Society for Endocrinology

Imperial College London

Imperial Pituitary Masterclass Meeting

Monday 28th November 2022

IMPERIAL PITUITARY MASTERCLASS MEETING 2022

Venue: Charing Cross Hospital, Imperial College Healthcare NHS Trust, London

09.00 – 09:30 Registration

SESSION 1

CHAIRS: Dr Alison Wren, Chelsea and Westminster Hospital, London
Dr Zane Jaunmuktane, University Hospital London.

09:30 – 10:00 *Acute presentations of pituitary disease.*
Dr Mona Waterhouse, Barts Heath NHS Trust, London.

10:00 – 10:20 *An unusual pituitary mass which improved with steroids and unmasked an unexpected diagnosis.*
L Yang, C Borysiewicz, F Sanderson, F Wernig.
Imperial College Healthcare NHS Trust, London.

10:20 – 10:40 *Cavernous ICA aneurysms and Cushing's disease: no stress, just go with the flow*
L Thurston, E Hatfield, D Peters, N Martin, L Dixon, A Gonstarova, R Nair, N Mendoza, K Meeran, A Dalton.
Imperial College Healthcare NHS Trust, London.

10:40 – 11:00 *The discovery and management of an ectopic pituitary tumour.*
C Shepherd[1], P Patel[2], K Muralidhara[1].
1. Kingston Hospital.
2. St George's University Hospitals NHS Foundation Trust, London.

11:00 – 11:20 *An atypical presentation of hypopituitarism.*
R Minhas[1], K Bashir[1], C Mitchell[1], YY Ling[1], G Tarigopula[1], M Al-Ansari[1], J Tomlinson[2], F Wernig[2]
1. Hillingdon Hospitals NHS Foundation Trust.
2. Imperial College Healthcare NHS Trust, London.

11:20 – 11:50 TEA & COFFEE BREAK

SESSION 2

CHAIRS: Dr Anna Crown (Brighton and Sussex University Hospitals NHS Trust)
Mr Nigel Mendoza (Imperial College Healthcare NHS Trust, London).

11:50 - 12:10 *The endocrine plumbing problem.*
H Ibrahim, R Herring.
Southend University Hospital.

12:10 - 12:30 *Challenging case of recurrent Cushing's disease after an episode of pituitary apoplexy.*
M Alameri [1], E Hatfield[1], N Martin[1], A N Di Marco [1], N Mendoza[1], R Nair[1], B Jones [1], M Gurnell[2], K Meeran[1], P Behary[1].
1. Imperial College Healthcare NHS Trust, London.
2. Department of Endocrinology, Cambridge University Hospitals.

12:30 – 12:50 *Endocrinopathies in patients on checkpoint inhibitors - why ongoing "checks" are essential*.
Z Mohsin, S Noronha, M Payne, G Tan and HE Turner.
Oxford Centre for Diabetes, Endocrinology and Metabolism, Oxford University Hospital.

12:50 - 13:10 *Hypopituitarism and hypothalamic obesity following craniopharyngioma: A chronic disease management perspective.*
J Makaronidis, A Pechir-Cerillo, M Adamo, R Batterham, S Baldeweg. University College London Hospitals, London.

13:10 – 14:10 LUNCH

SESSION 3

CHAIRS:
Dr Rochan Agha-Jaffar (Imperial College Healthcare NHS Trust, London)
Mr Ramesh Nair (Imperial College Healthcare NHS Trust, London)

14:10 - 14.40 *Challenges in pituitary surgery.*
Mr Arthur Dalton, Imperial College Healthcare NHS Trust.

14:40 – 15:00 *The role of surgery in improving fertility in a patient with prolactinoma.*
B Mahamud, J.Pollock, U.Farhana, B Hossain. H Rehmani, N Stajanovic, K. Ramachandran S Solomon, J Eisold, M al-Aaraj, B Abdeen,S.Sahota, G Mlawa.
Barking, Havering and Redbridge University Hospitals NHS Trust, Essex.

15:00 - 15:20 *A rare case of Sheehan's syndrome*
B Inayat, T Abdellatif, N Thorogood, K Bradley.
Bristol Royal Infirmary University Hospitals Bristol NHS Foundation Trust.

15:20 – 15:40 *Silent corticotroph pituitary adenoma: clinical presentation mimicking pituitary apoplexy.*
R Siddique[1], F Swords[1], Neil Dorward[2], R Ahluwalia[1].
1. Norfolk Norwich University Hospital.
2. University College London Hospital.

15:40 - 16:00 *Endocrinopathy behind the facemask – Follow up.*
S Nyunt[1], A Sharma[1], K Narula[1], C Mitchell[2], N Mendoza[1], YY Ling[2], ECI Hatfield[1], K Meeran[1], NM Martin[1].
1. Imperial College Healthcare NHS Trust, London.
2. Hillingdon Hospitals NHS Trust.

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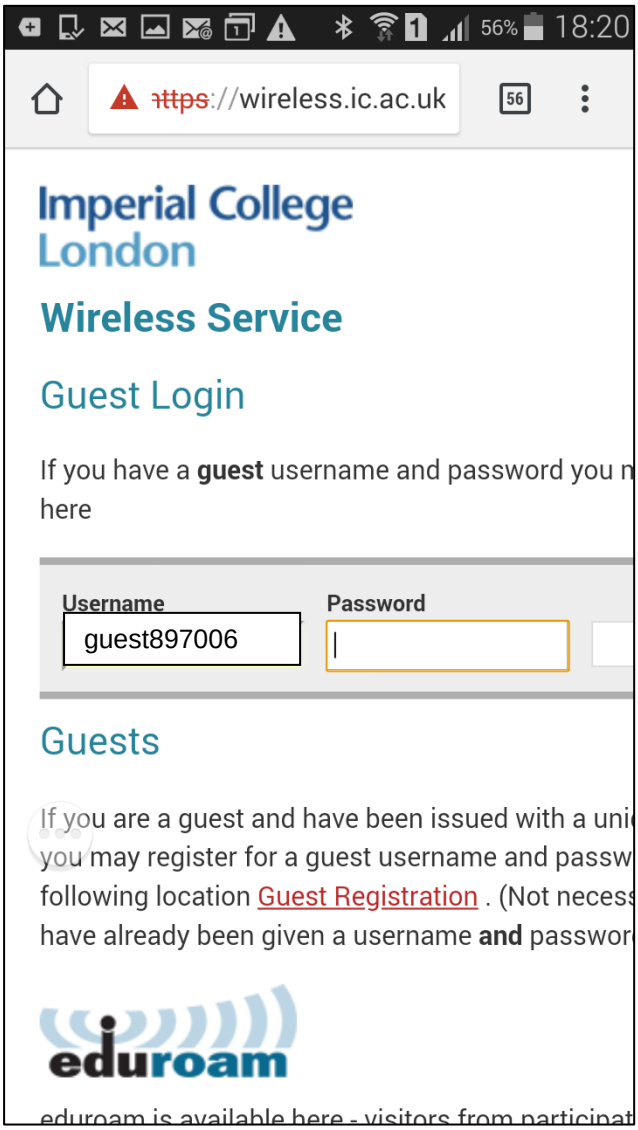

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An unusual pituitary mass which improved with steroids and unmasked an unexpected diagnosis.

L Yang, C Borysiewicz, F Sanderson, F Wernig.
Imperial College Healthcare NHS Trust, London.

A 49-year-old man developed generalised headaches, blurred vision, nausea and vomiting over one month and presented to his local hospital with progressive ptosis of the left eye associated with diplopia and dizziness on looking to the right. He had a background of hypertension but was otherwise fit and well. On examination, he had a left third nerve palsy with complete ptosis, dilated pupil and impaired adduction of the left eye. Biochemistry revealed hyponatremia with sodium 125 mmol/L. Subsequently a full pituitary profile was performed which showed TSH 1.8 mIU/L, free T4 4.4 pmol/L, prolactin 197 mIU/L, LH 0.7 U/L, FSH 4.2 U/L, testosterone < 0.3 nmol/L, random cortisol 18 nmol/L. A SST confirmed inadequate cortisol response with cortisol 19 nmol/L at baseline, 100 nmol/L at 30 minutes and 130 nmol/L at 60 minutes.

An urgent MRI head revealed a sellar and suprasellar cystic lesion measuring 15mm in maximal diameter expanding into the cavernous sinuses bilaterally, displacement of the optic chiasm and thickening of the infundibulum. The patient was commenced on pituitary hormone replacement including prednisolone 5mg which led to resolution of ptosis. A CT chest/abdomen/pelvis found no significant intra-thoracic or intra-abdominal pathology and he underwent FDG-PET CT which showed metabolic activity in the left thyroid gland only. He also had a lumbar puncture which found raised CSF white cell count consistent with an inflammatory response.

Dedicated MRI pituitary performed 16 days after presentation showed reduction in the size of the pituitary mass. Histology from trans-sphenoidal pituitary biopsy revealed non-caseating granulomas and Langerin staining suggestive of Langerhans Cells Histiocytosis/ Erdheim Chester disease. Subsequently, prednisolone was increased to 15 mg once daily and repeat MRI pituitary at 3 months and 5 months showed almost complete resolution of the cystic pituitary mass. Testosterone levels recovered and he was able to stop testosterone replacement around the same time.

In view of the FDG-avid thyroid nodule, US thyroid and FNA were performed which revealed a 2.8cm left thyroid mass with Thy3f Hurthle cell neoplasm on cytology. Diagnostic hemi-thyroidectomy confirmed a minimally invasive Hurthle cell follicular carcinoma (pT2, Nx, Mx).

The patient was referred to the Langherhans Cell Histiocytosis service who organised an interval FDG-PET CT 4 months after the previous scan. This revealed multiple new FDG-avid lymph nodes in the supraclavicular, paratracheal and hilar regions. A percutaneous lymph node biopsy found necrotising granulomatous inflammation, most likely due to mycobacterial or fungal infection. Repeat lymph node biopsy was performed and samples sent for TB cultures which returned showing fully sensitive acid-fast bacilli. Notably, a TB ELISPOT was performed at initial presentation and was positive however TB PCR was negative and therefore active TB was not pursued at that time. The patient has now completed one year of anti-TB therapy and has been weaned off prednisolone with continued reduction in size of the pituitary lesion. In this time, he has also become a father to identical twin girls!

Questions for discussion:

1. Why did steroids improve symptoms and shrink the pituitary mass?
2. What other differentials could have been considered?

Cavernous ICA aneurysms and Cushing's disease: no stress, just go with the flow

L Thurston, E Hatfield, D Peters, N Martin, L Dixon, A Gonstarova, R Nair, N Mendoza, K Meeran, A Dalton.
Imperial College Healthcare NHS Trust, London.

Case history:
A 38-year-old lady was referred to the Pituitary MDT (August 2021) with a strong suspicion of Cushing's syndrome. She had easy bruising, acne, hirsutism, and central adiposity on a background of newly diagnosed type 2 diabetes and hypertension. Her baseline cortisol was 750 nmol/L and failed to suppress following a low dose dexamethasone suppression test (159 mmol/L). Baseline ACTH was 82 ng/L. MR pituitary imaging demonstrated a macroadenoma, protruding into the sphenoid sinus with maximal dimension of 11mm. She went on to have IPSS which showed a significant central to peripheral gradient and she was commenced on metyrapone 500 mg daily.

Following discussion at the Pituitary MDT, she was admitted for trans-sphenoidal surgery. However, following the planning MRA, two right cavernous ICA aneurysms and one left ICA aneurysm were identified. Of concern, the largest (3mm right sided) projected into the tumour. Consequently, surgery was cancelled and the metyrapone dose was uptitrated to a 'block and replace' regime with prednisolone. Two weeks later, a clinical balloon occlusion test was undertaken which demonstrated that the aneurysms were not amenable to coil embolisation. It did however show satisfactory cross flow from the left internal carotid, should the right sided ICA need to be sacrificed. Following discussion at the Neurovascular MDT, the patient underwent insertion of a right ICA pipeline vantage flow diverting stent and was commenced on a minimum six-month course of dual antiplatelet therapy (aspirin and prasugrel) to enable closure of the aneurysms.

Three months later, a vaso-CT was performed which confirmed no stenosis or thrombosis within the stent, however, identified some residual flow within the aneurysm. Subsequent MR angiography (three months later) revealed a persistent right ICA aneurysm. Consequently, the decision was made to wait a further six months to allow sufficient time for complete closure of the aneurysm, (with cerebral catheter angiography for confirmation) prior to definitive transsphenoidal surgery.

During this time, the effects of Cushing's disease were managed, including uptitration of antidiabetic medication/insulin, antihypertensives, and the initiation of rivaroxaban for VTE prophylaxis. Regular biochemical monitoring was required to achieve the optimal doses of metyrapone (1g QDS) and prednisolone (4mg OD).

This case illustrates the challenges of managing ICA aneurysms prior to transsphenoidal surgery and the medical management of hypercortisolaemia over an extended 12-month period.

Questions for discussion:

1. Are there alternative medical options for long term management of hypercortisolism that we should have explored?
2. Should we have offered her a bilateral adrenalectomy as a more definitive cure given the difficulties around pituitary surgery?

The discovery and management of an ectopic pituitary tumour.

C Shepherd[1], P Patel[2] and K Muralidhara[1]
1 - Kingston Hospital NHS Foundation Trust.
2 - St George's University Hospitals NHS Foundation Trust.

Case History:
A 73-year-old woman of Chinese origin presented with a 6-year history of left sided blood-stained rhinorrhoea. Symptoms were precipitated on bending forward and exercise. She denied headaches or visual disturbance. Past medical history included a right sided total hip replacement and gallbladder polyps under surveillance. There was no personal or family history of malignancy. She is a non-smoker, does not drink alcohol and does not take any regular medications.

Investigations:
Nasal endoscopy did not reveal a bleeding point. Computed tomography (CT) found abnormal soft tissue within the right sphenoid sinus with dehiscence of the lateral and posterior wall of the sphenoid sinus. This also involved the cavernous sinus and partially, the pituitary fossa. Magnetic resonance imaging confirmed the size and findings; image 1. Visual fields were normal. CT thorax, abdomen and pelvis did not show any further measurable abnormality to indicate a primary malignancy. A full pituitary profile showed normal biochemistry. She was presented at a multi-disciplinary skull base meeting with the working diagnosis of a metastatic malignancy, pituitary tumour, chordoma or chondrosarcoma

Results and treatment:
The patient underwent successful complete extrasellar resection. Post-operative histology confirmed ACTH positivity with a Ki-67 proliferation index of 0.8%. The appearances in keeping with a pituitary neuroendocrine tumour. The immunophenotype consistent with a corticotroph adenoma, and T-pit differentiation. The tumour therefore represented a silent corticotroph adenoma.

Conclusion and points for discussion:
This lady presents with an ectopic sphenoid sinus pituitary adenoma (ESSPA), with an intact normal anterior pituitary gland. The unusual location and non-specific sinonasal symptoms can make the diagnosis challenging warranting a multi-disciplinary approach. Interestingly, she did not complain of visual, nerve or balance related issues which can occur with cavernous sinus involvement. She did not present with symptoms to suggest hormone activity but carrying out an anterior pituitary profile plus stimulation testing as necessary, is an important part of the work up. Detailed imaging studies are critical in looking at size and tumour margins when planning for surgery, as well as distinguishing the mass from a pituitary tumour extending into the sphenoid sinus. The bone destruction and remodelling suggest chronicity and expansion in a confined anatomic location, rather than aggression of the tumour. The immunohistochemistry highlights the epithelial and endocrine nature of the tissue, which is vital in confirming the diagnosis. Ongoing management will include surveillance imaging to monitor for regrowth.

Questions for discussion:
1. Where else can ectopic pituitary tumours occur?
2. How would you manage the patient if it was a prolactin secreting tumour?

An atypical presentation of hypopituitarism

R Minhas[1], K Bashir[1], C Mitchell[1], YY Ling[1], G Tarigopula[1], M Al-ansari[1], J Tomlinson[2], F Wernig[2]
1 - Hillingdon Hospital.
2 - Imperial College Healthcare NHS Trust.

Case History:
A 48-year-old male presented with headache and hyponatraemia. His past medical history included primary hypothyroidism aged 10 for which he was on levothyroxine. His brother had a Rathke's cleft cyst that was surgically removed. His blood test was as follows: sodium 125mmol/L,TSH 0.10mU/L, Ft4 10.2pmol/L, Ft3 2.6pmol/L, cortisol < 28nmol/L, prolactin 240mU/Testosterone <0.5nmol/L,FSH 1.8U/L,LH 0.3U/L. He was commenced on prednisolone 3mg and continued on levothyroxine 125mcg.

Initial CT Head showed suprasellar lesion with right sided mastoiditis. MRI pituitary showed sellar and suprasellar lesion with marked central cystic lesion and peripheral enhancing tissue, abnormal signal in retro-chiasmatic tracts. Visual field (VF) assessment showed left temporal superior visual field defect with normal right vision. The differential diagnosis was cystic macroadenoma, atypical hypophysitis or previous apoplexy. Pituitary MDT discussion advised to send off vasculitis screen that showed weakly positive c-ANCA, PR3 antibody positive at 7.3 IU/ml (normal range up to 1.9). All other antibodies were negative including negative TB Eli spot.

A repeat MRI pituitary in 6 weeks showed interval reduction in size with central cystic cavity collapsing down, likely an inflammatory process. A repeat VF assessment however, showed progression to bitemporal quadrantanopia. A diagnosis of ANCA-associated vasculitis was established, and his prednisolone dose was increased to 60 mg OD followed by a weaning regime. A pituitary biopsy was considered if no improvement in visual fields.

He then reported polyuria and water deprivation test was consistent with a diagnosis of cranial diabetes insipidus which was managed with desmopressin. He was also noted to have saddle nose deformity which is typical for Polychondritis in the context of ANCA-associated vasculitis. He was then commenced on rituximab. Subsequently, he showed good clinical and radiological improvement with his treatment and was able to return to work. He continues to be followed up in the vasculitis clinic.

Conclusion:
This case posed a diagnostic challenge given the absence of many systemic features of vasculitis. In addition, it highlights the delayed diagnosis of posterior pituitary dysfunction which was presumably unmasked by the high dose steroids. It also emphasizes the importance of the MDT approach to manage such cases.

Questions for discussion:
1. At what point should surgery be considered an option? Steroids VS surgery?
2. Was biopsy necessary in this case prior to starting immunosuppressive agents?

The endocrine plumbing problem.

H. Ibrahim and R. Herring.
Southend University Hospital NHS Foundation Trust.

We are presenting a case of 17-year-old gentleman who was referred to endocrine clinic for a year history of increased thirst, polyuria and tiredness. He was attending college studying to be a plumber.

On direct questioning, he was drinking about 4 litres of fluid during the day and 2L overnight. He was passing urine 5-6 times during the day and once at night. He was also being experiencing headaches that are often daily and got worse towards the end of the day. He described no visual disturbance and was not taking any regular analgesia. In addition, he has noticed decreased frequency of morning erections and increased tiredness. There is no relevant family history. Diabetes mellitus was excluded by his GP with a normal HbA1c.

Initial osmolalitities were suggestive of diabetes insipidus in addition a full pituitary profile was undertaken, which shined a 9 am cortisol of 34 nmol/L, IGF-1 23 nmol/L, FSH <0.21U/L, LH <0.22U/L, with a prolactin of 893 mU/L, total testosterone <0.4nmol/L, TSH elevated at 9.37mU/L, with a free T4 of 6.4pmol/L. At this point he was started on Hydrocortisone 10mg bd. He was requested urgent MRI pituitary and water deprivation test.

Formal water deprivation test confirmed cranial DI and hence he was started on DDAVP replacement. MRI of the pituitary gland demonstrated an avidly enhancing suprasellar mass (18mmX 9mmX 12mm) centred onto the pituitary infundibulum with resulting compression upon the anterior visual pathway. Formal visual field check showed evidence of bitemporal hemianopia being denser on the left side.

He was discussed in local neuroradiology MDT and the possible differentials put forward were optic glioma, hypothalamic tumour or germinoma. He was referred to tertiary centre at National Hospital for Neurology and Neurosurgery for further management. Repeat MRI scan of pituitary revealed uniformly enhancing lesion in the suprasellar region and involve the pituitary stalk, with differential diagnosis including lymphocytic hypophysitis, histiocytosis, sarcoidosis or neoplastic lesion. CSF fluid sampled and didn’t demonstrate malignant cells, and serum tumour markers HCG and AFP were within normal limits.

An initial biopsy performed by TSS and demonstrated normal anterior pituitary tissue only. A second biopsy favoured a diagnosis of lymphocytic infundibuloneurohypophysitis. Of concern he developed visual problem after the first biopsy and the vision significantly deteriorated following the second biopsy and he has been under the care of ophthalmology. The visual impairment has hugely impacted his quality of life. Examination revealed significant visual field deficit with complete loss of vision of lateral field vision on the left side, and only presence of vision in the top medial quadrant of the right side. He has been started on trial of high dose prednisone 40 mg daily 3 months following the second biopsy to see if it will help with shrinkage of the lesion and improving of his vision.

Question for discussion:

1. Should high dose steroids be considered early in inflammatory pituitary lesions?

Challenging case of recurrent Cushing's disease after an episode of pituitary apoplexy.

M Alameri [1], E Hatfield[1], N Martin[1], AN Di Marco [2], N Mendoza[3], R Nair[3], B Jones [4], M Gurnell [5], K Meeran[1] and P Behary[1]
1 - Department of Diabetes & Endocrinology, Imperial College Healthcare NHS Trust, London, UK.
2 - Department of Endocrine Surgery, Imperial College NHS Trust, London, UK.
3 - Department of Neurosurgery, Charing Cross Hospital, Imperial College NHS Trust, London, UK.
4 - Department of Radiology, Imperial College Healthcare NHS trust, London, UK.
5 - Department of Endocrinology, Wellcome-MRC Institute of Metabolic Science, University of Cambridge, and NIHR Cambridge Biomedical Research Centre, Cambridge University Hospitals, Cambridge, UK.

Introduction:
Both Cushing's disease (CD) and pituitary apoplexy are rare conditions. We report a case of recurrent CD following pituitary apoplexy of an ACTH-secreting pituitary adenoma, managed primarily with bilateral adrenalectomy.

Case presentation:
A 33-year-old female with a background history of type 2 diabetes, primary hypothyroidism and morbid obesity presented with a sudden onset severe headache, a complete left-sided ptosis and ophthalmoplegia involving cranial nerves III, VI and VI. A pituitary MRI revealed a large sellar mass with a small volume haemorrhage, extending into the left cavernous sinus, but without any optic chiasm compression. These findings were in keeping with a pituitary apoplexy. A random cortisol level was 513 nmol/L with an ACTH of 17 ng/L at presentation. The rest of her pituitary hormonal axis was intact and visual fields were normal. Further clinical examination and history from the patient of gradual weight gain, worsening acne, oligomenorrhoea and symptoms of proximal myopathy were suggestive of an underlying diagnosis of Cushing's. She was discussed at our Pituitary MDT and a conservative management was recommended. Approximately 2 weeks after her presentation, she was found to have a low morning cortisol at 72 nmol/L and low levels on a Cortisol Day Curve (CDC) (cortisol of 133, 49, 47 and 67 nmol/L), at which point she was started on prednisolone 3 mg.

However, 2 months later, another CDC suggested recovery of her ACTH-cortisol axis and prednisolone was stopped. We continued to monitor her closely both clinically and biochemically. Unfortunately, 4 months after her apoplexy, she became progressively hypercortisolaemic. This was confirmed on a number of tests: CDC with cortisol levels of 603, 311, 319 and 351 nmol/L, elevated late night salivary cortisol and a Low Dose Dexamethasone Suppression Test with an unsuppressed 48-hour cortisol level of 100 nmol/L. She was rediscussed at the Pituitary MDT where it was felt that despite resolution of the haemorrhage, there was no discrete pituitary target lesion seen on her MRI to direct surgery. A methionine PET (M-PET) was recommended to localise any functional lesion to aid surgery. The M-PET scan showed a left-sided tumour, extending posterior-laterally in the left cavernous sinus (more than what was initially suspected from previous imaging) with tracer uptake, suggestive that it was functional. This was not amenable to transsphenoidal surgery (TSS) as a curative option, as tumour could not be fully resected due to its location. Bilateral adrenalectomy +/- pituitary radiotherapy was suggested. Patient was treated with metyrapone before she underwent a bilateral adrenalectomy. At her 6-month post-surgery follow-up, she remained well with normalisation of her cortisol levels.

Conclusion:
Vigilant continued clinical and biochemical monitoring of patients' who experience apparent resolution of a functional pituitary tumour, following pituitary apoplexy is essential. This report highlights the importance of a MDT approach, with utilisation of functional scanning, such as M-PET, to aid decision-making in challenging cases.

Endocrinopathies in Patients on Checkpoint Inhibitors--why ongoing "checks" are Essential.

Z Mohsin, S Noronha, M Payne, G Tan and HE Turner.
Oxford Centre for Diabetes, Endocrinology and Metabolism, Oxford University Hospital.

We describe the case of a 67-year-old gentleman who was diagnosed with stage III melanoma in 2017 and commenced combination immunotherapy with Nivolumab and Ipilimumab a year later for metastatic disease. After the second cycle of immunotherapy, he developed subclinical hyperthyroidism with a suppressed TSH, a free T4 of 35.5munit/L (9.0-19.0) and a free T3 of 15.1pmol/L (2.6-5.7), likely secondary to thyroiditis.

Four weeks later, during routine bloods following the 3rd cycle of immunotherapy, he was found to have raised blood sugars of 30.4mmol/L with ketonemia and positive Glutamic Acid Decarboxylase (GAD) antibodies pointing to a new diagnosis of type I diabetes. The C-peptide level, measured at a later date, was undetectable. There was also progression of his thyroiditis to frank primary hypothyroidism with a TSH of 10.51munit/L (0.30-4.20), a free T4 of <5.2 munit/L (9.0-19.0 munit/L) and a free T3 of <1.5pmol/L (2.6-5.7). Thyroid peroxidase and thyroid receptor antibodies were not elevated. He was initiated on basal-bolus insulin therapy and 75 mcg of Levothyroxine daily.

Two weeks later, he was admitted for a short history of fatigue and bloods revealed hyponatremia of 122 mmol/L and a random cortisol of 44nmol/L. A 9:00 am cortisol measured approximately 2 weeks earlier was 641 nmol/L. The repeat cortisol level was 35 nmol/L with an ACTH level measured post discharge of <5 ng/L (0-46), suggesting secondary adrenal insufficiency. He was initiated on hydrocortisone.

Although the likely cause was hypophysitis, interestingly the MRI of his pituitary and CT of his adrenals were both normal. The rest of his pituitary profile was normal, although his prolactin levels transiently decreased from 204munit/L to 75 munit/L (70-410). In view of significant autoimmune toxicities experienced from the use of checkpoint inhibitors, maintenance immunotherapy was not administered, and he has been under clinical observation with surveillance scans. His disease remains well controlled with no evidence of progression for the last three and a half years and he retains a good quality of life with a performance score of 0.

This is a unique case as it describes the rare emergence of several endocrinopathies in a sequential pattern in a single patient. Nonetheless, this case describes the need for the clinician to be vigilant of autoimmune endocrinopathies that can arise from immunotherapy use which can be life-threatening and may impair the quality of life of the patient more than the original disease. Close collaboration between oncology and endocrinology is essential with patient education and careful monitoring being the cornerstone of successful management.

Hypopituitarism and hypothalamic obesity following craniopharyngioma: A chronic disease management perspective.

J Makaronidis, A Pechir-Cerillo, M Adamo, R Batterham and S Baldeweg.
Department of Diabetes and Endocrinology, University College London Hospitals NHS Foundation Trust.

We present the case of a 32-year-old male diagnosed with a craniopharyngioma in 2000 aged 10. He underwent drainage, followed by a craniotomy and tumour resection in 2001. He developed panhypopituitarism with diabetes insipidus (DI) in the post-operative period and was commenced on hormone replacement with hydrocortisone, levothyroxine, growth hormone, testosterone and desmopressin. The craniopharyngioma recurred in 2002 and he was treated with stereotactic radiotherapy. He developed accelerated weight gain, hyperphagia and food seeking behaviours.

The weight gain resulted in bow leg deformity and mobility difficulties, requiring a corrective osteotomy. He was referred to adolescent weight management services and several lifestyle interventions with energy restriction and exercise did not prevent further weight gain. In 2010, at the age of 20 and a BMI of 65.7kg/m² (179kg) he underwent a sleeve gastrectomy. He had no peri-operative complications, but had hypernatraemia and polyuria following the operation, managed with additional desmopressin. For an 8-month period his weight reduced, reaching a nadir of 145kg (BMI 54.7 kg/m²), which was followed by recurrence of hyperphagia, and weight regain. Between 2012-2017 he developed type 2 diabetes (T2DM), hypertension, hyperlipidaemia, obstructive sleep apnoea and his mobility deteriorated, resulting in being wheelchair bound. DI and polydipsia overlapped with hyperphagia, resulting in a complex management situation, from compulsive consumption of sugar-containing drinks. These events had an important emotional impact on his transition from adolescence to adulthood.

In 2018 he received a trial of liraglutide 3mg daily for weight management with no response. In 2021, at a BMI of 80 kg/m², revisional bariatric surgery was considered. In preparation, he engaged in an intensive lifestyle intervention programme and a multidisciplinary effort to optimise his medical management was adopted. In 2022 he underwent revisional bariatric surgery with a laparoscopic one-anastomosis gastric bypass at a pre-surgical BMI 70.2 kg/m² and 191kg. There were no complications and his sodium remained stable. At 4 months following the revisional procedure his weight had reduced to 185kg, T2DM was in remission and his mobility improved, no longer requiring a wheelchair.

Panhypopituitarism and hypothalamic obesity following craniopharyngioma carry a high risk of morbidity and mortality and represent incredibly complex management challenges in the long-term, as illustrated by this case.

Questions for discussion:

1. What should the health-related goals of the long-term care be for patients with these complex presentations?
2. How do we best evaluate the success of such interventions?

The role of Surgery in improving Fertility in a patient with Prolactinoma.

B Mahamud, J Pollock, U Farhana, B Hossain. H Rehmani, N Stajanovic, K Ramachandran, S Solomon, J Eisold, M al-Aaraj, B Abdeen, S Sahota, G Mlawa.
Barking, Havering and Redbridge University Hospitals NHS Trust, Essex.

Prolactinomas are the most common type of functional pituitary tumours, accounting for up to 60% of hormone-secreting pituitary tumours. Prolactinomas can be grouped as either those that cause increased prolactin hormone secretion and those that cause mass effect (Jan et al., 2007; Majumdar and Mangal, 2013; Mehta and Lonser, 2017).

An increase in prolactin secretion often causes concomitant inhibition of gonadotropin-releasing hormone (hypogonadism) which ultimately culminates in reproductive and sexual dysfunction, characterised by oligomenorrhea, galactorrhoea, osteoporosis and amenorrhea in women and decreased libido in men (Bonert, 2020; Zamanipoor Najafabadi et al., 2020). Macroadenomas which impinge on surrounding structures cause of mass effect symptoms characterised by headaches, vision loss, and hypopituitarism (Goldblum, 2018).

The majority of prolactinomas are microadenomas and inversely affect women more than men, with age of occurrence peaking between ages of 20 and 50 years (Zamanipoor Najafabadi et al., 2020).

The gold standard management for prolactinomas is through medical treatment with dopamine agonist (DA). However, in a number of patients management prolactinomas can become complex and resistant to medical therapy, these cases can be managed though surgical intervention (Glezer and Bronstein, 2019).

Case report:
31-year-old lady was referred by GP to Endocrine team clinic with 7 years history of amenorrhea. She had a blood test which showed prolactin level of 3700 and macroprolactin of 1525. She then had MRI scan of the brain which showed a 7mm pituitary adenoma.

She was then started on medical management in 2015, initially with Quinagolide, which brought down the level to about 1200 by January 2016. However, the patient reported several side effects at which point the Quinagolide was discontinued and switched to cabergoline. Her Prolactin level checked at that time was down to 1495 in February 2016. She was then switched to Bromocriptine to better control her Prolactin levels and however she reported developing adverse effect including nausea and headache, therefore, this medication was also stopped.

A surgical option was discussed with the surgical team and transsphenoidal excision of pituitary microadenoma (Stealth-guided transsphenoidal removal of pituitary adenoma in 2016) was offered to assist treatment with of amenorrhea and pregnancy. Her periods resumed and she was able to conceive in 2017, and she is currently a mother of a 4-year-old.

She started to experience irregular menstrual periods (amenorrhea) in January 2020 and second transsphenoidal surgery was offered in May 2022. She developed diabetes insipidus and secondary adrenal insufficiency following the second surgery.

Her post-operative prolactin level is now normal at 214 implying a biochemical cure.

Discussion:
Medical therapy is the first treatment for prolactinoma after excluding secondary causes of hyperprolactinemia (Zamanipoor Najafabadi et al., 2020). In most cases the use of DA which normalise prolactin levels, reverse hypogonadism, and promote tumour shrinkage. In over 90% of cases DA facilitates restoration of fertility (Szmygin, Szydełko and Matyjaszek-Matuszek, 2022). Surgical intervention of microprolactinomas is indicated for patients who are resistant to medical therapy or those that experience severe side effects due to treatment or if they develop neurological deficits or pituitary apoplexy (Ma et al., 2018; Donoho and Laws, 2019).

Recurrent and aggressive microprolactinomas often require surgical intervention. Surgical management of microprolactinomas is through transsphenoidal or, less frequently, a trans-frontal surgical approach. In 70-90% of cases adenectomy results in normalization of prolactin levels with little morbidity and no mortality (Jan et al., 2007).

Question for discussion:

1. Would you manage this patient microprolactinoma differently?

A rare case of Sheehan's syndrome

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Sheehan's syndrome is postpartum hypopituitarism caused by ischemic pituitary necrosis, usually the result of severe hypotension or shock caused by severe postpartum haemorrhage during or after delivery. It is a rare cause of hypopituitarism in developed countries owing to advances in obstetric care. It is still frequent in underdeveloped and developing countries. History of PPH, failure to lactate and cessation of menses are important clues to the diagnosis. Patients with SS mostly present with panhypopituitarism. Haematological abnormalities are common. Anaemia is believed to be due to deficient anterior pituitary hormones. The main radiological finding of SS is empty sella or partially empty sella. SS often evolves slowly and hence is diagnosed late. Early diagnosis and appropriate treatment is necessary to reduce the morbidity and mortality of patients.

We present a case of 47 year old Somalian lady with limited English, who presented in hospital with epigastric pain, with a working diagnosis of aortic dissection by ED and GI bleed by medicine. Bloods showed normocytic anaemia, Hb 99g/L (120 – 150), MCV 94.4f/L (83 – 100). ECG and CT angiogram aorta ruled out cardiac cause and plan was made for outpatient endoscopy. She was seen by Endocrine consultant on post take ward round who picked up her history of amenorrhoea since delivery of her 3rd child in 1998 which was complicated by major postpartum haemorrhage.

They requested haematinics and full pituitary profile to exclude long term Sheehan's syndrome. Results showed severe iron deficiency anaemia and panhypopituitarism, TSH 1.08 (0.27 – 4.2), FT4 <1.3 pmol/L (12 - 22), FT3 <1.5 pmol/L (3.1 – 6.8), prolactin 11 mIU/L (<699), LH 0.9 IU/L, FSH 2.9 IU/L, morning cortisol 115 nmol/L. She was diagnosed with Sheehan's syndrome with panhypopituitarism, and iron deficiency anaemia secondary to her profound hypothyroidism. She was commenced on hydrocortisone and Levothyroxine. MRI pituitary and bone scan was requested to look for bone health. Plan was made for outpatient clinic, to review for growth hormone replacement, HRT and whether anaemia is corrected after thyroid hormone replacement.

After the diagnosis, endocrine team discovered that sadly, she had investigations for infertility in the UK, back in 2008 where they picked up her history and diagnosed likely hypogonadotropic hypogonadism but had not ever considered her wider pituitary function. Good learning point for all endocrine teams about close working relationships with fertility services. Importance of excellent history and counselling her about newly diagnosed condition given language barrier for complex discussions. Many of her symptoms were just part of her difficult journey.

Questions for discussion:

1. Could this delay in diagnosis and management have been prevented?
2. What interventions can we take in future to help with early diagnosis and prompt management?

Silent corticotroph pituitary adenoma: clinical presentation mimicking pituitary apoplexy

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Background:
Silent corticotroph adenomas (SCAs) are a subtype of non-functioning pituitary adenomas (NFAs) that demonstrate positive staining for adrenocorticotropic hormone (ACTH) in the absence of overt Cushing's disease. SCAs are biologically more aggressive tumours than NFAs. We present a case of a large SCA presenting as pituitary apoplexy.

Case: A 48-year-old lady presented to her optician with 6 months of peripheral visual loss (predominantly on the left) with headaches. She was referred for a routine MRI. In the meantime, while on holiday in Columbia, she developed sudden worsening of headache and vomiting with deterioration in vision. She had an urgent MR Pituitary in Columbia and was recommended urgent surgery given MRI scan showed a large macroadenoma with pituitary apoplexy.

She preferred to fly back to UK. On her return, she presented to A&E with ongoing headache and visual loss. She was commenced on Prednisolone while in Colombia. She did not have any past medical history and was not on any regular medications. She has history of weight gain around 9 KG in one year but did not have any Cushingoid features. He visual fields showed bitemporal hemianopia. Bloods showed normal thyroid and gonadal axis. Prolactin was mildly elevated at 581 mIU/L (40-540) and IGF-1 level was low at 4.4 nmol/L (6.9-28).

She underwent urgent endoscopic transsphenoidal resection with uneventful post-operative recovery barring persistent headaches. Postoperative pituitary hormonal profile was satisfactory with a day 2 cortisol level of 581 nmol/L and Hydrocortisone was stopped. Postoperatively visual fields improved significantly.

Histology showed patchy ACTH expression with no Crooke's hyaline changes. Further classification based on transcription factors confirmed positivity for TPIT-lineage in keeping with a silent coticotroph macroadenoma with a low proliferative index.

Question for discussion:
1. What would be the recommended frequency and tests for post operative biochemical screening for silent corticotroph adenomas?

Endocrinopathy behind the facemask – Follow up

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A 44-year-old gentleman presented to the Emergency Department with a 2-week history of fever and rigors. Past medical history was unremarkable other than an earlier diagnosis of hypertension. He was noted to have new onset atrial fibrillation with rapid ventricular response; a diagnosis of hypertrophic obstructive cardiomyopathy (HOCM) was made on echocardiography. In addition, a vegetation was identified on the mitral valve. Treatment for infective endocarditis (Streptococcus oralis) was initiated and he was subsequently transferred to a specialist centre for mechanical mitral valve replacement surgery. Warfarin was commenced post operatively with target INR of 3.5-4.5. During the admission, a history of chronic headaches was investigated. MRI pituitary revealed a 3.8 x 1.9 cm macroadenoma with suprasellar extension and invasion of the right cavernous sinus.

He was further evaluated in the outpatient endocrine clinic. On removal of his facemask, examination revealed typical acromegalic features with supraorbital ridge prominence, significant underbite and macroglossia. Visual fields were normal to confrontation testing. Pituitary function showed: IGF-1 140.3nmol/l (range 8.5-31.0), 9am cortisol 352nmol/L (range 200-750), prolactin 1119mU/L (range 60-300), TSH 1.98mU/L (range 0.34-5.60), FSH <0.U/L (range 1.7-8.0), testosterone 8.7 (range 10.0-30.0). Acromegaly was confirmed with an oral glucose tolerance test. The patient was subsequently discussed in Pituitary MDT and commenced on monthly Lanreotide injections to induce tumor size reduction while awaiting pituitary surgery.

His IGF-1 remained high despite monthly Lanreotide and therefore Cabergoline was added for further medical management whilst planning his surgery, since this required close collaboration with his cardiothoracic team regarding his cardiac function and prosthetic valve in addition to extensive discussions with Haematology regarding plans for safe peri-operative anticogulation. He underwent successful transphenoidal pituitary surgery; subsequent MRI imaging revealed a good surgical resection with histology confirming a sparsely granulated somatotroph pituitary adenoma (Ki67 1%).

Despite a good surgical outcome, his post-operative course was complicated by repeated episodes of epistaxis requiring regular input from Haematology and ENT teams. Anticogulation was cautiously continued due to the high risk of mechanical valve related thrombosis. Control of epistaxis was eventually achieved following a return to theatre for ligation of the sphenopalatine artery. He was discharged once INR was within the therapeutic target range.

This case highlights the requirement for early diagnosis and treatment to prevent further complications as well as the need for individualisation of complex treatment decisions through the involvement of multiple specialties within multidisciplinary team working patterns.

Questions for discussion:

1. Would earlier diagnosis of his acromegaly have changed his cardiovascular outcome?
2. Does the audience have any experience of managing a patient undergoing pituitary surgery who was unable to stop their anticoagulation pre-operatively?