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BioScientifica Ltd
Euro House
22 Apex Court
Woodlands
Bradley Stoke
Bristol BS32 4JT, UK

Contact: Kate Openshaw
Tel: +44 (0)1454 642214
Fax: +44 (0)1454 642222
E-mail: info@bioscientifica.com
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Oral Communications

OC1

Bilateral adrenalectomy in a patient with congenital adrenal hyperplasia

Owais Chaudhri[1], Emma Hatfield[1], Katie Wynne[2], Fausto Palazzo[1], Sanjeev Mehta[1], Humera Shaikh[1], Tricia Tan[1], Niamh Martin[1] & Karim Meeran[1]
[1]Endocrine Unit, Department of Investigative Medicine, Imperial College Healthcare NHS Trust, London, UK; [2]Department of Endocrine Surgery, Imperial College Healthcare NHS Trust, London, UK.

We present the case of a 33-year-old woman diagnosed with congenital adrenal hyperplasia (CAH) due to classical 21-hydroxylase deficiency following a salt-losing crisis as a newborn. She had previously been controlled on dexamethasone 0.25 mg bd and fludrocortisone 100 mcg od, as evidenced by high-normal 17-hydroxyprogesterone (17-OHP) and low-normal testosterone levels (8.5 and 0.4 nmol/l respectively).

However, she had experienced significant symptoms of Cushing's syndrome on this replacement regimen (body mass index 34.6 kg/m^2, striae). A reduction in the dose of dexamethasone resulted in secondary amenorrhoea and distressing hirsutism refractory to treatment by conservative measures. Efforts to identify a steroid replacement dose that adequately suppressed adrenal androgen production without causing further progression of her iatrogenic Cushing's syndrome were unsuccessful.

The patient underwent a long synacthen test to characterise the level of residual adrenal cortisol synthesis. Cortisol levels were undetectable throughout, in the presence of a significant rise in 17-OHP levels (peak levels 54.6 nmol/l), confirming complete blockade of the 21-hydroxylase enzyme. Bilateral adrenalectomy was therefore considered as a treatment option to permit the use of lower doses of exogenous steroid and removing the source of excess androgens. She underwent an uncomplicated bilateral laparoscopic adrenalectomy in October 2007. One year later, she is maintained on hydrocortisone replacement (10 mg am, 5 mg lunchtime) and has gradually lost weight. Symptomatically, she has improved significantly.

Discussion will revolve around the use of adrenalectomy to overcome the difficult need to balance adequate suppression of adrenal androgen production vs weight gain in women with CAH.

OC2

Adrenocortical carcinoma presenting as Cushing's syndrome: 2 case reports

Adeel Ghaffar[1], Daniel Morganstein[1], James Kirkby-Bott[2], Jeannie Todd[1] & Fausto Palazzo[2]
[1]Department of Endocrinology and Metabolism, Hammersmith Hospital, Imperial College Healthcare NHS Trust, London, UK; [2]Department of Endocrine Surgery, Hammersmith Hospital, Imperial College Healthcare NHS Trust, London, UK.

A 69-year-old lady presented with pancreatitis, which was managed conservatively and resolved. However, imaging identified a 4 cm left adrenal lesion. She had no features of Cushing's syndrome, and was normotensive, but serum potassium was low. Renin:aldosterone ratio was normal. She failed a low dose dexamethasone suppression test (LDDST). She underwent laparoscopic adrenalectomy. Histology showed an adrenocortical carcinoma extending into the adjacent adipose tissue. Sixteen months postoperatively she failed an LDDST. A MRI adrenals showed a 1.5×1.4 cm soft tissue mass, thought to represent recurrence of the adrenocortical carcinoma. She underwent repeat surgery with removal of the recurrent tumour, the left kidney, the spleen and a segment of colon. The histology showed multifocal adrenocortical carcinoma involving the previous surgical bed. She was commenced on a course of mitotane. Twenty-two months after her initial surgery, imaging was performed in view of abdominal pain. This showed local recurrence and peritoneal deposits.

A 60-year-old man was referred to our centre with a diagnosis of ACTH independent Cushing's syndrome and a 7.6 cm right adrenal mass, after presenting with hypertension. A CT with contrast suggested that this was a benign lesion. He underwent an open adrenalectomy, as the lesion was closely applied to the liver. Histology identified an adrenocortical tumour with a high mitotic rate and a Ki67 index >5%. This was consistent with an adrenocortical carcinoma, and there was evidence of involvement of peri-adrenal fat. He subsequently underwent chemotherapy with mitotane.

Mitotane is a synthetic derivative of the insecticide DDT. It inhibits cortisone metabolism, and also has cytotoxic effects on the adrenal cortex. Its role in the management of adrenocortical carcinoma is unclear. Recent retrospective data suggest that it should be used as adjuvant treatment following surgery, and should not be solely reserved for recurrent disease. The rarity of adrenocortical carcinoma makes the undertaking of adequately powered randomised trials difficult.

OC3

An unusual case of hypertension

Binu Krishnan & Emma Bingham
Frimley Park Hospital, Frimley, Surrey, UK.

A 21-year-old female patient was referred from the eye clinic after she was noted to have bilateral papilloedema during a routine eye examination. She gave a 3 months history of intermittent headaches and fleeting episodes of profuse sweating and rash, unrelated to the headaches. She had been investigated by the GP with routine blood tests which were found to be normal.

On examination, she was noted to be tachycardic at 100 beats/min and hypertensive at 189/130 mmHg. She had erythematous maculopapular rashes over the lower forearm and hand bilaterally. Examination of the fundus confirmed bilateral papilloedema. ECG showed evidence of left axis deviation and left ventricular hypertrophy. CXR showed normal sized heart and clear lung fields. She was commenced on Nifedipine MR 20 mg bd. Ultrasound of the abdomen revealed a 4 cm right adrenal mass. MRI of the brain was reported as normal. An MRI of the adrenals and MIBG scan were arranged.

Nifedipine was stopped and she was commenced on Phenoxybenzamine 10 mg bd. As the patient remained well with improved control of her blood pressure, she was commenced on Propranolol 40 mg bd after 3 days.

The patient unfortunately had a cardiac arrest and died despite prolonged resuscitation. The coroner reported cause of death as acute pulmonary oedema secondary to malignant hypertension due to a right adrenal phaeochromocytoma. Results of 3×24 h urine catecholamines obtained thereafter showed noradrenaline levels almost 100 times more than the normal levels at 11 800/24 700/19 100 μg/24 h (normal range <100) and normetanephrine levels at 100/47/30 μg/24 h (normal range <3.3).

This case highlights the difficulty in treating these extremely rare tumours and the potentially fatal complications due to an adrenergic crises in these patients.

OC4

MEN1: the full house

Barbara McGowan, Annabel Fountain, Owais Chaudhri, Puja Mehta, Tricia Tan, Emma Hatfield, Niamh Martin, Jeannie Todd & Karim Meeran
Imperial College, London, UK.

This gentleman was diagnosed with hyperparathyroidism at the age of 34 and was found to have multiple manifestations of MEN1.

Hyperparathyroidism

He was diagnosed with hyperparathyroidism and underwent a parathyroidectomy with removal of 2 glands. Four years later calcium levels were raised once again and two further hyperplastic glands were removed, with cure of his hypercalcaemia.

Gastrinoma and other pancreatic islet cell tumours

He developed heartburn, nausea, vomiting and abdominal pain, and on upper GI endoscopy was found to have moderate duodenitis. He was started on lansoprazole with immediate relief of his symptoms. Gut hormones were elevated suggestive of a gastrinoma and other islet cell tumours. Imaging revealed multiple duodenal and pancreatic lesions. Calcium stimulation tests revealed no discrete gastrin releasing foci amenable to surgery.

Pituitary disease

He complained of impotence, reduced libido, weight gain and appeared Cushingoid. Cushing's disease and a prolactinoma were diagnosed on the basis of elevated urinary cortisols, failure to suppress cortisol on a low dose dexamethasone suppression test, inferior petrosal sinus sampling and elevated prolactin. Two pituitary lesions were seen on pituitary MRI. The patient underwent trans-sphenoidal hypophysectomy. He was started on hydrocortisone, DDAVP, thyroxine and testosterone replacement.

Adrenal lesions

A CT scan of the adrenals showed bilateral nodular adrenal enlargement. Phaeochromocytoma was excluded on the basis of normal catecholamine excretions.

Other lesions
He developed lipomas on his zygomatic arch and thigh and an angiofibroma on the nose.
Genetic testing
MEN1 was confirmed. One of his 3 children has an MEN1 mutation.
Conclusions
Our patient developed a full house of endocrine neoplasias. He presented a diagnostic and management challenge requiring a multi-disciplinary approach to ensure optimal treatment.

OC5

MEN 1 with adrenal Cushing's (a rare association)

K O Shaafi, S Russell & S Roberts
Chase Farm Hospital, Middlesex, UK.

A 36-year-old gentleman presented to his GP 2 years ago with hypertension and was commenced on valsartan. In January 2008, he was noted to have mildly deranged LFT and abdominal ultrasound scan showed mild fatty liver infiltration and bilateral renal stones. He was referred to Urology for investigation of renal stones and a CT IVU showed multiple renal calculi, a $6 \times 5 \times 5$ cm soft tissue enhancing lesion arising from the right adrenal gland and a 7 mm calcified density within the body of the pancreas of benign appearance. Adrenal protocol CT confirmed the adrenal lesion. At this stage he was referred to the endocrine team for assessment of the adrenal mass. On examination he had a plethoric facial appearance, centripedal obesity and gynaecomastia. Twenty-four hours urine collections for catecholamines and cortisol were normal on two occasions. An overnight dexamethasone suppression test and the low dose dexamethasone suppression tests were both positive, with suppressed basal ACTH. Routine bloods revealed elevated calcium 3.07 mmol/l with elevated PTH 16.3 pmol/l. Baseline pituitary profile revealed elevated prolactin 709 mU/l and normal gonadotrophins LH 2.3 IU/l, FSH 1.7 IU/l, testosterone 13.3 nmol/l.
He underwent laparoscopic right adrenalectomy. Histopathology confirmed cortisol producing adrenal adenoma. Since removal of the adrenal adenoma he has remained off antihypertensive medication. Postoperatively the cortisol level suppressed on low dose dexamethasone suppression test.
MRI pituitary showed 7 mm pituitary microadenoma and sesta Mibi parathyroid scan revealed increased uptake in the left inferior position suggestive of parathyroid adenoma, but there was also delayed uptake in the other three parathyroid glands. He is due for parathyroid surgery and he is likely to undergo four gland removal. We are currently in the process of investigating whether he has a pancreatic tumour with fasting gut hormones screen.
On review of the literature there are few reported cases of MEN I with concurrent adrenal adenoma producing cortisol. Forty percent of MEN cases are associated with adrenal nodular hyperplasia with no endocrinopathy. Therefore we would like to submit this interesting case for presentation at The Third Hammersmith Multidisciplinary Symposium.

OC6

Case report: prophylactic thyroidectomy for increasing calcitonin levels?

Katie Wynne, Anthony Goldstone, Fausto Palazzo, Jeannie Todd & Karim Meeran
Imperial College NHS Healthcare Trust, London, UK.

A 19-year-old man was referred to Endocrinology Clinic with a minimally raised calcitonin level. He had a past history of primary hyperparathyroidism resulting in renal colic aged 17 years, and underwent three gland parathyroidectomy aged 18 years. Histology demonstrated a single right lower parathyroid adenoma with normal thyroid biopsy. He has no family history of endocrine disease and is a non-smoker. His calcium has remained normal since with detectable PTH. Over 5 years there has been a progressive increase in his basal calcitonin level:

Date	Feb '04	Sep '05	Jul–Oct '06	Dec '06	Feb '07	Jul '08
Calcitonin (ng/l)	14.1	15.8	34.1–32.9	41.3	49.1	46.8
Normal (ng/l)	11.5*	11.5	18.9	18.9	18.9	18.9**

Peak calcitonin during a pentagastrin stimulation test was *152.0 in 2004 and **173.0 in July 2008.

His CEA, urinary catecholamines and fasting gut hormones have remained normal. Genetic screening for MEN-1 and MEN-2 (*RET* exons 1–20) was negative. Yearly thyroid ultrasound scans have been normal. An MRI neck scan was normal in 2004, and a Dimercaptosuccinic Acid (DMSA) scan was normal in 2006. He is now 24 years old. Despite the development of primary hyperparathyroidism at a young age and an increasingly elevated calcitonin level, there has been no histological evidence of C-cell hyperplasia, genetic evidence for increased risk of medullary thyroid carcinoma (MTC), nor radiological evidence of MTC. There are surgical risks of hypoparathyroidism and voice change with repeat neck surgery. Our Multidisciplinary Endocrine Meeting has concluded that there is no current indication for prophylactic thyroidectomy. The follow-up plan is for annual assay of calcitonin and neck ultrasound scans with fine needle aspiration of any thyroid nodules. In the absence of a suspicious nodule, it is open for discussion whether there is a level of basal or stimulated calcitonin which should prompt further consideration of prophylactic thyroidectomy.

OC7

Management of familial medullary thyroid cancer: not as simple as it seems

Leena Krishnan & James Ahlquist
Endocrine Unit, Southend Hospital, Southend on Sea, Essex, UK.

Optimal management of familial medullary thyroid cancer (MTC) involves early genetic diagnosis of affected individuals and prophylactic thyroidectomy. Patients diagnosed later may have biochemical evidence of metastases which are clinically silent. We report a family which illustrates the management difficulties which may arise with this approach. SC, a healthy 25-year-old man, presented for assessment for MTC. His mother had had a total thyroidectomy for MTC with no clinical evidence of metastases, but after surgery she had a raised calcitonin level (0.36 μg/l, normal <0.08 by RIA). Investigation has shown no definite localisation of metastases, serum calcitonin remains elevated at 169 ng/l (by ICMA), she has had no further treatment and she remains well. SC had a raised serum calcitonin level, and thyroidectomy confirmed MTC. After surgery the calcitonin remained elevated (8.8 μg/l by RIA) and he underwent bilateral neck dissection and external radiotherapy to the neck and mediastinum. He also remains well, with no clinical evidence of recurrence, but the serum calcitonin has risen over 8 years, currently 2735 ng/l (normal <18.9 ng/l by ICMA). *RET* proto-oncogene analysis was performed in 1999 to identify a marker for MTC in this family. No abnormality was found in exons 10 or 11, the two exons then known to harbour causative mutations. Further analysis identified a mutation in exon 15 (codon 891 TCG$>$GCG) in SC, his mother and his twin brother MC. This mutation was not known to be a disease marker at that time (though is now recognised as one), and careful discussion with the family was needed to explain the interpretation of this finding. MC underwent thyroidectomy, had C-cell hyperplasia and remains well with normal serum calcitonin. Other family members have been screened for the mutation; positive individuals have undergone thyroidectomy. This family highlights the issues which arise when a novel mutation, not yet recognised as a disease marker, is used to advise family members. Despite a multidisciplinary approach involving endocrinologist, surgeon, radiotherapist, geneticist and biochemist, optimal management of a patient with a raised serum calcitonin level after thyroidectomy is not clear. SC has received extensive treatment without obvious benefit, whereas his mother has had no further treatment and is well, but both have residual disease. Is there any further intervention which will help these individuals?

Poster Presentations

P1

Can random urinary 5-hydroxyindolacetic acid/creatinine results and 24 hour urinary 5-hydoxyindolacetic acid levels predict carcinoid patient survival?

Simon May[1], Robert Peaston[2] & Petros Perros[2]
[1]University of Newcastle upon Tyne, Newcastle upon Tyne, UK; [2]Freeman Hospital, Newcastle upon Tyne, UK.

The use of 24 h urinary 5-hydroxyindolacetic acid (5-HIAA) concentrations to detect carcinoid patients is a well established procedure while random urinary 5-hydroxyindolacetic acid/creatinine ratio (5-HIAA/cr) may also offer a simpler approach in the diagnosis of carcinoid tumours. However, there is uncertainty regarding the ability of these tests to offer a prognostic indicator for patients.
We retrospectively reviewed the initial paired urinary 5-HIAA/cr and 24 h urinary 5-HIAA results for 176 carcinoid patients diagnosed between 2005 and 2008. The mortality and dates of death for this population were investigated and Kaplan–Meier survival plots (0, 180, 360, 540 and 720 days) created for different values of the two tests. During this time the mortality status of 137 patients were collected, of which 28 (20.4%) had died at the end of the study.
For both the 24 h urinary 5-HIAA and spot urinary 5-HIAA/cr tests survival rates decreased with increasing values in the respective initial tests. However, in both tests they did not become significant until 720 days follow up and it was only for the highest value groups. The initial 24 h urinary 5-HIAA group for values $\geqslant 100$ had a 0.61 (CI 0.43–0.77) survival at 720 days compared to a survival rate of 0.86 (CI 0.74–0.94) for the $\leqslant 20$ and 0.86 (CI 0.74–0.94) for the 21–99 value groups. Initial spot urinary 5-HIAA/cr patients with an initial value of $\geqslant 10$ had a 720 day survival of 0.52 (CI 0.3–0.7) compared to a 0.92 (CI 0.80–0.97) survival for values $\leqslant 1$ and a survival rate of 0.86 (CI 0.74–0.94) for the 2–9 group.
This data suggests that differing values for both 24 h urinary 5-HIAA and spot urinary 5-HIAA/cr tests may predict different survival rates at 2 years follow up in carcinoid patients.

P2

A comparsion of random urinary 5-hydroxyindolacetic acid/creatinine results with 24 hour urinary 5-hydroxyindolacetic acid values in carcinoid patients

Simon May[1], Robert Peaston[2] & Petros Perros[2]
[1]University of Newcastle upon Tyne, Newcastle upon Tyne, UK; [2]Freeman Hospital, Newcastle upon Tyne, UK.

Although 24 h urinary 5-hydroxyindolacetic acid (5-HIAA) concentration is a well established test for the detection and monitoring of carcinoid tumours, compliance, adequacy of collection and the influence of dietary sources of serotonin are problems associated with this approach. Recently, a random urinary 5-hydroxyindolacetic acid creatinine ratio (R/5-HIAA/cr) test has become available which can be used in outpatient departments, however its correlation to 24 h 5-HIAA (24/5-HIAA) values remains uncertain.
We retrospectively analysed 1113 paired 24 h urinary 5-HIAA and spot urinary 5-HIAA/cr from 176 patients (mean age 61 (S.D. 12.99, 52% male) with carcinoid tumours diagnosed between 2005 and 2008.
The correlation between the two tests was examined using Pearson's product moment correlation co-efficient (r). Overall a relatively strong correlation existed between the tests ($r=0.79$, $P<0.0001$). When the results were limited to the first paired sample from each patient (to remove the confounding effect of repeated measurements) the correlation for the overall sample ($r=0.83$, $P<0.0001$), for men ($r=0.87$, $P<0.0001$) and for women ($r=0.81$, $P<0.0001$) remained strong. Interestingly, the correlation between the tests was stronger for ages <55 ($r=0.99$, $P<0.0001$), 55–64 ($r=0.92$, $P<0.0001$) and >75 ($r=0.93$, $P<0.0001$). However, the 65–75 group had a reduced correlation ($r=0.41$, $P<0.05$). Whilst this studied highlights the poorer correlation in the 65–75 age range it is not clear why these results were observed.
In conclusion, we believe that there is a associated correlation between these two tests and that random urinary 5-HIAA/cr tests collected in the out-patients could be substituted for 24 h urinary 5-HIAA tests in carcinoid patients.

P3

A genetic cause for primary amenorrhoea

Marcus Martineau & Masud Haq
Northwick Park Hospital, London, UK.

Primary Amenorrhoea is usually the result of a genetic or anatomical abnormality. Androgen insensitivity syndrome (AIS) is an uncommon cause in which individuals with a 46XY male karyotype are resistant to testosterone due to a defect of the androgen receptor.
A 16-year-old female of non-consanguineous parents presented with primary amenorrhoea. There was no family history of delayed puberty. She was of normal female appearance (height $5'9''$, BMI 19 kg/m^2). However, both axillary and pubic hair was absent and she had underdeveloped breasts (Tanner Stage 2). External genitalia appeared normal with no clitoromegaly. Her vaginal canal was 5 cm in length.
Biochemical screening revealed elevated levels of LH 38.4 U/l (3–16), FSH 12.9 U/l (0.5–8) and testosterone 17.7 nmol/l (1–2.5) but low levels of serum oestradiol 138 pmol/l. Androstenedione, DHEAS, 17-OH progesterone, prolactin and thyroid function were all normal. A pelvic US confirmed absence of both uterus and ovaries. Subsequent MRI failed to identify any obvious gonadal tissue. Chromosomal screening confirmed a 46XY karyotype.
The findings were consistent with a diagnosis of complete androgen insensitivity syndrome (AIS), an X-linked recessive disorder with an incidence of approximately 1 of 20 000 births. The patient received psychological counselling and support from the AIS Support Group (AISSG, Registered UK Charity) and has coped well with her diagnosis returning to full time education. She has been commenced on low dose oestrogen to enhance her breast development and is being treated with non-surgical pressure dilation to her vaginal canal. Surgical exploration is planned to identify and remove remnant gonadal tissue.

P4

Management of a phaeochromocytoma in pregnancy

Barbara McGowan[1], Catherine Williamson[1,2], Karim Meeran[1], Anita Banerjee[1,2], Lee Min[1,3], Bill Fleming[1,3], Duncan Bassett[1] & Tricia Tan[1]
[1]Department of Investigative Medicine, Imperial College, London, UK; [2]Institute of Reproductive and Developmental Biology, Hammersmith Hospital, London, UK; [3]Department of Surgery, Hammersmith Hospital, London, UK.

A 30-year-old lady was transferred to our hospital at 37 weeks of pregnancy. Hypertension was noted at 24 weeks of gestation and managed with methyldopa and labetalol. Despite treatment, BP was labile with a systolic of 90–220 and diastolic of 50–129 mmHg. She reported occasional palpitations but not chest pain or shortness of breath. The patient had a history of migrainous headaches but not hypertension prior to pregnancy. Her brother had previously had an operation for a benign abdominal lesion. Abdominal ultrasound demonstrated a 5.2×5.7 cm mass superio-posteriorly to the right kidney. Twenty-four hour urine noradrenaline excretion was grossly elevated at 4470 nmol/24 h (non-pregnant normal range 0–560 nmol/24 h). Adrenaline and dopamine levels were normal. A diagnosis of a right-sided phaeochromocytoma was made. On admission, labetalol and methyldopa were stopped. She was hydrated with IV fluids and treated with phenoxybenzamine 0.5 mg/kg over 2–4 h for 3 consecutive days. She developed a tachycardia and was commenced on propranolol 40 mg tds. An elective Caesarean section at $37\frac{1}{2}$ weeks performed under continued IV alpha and beta blockade resulted in the delivery of a healthy and normotensive baby girl. Intravenous phenoxybenzamine was discontinued 2 days after delivery and oral phenoxybenzamine commenced. She was discharged home 5 days later on phenoxybenzamine 10 mg tds and propanolol 40 mg tds. Post-operatively she remained asymptomatic with lying and standing BPs of 130/70 and 115/80 respectively. An MIBG scan 3 weeks later showed avid uptake consistent with a right-sided phaechromocytoma. Calcium and calcitonin were normal. Adrenalectomy was performed 5 weeks post-partum. She remains normotensive after withdrawal of antihypertensive medication, and catecholamine excretion levels remained normal. Genetic testing revealed that both she and her brother carry a germline mutation in SDH-B.

P5

Temporary extracorporeal jugulo-femoral venous bypass under local anaesthesia to relieve SVC obstruction prior to total thyroidectomy

Mary Sligo, Duncan Farquhar-Thomson, David Cove & Nicholas Lagattolla
Dorset County Hospital, Dorchester Dorset, UK.

The induction of general anaesthesia and initiation of positive pressure ventilation in cases of superior vena caval (SVC) obstruction carries an unacceptably high risk of cerebral venous congestion, and with it, the risk of cerebral oedema and death. Two similar cases have been dealt with successfully in our unit: both had

thyrotoxic retrosternal multinodular goitres and SVC obstruction, and both had tracheal compression mandating surgery.
Following control of the hyperthyroidism by our medical colleagues (without improvement of the symptoms of compression), the patients were submitted to surgery. The patients were fully prepared for thyroidectomy with additional exposure of their chest, abdomen and groins. Under local anaesthesia, the left internal jugular vein and the left sapheno-femoral junction were exposed and controlled. After full heparinisation, an 8 mm diameter armoured PTFE graft was anastomosed to each using 5/0 prolene sutures, the graft remaining extracorporeal. Duplex was used to insonate the ipselateral common femoral vein before and after clamp removal confirming considerably augmented venous return via the graft.
With the SVC obstruction thus considerably relieved, we enabled safe induction of general anaesthesia and positive pressure ventilation. Both patients underwent uneventful total thyroidectomy though cervical incisions alone, with minimal blood loss. The grafts were removed at the end of the procedure. Both patients recovered without mishap with compression symptoms and signs abolished.
We recommend this simple technique over the insertion of endovenous stents, which can be difficult to place satisfactorily in the presence of massive compressing goitres, in the situation of SVC obstruction caused by retrosternal goitres requiring surgery.

P6

This abstract appears as OC4.

P7

This abstract appears as OC6.

P8

Clinical diagnosis of phaeochromocytoma leads to correct perioperative management despite negative biochemical and functional investigations

Annabel Fountain[1], Jeannie Todd[1], Karim Meeran[1], Fausto Palazzo[1] & Stephen Robinson[2]
[1]Hammersmith Hospital, London, UK; [2]St Mary's Hospital, London, UK.

We present a 44-year-old female referred to us with a two year history of episodic palpitations, chest tightness, headaches and pallor associated with hypertension. She had previously been extensively investigated by neurologists and cardiologists including MRI of the brain, renal ultrasonography, 24 h tape and echocardiography – all normal. Given the history, a CT of the adrenals was performed in 2007 which revealed a 1.5 cm nodule in the right adrenal with abnormal enhancement. The left adrenal was normal. Several 24-h urine collections for catecholamines were done to investigate the possibility of phaeochromocytoma and revealed normal levels apart from a borderline urinary adrenaline on one occasion. Twenty-four hours urinary free cortisol and overnight dexamethasone suppression tests were also normal. As she had persistent and troubling symptoms, CT scan was repeated in February 2008 which showed that the adrenal nodule had increased to 2.2 cm over the preceding 9 months. MIBG scan was performed to assess activity and showed no abnormal uptake. MRI in May 2008 confirmed abnormal enhancement and the nodule was now 3 cm in diameter. With the mass enlarging and the symptomatology it was elected that this lady should undergo right laproscopic adrenalectomy. Despite the lack of positive biochemical and functional imaging findings for a phaeochromocytoma, the history led us to recommend prior alpha-blockade in the form of oral phenoxybenzamine and atenolol and perioperative intravenous phenoxybenzamine. This lady underwent right adrenalectomy on 27th May 2008. Intraoperatively it was noted by the anaesthetist that her blood pressure was labile when the tumour was being handled. Histology showed a well-circumscribed, encapsulated, nodular central lesion which stains positively for chromagranin, synaptophysin and PGP9.5, negative for inhibin and Melan-A, therefore diagnostic for phaeochromocytoma. In conclusion, this case highlights the importance of the clinical history when making a diagnosis as biochemical and functional testing are not 100% reliable. We recommend that with a clinical history typical for phaeochromocytoma despite lack of biochemical and functional evidence, alpha-blockade should be considered prior to surgery.

P9

Challenges in the management of Cushing's syndrome in the severely ill patient

A E C Fountain[1], B M C McGowan[1,2], O Chaudhuri[1], S Saha[1], B C T Field[1,2], W Dhillo[1], J F Todd[1], A P Goldstone[1], N M Martin[1,2], K Meeran[1,2] & T Tan[1]
[1]Hammersmith Hospital, London, UK; [2]Charing Cross Hospital, London, UK.

We present a 57-year-old female with Cushing's syndrome characterised by new type 2 diabetes, hypertension, weight gain, bruising, proximal myopathy and depression. She also had poorly-healing cellulitic ulcers on both legs. *Investigations*: hypokalaemia and ACTH-dependent Cushing's syndrome. Low dose dexamethasone suppression test: $T=0$ ACTH 85 ng/l, cortisol 907 nmol/l, $T=48$ h cortisol 807. High dose dexamethasone suppression test failed to suppress cortisol $<50\%$ baseline, with $T=0$ cortisol 1372 and $T=48$ h cortisol 825. MRI pituitary: left sided microadenoma. CT chest, abdomen and pelvis: no potential ectopic source of ACTH. In view of significant hypokalaemia, and failure to suppress cortisol during a HDDST, an ectopic ACTH source remained a differential diagnosis to Cushing's disease. Inferior petrosal sinus sampling was not possible because she was too unwell: 1. Psychiatric problems: paranoia, agitation, obsessive-compulsive behaviour, anorexia and severe. 2. Sepsis (cellulitis and perforated sigmoid diverticulum): requiring laparatomy, which in turn was complicated by wound dehiscence. 3. Inadequate control of cortisol production despite maximal ketoconazole and metyrapone treatment, cortisol levels remained >1000 nmol/l. She needed continuous intravenous potassium replacement. We turned to subhyponotic etomidate administered on ITU. Initially this was successful in reducing serum cortisol to around 200 nmol/l but, within 2 weeks, her cortisol production escaped from etomidate control (5 mg/h). We added ketoconazole to the etomidate with still inadequate control of cortisol (approximately 500 nmol/l). Bilateral adrenalectomy as an emergency treatment was considered but it was felt that her previous peritonitis and impaired wound healing would jeopardize it's success. It was decided to perform trans-sphenoidal hypophysectomy despite the lack of a definitive diagnosis. Histology confirmed a corticotroph adenoma. Serum cortisol level off hydrocortisone in the early post-operative period was 168 nmol/l. This case highlights the following points: A. The use of etomidate as an inhibitor of steroidogenesis, and its place in the management of Cushing's syndrome. B. The timing of surgery for Cushing's syndrome, in particular how long should biochemical control of hypercortisolism be maintained before surgery? C. The inaccuracy of the high dose dexamethasone suppression test.
Hypophysectomy despite the lack of a definitive diagnosis. Histology confirmed a corticotroph adenoma. Serum cortisol level off hydrocortisone in the early post-operative period was 168 nmol/l.
The presentation will discuss the following points:
A. The use of etomidate as an inhibitor of steroidogenesis, and its place in the management of Cushing's syndrome.
B. The indication and timing of surgery for Cushing's syndrome, in particular how long should biochemical control of hypercortisolism be maintained before surgery?
C. The Mental Capacity Act and how this legislation affects the management of ill patients with neuropsychiatric manifestations of Cushing's syndrome.

P10

Positive correlation between radioisotope and CT/MRI imaging techniques in functioning adrenal adenomas may obviate the need for invasive adrenal vein sampling

Annabel Fountain, Vladimir Vaks & Alison Wren
Chelsea & Westminster Hospital, London, UK.

A 48-year-old Afro-Caribbean female was referred from Accident & Emergency in December 2007 with low serum potassium. She had been diagnosed elsewhere with hypertension due to primary aldosteronism at the age of 28 but was then lost to Endocrine follow-up. She reported intolerance to spironolactone – blood pressure was controlled with Amiloride until 1999 and then Amlodipine. Questioning in clinic, January 2008, revealed that she had had extensive investigations when previously assessed including CT, MRI and radioisotope scans, but these were

unavailable. In March 2008, she complained of tiredness and weight gain, easy bruising and oligomenorrhoea over the preceding year. *Investigations 2008*: aldosterone:renin ratio > 4200. *MRI abdomen*: 3 cm right adrenal adenoma, *Iodocholesterol scan*: increased tracer uptake in the region of the upper pole of the right kidney with no tracer uptake of the left adrenal consistent with hyperfunctioning right adrenal adenoma. *Low dose dexamethasone suppression test (LDDST)*: $T=0$ 304 nmol/l, $T=+24$ 251 nmol/l, $T=+48$ 232 nmol/l with suppressed ACTH < 5 ng/l, supportive of adrenal Cushing's syndrome. Twenty-four hours urinary cortisols were raised: 328 and 461 nmol/l, 24 h urinary catecholamines were normal. It was recommended that the 3 cm adenoma be removed surgically. The gold standard investigation for lateralising an adrenal source of aldosterone or cortisol is adrenal vein sampling (AVS). However, as the iodocholesterol scan showed suppression of the contralateral adrenal gland, it was extremely unlikely that the source of aldosterone and cortisol was the left adrenal. The patient was not submitted for (AVS) as she was extremely anxious and did not wish to undergo invasive testing. She underwent right adrenalectomy 23.09.2008, with steroid cover. Histology confirmed a benign adrenal cortical adenoma. She was discharged on hydrocortisone 10, 5, and 5 mg, fludrocortisone 50 mcg daily and amlodipine 10 mg od. She no longer requires potassium replacement. She will undergo Short SynACTHen test on 22.10.2008 to establish whether she can discontinue her hydrocortisone therapy. If she passes this, she will have a LDDST to confirm cure of her Cushing's syndrome. This case illustrates that, although adrenal vein sampling is the gold standard, the presence of localising imaging of more than one modality may be sufficient to proceed to surgery.

P11

Treatment resistant acromegaly

Paul Grant
Royal Sussex County Hospital, Brighton, UK.

The issue of treatment-resistant pituitary tumour growth remains relatively under-explored. We describe the case of a gentleman whose diagnosis and management involved several medical disciplines and his management proved challenging over the long term.

A 47-year-old gentleman who was seen by a variety of specialists before and after diagnosis for the complications of his aggressive acromegaly.

Initially seen in 1997 by an Occupational Health Physician as he complained of enlargement of both hands making his work difficult. He was subsequently referred on to a Urologist because of erectile dysfunction. The urologist queried the diagnosis of diabetes and arranged for him to have an OGTT and see a Diabetic nurse specialist. The DSN found that he had impaired glucose tolerance and gave diet and lifestyle advice.

He was subsequently reviewed by his General Practitioner who thought that he may have an underlying endocrinopathy and referred him on to see a Cardiologist for features of heart failure, and…

An endocrinologist who diagnosed him with Acromegaly. He was found to have a large pituitary macro-adenoma which had eroded the pituitary fossa and cavernous sinus.

He was seen by a Neurosurgeon who performed two trans-sphenoidal debulking operations with mixed results. He went on to have Radiotherapy administered by Nuclear Medicine.

Following these treatments he was still found to have active disease with elevated IGF-1, despite medical management with Octreotide. The patient subsequently declined further radiotherapy due to concerns about cognitive decline and his tumour was not felt to be amenable to further neurosurgery. He is due to be considered for treatment with Pegvisomant (a growth hormone receptor antagonist).

There exists a clear need to study the causes and management of treatment-resistant Acromegaly, particularly in terms of tumour progression. Well-designed MRI studies in patients potentially at risk from an aggressive tumour would be very useful to verify the true rates of therapeutic failure across all treatment modalities.

P12

Cushing's syndrome secondary to adrenocortical carcinoma without metastases at diagnosis

Alexander Miras[1,2], Fausto Palazzo[1,2] & Safdar Naqvi[1,2]
[1]St Peter's Hospital, Chertsey, Surrey, UK; [2]Hammersmith Hospital, London, UK.

A 60-year-old gentleman of previously good health presented with a 6 months history of resistant hypertension, newly diagnosed Diabetes Mellitus, worsening obesity and proximal muscle weakness. Clinical examination revealed a buffalo hump, truncal obesity, paper thin skin with bruising and proximal muscle wasting. His biochemistry showed elevated midnight cortisol levels, no suppression of cortisol post high dose dexamethasone and an undetectable ACTH. Accompanying abnormalities included low gonadotrophin levels, persistent hypokalaemia and derangement of the liver enzymes. A CT scan of his chest, abdomen and pelvis showed two masses in the right adrenal measuring $7.2\times6.2\times5.7$ cm and 2.8 cm in diameter respectively. There was no evidence of surrounding structure invasion but considerable intraabdominal and intrahepatic fat was noted. The diagnosis of ACTH independent Cushing's syndrome secondary to adrenal tumours was made. The patient was initially treated with Ketoconazole but due to further deterioration of his liver function was switched to Metyrapone which was titrated to suppress cortisol levels for 6 weeks. Oral hypoglycaemic agents and potassium replacement were also added to the treatment regime. A laparoscopic adrenalectomy was attempted but due to the size of the adrenal masses the procedure was converted to an open one. The right adrenal was successfully excised and there was no intraoperative evidence of surrounding structure invasion. Three weeks post operatively the doses of his antihypertensives were reduced, the oral hypoglycaemic agents stopped and he had normal cortisol responses. Histology of the excised mass was compatible with adrenal cortical carcinoma. He was started on Mitotane. His prognosis is expected to be good in the absence of metastatic spread which is found at presentation in 75% of patients with adrenal carcinoma.

P13

Unusual hypoglycaemia: real or factitious?

Sanjeev Sharma, Francesca Swords & Nicoletta Dozio
Norfolk & Norwich University Hospital, Norwich, UK.

Factitious hypoglycaemia is characterised by high insulin levels but accompanied with low Proinsulin and C-peptide levels and a negative sulfonylurea screen

We present a 54-year-old woman who was initially diagnosed with type 2 diabetes in 2002 and by 2004, she was converted to insulin treatment due to poor tolerance to Metformin. She also had a previous history of Manic depressive psychosis and treated with lithium.

In the months prior to her admission, due to persistent hypoglycaemic episodes, her insulin requirement had to be dramatically reduced from a total of 60 to 15 units/day.

She was admitted with repeated, severe episodes of neuroglycopenia, and after stopping all forms of insulin, was monitored as an in-patient. Her 4-hourly blood glucose level remained between 3 and 7 mmol/l and was interspersed with further hypoglycaemic episodes. During one such episode, her blood glucose was as low as 1.4 mmol/l but her concomitant plasma insulin levels was < 2 pmol/l (Perkin Elmer insulin immunoassay; normal = 0–60 pmol/l) and C-peptide was 46 pmol/l (174–960 pmol/l). Urinary screen for sulfonylurea was negative. Insulinoma investigated with a dynamic CT scan of the pancreas and IFG-2:IGF-1 ratio 2.2 (normal: < 10). She bafflingly continued to have dramatic and sustained hypoglycaemia despite not receiving any insulin. At a subsequent hypoglycaemic episode with blood glucose of 1.2 mmol/l, standard methods did not detect any insulin but an alternative assay method (Delfia insulin assay) showed insulin Aspart levels of > 200 pmol/l.

Subsequent follow up revealed that she had been working at the same mental care home, where-in she previously was a patient. Her hypoglycaemic symptoms have now ceased and she is doing well on Metformin alone. At this modern age of insulin analogues, this fascinating story reminds us the need of awareness about the complexities of insulin assays, and their importance in diagnosing factitious hypoglycaemia.

P14

This abstract appears as OC7.

P15

An unusual case of a gland in the neck

Tee Wei Siah, Binu Krishnan, Emma Bingham & Jennifer Tringham
Frimley Park Hospital, Surrey, UK.

A 23-year-old man initially presented to his GP with palpable 'gland in his neck' for 9 years. He was otherwise well with no medical problems. He has a family history

of hypothyroidism. Examination revealed right submandibular gland enlargement and lymph nodes in the anterior cervical region bilaterally. His TPO antibody was negative and the only abnormal result was a raised TSH of 12.7. He was commenced on 50 μg of thyroxine daily. Ultrasound of the neck showed no normal thyroid gland and an abnormal oval lesion beneath the strap muscles underlying the normal right submandibular gland. This was confirmed on CT scan, which showed another swelling at the base of the tongue. Technetium Thyroid scan showed increased uptake in the region of the right submandibular gland and at the base of the tongue with absence of uptake in the usual region of the thyroid. This confirmed the presence of ectopic thyroid tissue. Ectopic thyroid tissue has been reported to be found at the foramen caecum and along the thyroglossal duct, where its localization can be explained by the embryogenesis of the thyroid gland. The presence of thyroid tissue outside this pathway has been reported in the submandibular region, parotid gland, gallbladder, skin, liver, trachea, mediastinum, heart, lung, duodenum, iris, and adrenal glands and these cannot be explained readily by embryogenesis. It has been suggested that the possibilities for the presence of thyroid tissue in the submandibular region include abnormal migration during the course of embryonal development, spread of tissue during surgery on a normally located thyroid gland and metastasis of thyroid carcinoma. Malignancy in this patient is unlikely because of a negative family history for thyroid cancer and the young age of the patient. This case represents an unusual description of thyroid tissue at right submandibular region and foramen caecum.

P16

Primary hyperparathyroidism and pregnancy

James Kirkby-Bott[1], Catherine Williamson[2], Fausto Palazzo[1], Anita Banerjee[1], Karim Meeran[1] & Tricia Tan[1]
[1]Hammersmith Hospital, London, UK; [2]Queen Charlotte and Chelsea Hospital, London, UK.

Primary hyperparathyroidism (HPT) in pregnancy is an uncommon phenomenon, mostly occurring in the 2nd or 3rd trimester. HPT in pregnancy may cause complications affecting both the mother (renal stones, pancreatitis) and fetus (neonatal tetany, seizures, intra-uterine growth retardation and preterm labour). We report two recent cases that highlight the potential risks.

Results

The patients, 37 and 35 years old at presentation respectively, both presented with symptomatic hypercalcaemia. Diagnosis was proven biochemically. One had undergone localisation studies at another centre pre-pregnancy and fallen pregnant during work-up.

Patient 1 only had pre-op ultrasound localisation showing an enlarged right inferior gland. Patient 2 who presented pre-pregnancy had had a negative sestaMIBI performed at another centre and an ultrasound showing two likely adenomas. At surgery 5 gland hyperplasia was found. Patient 1 underwent minimally invasive parathyroidectomy and a 4.8 g adenoma was removed. She has persistent hyperparathyroidism and re-localisation *post partum* localises a left inferior gland. She is awaiting a bilateral re-exploration. Both patients are undergoing genetic screening. Both went on to have uncomplicated deliveries and healthy babies.

Conclusions

HPT in pregnancy typically affects a young age group where multiple gland disease and inherited syndromes are more common. Localisation studies with USS without sestaMIBI may be misleading. Given the limitations of localisation, a bilateral exploration in the second trimester +/− use of intra-operative PTH assay is advisable. Young women being investigated for hypercalcaemia should be advised to avoid pregnancy until fully investigated and treated. An MDT with endocrinologists, endocrine surgeons, obstetric physicians, radiologists and geneticists is required for optimal management.

P17

A case of frontal ethmoid sinus mucocele: a late complication of pituitary surgery?

Elaine Hui[1], Anjali Amin[1], Sandro Chiti-Batelli[2], Pooja Patel[3], Nick Linton[4] & Jonathan Valabhji[1]
[1]Department of Metabolic Medicine, St Mary's Hospital, Imperial College Healthcare NHS Trust, London, UK; [2]Department of Otolaryngology-Head-Neck Surgery, St Mary's Hospital, London, UK; [3]Department of Neurology, St Mary's Hospital, London, UK; [4]Department of Cardiology, St Mary's Hospital, London, UK.

A 69-year-old Iraqi lady presented with a 4-day history of diplopia, headache and right eye swelling. She had a pituitary adenoma diagnosed in Beirut when she was 20 years old and underwent trans-frontal surgeries and radiotherapy twice and further transsphenoidal surgery in 1980. She was on prednisolone 2 mg + 1 mg and thyroxine 50 mcg daily.

She initially presented to the ophthalmology clinic. Examination revealed a pupil-sparing 3rd nerve palsy of the right eye, with complete ptosis, proptosis and reduced visual acuity (6/9). She was referred to the medical team. CT head demonstrated a 2 × 1.5 cm homogeneous soft tissue mass in the medial aspect of the right orbit. MRI revealed a well-circumscribed mass arising within the anterior ethmoid air cells extending to the right frontal sinus. The pituitary fossa was enlarged but empty. IGF-1, prolactin and free T4 levels were normal. A multidisciplinary discussion involving radiologists, ophthalmologists, endocrinologists and otolaryngologists concluded that the MRI appearance was suggestive of a frontal ethmoid mucocele. Evacuation of the right frontal mucocele was performed by the otolaryngologists via endoscopic approach with antibiotics and steroid cover. She had complete resolution of the diplopia, ptosis and proptosis and visual acuity improved (6/6) post-operatively. Unfortunately, she had uncontrolled atrial fibrillation post-operatively and a positive troponin I of 0.09 μg/l (<0.04 μg/l). She was treated as acute coronary syndrome and subsequent coronary angiography did not show any significant coronary artery disease. She was discharged without further complication.

Mucoceles are slow-expanding cystic lesions with respiratory epithelium containing mucus caused by obstruction of sinus ostium[1]. They can extend intraorbitally and intracranially[2]. Mucocele formation is a rare complication of transsphenoidal surgery. There is no reported case following trans-frontal surgery. We reported a case of frontal ethmoid mucocele presented 50 years after trans-frontal surgery to a pituitary adenoma.

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P18

This abstract appears as OC1.

P19

Young hyperparathyroidism: to localize or not

Sarah Skennerton, George Tharakan, Niamh Martin, Karim Meeran & Daniel Morganstein
Imperial College Healthcare NHS Trust, London, UK.

A 37-year-old lady was referred to the endocrine clinic with a raised serum calcium of 2.73 mmol/l in the presence of an inappropriately raised parathyroid hormone of 8.7 pmol/l. Calcium:creatinine clearance ratio was elevated at 0.018, excluding familial hypercalcaemic hypocalciuria. Past medical history revealed an episode of renal stones 17 years ago but a renal ultrasound excluded nephrocalcinosis.

A diagnosis of primary hyperparathyroidism was made.

Sporadic hyperparathyroidism is due to a single adenoma in around 80% of cases so pre-operative localization studies are frequently performed to enable minimally invasive approaches. However the patient's young age of onset raises the possibility of an inherited predisposition, for example MEN 1 or 2.

Parathyroid glandular involvement is more diffuse in MEN than in sporadic hyperparathyroidism and removal of all four glands is therefore the preferred option. Patients are however rendered permanently hypoparathyroid and dependent on lifelong alphacalcidol with calcium supplements. Therefore the patient was referred for genetic and biochemical screening for MEN, as a positive result would dramatically alter the surgical management and render pre-operative localization redundant.

This case study highlights the importance of considering MEN1 or 2 syndromes in young patients presenting with primary hyperparathyroidism prior to localization studies or surgery.

P20

Lymphocytic hypophysitis secondary to a ruptured Rathke's cleft cyst (RCC): a diagnostic and management challenge

Puja Mehta[1], Frederico Roncaroli[2], Amrish Mehta[2], Maneesh Bhojak[2], James Lawrence[3], Emma Hatfield[2], Karim Meeran[1] & Waljit Dhillo[2]
[1]Hammersmith Hospital, London, UK; [2]Charing Cross Hospital, London, UK; [3]Salisbury District Hospital, Salisbury, UK.

Hypophysitis describes inflammatory pituitary lesions which can be classified into lymphocytic (autoimmune), granulomatous or xanthomatous. Rathke's cleft cysts (RCC) are usually assymptomatic, benign tumours derived from remnants of Rathke's pouch. We present a case of lymphocytic hypophysitis secondary to a ruptured RCC and the first reported case of post-surgical recurrence.

A 34-year-old female presented with secondary amenorrhoea and fatigue. There were no features of infection, autoimmunity or granulomatous disease. Investigations revealed panhypopituitarism and she was prescribed replacement corticosteroids, thyroxine and HRT. She subsequently developed diabetes insipidus and was commenced on desmopressin. MRI scans showed expansion of a peripherally enhancing pituitary mass abutting the optic chiasm and transsphenoidal hypophysectomy was performed. Post-operative imaging demonstrated initial tumour debulking and then re-growth at 14 months with chiasmal compression, requiring a second hypophysectomy. Review of the histology confirmed a lymphocytic hypophysitis with underlying ruptured RCC. Her most recent MRI scan (7 months after the second hypophysectomy) demonstrated further expansion of the pituitary mass, and a trial of high-dose prednisolone has been commenced, with repeat MRI planned to assess response. If there is a sub-optimal response, we will consider further surgery and/or radiotherapy.

The contents of a ruptured RCC is thought to drive a sterile inflammatory or autoimmune process. There have been nine cases in the literature of hypophysitis associated with a ruptured RCC, only three of these cases involved lymphocytic infiltrates. We describe the fourth reported case, who required multidisciplinary team management. Our patient is the first case to exhibit recurrence after surgical debulking. Treatment options include high dose steroids, surgery and radiotherapy, although there is no consensus in the literature regarding management.

P21

This abstract appears as OC2.

P22

Primary adrenocortical insufficiency despite a 'normal' short synacthen test

S R Mehta, B C T Field, O B Chaudhri, H Shaikh, D L Morganstein, N M Martin, E C I Hatfield & K Meeran
Endocrine Unit, Department of Investigative Medicine, Imperial College Healthcare NHS Trust, London, UK.

A 60-year-old gentleman who had previously undergone a right nephrectomy for renal cell carcinoma was admitted electively for a left adrenalectomy due to metastatic disease. Prior to this he had been treated with immunotherapy (Sunitinib) and radiotherapy for pulmonary and bony metastases respectively. He was given perioperative cover with hydrocortisone. A short synacthen test (SST) performed the morning after discontinuing hydrocortisone showed a baseline cortisol of 406 nmol/l rising to 469 nmol/l at 30 min and 555 nmol/l at 60 min. He was clinically euadrenal, so it was decided that he did not require further hydrocortisone. He was discharged home 3 days later. Three weeks later, he presented with profound nausea and tiredness. On examination he had postural hypotension. Plasma sodium was 129 mmol/l, potassium 4.4 mmol/l and random cortisol 689 nmol/l. He was started on hydrocortisone 20 mg, 10 mg, 10 mg and fludrocortisone 100 mcg once daily. His symptoms resolved over the next 48 h, and the hydrocortisone was reduced to 10 mg, 5 mg, 5 mg prior to review in endocrinology out-patients. At out-patient review, it was noted that the baseline ACTH from his earlier SST was elevated at 52.0 ng/l suggesting subclinical hypocortisolaemia. The dose of his hydrocortisone was increased to 15 mg, 10 mg, 5 mg, as he is about to start further immunotherapy (Bevacizmab). We present a case of what we believe to be adrenal toxicity due to Sunitinib, with clinical findings and investigations suggesting predominantly mineralocorticoid deficiency. Adrenal toxicity with Sunitinib has been reported previously in animals but not humans. Such subclinical toxicity may be difficult to detect unless unmasked by physiological stressors. We highlight the importance of knowing both a baseline ACTH and peak cortisol when interpreting a SST, and also the difficulty in determining what is an appropriate random cortisol in an acutely unwell patient. Another potential explanation for the discordance between cortisol results and his clinical state is abnormal cortisol binding globulin levels, which must be considered.

P23

The need for a coordinated multidisciplinary approach in the management of morbid obesity

Leena Mukherjee, Sufyan Hussain, John Flood & Rashmi Kaushal
West Middlesex University Hospital, London, UK.

We describe the case of a 22-year-old South Asian male presenting to Endocrine Services with morbid obesity (BMI > 50), sexual immaturity and agoraphobia. Born at full term following an uncomplicated pregnancy, he reached all developmental milestones appropriately. His problems began aged 15 after witnessing a murder, with reactive depression and hyperphagia. By the age of 18, he weighed over 140 kg, had poor self-image and suicidal ideation. He was managed initially by several multidisciplinary teams, including the Eating Disorders Psychologists, Psychiatrists and Dieticians, although the emphasis was on only his psychiatric condition with little communication between the specialties.

On referral to the Endocrine Service, he was clearly hypogonadal with a BMI of 64. Investigations confirmed hypogonadotrophic hypogonadism, (LH 1 IU/l, FSH 5 IU/l, Testosterone 0.6 nmol/l) with a normal karyotype. The remainder of his pituitary and adrenal function was normal. An MRI of the pituitary was also normal. His serum leptin was 63.4 ng/ml, insulin 116 pmol/l, pro-insulin 12 pmol/l and split pro-insulin 10 pmol/l.

Despite being known to several multidisciplinary teams for his co-morbidities, his weight continued to soar. He developed severe sleep apnoea, hypertension, arthritis and finally turned to heavy alcohol consumption.

It became clear that the only way forward was to establish organised communication between the teams. With diabetic education and anti-obesity medication he was able to lose 20 kg in weight. He was then referred to a tertiary Obesity Clinic weighing 177.6 kg (BMI 69). With organised contribution from all the specialties involved, a gastric banding procedure was eventually performed, resulting in a 12-month weight loss of 40.6 kg (BMI 53.5).

At 28 years of age, his management is currently shared by Endocrinology and Gastrointestinal Surgery. Plans for an jejunoileal bypass have been agreed with support from the Morbid Obesity team consisting of a Dietician, Psychologist, Consultant Physician, Psychiatrist and Fitness Instructor.

P24

Surveillance of a slowly progressive non-benign adrenal incidentaloma

Debbie-Ann Charles, Yuk-Fun Liu, Audrey Jaques & Paul Carroll
Guy's and St Thomas' Hospital NHS Foundation Trust, London, UK.

Adrenal incidentalomas are tumours of the adrenal incidentally discovered during evaluation for non-adrenal disease. The National Institute of Health consensus definition usually excludes lesions found during evaluation for malignancy. We present a case of an initially non-secretory adrenal incidentaloma that progressively increased in size over 6 years and became hormonally active.

A 78-year-old hypertensive male was found to have a right adrenal mass during investigation for a rectal carcinoma. He underwent a successful anterio-posterior resection requiring no adjuvant therapy. Initial surveillance under the surgeons involved 6 monthly abdominal computed tomograms (CT). Over a 2 year period the mass increased in size from 2 to 3 cm in longest diameter. Neither unenhanced CT nor chemical shift MRI (CSI) was compatible with a benign adenoma. He was therefore referred to endocrinology.

He was asymptomatic and normotensive on amlodipine and doxazosin. Initial hormonal evaluation showed that his cortisol circadian rhythm was preserved, 48 h dexamethasone suppression test suppressed to 59 nmol/l and electrolytes and aldosterone:renin ratio was normal. Urinary catecholamines were not elevated. Over the next 4 years, he remained completely asymptomatic. Follow-up imaging demonstrated a well circumscribed mass that slowly but progressively attained a size of 3.4×2.9 cm with a necrotic centre. There was no loss of signal intensity on CSI. Periodic hormonal evaluation showed normal cortisol, androgens and aldosterone production. At his 6 year follow-up urinary catecholamines and metanephrines were elevated. MIBG showed persistent uptake in the right adrenal. Adrenalectomy was performed and histology confirmed a phaeochromocytoma. Unless there is clear radiological confidence that a benign adenoma is present surveillance and re-assessment of functionality is warranted.

P25

Early report of ultrasound vocal cord assessment for patients undergoing cervicotomy

James Kirkby-Bott, Amanda Mortier, Gavin Royle & K Dewbury
Southampton University Hospitals Trust, Southampton, UK.

Introduction
The majority of dedicated endocrine surgery centres in the UK and abroad routinely perform pre-operative vocal cord checks prior to cervicotomy. Ultrasound scanning is increasingly used as routine imaging in the work up of patients being assessed with thyroid and parathyroid disease. We report on the additional use of USS as a method of performing pre-operative vocal cord checks.
Method
We report the early results on 32 patients undergoing USS as an assessment of thyroid status, USS guided FNAC and assessment of lymph node status prior to thyroidectomy. In addition to this all patients had vocal cord function assessed. Whilst in the supine position the vocal folds were assessed during quiet respiration and phonation. The patients then underwent naso-endoscopy to validate the USS vocal cord findings.
Results
Thirty-two patients (26 female 6 male) underwent thyroid surgery. None had thyroid cancer. Thirty patients vocal folds were seen on USS to move normally; this corresponded with normal vocal cord movement on naso-endoscopy. Two patients had calcified thyroid cartilage making USS interpretation too difficult to be reliable.
Conclusion
USS assessment of vocal cord function appears to be possible and feasible in the great majority of cases. Greater numbers are needed to confirm the accuracy of USS in abnormal vocal cord movement and the proportion of patients in whom USS vocal cord assessment is successfully undertaken. However these findings would suggest it is a useful means of assessing vocal cord function prior to cervicotomy in most patients.

P26

Manubrial split provides adequate exposure for mediastinal exploration for parathyroid adenoma

Edward Lake, James Kirkby-Bott, Fawzia Imtiaz, James Jackson & Fausto Palazzo
Hammersmith Hospital, London, UK.

Introduction
The traditional approach to mediastinal ectopic parathyroid adenomas has been through a median sternotomy. With improved localization techniques it has become possible to use less invasive approaches to access the mediastinum. We present our recent experience of three cases of mediastinal parathyroid adenomas successfully treated using a manubriotomy approach which provides good access to the superior and anterior mediastinum.
Method
All patients had pre-operative localization with sestamibi and USS. Where MIBI highlighted a mediastinal gland SPECT MIBI and contrast CT imaging were used to confirm the adenoma's position in the superior/anterior mediastinum.
Results
The diagnosis of primary hyperparathyroidism (HPT) was established using standard departmental protocols. In all three cases the glands were seen on MIBI and CT and localised to a mediastinal compartment. At surgery removal of the thymus and all other fatty tissue in the superior/anterior mediastinum was needed. Frozen section was used to confirm the presence of parathyroid tissue. In all cases parathyroid tissue was demonstrated on permanent section histology and serum calcium and PTH levels promptly returned to within the normal range. All patients made swift recovery with little analgesic requirement.
Conclusion
Although case reports of mediastinoscopic and thoracoscopic removal exist careful and meticulous dissection of the mediastinal compartments is needed to ensure removal of all parathyroid tissue. Traditionally this has been performed using a median sternotomy, but manubriotomy appears to provide a safe curative approach with a short recovery.

P27

Unusual case of hyperpigmentation

Heba El-Gayar & Owais Chaudhri
Imperial College, London, UK.

Cutaneous pigmentation results from the synthesis of melanin by the melanocytes. Its distribution pattern in the surrounding keratinocytes determines the actual colour of the skin. ACTH and α-MSH are equipotent at the melanocortin-1 receptor (MC-1R) that is expressed on the cell surface of melanocytes. Activation of these receptors stimulates both proliferation of melanocytes and melanin synthesis.
We present the case of a 66-year-old lady. Originally from Ghana, she has lived in the UK for over 40 years. She presented with an 18 months history of generalised hyperpigmentation. Cortisol day curve and a random ACTH were both normal. A skin biopsy showed a normal number of melanocytes but with hyperpigmentation of the dermis and epidermis. Levels of α-MSH levels were undetectable in the plasma. MRI pituitary was normal. Imaging revealed an octreotide avid lesion within the mid zone of the left lung. Unfortunately CT thorax following the octreotide scan did not show any corresponding lesion.
Her skin continued to darken. She went back to Ghana for a holiday where unfortunately she was shunned by her family, as there was a belief that she was cursed. She is extremely desperate for a cure however but is not keen on having surgery.
Discussion
Our α-MSH assay is validated for *in vitro* release from hypothalamic explants. Its accuracy on plasma samples is unknown. An α-MSH secreting tumour therefore cannot be excluded. Other options for therapy include octreotide or surgery. However, the success of therapy will be difficult to judge as any improvement in this lady's skin colour is likely to be slow.

P28

A case of frontal ethmoid sinus mucocele: a late complication of pituitary surgery

Elaine Hui, Anjali Amin, Sandro Chiti-Batelli, Pooja Dassan, Nick Linton & Jonathan Valabhji
St Mary's Hospital, London, UK.

A 69-year-old lady presented with a 4-day history of diplopia, headache and right eye swelling. She had a pituitary adenoma diagnosed in Beirut when she was 20 years old and underwent trans-frontal surgeries and radiotherapy twice and further transsphenoidal surgery in 1980. She initially presented to the ophthalmology clinic. Examination revealed a pupil-sparing 3rd nerve palsy of the right eye, with complete ptosis, proptosis and reduced visual acuity (6/9). She was referred to the medical team. CT head demonstrated a 2×1.5 cm homogeneous soft tissue mass in the medial aspect of the right orbit. MRI revealed a well-circumscribed mass arising within the anterior ethmoid air cells extending to the right frontal sinus. The pituitary fossa was enlarged but empty. IGF-1, prolactin and free T4 levels were normal. A multidisciplinary discussion involving radiologists, ophthalmologists, endocrinologists and otolaryngologists concluded that the MRI appearance

was suggestive of a frontal ethmoid mucocele. Evacuation of the right frontal mucocele was performed by the otolaryngologists via endoscopic approach. She had complete resolution of the diplopia, ptosis and protosis and visual acuity improved (6/6) post-operatively. Unfortunately, she had uncontrolled atrial fibrillation post-operatively and a positive troponin I of 0.09 μg/l (<0.04 μg/l). She was treated as acute coronary syndrome and subsequent coronary angiography did not show any significant coronary artery disease. She was discharged without further complication.
Mucocele is a slow-expanding cystic lesion with respiratory epithelium containing mucus caused by obstruction of sinus ostium and can extend intraorbitally and intracranially. It is a rare complication of transsphenoidal surgery. We report a case of frontal ethmoid mucocele presented 50 years after trans-frontal surgery to a pituitary adenoma.

P29

This abstract appears as OC3.

P30

Atypical location and treatment for a rare neuroendocrine tumour
Heba El-Gayar, Daniel Morganstein & Karim Meeran
Imperial College, London, UK.

Somatostatinomas are rare neuroendocrine tumors usually arising from the pancreas and duodenum. Symptoms include hyperglycemia, cholelithiasis, diarrhea and steatorrhoea. Treatment with somatostatin (SST) analogue may appear paradoxical, but can lower SST levels and improve symptoms.
Case
A 60-year-old gentleman presented with diarrhoea, opening his bowels between 12 and 13 times a day causing him to stop working and become depressed. Repeated fasting gut hormone profiles showed an isolated elevated SST levels. CT abdomen did not show any pancreatic lesions and colonoscopy was normal. Octreotide scan revealed a well defined lesion in the base of the right lung, confirmed on a CT thorax. Visceral angiography with calcium stimulation and sampling from the femoral artery revealed elevated SST in all samples (>400 pmol/l). SST levels taken from the right atrium and femoral artery, showed a higher level in the arteries (venous 152 pmol/l, arterial 185 pmol), suggesting a pulmonary source. However, the surgical risks of resection were thought to be high. He had a trial of Octreotide therapy without benefit. Four years later, he was treated with a combination of Lanreotide 30 mg every 14 days, and Octreotide 50 mcg TDS, resulting in a dramatic improvement in symptoms. He now opens his bowels once or twice a day. His symptoms worsened when Octreotide was withdrawn. He also noticed two days prior to the lanreotide injection be opens his bowels more frequently.
Discussion
There are 5 subtypes of somatostatin receptors (SSTR). All five SSTRs bind to the natural SST. Two different SST analogues are used clinically, octreotide and lanreotide. These analogues bind principally to the receptor subtype 2 and 5. Although most studies have shown no major difference between octreotide and lanreotide in terms of receptor affinity or biological activity, this case illustrates that combination therapy may have clinical benefits.

P31

Diagnostic cut-off for calcitonin: is 10 ng/l still valid?
Radha Ramachandran[1], Patricia Benfield[2], Sara White[1], Richard Chapman[2], Karim Meeran[1], Mandy Donaldson[2] & Niamh Martin[1]
[1]Department of Investigative Sciences, Imperial College Healthcare NHS Trust, London, UK; [2]Supraregional Assay Service, Imperial College Healthcare NHS Trust, London, UK.

Aim
Population studies have shown that basal calcitonin concentrations are below 10 ng/l in the normal population. Ten nanograms per liter is used as a diagnostic cut-off by most laboratories and patients with levels higher than this are offered a pentagastrin stimulation test to exclude medullary thyroid carcinoma. However, these guidelines were adopted from studies that used the Cisbio Immunoradiometric assay for measuring calcitonin. Most laboratories have now moved to chemiluminescent assays. Our laboratory uses the Diasorin Liaison chemiluminescent. We assessed the validity of continuing to use 10 ng/l as a diagnostic cut-off for our assay.
Methods
Sixty-one healthy, volunteers (35 female and 26 male) were recruited (07/Q0406/18). Age ranges were 21–64 years (mean = 39.9) for males and 19–58 years (mean = 30.7) for females. A single basal blood sample was collected into plain tubes and allowed to clot. Samples were immediately centrifuged, stored at −20 °C and assayed using the Diasorin Liason Calcitonin assay.
Results
The manufacturers quote a reference range of <5.5 ng/l for females and <18.9 ng/l for males. Our study showed that the upper limit for males (22 ng/l) is similar to the manufacturer's range. However, the value for females (10.8 ng/l) is double the recommendation but is in keeping with observations made in our clinical practice and similar to the reference ranges quoted by manufacturers of most other calcitonin assays. Two out of 35 females and 7 out of 26 males had levels above 10 ng/l.
Conclusion
Our study revealed that 6% of females and 27% of normal males had levels greater than 10 ng/l. This suggests that adhering to a threshold of 10 ng/l for pentagastrin stimulation test will result in a significant number of patients with normal results being subjected to a pentagastrin test unnecessarily. We therefore recommend that the old diagnostic cut-off of 10 ng/l be reviewed.

P32

Primary hyperparathyroidism presenting in pregnancy
Ali Abbara, Varunika Lecamwasam, Kevin Baynes, Richard Bell, Thomas Kurzawinski, Mahadevan Neila, Akkib Rafique & Faris Kubba
Ealing Hospital, London, UK.

A 25-year-old pregnant lady presented to the accident and emergency department at 16 weeks gestation with intractable vomiting, weight loss and lethargy. She reported a 2 months history of hyperemesis gravidarum managed in the community prior to admission.
She had no past medical history and her only medication was of a Polish antenatal vitamin containing 400 IU of Vitamin D (D2). Her blood biochemistry revealed a markedly raised corrected calcium at 3.57 mmol/l (2.15–2.55), inappropriately ↑PTH at 17.6 pmol/l (1.6–6.9), ↓Phosphate 0.7 mmol/l (0.87–1.45), ↑Vitamin D 140 nmol/l (50–80). Fractional excretion of Calcium on a 24 h urine collection was 0.03 (>0.01) excluding familial hypocalciuric hypercalcaemia.
She was treated with intravenous normal saline (4 l/day) for 4 days but unfortunately her calcium only marginally decreased to 3.17 nmol/l. She was reviewed by the obstetricians and no foetal complications were detected. An ultrasound of her parathyroid glands revealed a right inferior pole parathyroid adenoma 1.5 by 1 cm. At 20 weeks gestation, she was referred to a specialist endocrine surgeon and underwent a minimally invasive parathyroidectomy with rapid intraoperative PTH measurement. The operation was successful with a postoperative PTH of 3 pmol/l and cCa 2.23 with no complications. Given that she has presented with hyperparathyroidism at such a young age, she will clearly require screening for MEN-1.
The prevalence of primary hyperparathyroidism in the general population is 0.15% and a quarter of these cases occur in women of childbearing age, however there are only an estimated 200 cases reported in the literature thus far. Symptomatic hyperparathyroidism in pregnancy is ideally managed with surgery in the second trimester. Regardless of whether the hyperparathyroidism is managed medically or surgically, the pregnancy is regarded as high risk and a high degree of vigilance is indicated with a clear multidisciplinary approach. Had the ultrasound scan been negative (sensitivity <50%) in this lady with no clear surgical target, the management may have been even more complicated, especially since sestamibi scanning is contraindicated in pregnancy.

P33

Pituitary stalk haemorrhage

Varunika Lecamwasam, Monica Whittle & Mark Edwards
Hillingdon Hospital, London, UK.

We report the case of a 59-year-old male who presented complaining of several weeks of tiredness, poor appetite and dizziness on standing. He had been treated with flucloxacillin by his GP for otitis externa one week earlier.

In 1982, he was diagnosed with nasopharyngeal carcinoma in Hong Kong, which was treated by radical dissection and radiotherapy.

On examination, his standing and lying blood pressure were 150/100 and 110/90 respectively. There was no mastoid or sinus tenderness. Hormonal evaluation showed a free thyroxine 17.2 pmol/l (normal range 9–22.7 pmol/l), TSH 0.2 mU/l (NR 0.35–5), random cortisol <50 nmol/l (NR >100), ACTH <5 ng/l, FSH 1.7 U/l (NR 3–20), LH 0.1 U/l (NR 1.2–8.6), testosterone <0.6 nmol/l (NR 6.1–27.1) and a prolactin level of 474 mU/l (NR <100). He was immediately commenced on hydrocortisone followed by thyroxine for treatment of hypopituitarism.

MRI of the head on admission revealed a nasopharyngeal mucocoele and a haemorrhagic lesion in the pituitary stalk. He was then transferred to a neurosurgical unit where he underwent biopsy of this lesion via an endoscopic sphenoidectomy. This revealed no recurrence of previous nasopharyngeal tumour. He also had a whole body CT scan which only revealed a small nodule on the left lobe of thyroid gland.

A repeat Pituitary MRI four months later revealed the pituitary stalk haemorrhage had completely resolved.

Pituitary apoplexy is commonly due to ischaemia or necrosis for example infarction of a pituitary adenoma. We believe this is the first report and MRI images of pituitary stalk haemorrhage.

P34

A potential complication of radiotherapy for acromegaly

Varunika Lecamwasam, Ali Abbara, Richard Bell, Kevin Baynes & Akkib Rafique
Ealing Hospital, London, UK.

Case report

A 40-year-old lady was diagnosed with acromegaly in 1993 after presentation with clinical symptoms. Imaging showed a 1 cm pituitary lesion and she underwent transphenoidal hypophysectomy in 1993. Post-operatively she was rendered hypopituitary, but still had biochemical evidence of active acromegaly. She proceeded to external beam radiotherapy and was started on bromocriptine medical therapy.

On routine review in 2006, her acromegaly was biochemically quiescent, but a surveillance pituitary MRI revealed an asymptomatic incidental 1.5 cm right parafalcine tumour (Fig. 1). This had features consistent with a meningioma and was treated conservatively.

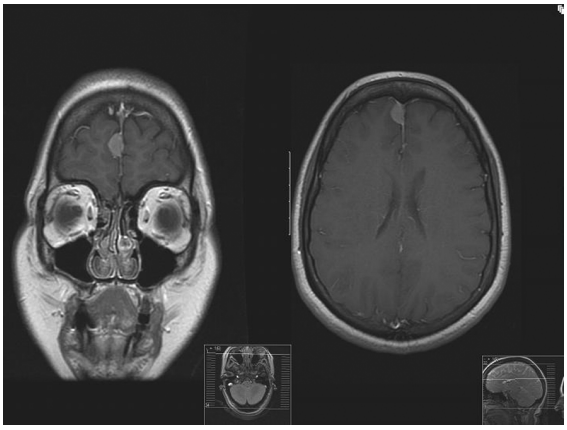

Figure 1 Gadolinium enhanced MRI Brain 1.5 cm right petrous right parafalcine tumour cavernous posteriorly to abut the anterior surface of the pons/mid-brain.

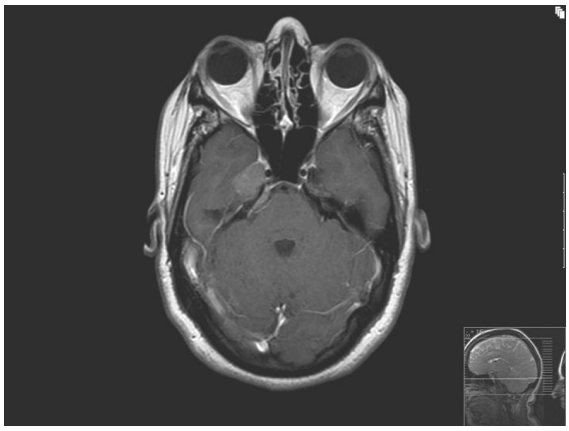

Figure 2 Meningioma arising from the apex adjacent to the sinus.

In 2007, she complained of right facial neuropathic pain. On clinical examination she had reduced sensation of the ophthalmic and maxillary division of 5th cranial nerve with loss of corneal reflex & loss of right tongue sensation. Repeat MRI imaging showed a new lesion arising lateral to the cavernous sinus from the dural tail of the petrous bone (Fig. 2). She underwent an orbito-zygomatic craniotomy and excision of this lesion. Histology confirmed an atypical meningothelial meningioma with brisk mitotic activity with 80% of cells expressing progesterone receptor.

Clinical learning point

Hypopituitarism secondary to radiotherapy is common and well described. It is likely that this patient had radiotherapy-related meningiomas since they occurred within the field of the radiotherapy beam and at an appropriate time interval.

As of yet, there are no conclusive prospective studies with long term follow up and adequate patient numbers to fully assess the excess risk of non-pituitary cerebral malignancies secondary to radiotherapy.

This potential late occurrence should be considered for patients who have had radiotherapy for pituitary disease.

P35

An unusual case of primary infertility

Anjali Amin, Stephen Robinson & Lisa Webber
St Mary's Hospital, London, UK.

We present a case of a 29-year-old lady who presented with subfertility. She had had a 2 years history of oligomenorrhoea with highly irregular menses. She had symptoms of depression and described tunnel vision. Clinically, she was euendocrine with no features of hormonal excess or deficiency. She was not hirsute. Visual field testing demonstrated a severe left superior temporal quadrantopia.

Laboratory investigations demonstrated an oestriadol level ranging between 5000 and 6000 pmol/l, in the context of a FSH of 15 U/l. Her prolactin was mildly elevated at 600 mU/l. A short synacthen test was consistent with pituitary adrenal failure (0 min 219, 30 min 411, 60 min 444). Her free T_4 was 10.3. A MRI of the pituitary gland revealed a large homogenous adenoma with chiasmal compression.

A diagnosis of a pituitary adenoma secreting FSH with ovarian hyperstimulation was made. She was commenced on hydrocortisone 30mg daily. It was felt that any tissue secreting FSH should be removed through surgery in order improve her chances of fertility. She elected to have her pituitary surgery abroad. She had a successful transphenoidal resection of the pituitary tumour, which was complicated by a post-operative CSF leak. Following her surgery, her FSH had dropped to 4.9 U/l with an undetectable oestradiol level. An insulin stress test showed good hypoglycaemia with a peak cortisol of 472 nmol/l at 90 min and peak growth hormone of 1.5 mU/l at 60 min.

She did not have a return of her menses, which was consistent with her hypopituitary status. She is still trying to conceive and is being considered for fertility treatment with GnRH treatment, however there are several factors which have made this problematic. Firstly, her BMI is over 35 and secondly her partner lives abroad.

In summary, this is a 29-year-old lady with a FSH secreting pituitary tumour, who presents as an unusual cause of primary infertility.

P36

A case of insulin allergy: something to zinc about

Katherine Simpson, Michael Clements, Arla Ogilvie, Sharon Carter, Chris Feben, Kapila Batta & Alan Rubin
Watford General Hospital, Watford, Hertfordshire, UK.

A 50-year-old man with poorly controlled type 2 diabetes mellitus on oral hypoglycaemics was under review as a diabetic outpatient. In April 2007, he developed idiopathic thrombocytopaenic purpura which, on a background of probable diabetic nephropathy, precipitated end-stage renal failure. He was treated with oral steroids and was established on haemodialysis. To achieve better glycaemic control, he was converted to twice daily insulin. His treatment dose of prednisolone was decreased to 10 mg in August 2007. In October 2007, he started to notice discrete firm nodules appearing at the sites of insulin injection and his insulin requirements increased. In December 2007, he discontinued his prednisolone and following each insulin injection he noticed a large wheal appearing within a few minutes at the injection site. His eosinophil count was noted to be high, with a peak of 12.01×10^9/l and an IgE RAST test to insulin was strongly positive. A biopsy of a nodule was performed which revealed dense eosinophilic infiltrates. He was subsequently reviewed by the diabetes team with a preliminary diagnosis of insulin allergy. Different insulin preparations were tried as well as exenatide. His eosinophil counts are charted and clearly demonstrate a response to the different diabetes treatments and reducing steroid dose. Since the insulin types and their additives were easily determined, we were able to try alternative insulin regimes until the reactions stopped. From these observations, we were able to deduce that this patient suffers from a zinc allergy, a common additive used to form hexamers in commercial insulins.

P37

An unusual case of hypercalcaemia

Anjali Bala, Belinda Sandler, Felicity Kaplan & Sunil Kaniyur
Lister Hospital, Stevenage, UK.

We describe the case of a 70-year-old lady presenting with marked, symptomatic hypercalcaemia.
Past medical history was of insulin-treated Type 2 Diabetes, macrovascular disease and chronic renal impairment.
On admission, corrected calcium was noted to be 3.53 mmol/l with a paired PTH within normal range (4.3 pmol/l, range 1.6–9.3). Further questioning revealed a history of weight loss but no other clinical features specifically suggestive of malignancy. There was no personal or family history of endocrinopathy, and the patient was not taking any relevant medication.
Aside from clinical evidence of dehydration, examination was unremarkable and in particular, there was no evidence of finger clubbing, lymphadenopathy, breast or abdominal masses.
Investigations conducted including liver function tests, tumour markers, myeloma screen and bone scan were normal. A serum ACE was found to be moderately elevated at 113 IU/l (10–70) in the absence of overt respiratory, cutaneous or musculoskeletal pathology.
Following hydration and intravenous bisphosphonate therapy, corrected calcium remained elevated.
Although PTH was not elevated or indeed at the upper end of normal, a lack of clear malignant aetiology prompted a request for an ultrasound scan of the parathyroids. This displayed a right parathyroid lesion which corresponded to an area of increased uptake on subsequent Sestamibi imaging. The patient was referred for surgery in view of symptomology and degree of hypercalcaemia.
Surprisingly, histology showed a parathyroid adenoma with granuloma suggestive of sarcoidosis, as well as an area of thyroid tissue with features of granulomatous thyroiditis, also favouring sarcoidosis. These findings correlated with the elevated ACE and a subsequent high-resolution CT chest which showed evidence of nodular interstitial disease predominantly in the mid and upper zones. The final diagnosis was therefore unexpected – sarcoidosis with granulomatous infiltration of the parathyroid gland resulting in hypercalcaemia.

P38

Rapid management of hypercalcaemic crisis: a multidisciplinary approach

Alero Adjene, James Donaldson & Keith Steer
North West London Hospitals NHS Trust, London, UK.

A 69-year-old man with longstanding ulcerative colitis presented for annual review by the gastroenterologists. He complained of lower back pain for six months and a recent history of anorexia, constipation, thirst and urinary frequency.
His serum calcium was 5.12 mmol/l, phosphate 1.77 mmol/l, urea 19.1 mmol/l and creatinine 252 μmol/l.
He was admitted, rehydrated with 5 l of intravenous normal saline over 24 h and given pamidronate 90 mg, but remained unwell and hypercalcaemic.
The endocrinologists were informed of his admission. On examination, a mass was palpable in the right anterior cervical triangle and an urgent parathyroid hormone (PTH) level requested. His PTH was 192.9 pmol/l and a neck ultrasound was performed that morning; this identified a heterogeneous nodule ($4.1 \times 3.8 \times 2.3$ cm), separate to the thyroid, thought to represent a right superior parathyroid adenoma. The size of the nodule and the PTH level raised concerns of a parathyroid carcinoma. After liaising with the surgeons, he had a parathyroidectomy that afternoon and required intensive care post-operatively for renal support.
His recovery was uneventful with no significant hypocalcaemia. The pathologists reported a parathyroid neoplasm of uncertain malignant potential.
Following the diagnosis of parathyroid carcinoma, he is seen regularly. His calcium levels are within normal limits, but his PTH remains elevated (29.8 pmol/l at his last visit) and he is vitamin D deficient.
Parathyroid carcinoma is a rare cause of PTH-related hypercalcaemia. Histopathological distinction between benign and malignant parathyroid tumours is difficult.
Surgery is the most effective treatment in cases of severe hypercalcaemia and the timely collaboration between the different specialties in this case was essential to this man receiving prompt and appropriate treatment.

P39

Renal artery stenosis and possible coexisting Conn's adenoma

K O Shaafi & S Russell
Chase Farm Hospital, Middlesex, UK.

A 47-year-old lady presented with a long history of resistant hypertension. Her GP referred her for further investigation to a cardiologist who found a smaller right kidney on ultrasound scan and an elevated rennin level. Renal artery MRA revealed right renal artery stenosis. On September 2008, she underwent right renal artery stenting and was advised to stop taking the antihypertensive medications (doxazosin and amlodipine). A week later she was admitted with headache, vomiting and severe hypertension. She was referred to our endocrine department and on review of her notes it was clear that she had very long standing hypokalaemia with potassium ranging between 2.5 and 3.5 mmol/l over the past 5 years. Despite recommencement of her antihypertensive (doxazosin 8 mg b.d) her blood pressure remained difficult to control (220/124). A repeat CT with Doppler flow examination post stent insertion revealed that the proximal segment of the right main renal artery was patent and appeared to show contrast. However, there was no obvious contrast seen within the stent. Distal to the stent the contrast was seen in the vessel and the right kidney showed uniform complete enhancement. The Doppler examination showed forward diastolic flow with patent Doppler signal. Incidentally there was a 10 mm lesion in the left adrenal gland.
Her plasma renin activity remains high and a postural Aldosterone Renin ratios were as follow: 09:19 am renin 10 aldosterone 330, 10:10 am renin 15 aldosterone 1210 and 12:55 pm renin 22 aldosterone 370. UE and LFT were normal as well as the rest of routine blood tests.
She was recently commenced on spironolactone (in addition to doxazosin 8 mg b.d) titrating the dose gradually to 100 mg b.d. This has resulted in improvement in the BP readings. We are contemplating adrenal venous sampling for Conn's adenoma and possible MRI of the adrenal gland. Also she needs a repeat renal artery MRA to rule out possible blockage of the stent (possible 6 weeks post stent insertion).
This is a case of possible adrenal aldosterone-secreting adenoma with coexisting active renal artery stenosis in a hypertensive middle-aged woman. The concomitance of the two lesions was previously reported in the literature only in five patients. Therefore we would like to present this interesting case to the Third Hammersmith Multidisciplinary Meeting principally to discuss the following points:

1. Is this likely to be Conn's coexisting with RA stenosis (is hypokalaemia is feature of RA stenosis)
2. Shall we proceed with adrenal venous sampling.
3. The confounding effects on biochemistry when both conditions coexist.

P40

This abstract appears as OC5.

P41

A diabetic foot dilemma resolved through the use of the diabetic foot MDT

K F Styles, C M Burns, H Shaikh & J Turner
Endocrine Unit, Department of Investigative Medicine, Imperial College NHS Trust, London, UK.

A 63-year-old type 2 diabetic gentleman with paranoid schizophrenia presented in October 2008 with systemic sepsis arising from multiple neuropathic foot ulcers. Foot MRI confirmed extensive osteomyelitic change. This admission followed two previous similar episodes which culminated in surgical debridement of the ulcers and amputation of the second and third toe of his left foot. Post-operatively, as his condition improved he was unable to comply with IV antibiotics, VAC dressings and off-loading of his feet due to his schizophrenic delusions re-emerging.

On his admission, he tolerated IV antibiotics fleetingly during the acute phase of his illness. However as IV access became problematic, he was subsequently switched to oral Ciprofloxacin and Clindamycin. He was discussed at the Diabetic Foot MDT. It was felt that bilateral below knee amputations would be an option which, given our patient's lack of capacity, would need further discussion with the next of kin and his psychiatrist to ascertain the patient's best interest. In the interim, we continued with oral antibiotics and closely monitored his clinical state. His condition deteriorated and he was discussed in further MDT and case conference settings with a Consultant Diabetologist, Vascular Surgeon, Microbiologist, Psychiatrist, Next of Kin, Community Psychiatric Nurse and Nursing Home Manager present. It was concluded that due to this patient's inability to tolerate IV antibiotics and his poor rehabilitation potential combined with his poor quality of life and prognosis, it was in his best interest to be treated palliatively. To this end we withdrew antibiotics and discharged him to his nursing home with community palliative support.

This case highlights the vital importance of regular diabetic foot multidisciplinary meetings in tailoring management to meet individual patient needs.

Author index (2008)

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