year-old man initially diagnosed with hypertension aged 29. Following a presentation with collapse and uncontrolled hypertension in 2012, he underwent biochemical investigations at his local endocrine centre and was diagnosed with Conn's syndrome. Adrenal venous sampling confirmed hyperaldosteronism with lateralisation to the right adrenal gland, whilst computerised tomography imaging revealed a 16 mm adrenal adenoma. He subsequently underwent laparoscopic resection of the right adrenal gland in 2012. Whilst surgery was successful in treating his hypertension, he developed post-operative complications including pain and haematoma which resulted in further surgical procedures locally in 2013 following which he was discharged on no antihypertensive therapy.

Later in 2013, his hypertension recurred along with symptoms of headache and right flank pain. As these were to resistant to treatment, he was in 2016 referred to a tertiary surgical endocrine centre for further assessment. Magnetic resonance imaging revealed a lesion in his right adrenal bed and further revealed a left sided lesion that was felt to be a benign adrenal adenoma. Due to persistent pain and symptoms, he underwent surgical re-exploration of the previously operated site which resulted in the excision of a right sided mass adherent to the inferior vena cava. On histology, this was found to be a benign 'Surgicel granuloma', which was an inflammatory mass formed around absorbable surgical material used to control bleeding during his initial adrenal resection.

Between 2016 and 2019, he nevertheless remained hypertensive with a blood pressure in the community of around 180/130 mmHg and symptoms of ongoing right sided pain, nausea and headaches. On review in endocrine clinic in Aug 2019, his BP was 206/138 and potassium was low at 3.2 mmol/l despite being on 5 anti-hypertensive agents including eplerenone, lisinopril, indapamide, doxazosin and amlodipine. Between August and October 2019, these agents were adjusted in a stepwise fashion to a combination of verapamil, hydralazine and doxazosin to allow for assessment of plasma aldosterone and renin. On this regimen, his aldosterone:renin ratio was 112 (morning result) and 100 (afternoon result) (aldosterone 280 and 310 pmol/l; renin 2.5 and 3.1 nmol/L/hr). These results excluded a diagnosis of Conn's recurrence. Other endocrine causes of hypertension were also ruled out and he is currently awaiting admission to assess for medication compliance and optimisation of anti-hypertensive therapy.

Z024

Paraganglioma Crisis: the challenges of a silent neuroendocrine neoplasm

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Case Presentation: A 47-year-old female presented with sudden visual loss and blurring associated with severe hypertension. She had a six-week prodrome of feeling generally tired with low energy and palpitations as well as significant weight loss (>25kg) over two years. She was otherwise normally fit and well with no past medical or drug history of note, aside from anaemia. On clinical examination, she was alert, orientated and apyrexial with a blood pressure of 224/147 and normal heart rate. Her chest was clear, abdomen soft and non-tender with a right-sided palpable mass, and her neurology intact. There was no meningism, stigmata of endocrinopathy or diaphoresis elicited. On ophthalmologic evaluation, she had bilateral retinopathic changes with macular oedema. She was promptly commenced on calcium channel and angiotensin II receptor blockers for blood pressure control but remained severely hypertensive with no response.

She was hyperglycaemic on admission and mildly hypokalaemic with a raised urea level and deranged liver function. She was biochemically euthyroid, had a satisfactory cortisol level and raised renin activity with normal aldosterone levels. She had severe concentric left ventricular hypertrophy on echocardiography. Abdominal ultrasound showed a 21 x 18 cm mass that filled the entire upper abdomen extending from the vertebral column to the anterior abdominal wall and MRI confirmed a large retroperitoneal mass which did not appear to involve the adrenal glands. Moreover, her plasma and urinary catecholamine status was significantly abnormal; the plasma Normetadrenaline level was 69,106 (normal range < 1180) and Metadrenaline 99,134 (normal range < 510). Her neuroendocrine secretory proteins, Chromogranin A and B levels were also raised. Further differentiation with Ga68 DOTATATE PET CT revealed that the mass likely represented an extra-adrenal paraganglioma.

Conclusion: This case represents the diagnostic challenges from such a rare neuroendocrine tumour. Abdominal paraganglioma tend to be functional and slow-growing, and in this case presented as an inoperable mass, making them complex to medically manage. It is a vital diagnosis not to miss that may initially masquerade with few paroxysmal symptoms or undiagnosed hypertension, before acutely presenting as a crisis.

Z025 and OC003

A Case Report of Concomitant Diagnosis of Multiple Myeloma and Primary Hyperparathyroidism

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Introduction: Primary Hyperparathyroidism and multiple myeloma are two of the most common causes of hypercalcemia but the concomitant diagnosis of both in one case is rare.

Primary hyperparathyroidism (PHPT) has prevalence estimates of one to seven cases per 1000 adults [1-2] It is believed to be the most common cause of hypercalcemia.

Abstract: We describe a case of 45 years old Jamaican female who presented with left sided chest pain for a month. CXR showed a pathological left clavicular fracture with a lytic lesion. She was noted to have a serum Ca of 3.26 mmol/L.
Investigations: Corrected Ca 3.26 mmol/L (2.20-2.60 mmol/l), Phosphate 0.60mmol/L (0.87-1.45 mmol/L), PTH 16.8pmolL (1.6-6.9 pmol/L), Vitamin D 26nmol/L (Insufficient if 25-75nmol/L); Paraprotein not detected, kappa light chains 44 mg/L (3.3-19.4 mg/L), lambda light chains 8.5 mg/L (5.7-26.3 mg/L), kappa lambda ratio 5.2 (0.26-1.65), urine-BJP negative and beta-2 microglobulin 4.3mg/L (0.26-1.65), LDH 180 IU/L (135-214 IU/L).

Bone marrow: 80% plasma cells on trephine with findings consistent with high risk myeloma.

CT CAP: expansile lytic lesion in left clavicle, probable pathological fracture of right fourth rib, multiple lytic lesions in bones and calculus in right kidney. MRI spine showed abnormal signal in multiple vertebrae.

US Neck: 1.2 low density structure highly suspicious of enlarged parathyroid gland.
Sestamibi: adenoma in the region of the upper pole of the left thyroid lobe.
PET scan post-chemotherapy showed response to treatment with low-grade mild activity in left clavicle only.

Management: Patient was referred for Parathyroidectomy.
Patient was diagnosed with Oligo-secretory Myeloma, revised ISS stage two and primary hyperparathyroidism.
She completed 6 cycles of chemotherapy and received monthly Zoledronic acid. She is awaiting stem cell transplant.
Patient was referred for Parathyroidectomy.

Conclusion: Hypercalcemia as a presenting symptom of concomitant MM and Hyperparathyroidism is rare with 30 reported cases (Hussain *et al* 2013)[3]. Majority were female with age ranging from 45-92 years. Our patient is the youngest to our knowledge.

Parathyroidectomy, chemotherapy and radiotherapy have been used for treatment with variable success. The prognosis has been generally poor with 28% dying within 5 years of diagnosis.

References

1. Writing Group for the SEARCH for Diabetes in Youth Study Group, Dabelea D, Bell RA, et al., editors.Incidence of diabetes in youth in the United States. JAMA. 2007;297(24):2716–2724 [PubMed] [Google Scholar]
2. Lawrence JM, Lukacz ES, Nager CW, Hsu JW, Luber KM. Prevalence and co-occurrence of pelvic floor disorders in community-dwelling women. Obstet Gynecol. 2008;111(3):678–685 [PubMed]
3. Hussain N., Khan M., Natarajan A., Mohammedabdul M., Mustafa U., Yedulla K., and al. A case of multiple myeloma coexisting with primary hyperparathyroidism and review of the literature *Case Rep Oncol Med* 2013; 2013:420565

Z026

Phaeochromocytoma presenting as an acute coronary syndrome

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A 48-year-old man presented to the Emergency Department with a one-day history of central crushing chest pain and palpitations. On presentation his initial ECG showed tachycardia with ST depression in lateral leads. Blood results showed positive and dynamic troponin levels and he was initiated on treatment for an acute coronary syndrome (ACS) with dual antiplatelet therapy (DAPT), fondaparinux plus a beta blocker. There was no significant past medical history or family history of ischaemic heart disease. He was a smoker and had a moderate intake of alcohol weekly.

Subsequent investigations demonstrated inferior wall hypokinesis with preserved systolic function and unobstructed coronary arteries. In view of the angiographic findings, the working diagnosis was myopericarditis likely secondary to a viral lower respiratory tract infection (LRTI). However, despite high dose beta blockers and treatment of a suspected LRTI the patient remained tachycardic and sweaty.

He underwent an ultrasound of the abdomen for investigation of a potential underlying myeloproliferative disorder on account of thrombocytosis on his blood results. The scan demonstrated a large solid and cystic adrenal mass suspicious for a phaeochromocytoma. MRI abdomen reported a heterogeneous mass involving the medial limb of the left adrenal gland measuring 5.3 x 6 x 5cm. 24-hour urinary normetanephrines and metaneprhines were elevated at 9860 nmol and 21547.5 nmol respectively.

He was seen by the endocrinology team and alpha blockade with doxazosin was initiated followed by addition of the beta-blocker propranolol. The patient was referred to the endocrine surgeons and successfully underwent a laparoscopic adrenalectomy. Histology confirmed a composite phaechromocytoma and ganglioneuroma, Ki-67 of 2-3% and immunohistochemistry demonstrated cells expressing synaptophysin and chromogranin. Analysis of 10 phaeochromocytoma and paraganglioma susceptibility genes did not detect any pathogenic variant.

This case demonstrates that phaeochromocytomas can mimic a number of cardiac conditions including acute coronary syndrome. Elevated plasma catecholamines may cause myocardial injury by a direct toxic effect on myocytes and / or diffuse coronary vasospasm. Phaeochromocytoma should be considered in the differential diagnosis of patients presenting with chest pain, uncontrolled blood pressure, palpitations and sweating.

Z027

An Addisonian Crisis following recent Diagnosis and Treatment for Tuberculosis

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Introduction: Addison's was first described in patients with adrenal tuberculosis (TB) and is the most commonly involved endocrine organ in TB[1]. It can cause adrenal insufficiency in a number of ways. Here we present a patient who presented with an Addisonian crisis following recent commencement on anti-TB treatment.
Case Presentation

A 48-year-old Indian male presented with a 3-day history of fatigue and dizziness, with an acute onset of abdominal pain and vomiting 24 hours previous to presentation. He had recently been diagnosed with TB, and one week previously had commenced anti-TB treatment (rimfampicin, pyrazinadmide, pyridoxine and ethambutol). His initial imaging 2 months previously had shown bilateral adrenal masses, and a CT adrenal showed diffuse enlargement of both adrenals with poor enhancement on the portal venous phase, in keeping with TB adrenalitis.

On further questioning, this patient's wife had commented that his skin appeared darker, with friends and family members asking if he had been abroad. His past medical history included mild depression, and his surgical history included appendectomy. He was taking mirtazapine 45mg OD.

On initial assessment, he had a heart rate of 129 bpm, blood pressure of 111/75 mmHg, a respiratory rate of 29 breaths per minute, and oxygen saturations of 98% on air. He was mildly febrile and had a GCS of 15. Cardiovascular, respiratory and abdominal examinations were unremarkable. An initial VBG showed a pH of 7.405, bicarb of 24.5 mmol/L, lactate of 0.8 mmol/L, glucose of 3.4 mmol/L, potassium of 3.8 mmol/L a sodium of 115 mmol/L. Formal bloods showed a sodium of 117 mmol/L with a plasma osmolality of 243 mOsmol/kg, urine osmolality of 524 mOsmol/kg and urine sodium of 95 mmol/L. The initial post take plan was to give slow intravenous fluids to treat for hypovolaemic hyponatraemia, stop the mirtazapine, and complete the remaining hyponatramia screen. His sodium dropped further to 113 mmol/L and a short synacthen test was performed; basal cortisol: 132 nmol/L, cortisol at 30 minutes 130 nmol/L and cortisol at 60 minutes 126 nmol/L. His ACTH was 869 ng/L.He was then given hypertonic saline and commenced on hydrocortisone 20mg/10mg/10mg, and his anti-TB medications stopped. His sodium had normalised to 133 mmol/L 4 days later, and fludrocortisone 100mcg once a day was then started. Due to the temporal relationship between starting anti-TB medications and the onset of the Addisonian crisis, it was surmised that rifampicin (a known potent hepatic enzyme inducer) had increased the metabolism of this patient's endogenous cortisol. This precipitated a crisis in a patient with a background of probable subclinical adrenal insufficiency secondary to tuberculosis infiltration.

This was later evidenced by a hydrocortisone day curve several weeks, established on rifampicin and hydrocortisone 20mg + 10mg + 10mg: 09:00 cortisol 59 mmolL, 11:00 cortisol mmol/l, 13:00 244 mmol/L, 15:00 420 mmol/L, 17:00 161 mmol/L.

Discussion: TB and its treatment can affect the adrenal gland in multiple ways; including direct infiltration, and increased cortisol metabolism secondary to enzyme induction from TB treatment. Rifampicin is known to increase cortisol breakdown to 6beta- hydroxycortisol by selectively upregulating liver microsomal cytochrome P450IIIA and this can be achieved by administering for as little as 5 days[2]. Our patient had radiological evidence of adrenal infiltration on initial imaging but had not had baseline biochemistry including an early morning cortisol performed.
Learning points from this case therefore include prompt assessment of adrenal reserve prior to commencing anti-TB medications, especially if there is radiological evidence of adrenal involvement, and the need to ensure prompt administration of glucocorticoids in the acute setting.

References

1. Kelestimur. F. The endocrinology of adrenal tuberculosis: The effects of tuberculosis on the hypothalamo-pituitary-adrenal axis and adrenal function. *J Endocrinol. Invest.* 27: 3800-386, 2004
2. Ged C et al. The increase in urinary exretion of 6 beta-hydroxycortisol as a marker of human hepatic cytochrome P450IIIA induction. *Br J Clin Pharmacol* 1989; 28: 373-87.

Z028

Synchronous papillary (pT1a(m)N0) and medullary (pTxN1b) thyroid cancer – a diagnostic pitfall

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A 34-year-old male with a neck pain and left side submandibular lymphadenopathy admitted to otorhinolaryngology (ORL) outpatient clinic. Fine needle aspiration biopsy (FNAB) of enlarged lymph node was made. With the initial diagnosis of malignant salivary gland neoplasm were sent for magnetic resonance (MRI) (Figure 1A) which revealed pathological lymph nodes with no abnormalities in salivary glands. Additionally, PET with 18F-FDG was done (Figure 1B) - it showed active metabolic process in several lymph nodes of the upper and middle part of the neck. Subsequently, bilateral tonsils, pharyngeal lymphatic tissue and enlarged left side (II group) lymph node were removed. Histopathological examination of the specimens demonstrated macro metastasis of medullary thyroid carcinoma (MTC) to the lymph node with positive staining of synaptophysin, chromogranin A, calcitonin and Ki 67-7%. Pheochromocytoma was ruled out. Serum calcitonin level was 300 ng/ml. After interdisciplinary decision, radical thyroidectomy with bilateral and central lymphadenectomy was performed. Specimen was evaluated independently by two experienced histopathologists, the result was: multifocal papillary thyroid cancer in the thyroid gland (pT1a(m)N0) and left sides lymph nodes (group II-IV) 7/29 and central (group VI) 2/4 positive for MTC metastases; but surprisingly primary MTC tumor was not found (pTxN1b) (Figure 1C). The post-operative period was uneventful. On subsequent follow-up visit RET and BRAF mutation were excluded, the patient was doing well. Calcitonin level in 6-month observation is below 2 pg/ml.

The simultaneous occurrence of MTC and PTC is a very rare phenomenon. It usually observed in two main settings: a mixed tumor (dual differentiation) or a collision tumor (with two separate and different components) [1]. The current cases belong to even different category with two independent malignancies with unknown primary site of one of them.

Z029 and OC004

Case of possible non-secretory metastatic Phaeochromocytoma

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A 46-year-old gentleman was diagnosed with right sided (6cm) sporadic phaeochromocytoma in 2010 at the age of 36. He had two-year history of palpitations, sweating, hypertension and multiple syncopal episodes. He underwent right adrenalectomy with partial nephrectomy at a separate London teaching unit. Histology showed completely excised phaeochromocytoma, positive for SDH staining with negative blood genetic analysis in 2011.He has been under surveillance at Royal Free since January 2017 with monitoring of 24 hours urinary metanephrines. He is currently not on any medication.

He had no significant past medical history of note. There was a family history of hypertension in both parents, and his mother had stroke at the age of 66. His father had MI in his 40s and died of heart failure at the age of 63. His older brother has lymphoma.

His BP was 145/86mmHg, heart rate 73/minute and BMI 26. There were no symptoms suggestive of adrenal hormone excess. He has had normal 24-hour urinary metanephrine since 2012.

Biochemical profile (09/2019)	Result	Normal Range
24 Hr Urine Metadrenaline	0.43	0-1.2
24 Hr Urine Normetadrenaline	1.18	0-3.3
24 Hr Urine 3-Methoxytyramine	0.69	0-2.5
Free T4 Plasma	14.4 pmol	12-22
Thyroid Stimulating Hormone Serum	1.41 munit/L	0.3-4.2
Cortisol at 9am	252 nmol/L	172-497

He developed right abdominal pain and had been diagnosed with acute appendicitis. He underwent laparoscopic appendicectomy in June 2019 at Barnet Hospital with no surgical complication. A CT scan revealed incidental finding of left retroperitoneal mass in left suprarenal area, measuring 32mm.

Ga68 Dotatate PET CT showed lobulated retroperitoneal masses in the left suprarenal region and left para-aortic space at the level of the left renal vein which demonstrated low grade Dotatate uptake (less than background liver). These may represent recurrent phaeochromocytoma, a paragangliomas or malignant/metastatic lymphadenopathy. A subsequent FDG PET CT demonstrated increased FDG activity in cluster of nodes centred to left of coeliac axis.

The adrenal MDT discussed management of an enlarging nodal mass possibly invading left renal vein, which is thought to be likely metastatic from previous phaeochromocytoma. Surgical management would require open resection with left nephrectomy. CT guided biopsy has been arranged prior to surgical intervention to achieve histological diagnosis. These results would be available for the meeting.

Z030

An unusual case of SIADH in patient with HTLV-1 positive infection

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Introduction: Human T-Cell Leukaemia virus type-1 (HTLV-1) is retrovirus that affects 20-30 million people worldwide with significant endemic foci in the Caribbean, southern Japan, central and South Africa, and South America. It is commonly associated with leukaemia/lymphoma, myelopathy (spastic paraparesis) and less commonly endocrine and metabolic disorders, notably the syndrome of inappropriate antidiuretic hormone secretion (SIADH). Herein, we present a rare case of SIADH associated with HTLV-1 infection[1].

Case Report: We report 57-year-old Afro-Caribbean man with HTLV-1 infection presenting with syndrome of inappropriate antidiuretic hormone and myeloproliferative disorder.

Clinical Case: Patient presented with 1-week history of confusion, falls and increased stiffness in lower limbs. Examination revealed mild cognitive impairment with increased stiffness in the lower limbs and bilateral crackles.
Laboratory studies on admission Sodium levels of 112 mmol/L (135-145) with raised platelets of 1539 10^9/L (150-400) WBC 17 10^9/L (4-11), positive urinary pneumococcal antigen. Urine osmolality was 285 mosm/kg, urinary sodium of 87mmol/L, TFT and Cortisol were normal in keeping with diagnosis of SIADH. Hypertonic saline 1.8% was given in High dependency unit, despite management of Pneumococcal pneumonia with antibiotics and improvement of inflammatory markers, the hyponatremia persisted, Tolvaptan was given after failure of fluid restriction and resulted in resolution.
CT CAP and 18FDGPET showed splenomegaly with splenic infarcts only.
HTLV-1 serology was positive which explained the spastic paraparesis (HTLV-1-associated myelopathy/tropical spastic paraparesis). JAK2 was positive suggesting myeloproliferative disorder and with platelets persistently above 1000 x10^9/L, he was started on hydroxycarbamide.

Discussion: Although pneumococcal pneumonia can cause SIADH, the persistence of severe hyponatraemia for several weeks after resolution of pneumonia was suggestive of another pathology, such as HTLV-1. HTLV-1 infection has been associated with SIADH in two reports. In both cases there was CNS involvement with HTLV-1 and one patient had T cell leukaemia whereas our patient has myelopathy, but no leukaemia. This case adds to the literature, however further reports are needed to see whether there is a causal association.

References

1. Cresio Alves MD,PhD , Endocrine and metabolic disorders in HTLV-1 infected patients , The Brazilian Journal of infectious Diseases Volume 14, Issue 6, November –December 2010 ,Pages 613-620.
2. Uta Nishiwaki1, Syndrome of inappropriate antidiuretic hormone secretion associated with adult T-cell leukaemia/lymphoma, British Journal of Haematology, 2014, 166, 155

Z031

Investigating hypokalaemia – don't be Conn'd

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Case history: A 28-year-old male presented to A&E with a three-day history of diarrhoea and vomiting in April 2016. Blood tests showed severe hypokalaemia (2.3 mmol/L). Despite multiple infusions of saline with potassium chloride, serum potassium levels were slow to correct and consequently the patient self-discharged. A year later he represented to A&E with a frontal headache, a blood pressure of 220/110 mmHg and hypokalaemia confirmed on serum testing (2.5 mmol/L). Following initiation of oral potassium replacement and antihypertensive therapy (Amlodipine 10 mg OD and Ramipril 2.5 mg OD) he was discharged home however was lost to follow-up (did not attend). The following year, after a call from his GP concerning ongoing hypokalaemia, he was referred to Endocrinology for further assessment. Subsequent to biochemical correction of his hypokalaemia (2.5 mmol/L), Renin and Aldosterone testing was undertaken.

Results: Plasma Renin Activity <0.2 nmol/L/h, Aldosterone 1820 pmol/L, ratio >9100 supporting a diagnosis of primary aldosteronism. At that time his renal magnetic resonance (MR) angiogram (May 2018) was reported as normal (with both adrenal glands within normal limits and no mass lesions). As a result, he was referred for Adrenal Venous Sampling (AVS) in January 2019, results were as follows: Cortisol levels in the right and left adrenal veins (10,229 nmol/L and 8235 nmol/L respectively) indicated correct siting of the catheter. The Aldosterone/ Cortisol ratio (ACR) in the left adrenal vein was suppressed (0.4) whilst that in the right adrenal vein is markedly elevated (101) suggesting a predominantly unilateral secretion of Aldosterone from the right adrenal gland. The lateralisation index ($ACR_{dominant}$/ $ACR_{contralateral}$) was 253 and the contralateral suppression index ($ACR_{contralateral}$/ ACR_{IVC}) was 0.03, further supporting a diagnosis of unilateral disease.

Management: post-AVS he was commenced on Spironolactone which was uptitrated until serum potassium levels were >3.0 mmol/L. Blood pressure was managed with Amlodipine 10 mg OD and Doxazosin 8 mg BD. Dedicated adrenal MR imaging in May 2019 demonstrated a 22 mm lipid rich adenoma in the right adrenal gland; with a subsequent review of the original imaging confirming the presence of the same (previously missed) adenoma. As a result of the above investigations he has been referred for a right-sided laparoscopic adrenalectomy.

Discussion: This case highlights three important points.
1) Appropriate targeted investigation to ascertain the aetiology of hypokalaemia.
2) A corroborative evidence base reaffirming current Endocrine Society guidelines as to the appropriateness of when AVS is indicated. 3) Where possible the need for a multidisciplinary team approach pertaining to the management of adrenal disease – involving an (interventional) radiologist, endocrinologist and endocrine surgeon in pursuit of optimal patient care and avoidance of unnecessary and potentially harmful investigations.

Z032 and OC001

A Surgical Treatment for Cardiomyopathy

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We present a case of refractory hypertension and severe cardiomyopathy, which highlights the importance of considering the differential diagnosis of primary aldosteronism.

The case: A 57-year-old male was referred to the local cardiology unit with heart failure. He had an elevated blood pressure (180/90), despite treatment with multiple anti-hypertensives. An echocardiogram demonstrated severe concentric left ventricular hypertrophy (LVH).

Over the next five years, he became almost house bound as his hypertension and heart failure worsened and eventually required a pericardial window for significant effusion. He was extensively investigated by the cardiologist (including endomyocardial biopsy and genetic testing) but no cause for cardiomyopathy was found. A FDG-PET scan did show an incidental finding of a 2cm right adrenal nodule.

A referral to our endocrinology department was made, an elevated aldosterone renin ratio (ARR) of >25,123 (normal range <1200) was found. Years prior an elevated ARR had been identified at a peripheral hospital but as no adrenal pathology had been seen on imaging, this finding was not identified as a priority in the investigation of worsening cardiomyopathy at that time.

A repeat CT adrenal showed bilateral adrenal nodules (3cm on the right, 2cm on the left, Hounsfield units 20). Adrenal vein sampling was not possible as his interfering medications could not be weaned safely without cardiac decompensation. Instead 11-C Metomidate scanning demonstrated bilateral uptake but with an obvious dominant hot nodule on the right. A right laparoscopic adrenalectomy was performed.
The surgery had a successful outcome. His symptoms markedly improved three months postoperatively and his aldosterone and renin also normalised. Three years later, he remains normotensive (on amiloride and amlodipine only), with unlimited exercise tolerance. His echocardiography showed significant LVH regression.

Discussion and key learnings:

- Primary aldosteronism is under recognised, now described as >5% of HT patients and up to 20% in patients with resistant HT.[1-3]
- Clinicians should particularly consider screening those who meet the criteria suggested by the endocrine society guidelines.[1]
- Given hypertension can be cured/improved with treatment, a timely and accurate diagnosis is imperative.

- The novel PET/CT scan using the 11C-metomidate molecular tracer that binds adrenal steroidogenic enzymes can be used as an adjunct and/or alternative lateralisation modality.[4]

References:

[1.] Funder JW, Carey RM, Mantero F, Murad MH, Reincke M, Shibata H, et al. The Management of Primary Aldosteronism: Case Detection, Diagnosis, and Treatment: An Endocrine Society Clinical Practice Guideline. J Clin Endocrinol Metab. 2016;101(5):1889-916.

[2.] Rossi GP, Bernini G, Caliumi C, Desideri G, Fabris B, Ferri C, et al. A prospective study of the prevalence of primary aldosteronism in 1,125 hypertensive patients. J Am Coll Cardiol. 2006;48(11):2293-300.

[3.] Mulatero P, Stowasser M, Loh KC, Fardella CE, Gordon RD, Mosso L, et al. Increased diagnosis of primary aldosteronism, including surgically correctable forms, in centers from five continents. J Clin Endocrinol Metab. 2004;89(3):1045-50.

[4.] O'Shea PM, O'Donoghue D, Bashari W, Senanayake R, Joyce MB, Powlson AS, et al. (11) C-Metomidate PET/CT is a useful adjunct for lateralization of primary aldosteronism in routine clinical practice. Clin Endocrinol (Oxf). 2019;90(5):670-9.

Z033

Graves thyrotoxicosis presenting late in pregnancy: Additional challenges, considerations and multidisciplinary teammates

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Abstract: A 35-year-old G4P3 woman presented at 29+4 weeks with chest pain, neck swelling and abdominal tightening. She had similar previous presentations. On further questioning she had been unwell for months feeling anxious, tremulous and excessively sweaty. Examination revealed tachycardia, a tremor and a goitre with a thyroid bruit. Eye examination revealed normal eye movements with reported pain and diplopia in all directions of gaze, lid lag and exophthalmos. Investigations revealed fT4 59.5 (9-23) pmol/L, T3 >46.1 (2.5 – 5.7) pmol/L and Thyroid Stimulating hormone <0.01 (0.3-4.2 milliunit/L. Her TSH receptor antibodies later came back positive. She was diagnosed with thyrotoxicosis, secondary to Graves' disease and threatened pre term labour. She started carbimazole 20mg tds and propranolol 40mg tds. She was switched to propylthiouracil 100mg TDS after developing a blistering rash. She remained thyrotoxic throughout pregnancy, complicated by non-compliance, inability to attend ophthalmology clinic and repeat admissions with threatened preterm labour. She delivered a healthy 2150g baby at 33+4 weeks. 19 days postnatally she attended ophthalmology clinic) where vision threatening thyroid eye disease was diagnosed. Despite receiving 3 days of 1g IV methylprednisolone her vision deteriorated and she required an emergency endoscopic bilateral orbital decompression. In view of ongoing difficulties with compliance and unsuitability for radioactive iodine due to both her thyroid eye disease and her young children, a thyroidectomy is planned.

Discussion: Thyroid disease is relatively frequently encountered in pregnancy; however, this is often in the context of pre-existing thyroid disease or mildly abnormal thyroid function tests. It is much less common for Graves to present in pregnancy and it is unusual for Graves thyrotoxicosis to be this severe in the third trimester. In this case her severe disease and difficulty controlling her Graves led to adverse obstetric (preterm delivery) and ophthalmological (vision threatening disease requiring surgery) outcomes. During pregnancy and postnatally she has required input from a large multidisciplinary team which included obstetric physicians, obstetricians, endocrinologists, ophthalmologists, ENT and endocrine surgeons. This medically complex patient presenting late in pregnancy with symptomatic thyrotoxicosis and threatened pre term labour demonstrates the importance of the multidisciplinary team and good communication and teamwork.

Z034

Development of primary hyperparathyroidism after cardiothoracic surgery: a case of ectopic mediastinal parathyroid adenoma

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Case: A 54-year-old male was referred to endocrinology services after developing hypercalcaemia following coronary artery bypass graft (CABG) 5 months earlier. Post procedure bloods revealed a rapid increase in calcium (corrected) from 2.17mmol/L (Day 1 post-surgery) rising to 2.63mmol/L within 5 days with a raised PTH of 11.1 pmol/L consistent with primary hyperparathyroidism. He remained asymptomatic with no end-organ damage (no nephrocalcinosis or osteoporosis). Ultrasound of the parathyroid glands did not reveal an adenoma. Sestamibi SPECT-CT revealed a 6mm ectopic parathyroid in the anterior mediastinum. After Endocrine multidisciplinary discussion, he will undergo thoracoscopic procedure for a likely ectopic mediastinal parathyroid adenoma.

Discussion: Ectopic mediastinal parathyroid adenomas (MPAs) are relatively uncommon causes of primary hyperparathyroidism. Due to the common third arch origin of the inferior parathyroid gland with the thymus, they have a more extensive embryological migration pattern. Thus, inferior parathyroid glands are more frequently ectopic than superior glands and mostly found within or near the thymus gland.

Definitive management may be challenging since MPAs are not accessible by cervical incision, and removal may require a sternotomy or minimally invasive video-assisted thoracoscopic surgery (VATS). There are few reported cases of ectopic MPA removal in previous CABG but with risk of gland division, and increased chance of encountering scar tissue and adhesions. Accurate imaging to localise the glands is essential.

There are no official recommendations in existing guidelines for ectopic parathyroid adenomas. MDT debate includes the option of conservative management and surgery due to the risk/benefit analysis. The patient in our case has a long-life expectancy and is likely therefore to benefit from surgery.

Z035

Management strategies for TSH secreting pituitary adenomas (TSHomas)

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Background: TSHomas are rare, and our limited experience can result in diagnostic and treatment challenges. Here we describe two cases to highlight how individualising patient management can lead to successful outcomes.

Case 1: A 35-year-old lady presented with amenorrhoea following discontinuation of the oral contraceptive pill. After a single progesterone challenge, she fell pregnant and successfully breastfed, however her amenorrhoea persisted. She also noted increased tiredness, anxiety and palpitations. Her main desire at the time was pregnancy. She is otherwise fit and well, not taking any regular medications, and there is no family history of thyroid problems. She was clinically euthyroid with no evidence of eye disease or goitre, and visual fields were intact to confrontation.

Initial work-up showed raised thyroid hormones (FT4 29 pmol/L[NR 9-23] and FT3 13.8 pmol/L [NR 2.5-5.7]) with unsuppressed TSH (5.4 milliunit/L [NR 0.3-4.2]). There was no evidence of co-secretion of other pituitary hormones. The abnormality persisted despite excluding assay interference and non-thyroidal illness; and central hyperthyroidism was suspected. An MRI demonstrated a pituitary macroadenoma (17 mm), with excavation of the sellar floor and extension into the suprasellar space, but no contact with the optic chiasm. Markers of peripheral thyroid action including sex hormone binding globulin and alpha-glycoprotein subunit were raised. Dynamic testing revealed a blunted response to thyrotropin releasing hormone.

The results were discussed at the Pituitary Multidisciplinary meeting, and a diagnosis of TSHoma was made with a plan for surgical removal. To optimise thyroid function pre-operatively, lanreotide was administered; however, a single dose induced intolerable diarrhoea, and hence it was discontinued. Surgery was performed successfully 8 weeks later; however, her diarrhoea persisted, and she was admitted with hyponatraemia. The differentials included hypovolaemia, post-op diabetes insipidus and cortisol deficiency. Although she was treated temporarily with fluids and hydrocortisone, it was felt her diarrhoea was due to the recent lanreotide. Her post-op pituitary hormone axes were preserved, and there was no residual tumour on MRI. Her menses have returned and she remains well off hormone replacement.

Case 2: A 65-year-old lady with confirmed TSHoma in 2002, declined surgery in view of her neuropathy and reduced mobility related to underlying Charcot-Marie-Tooth disease. As a consequence, she was commenced on monthly octreotide LAR injections (20mg subcutaneously). Her baseline TFTs were: TSH 3.5 milliunit/L, FT4 20.9pmol/L and FT3 12.8 pmol/L. Within two years of treatment, the tumour had shrunk in size and she was rendered euthyroid. Her annual follow up remained unremarkable with no side effects, and normal liver function. In 2008, she developed type 2 diabetes (HbA1C 6.9%) and was commenced on oral therapy. She has responded well to octreotide for nearly 18 years and her latest TFTs are: TSH 2.83 milliunit/L, FT4 17.1 pmol/L and FT3 3.1 pmol/L.

Z036

The “Jekyll and Hyde” of Cabergoline therapy

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A 57-year-old gentleman presented to Charing Cross Hospital with a five-month history of gynaecomastia, low libido and energy levels. Following investigations, he was found to have an elevated prolactin of 2391 milliunit/L with secondary hypogonadism with a testosterone of 4.5nmol/L. A pituitary MRI showed a 11X9 mm pituitary adenoma in the right inferior aspect of the gland, with no compression of the chiasma.

He was started on cabergoline 500 mcg/weekly and subsequently shown a good clinical response with a prolactin level within the normal range and a testosterone level of 18.9 nmol/L.

However, within 5 months of starting cabergoline, he presented to A&E, every anxious and wanted to be checked for HIV. Further history revealed that he has been engaging in unsafe sexual activities with multiple partners over the last couple of months, despite being married. This behaviour was out of keeping for him and causing significant tension at home with his husband. The latter called the endocrine team to relate his concerns regarding the change in behaviour of the patient.
We arranged to review the patient urgently in clinic and discuss stopping cabergoline as we felt this was a case of cabergoline-induced sexual disinhibition.

The patient stopped cabergoline but re-presented to us complaining of recurrence of his previous symptoms of gynaecomastia, poor sex drive, low energy levels and low mood, to the point that he was unable to work. He wanted to be restarted on cabergoline and was not fully appreciative of the negative effects of cabergoline on his behaviour. We discussed his case in our Pituitary MDT and the decision was to refer him to Neurosurgeons for consideration of trans-sphenoidal surgery. In the meantime, he was closely monitored in clinic and as his testosterone levels trended down, off cabergoline, we were able to start him on testosterone replacement (Tostran 2% gel). At the latest clinical review, his testosterone levels were in the normal range and his symptoms have improved.

This is a challenging case of cabergoline-induced hypersexuality, which needed to be handled sensitively and required careful MDT management. Third-party corroboration of any manifestation suggestive of an impulse control behaviour following dopamine agonists is useful. It is believed that over-stimulation of the mesolimbic dopamine ‘reward’ areas may be involved.

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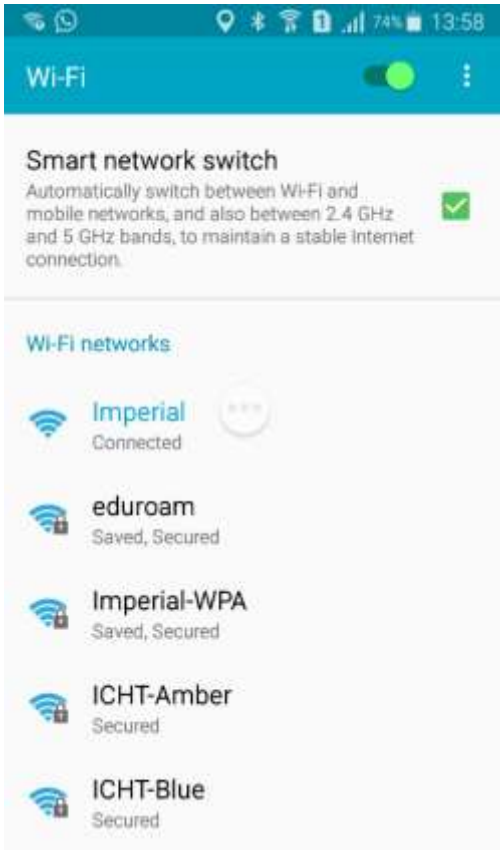

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