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Twenty Second Clinicopathological Conference on Pituitary Disease

Monday 3rd February 2020

Cavendish Conference Centre, 22 Duchess Mews, London, W1G 9DT

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AGENDA

08:30 Registration

09:25 **Welcome and Introduction**
Prof Stephanie Baldeweg, Consultant Endocrinologist, University College London Hospitals

09:30 **Key Note Lecture: The Pituitary in 2019**
Prof Will Drake, Consultant Endocrinologist, QMUL

Forum 1 – Case Presentations, Theme: Functional tumours
Chairs: Prof Stephanie E Baldeweg & Mr Neil Dorward

10:00 **A case of an impressive giant prolactinoma**
Dr Milanka Wattegama, Honorary Fellow in Endocrinology, Department of Endocrinology, Queen Elizabeth Hospital, University Hospitals Birmingham NHS Foundation

10:15 **Cabergoline: The Ethics of Side Effects**
Dr Risheka Ratnasabapathy, ST7 Registrar, Imperial College Healthcare NHS Trust

10:30 **A Case of Cyclic Cushing's Disease - Cortisol Surfing; Catching the Cortisol wave!**
Dr Mark Sutton-Smith, ST7, Imperial Centre for Endocrinology, Imperial College Healthcare NHS Trust, Hammersmith Hospital, London

10:45 **Acromegaly – what next?**
Dr Waiel Bashari, Clinical Research Fellow in Endocrinology, University of Cambridge

11:00 Exhibition, Refreshments and Poster Display

11:30 **Radiation damage and vision**
Dr Gordon Plant, Consultant Neurophthalmologist, NHNN

Forum 2 – Case Presentations, Theme: Pituitary all sorts
Chairs: Dr Umasuthan Srirangalingam & Mr Hani Marcus

12:00 **Identification of a cause for Diabetes Insipidus many years after presentation**
Dr Rakshit Kumar, ST7 Specialist Registrar in Diabetes & Endocrinology, Department of Diabetes & Endocrinology, Guys & St Thomas' NHS Foundation Trust

12:15 **Challenging Craniopharyngioma...Management Dilemma**
Dr Ziad Hussein, Clinical Research Fellow, University College London Hospitals NHS Foundation Trust

12:30 **PET-guided endoscopic transsphenoidal surgery can be an effective treatment option for patients with persistent acromegaly due to previously deemed unresectable lateral disease**
Mr Angelos Kolias, Clinical Lecturer in Neurosurgery, Addenbrooke's Hospital and University of Cambridge

12:45 **Delayed pituitary metastasis of renal cell carcinoma: a highly vascular phenomenon. A case report and literature review**
Mr Richard Moon, Specialist Trainee in Neurosurgery, Southmead Hospital, Bristol

13:00 **Steroid news**
Prof John Wass, Consultant Endocrinologist, Oxford

13:10 Exhibition, Refreshments and Poster Display

14:00 **The Pituitary Foundation**
Mrs Menai Owen-Jones, CEO, The Pituitary Foundation

14:15 **Nelson's syndrome: outcome after primary treatment**
Dr Niki Karavitaki, Senior Clinical Lecturer in Endocrinology & Honorary Consultant Endocrinologist, Queen Elizabeth Hospital Birmingham

Forum 3 – Case Presentations, Theme: Pituitary apoplexy
Chairs: Dr James Ahlquist & Miss Joan Grieve

14:55 **Pregnancy with pituitary apoplexy: Time to Act**
Dr Aiyappa Biddanda, ST3, Basildon and Thurrock University Hospital NHS Trust

15:10 **Sheehan like syndrome in males: A case series of three patients developing pituitary infarction following hypotension**
Dr Ambreen Qayum, SpR Endocrinology and Diabetes, Imperial College Healthcare NHS Trust

15:25 **Peculiar pathology of a sellar lesion and management dilemmas**
Dr Athanasios Fountas, Post-CCT Fellow, Queen Elizabeth Hospital, University Hospitals Birmingham NHS Foundation Trust

15:40 Exhibition, Refreshments and Poster Display

16:00 **Senescence markers in relation to the hormone-producing pituitary tumour cells**
Prof JP Martinez-Barbera, Professor of Developmental Biology and Cancer, UCL

Forum 4 – Case Presentations Theme: Macroadenomas
Chairs: Prof Pierre-Marc Bouloux & Mr Michael Powell

16:30 **Pituitary macroadenoma and a colloid cyst of the third ventricle: dilemmas and options**
Dr Bessie Kwok, Junior Clinical Fellow in Endocrinology, St Bartholomew's Hospital

16:45 **Management of suprasellar pituitary macroadenomas with temporal extension**
Mr Jarnail Bal, RCS Skull Base Fellow, Queens Hospital

17:00 **Poster and case presentation prizes**
Prof Stephanie Baldeweg, Consultant Endocrinologist, University College London Hospitals

17:15 Close

FACULTY

PANEL & ORGANISERS

FACULTY

Dr James Ahlquist, Consultant in Endocrinology and Diabetes, Southend Hospital

Prof Stephanie E Baldeweg, Consultant Endocrinologist, University College London Hospitals

Prof Pierre-Marc Bouloux, Consultant Endocrinologist, Royal Free London

Mr Neil Dorward, Consultant Neurosurgeon, National Hospital for Neurology and Neurosurgery

Prof Will Drake, Consultant Endocrinologist, Queen Mary University of London

Miss Joan P Grieve, Consultant Neurosurgeon and Clinical Lead for Neurosurgery, National Hospital for Neurology and Neurosurgery

Dr Ziad Hussein, Clinical Research Fellow, University College London Hospitals NHS Foundation Trust

Dr Niki Karavitaki, Senior Clinical Lecturer in Endocrinology, University Hospitals Birmingham

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Prof JP Martinez-Barbera, Professor of Developmental Biology and Cancer, UCL GOS Institute of Child Health

Mrs Menai Owen-Jones, CEO, The Pituitary Foundation

Dr Gordon Plant, Consultant Neurophthalmologist, NHNN

Mr Michael Powell, Consultant Neorosurgeon, National Hospital for Neurology and Neurosurgery

Prof John Wass, Consultant Endocrinologist, Oxford

ORGANISING COMMITTEE

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Consultant Neurosurgeon, Sheffield Teaching Hospitals

Dr Umasuthan Srirangalingam
Consultant in Endocrinology & Diabetes, University College London Hospitals

Mr Nick Thomas
Consultant Neurosurgeon, King's College Hospital, London

1.) A case of an impressive giant prolactinoma

Authors

Dr Milanka Wattegama[1,2,3,] Dr Athanasis Fountas[1,2,3,] Mrs Miriam Asia[1,] Dr Swarupsinh Chavda[4,] Mr Georgios Tsermoulas[5,] Miss Ruchika Batra[6,] Dr Niki Karavitaki[1,2,3]
[1]Department of Endocrinology, Queen Elizabeth Hospital, University Hospitals Birmingham NHS Foundation, Birmingham, United Kingdom, [2]Center for Endocrinology, Diabetes and Metabolism, Birmingham Health Partners, Birmingham, United Kingdom, [3]Institute of Metabolism and Systems Research, College of Medical and Dental Sciences, University of Birmingham, Birmingham, United Kingdom, [4]Department of Radiology, Queen Elizabeth Hospital, University Hospitals Birmingham NHS Foundation, Birmingham, United Kingdom, [5]Department of Neurosurgery, Queen Elizabeth Hospital, University Hospitals Birmingham NHS Foundation , Birmingham, United Kingdom, [6]Department of Opthalmology, Queen Elizabeth Hospital, University Hospitals Birmingham NHS Foundation , Birmingham, United Kingdom

Biography:

Dr. Milanka Wattegama is an international clinical fellow currently working as an honorary fellow in endocrinology at the Queen Elizabeth Hospitals Birmingham.
She completed her MBBS with first-class honors and obtained her MD in Internal Medicine from the University of Colombo, Sri Lanka. She completed her MRCP(UK) in January 2018. She is in the process of completing her specialist training in Endocrinology in Sri Lanka.
Her interests are Neuro-endocrinology, reproductive endocrinology and diabetes.
She has numerous papers In local and peer-reviewed journals.

Abstract

A 21-year-old male complained of reduced vision in the left eye for one year. He was seen by his optician and bitemporal hemianopia was detected. He was referred to Ophthalmology and subsequent brain MRI showed a 6x7x6.5cm heterogenous sellar mass with a multicystic component and a central solid component; the lesion extended to the cavernous sinuses (encasing the left internal carotid artery which appeared to be narrower compared to the right), suprasellar area with chiasmal compression, left lateral and anterior cranial fossa. There was displacement and encasement of the anterior cerebral arteries, compression of the frontal horns of both lateral ventricles, compression of left temporal lobe, and compression and stretching of the genu of corpus callosum. The tumour was considered to be a craniopharyngioma and the patient was referred urgently for neurosurgical management.
On further questioning, he confirmed temporal headaches and no changes in behaviour. He also reported low energy levels, erectile dysfunction and absence of facial hair. Neuro-ophthalmology assessment revealed severe left optic nerve dysfunction with reduced visual acuity, constricted Goldmann visual fields and significantly reduced colour vision. There was also mild right nerve dysfunction. He was obese with sparse facial and trunk hair and had mild provoked right galactorrhoea. His testes were 25 ml (left) and 20ml (right). Biochemistry revealed prolactin 229,503mU/L (83-325mU/L) consistent with prolactinoma. There was also, hypogonadotropic hypogonadism with normal TSH, ACTH reserve and normal IGF-I and calcium levels.
He also had microcytic hypochromic anaemia. He was commenced on cabergoline 250mcg twice weekly with gradual up titration. One month later, he had excellent visual acuities and full Goldmann visual fields with prolactin at 7853 mU/L. Pituitary MRI four months later showed reduction in the size of the tumour (4.7 x 3.8cm) with better visualization of the optic chiasm. Nine months since diagnosis, he was on cabergoline (2 mcg weekly) with no adverse effects on it; he had achieved normal prolactin and he was on testosterone replacement due to persistent hypogonadotropic hypogonadism. He had no family history of pituitary adenoma and genetic testing for AIP mutations was negative.
Giant prolactinomas (measuring >4 cm) are a rare entity. Diagnosis can be triggered due to acute neurological deterioration despite long-standing, subtle endocrine manifestations in the background. In our case, the imaging features of the tumour and particularly the significant cystic component, lead initially to the suspicion of craniopharyngioma and referral for surgery but hormonal assessment guided the optimal management approach. Despite their often-impressive size, extensions and invasiveness, giant prolactinomas show optimal response to dopamine agonists in most cases (usual dose of cabergoline in the range of 1.5 -5 mg week); normal prolactin is achieved in >60% of cases and 50% reduction in adenoma size is seen in >50% of patients within 6-12 months. Visual field improvement is detected in most patients on cabergoline within a few days after starting treatment. Our patient had a remarkable responsiveness to a relatively low dose of cabergoline making surgical intervention (combined or not with radiotherapy) not necessary.

2.) Cabergoline: The Ethics of Side Effects

Authors

Dr Risheka Ratnasabapathy[1,] Dr Ambreen Quyam[1,] Dr Bijal Patel[1,] Dr Preeshila Behary[1,] Dr Shamaila Zaman[1,] Dr Niamh Martin[1]
[1]Imperial College Healthcare NHS Trust

Biography:

Risheka Ratnasabapathy is a ST7 registrar who practices in both Diabetes and Endocrinology as well as General Internal Medicine. She trained at Imperial College London where she also recently completed her PhD.

Abstract

Introduction: Dopamine agonist therapy has long been used to treat prolactinomas for symptomatic relief, biochemical recovery and radiological shrinkage of pituitary tumours. As clinicians we are proficient at screening for sided effects but are we holistic enough in our approach? We present two cases of the damaging behavioural consequences of Cabergoline treatment for prolactinomas.

Case 1: A 49 year old Colombian father of two, in a long term steady relationship, presented with a tonic clonic seizure in April 2018. MRI showed a large 2.3 x 2.5 x 2.5cm sellar and suprasellar mass with cystic and haemorrhagic components and compression of the optic chaism and an unrelated right frontal tumour, histologically an oliogodendroglioma which was debulked and undergoing active surveillance. Initial prolactin levels were 50,205 and the remainder of his pituitary profile showed TSH 0.75, T4 9.5, ACTH 6.9, cortisol <28, LH 0.9, FSH 1.3, testosterone 1.7, GH <0.05. He was commenced onto cabergoline 500mcg twice weekly and 4mg prednisolone once daily. He responded well biochemically to Cabergoline with prolactin falling from 50,205 to 816 over 5 months. Eighteen months later, he reported a change in his sexual behaviour. He was using escort services and was engaging romantically with work colleagues for the last 1 year. In light of this disinhibited behaviour, the Cabergoline was discontinued in November 2019 and his repeat prolactin levels in December 2019 were 583 with a testosterone of 12.

Case 2: A 56 year old gentleman who was in a long term relationship with his male partner, presented with erectile dysfunction in October 2018. His prolactin was 3,464 with testosterone of 4.5. The remainder of his pituitary profile revealed TSH 1.07, T4 11.3, cortisol 189, LH 1.8, FSH 2.1, Testosterone 4.5 and SHBG 31. An MRI Pituitary revealed a 11 x 9 cm pituitary adenoma with no impingement of the optic chiasm. He was commenced onto Cabergoline 250mcg once weekly. His prolactin fell from 2464 to 18 within 5 months, with testosterone levels of 18.9. In October 2019, accompanied by his concerned partner, he described a change in personality, and hypersexuality. Cabergoline was stopped in light of his disinhibited behaviour. However, the patient was anxious about a return of his previous low mood, low energy and gynaecomastia. Testosterone therapy (Tostran 2% 10mg) was commenced. His prolactin in November 2019 was 1942.

Discussion: These two cases raise important physiological and ethical questions. Firstly, how much of the observed hypersexual behaviour is a direct side effect of dopamine agonist therapy and how much is a behavioural consequence of the return of ‘normal’ prolactin and testosterone levels? Secondly, when an otherwise effective treatment results in behavioural side effects such as the collapse of a marriage or gambling, whose ‘fault’ is it when huge debts emerge and relationships fail? One must exert caution when prescribing dopamine agonist therapy to avoid social harm to patients and this must be balanced with depriving patients of effective non-invasive treatments by being mindful of the power of suggestion bias when screening for side effects.

3. A Case of Cyclic Cushing's Disease - Cortisol Surfing; Catching the Cortisol wave!

Authors

Dr Mark Sutton-Smith[1], Dr Zeeshan Yasin[1], Dr Hessa Boharoon[1], Dr Idowu Oluwagbemiga[1], Mr Ramesh Nair[2], Dr Jeannie F. Todd[1]
1Imperial Centre for Endocrinology, Imperial College Healthcare NHS Trust, Hammersmith Hospital, London, United Kingdom,
[2]Department Neurosurgery, Imperial College Healthcare NHS Trust, Charing Cross Hospital, London, United Kingdom

Biography:
ST7, Imperial Centre for Endocrinology, Imperial College Healthcare NHS Trust, Hammersmith Hospital

Abstract

A 28-year-old woman presents with one-year history of marked weight gain of 18 kg, increasing hirsutism and night sweats. Clinically, hyperandrogenism and Cushing's syndrome were suspected. A diagnosis of Polycystic ovarian syndrome was made based on Rotterdam criteria (hyperandrogenism and polycystic ovaries on ultrasound) and Metformin was initiated.

Initial testing for Cushing's syndrome demonstrated a high urinary free Cortisol (306 nmol/L) with incomplete Cortisol suppression on an overnight dexamethasone suppression test (Cortisol 60 nmol/L). Plasma metanephrines and Magnetic Resonance Imaging (MRI) of the adrenals were normal. However, a follow up low dose dexamethasone suppression test (48 hour Cortisol <28 nmol/L) and repeat overnight dexamethasone suppression test (Cortisol 44 nmol/L) were normal; casting doubt on the diagnosis of Cushing's syndrome. Clinical suspicion for Cushing's syndrome remained high as she continued to gain weight and experienced cyclic symptoms of rapid weight gain, easy bruising and emotional lability. In addition, serial midnight salivary Cortisol and Cortisone measurements were elevated ranging 3.5-7.4 nmol/L (normal <2.6 nmol/L) and 12.8-31.3 mol/L (normal < 18 nmol/L), respectively. Later a follow-up overnight dexamethasone suppression test showed no suppressibility of Cortisol (84 nmol/L).

The clinical suspicion for Cushing's syndrome was reinvigorated and Cyclic Cushing's syndrome was now suspected. A pituitary MRI showed a 3mm right sided microadenoma and inferior petrosal sampling clearly confirmed a central ACTH source. Endoscopic transsphenoidal pituitary tumour resection was scheduled and performed using Brainlab image guidance. There were no peri-operative complications and a Day 4 Cortisol (<28 nmol/L) was consistent with surgical remission. Histology of the pituitary lesion showed clear features of a Corticotroph adenoma. A follow up six-week post-operative insulin tolerance test confirmed secondary hypoadrenalism (peak Cortisol 112 nmol/L) and prednisolone was started. Later prednisolone was switched to hydrocortisone due to an undesirable side effect profile. Post operatively patient symptomology has now resolved with diminishment of Cushingoid features over time. She is having periods every 28 days (previously 26 days) and has plans for pregnancy in the coming year.

Cyclic Cushing's syndrome is a rare, difficult to diagnose, form of Cushing's syndrome. It is often overlooked and is characterised by a cyclic pattern of normal Cortisol interspersed with episodes of hypercortisolaemia. Intercycles periods can range from days to months. With the advent of the obesity epidemic it is becoming increasingly important for clinicians to remain vigilant when clinical suspicion of Cushing's syndrome is high despite conflicting Cushing's syndrome screening results. We recommend the use of the various Cortisol screening tests in a temporal fashion and make use of a symptom diary whenever Cyclic Cushing's syndrome is suspected as the Cortisol Wave can be easily missed.

4.) Acromegaly – what next?

Authors

Dr Waiel Bashari[1,2], Dr Russell Senanayake[1,2], Dr Olympia Koulouri[1,2], Dr James MacFarlane[2], Sr Laura Serban[2], Dr Andrew Dean[2], Dr Dominic O'Donovan[2], Mr Angelos Kolias[1,2], Mr Neil Donnelly[2], Mr Richard Mannion[2], Professor Mark Gurnell[1,2]
[1]University of Cambridge, Cambridge, United Kingdom, [2]Addenbrooke's Hospital, Cambridge, United Kingdom

Biography:
Waiel Bashari is a Clinical Research Fellow in Endocrinology, currently undertaking a PhD in Clinical Biochemistry with the University of Cambridge. He is supervised by Professor Mark Gurnell, and is a member of the endocrine molecular imaging group, where his focus is on the evaluation of 11C-methioinine PET as a functional imaging technique in pituitary disease. He has recently published a comprehensive review of modern imaging of pituitary adenomas and is preparing several primary manuscripts and other reviews based on his PhD studies. He was awarded the prize for best poster for the Neuroendocrinology, Pituitary and Neoplasia section at the British Endocrine Societies 2019 conference in Brighton.

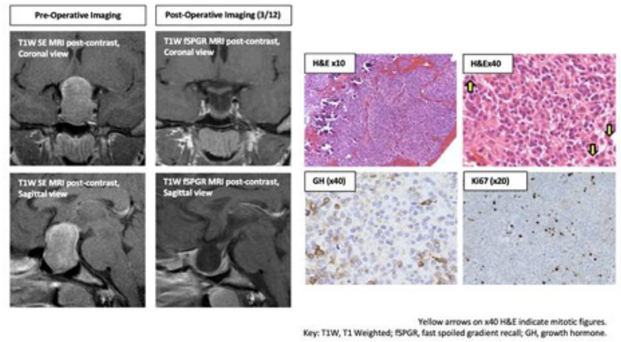
Abstract

Background
Modern acromegaly management often requires a multimodal approach, but with transsphenoidal surgery (TSS) remaining the primary intervention in the majority of patients. Medical therapy [e.g. somatostatin analogue (SSA), dopamine agonist (DA), pegvisomant] are effective adjunctive treatment options in cases with persistent disease following TSS, but typically must be continued long-term to control residual growth hormone (GH) and/or insulin-like growth factor 1 (IGF-1) hypersecretion. Radiotherapy may be considered in those with difficult to control disease (especially when there are concerns regarding tumour growth), in patients who wish to avoid long-term medical therapy, or in a subgroup for whom primary surgery is not possible or preferred. In recent years, there has been increasing interest in the possibility of individualised decision-making based on clinical, biochemical, radiological, histological and genetic parameters.

Case Report
We describe the case of a 25-year-old woman who presented to her local hospital with visual loss that had developed over a 2-3 month period. Aside from occasional headaches she was otherwise well. Ophthalmic assessment demonstrated a dense bitemporal field defect. An urgent MR scan revealed a large sella-based lesion (Figure) in keeping with a macroadenoma, and she was transferred as an emergency to our centre. On admission, she was noted to have numerous clinical features of acromegaly, which was confirmed biochemically with the finding of an elevated IGF-1 (72.8 nmol/L; upper limit of normal =43.2). Trans-sphenoidal surgery (TSS) was undertaken, with good tumour clearance and no postoperative complications (Figure). Histology confirmed a somatotroph adenoma, with patchy granular staining for GH on immunohistochemistry. Concerningly, a high proliferation index was observed (MIB-1 =15%) along with numerous mitotic figures (Figure). Postoperative assessment demonstrated: an excellent visual response; retention of normal anterior pituitary function; and early normalisation of serum IGF-1 (33.8 nmol/L). 9am GH was 0.73 mcg/L; however, GH nadir in an oral glucose tolerance test (OGTT) was 0.83 mcg/L. The patient was referred for genetic assessment.

Repeat pituitary MRI at 3 months postoperatively confirmed an excellent surgical result, with only a thin rim of enhancing soft tissue within the sella. After careful consideration, a decision was taken to undertake close surveillance and the patient remained clinically well with a normal IGF-1 until month 10. At this point clinical and biochemical evidence of recurrent disease became evident and repeat MRI identified a nodule posterior to the insertion of the infundibulum, which correlated with an area of intense focal tracer uptake on 11C-methionine PET-CT/MRICR.

Discussion
We would welcome the panel's thoughts on further management in this patient – in particular which treatment modality (modalities) would they now recommend? In addition, in light of the subsequent clinical course following primary surgery, and based on the histological findings, should adjunctive therapy have been considered at an earlier stage?



Yellow arrows on x40 H&E indicate mitotic figures.
Key: T1W, T1 Weighted; fSPGR, fast spoiled gradient recall; GH, growth hormone.

5.) Identification of a cause for Diabetes Insipidus many years after presentation

Authors

Dr Rakshit Kumar[1,] Dr Paul Victor Carroll[1,] Dr Mamta Joshi[1]
[1]Department of Diabetes & Endocrinology, Guys & St Thomas' NHS Foundation Trust, , United Kingdom

Biography:
ST7, Specialist Registrar in Diabetes & Endocrinology

Abstract

A 35 year old Caucasian male was assessed for symptoms of thirst and polyuria in 2012. Symptoms had started in 2005 but had gradually worsened leading to a review with the General Practitioner and referral for consideration of endocrine pathology. The patient estimated fluid intake of 8 to 10 litres per day with >5 episodes of nocturnal polyuria. The past medical history included scalp psoriasis and localised Ocular Melanoma in 2003, treated with enucleation of the orbit with no systemic therapy. Systemic examination was unremarkable apart from BMI of 31 kg/m2. Investigations in 2012 showed plasma osmolality 297 (285-295 mOsmol) and Sodium of 143 (135-145 mmol/L). Anterior pituitary function, calcium and glucose related tests were satisfactory. Water Deprivation test confirmed isolated Cranial Diabetes Insipidus (CDI). MRI of the pituitary was unremarkable with preserved posterior pituitary bright spot and normal appearance of infundibulum and hypothalamus.

Symptoms improved with Desmopressin. Surveillance MRI pituitary after 24 months did not show progressive pathology. Over next 6 years, he had adequate symptom and biochemical control of CDI with normal range pituitary hormones. In 2018, his diagnosis was revisited with a plan to repeat MRI scan to ensure there is no neoplastic or infiltrative process. The MRI scan showed nodular thickening of the pituitary stalk, which was not seen on earlier images. Hence, (with a history of melanoma and developing anatomical abnormality) FDG PET study was organised. Serum tests for Germinoma and IgG4 related hypophysitis remained unremarkable.

The FDG PET study showed a small focal lesion, on the top of the pituitary stalk, not consistent with metastasis. There were bilateral femoral head areas of avidity, reported as consistent with metastases. The patient mentioned thigh pain for the preceding three months.

To make a diagnosis bone biopsy and whole body MRI were performed. The biopsy confirmed a diagnosis of Langerhans' Cell Histocytosis (LCH) involving bilateral femoral bone, with largest lesion of 7cm. This required urgent prophylactic surgery to prevent pathological fracture, followed by local radiotherapy to femur and systemic chemotherapy with Cladribine. We attribute the Cranial DI to LCH, reflecting multi-site disease.
The presentation of LCH as isolated CDI is rare and most patients present with pituitary disease as part of multi-site disease. This patient presented with CDI many years before the causative diagnosis was made. If the pituitary stalk LCH increases in volume, we will consider low dose External Beam Radiotherapy (EBRT).

This case emphasises need for detailed investigation of the patient presenting with CDI including whole-body functional imaging. The clinical context and presence of bone LCH allowed us ultimately make a diagnosis. LCH should be considered in patients presenting with 'isolated' DI and advances in imaging may improve the diagnostic yield in these patients.

6.) Challenging Craniopharyngioma...Management Dilemma

Authors:

Dr Z Hussein[1,] Prof SE Baldeweg[1,] Mr H Marcus[2,] Dr N Fersht[1,] Mr N Dorward[2]
[1]University College London Hospital, London, United Kingdom, [2]National Hospital for Neurology and Neurosurgery, United Kingdom

Biography:

Clinical Research Fellow, University College London Hospitals NHS Foundation Trust
Speciality Registrar in Endocrinology, Sheffield Teaching Hospitals NHS Foundation Trust

Abstract

Introduction
Craniopharyngiomas (CPs) are benign intracranial epithelial tumours of unpredicted growth potential, their location and infiltrative tendency to the surrounding structures can cause severe endocrine, visual and hypothalamic complications. The optimal management of these tumours remains challenging and often patients suffer from complex morbidities and quality of life impairment.

Case report
We report a 30-year-old patient with craniopharyngioma. He was diagnosed aged 9 with initial presentation of growth failure, underwent stereotactic aspiration and received 50.1 Gray of intensity-modulated radiotherapy in 2000. Consequently, he developed anterior pituitary insufficiency. Regular ophthalmology assessment showed normal visual acuities (6/6) and visual fields but pale optic discs which were stable. Over a decade of radiological follow-up, the tumour continued to be confined to the suprasellar space, indented the third ventricle, and was inseparable from the optic chiasm; however, no compression of the anterior visual pathway was demonstrated.
He was referred to the adult endocrinology services in 2010. Subsequent periodic MRI scans showed gradual regression in the neoplasm size with less indentation of the third ventricle. The patient continued on hormone replacement therapy including growth hormone, clinical and biochemical assessments were satisfactory. He achieved well at university and gained a high degree in Architecture.
18 years post initial therapy, surveillance scan demonstrated unexpected progression in craniopharyngioma size particularly the suprasellar component, reaching the interthalamic connexus and splaying the optic chiasm further. Meanwhile, the patient experienced deterioration in his vision which was confirmed on formal assessment.
Surgical debulking via extended endoscopic transsphenoidal approach was attempted to decompress the optic apparatus; however, the optic nerve was found to be very tenuous and attached to the tumour therefore the procedure was abandoned. Repeat imaging 6 months later showed a further increase in craniopharyngioma size.
There have been case reports of successful treatment of CPs with BRAF inhibitors; unfortunately, our patient was not found to have BRAF mutation. After several MDT discussions, reirradiation was considered to be potentially risky intervention especially for visual outcome; conservative approach was deemed to be the best option at this stage with close monitoring of tumour size. Furthermore, PCV chemotherapy regime (procarbazine, CCNU and vincristine) is a possible option in managing this tumour if more growth is shown in the future.

Discussion points:
- The role of growth hormone replacement in growing craniopharyngioma
- The role of further surgical excision/radiotherapy in managing this patient
- Is there an efficient medical treatment for craniopharyngioma?
- The use of pre-operative optic nerve tractography to better identify the thinned optic nerves, and intra-operative visual evoked potential to monitor optic nerve function

7.) PET-guided endoscopic transsphenoidal surgery can be an effective treatment option for patients with persistent acromegaly due to previously deemed unresectable lateral disease

Authors

Mr Angelos Kolias[1], Dr Waiel Bashari[2], Dr Russell Senanayake[2], Dr Olympia Koulouri[2], Dr James MacFarlane[2], Mr Daniel Gillett[3], Dr Heok Cheow[3], Dr Iosif Mendichovszky[4], Dr Andrew Powlson[2], Mr James Tysome[5], Mr Thomas Santarius[1], Mr Neil Donnelly[5], Professor Mark Gurnell[2], Mr Richard Mannion[1]
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Biography:
Angelos Kolias is a Clinical Lecturer in Neurosurgery in the Department of Clinical Neurosciences, Addenbrooke's Hospital and University of Cambridge, Cambridge, UK. His interests include neurotrauma, anterior skull base/pituitary surgery and spine surgery. He is also interested in the methodology of clinical neurosurgical research (particularly trials) and global neurosurgery. Currently he is working on i) the development of a core outcome set for pituitary surgery and ii) the implementation and evaluation of PET-guided surgery for pituitary adenomas. These are collaborative projects with Mr Richard Mannion (lead for pituitary surgery) and the academic endocrinology group led by Professor Mark Gurnell (lead for pituitary endocrinology).

Abstract

Aim
We have introduced functional / molecular imaging [with 11C-methionine Positron Emission Tomography Computed Tomography (PET/CT) co-registered with MRI (Met-PET-MRICR)] in order to facilitate more accurate localisation of primary and recurrent pituitary adenomas. Here, our aim was to determine if revision transsphenoidal surgery (TSS), guided by Met-PET/MRICR), can lead to remission in patients with persistent acromegaly due to post-operative lateral sellar / para-sellar disease remnant.

Methods
We identified 8 patients with persistent acromegaly following primary TSS, in whom further surgery had initially been discounted due to suspected lateral sellar / para-sellar disease. All patients underwent Met-PET/MRICR. Scan findings were used by the pituitary multidisciplinary team (MDT) to inform decision-making. Revision TSS was performed with wide lateral exploration as guided by the findings of Met-PET/MRICR.
We routinely use a binostril technique with two surgeons working together after the nasal stage of the procedure. In the cases described in this paper, particular care was taken to extend the opening laterally towards the cavernous sinus on the side identified by the pre-operative Met-PET/MRICR. Extensive exploration of the lateral sellar / para-sellar areas was performed with the use of 0° / 30° endoscopes. In cases where no abnormal tissue was seen, exploration of the whole fossa was performed, and biopsies were taken for histological assessment as necessary.

Results
Met-PET/MRICR demonstrated lateral sellar /para-sellar tracer uptake in areas of suspected residual disease in all included patients. Physiological uptake was also seen within the normal pituitary gland in several cases.
At surgery, in five patients, tumour was identified and resected, although histology confirmed somatotroph tumour in only four cases.
We did not identify grossly abnormal tissue in the other three patients but proceeded to thorough exploration and took specimens from areas that were thought to have equivocal tissue focusing on the side identified as abnormal by the Met-PET/MRICR.
Importantly, all patients achieved significant post-operative clinical and biochemical improvement [Insulin-like growth factor (IGF-1) < 1.2 x the upper limit of normal (ULN) in all cases, and fully normalised in six patients]. These findings have been maintained for up to 28 months (mean follow-up 13 months). No patient suffered any additional pituitary deficit or other complications.

Conclusion
Met-PET/MRICR was helpful in the evaluation of residual lateral sellar / para-sellar disease in acromegaly patients, and enabled us to proceed with re-do targeted surgical intervention.
We recognise that lateral sellar / para-sellar disease, during primary or re-do surgery, can be challenging and may be associated with a higher rate of complications. Our series has demonstrated that this management strategy is safe but the sample size is limited. Stereotactic radiosurgery may seem like an attractive option, due to the avoidance of the potential surgical risks. However, the latency period can be long, with a recent systematic review estimating an overall mean biochemical remission of 44.3% at a mean follow-up of 59 months, with the median time to remission at 41.5 months (1).

Reference
1. Updates in outcomes of stereotactic radiation therapy in acromegaly. Gheorghiu ML. Pituitary, 2017. 20:154–168.

8.) Delayed pituitary metastasis of renal cell carcinoma: a highly vascular phenomenon. A case report and literature review

Authors

Mr Richard Moon[1], Mr Will Singleton[1], Dr Paul Smith[2], Dr Kathryn Urankar[3], Dr Alison Evans[4], Mr Adam Williams[1]
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Biography:
Richard Moon is a specialist trainee in Neurosurgery in Bristol. He graduated from the University of Cambridge prior to completing Academic Foundation training in Liverpool.

Abstract

Tumour metastasis to the pituitary is rare, most commonly reported with either breast or lung cancer metastasising to the neurohypophysis. Pituitary metastases of renal cell carcinoma (RCC) are by contrast infrequently described. We report the case of a renal cell carcinoma pituitary metastasis 15 years after radical nephrectomy for primary disease and a review of the published literature.

A 69 year woman presented with cerebellar ataxia, migraines and retro-orbital headache. Past medical history included renal cell carcinoma treated by radical nephrectomy 15 years previously. There was no visual field deficit and screening bloods demonstrated hypocortisolaemia (9am cortisol <30 nmol/L) with moderately raised prolactin (1651 mU/L). Magnetic resonance imaging demonstrated a large sellar mass with suprasellar extension and prominent flow voids, with a radiological diagnosis most in keeping with pituitary macroadenoma. The patient underwent an endoscopic endonasal transsphenoidal adenectomy that was complicated by significant intraoperative haemorrhage from an unusually highly vascular tumour. Pathological examination of the tumour supported a diagnosis of metastatic clear cell renal carcinoma.

Literature review identified 41 published cases of RCC pituitary metastasis from 36 articles since 1984. The mean age at time of diagnosis with pituitary metastasis was 59.5 years (range 35-81 years, 73% male). Thirteen patients reported headache at the time of diagnosis, 29 reported visual disturbance and 30 had clinical or biochemical evidence of endocrine disturbance. Only seven patients were diagnosed with concurrent distal metastases. Pituitary metastasis was the initial presentation of RCC in 10 patients. The median time from RCC diagnosis to pituitary metastasis was 1 year (range 0-27 years), in contrast to the case presented. Surgical resection was performed for 28 patients, of which half reported a highly vascular tumour.

There is a paucity of literature reporting a delayed presentation of RCC pituitary metastasis. This case highlights the potential for delayed metastasis to the pituitary masquerading as a pituitary macroadenoma. Imaging findings consistent with a rich vascular supply should bring the diagnosis of RCC metastasis into the differential and are important to note when planning surgical resection

9.) Pregnancy with pituitary apoplexy: Time to Act

Authors

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Biography:
Aiyappa is an ST3 in North central London currently based in Basildon and Thurrock University Hospital NHS Trust.

Abstract

32F was referred to the endocrine service at Darent Valley Hospital as she developed amenorrhoea and galactorrhoea. Patient had normal menstrual cycles until the age of 17 when she started taking a combined low dose oestrogen contraceptive pill. This she stopped in March 2018 as she wanted to conceive.

May 2018: The patient failed to menstruate and developed galactorrhoea in Her Serum prolactin levels were raised at 1454 with TSH 1.26 and FT4 10

September 2018: Endocrine review and MRI Pit requested, possible prolactinoma.

October 2018: MRI Pituitary: Pituitary Macroadenoma 11.6 mm in keeping with prolactinoma with apoplexy. Patient reported severe headache in September (after her endocrine clinic appointment) which she put down to a migraine (though has no h/o migraine). Anterior Pituitary profile: 0900 am Cortisol 302, FSH 3.1, LH0.5, Prolactin 1558.

November 2018: Commenced on Cabergoline 25 mcg twice weekly. Advised to stop Cabergoline on conception. Follow up MRI in 3/12.

January 2019: MRI pituitary macro-adenoma remains the same size though the size of haemorrhage reduced.

February 2019: Resolving apoplexy, Resolved galactorrhoea, restored menstruation, Oestrogen 194, LH1.5, Prolactin 184(70-566).

June 2019: Endocrine clinic:- Pregnant and cabergolin stopped at conception. Prolactin 626, TSH 2.15, FT4 10.5, HCG: 1259. Visual fields on confrontation normal. Patient counselled to seek help if she develops headache, neck stiffness, visual defects.

November 2019: Presented with severe headache, no neck stiffness, no new neurology. Prolactin 2854 mU/L , Bioactive Monomeric Prolactin 3146 mU/L , Cortisol 255 nmol/L (185 - 624) , TSH 1.2 mU/L (0.3 - 5.0) ,Free T4 7.7 pmol/L (7.9 - 16.0) MRI Pituitary: Pituitary apoplexy is evident. Fluid fluid level with haemorrhage within a pituitary macro adenoma noted. The craniocaudal dimension is 1.4 cm. No optic chiasma, optic nerve compression.

Patient commenced on Cabergoline 250 mcg twice a week and discharged home to have OP follow up.

1. If a patient has pituitary apoplexy on two occasions, should she have caesarean rather than trial of labour? If a patient has a pituitary macro-adenoma can she be routinely be offered caesarean section if there is pituitary complication?
2. Is cabergoline indicated though out pregnancy in this scenario? What Counselling is required for this lady ?

10.) Sheehan like syndrome in males: A case series of three patients developing pituitary infarction following hypotension.

Authors

Dr Ambreen Qayum[1], Dr. Shamaila Zaman, Dr. Sanas Mohammad, Dr. Jeannine F Todd, Prof. Karim Meeran
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Biography:
SpR Endocrinology and Diabetes, Imperial College Healthcare NHS Trust

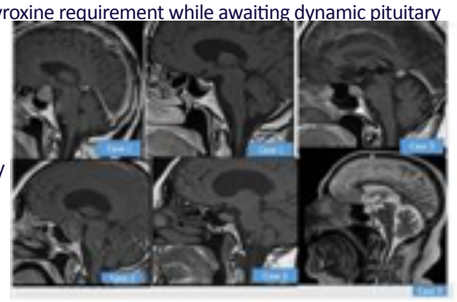
Abstract

Pituitary apoplexy caused by pure infarction is rare. Here we present three cases of pituitary macroadenoma infarction following hypotension.

1. A 84year old gentleman had a 2 X 2 cm pituitary macroadenoma causing chiasmal compression and a bitemporal hemianopia. He had a prolactin of 1095mU/l and panhypopituitarism and was on prednisolone, thyroxine and testosterone replacement. Before surgical intervention of the macroadenoma, he sustained a fall and broke his dominant side shoulder, which required internal fixation but unfortunately was complicated by an infected prosthesis. He required six further operative interventions on his shoulder under general anaesthesia and following a four hours procedure, reported headache, feeling unwell and a worsening of vision, which resolved spontaneously. His prolactin which had been 1095 fell to 48mU/l and a three months interval MRI showed a reduction in size of the pituitary macroadenoma with no chiasmal compromise. Pituitary MRI two years later shows further marked reduction in the size of pituitary gland with only a slender amount of tissue in the sella. He remains on prednisolone and thyroxine replacement and his prolactin remains low.

2. A 68-year old man underwent elective coronary artery bypass grafting (CABG). Post operatively he complained of severe headache, blurred vision and had a left sided third nerve palsy. CT head and MRI pituitary confirmed a 3.5cm mixed solid, cystic pituitary macrodenoma with significant suprasellar component stretching the optic chiasm. Pituitary profile one day following CABG showed TSH 0.89 (NR 0.3-4.2 mU/L), T3 2.7 (2.5-5.7pmol/L), T4 8.1(NR 9.0-23.0 pmol/L), ACTH 10.5 ng/L, 3 pm cortisol 125 , LH 1.3 (2.0-12.0 u/L), FSH 2.2 (1.7-8.0 u/L), GH 0.17 ng/L, prolactin <13 mU/L. He was started on prednisolone replacement. Because of his recent CABG, timing of neurological surgical intervention was considered carefully. On review in neurosurgery outpatients, he reported spontaneous improvement in his vision. Three weeks later, pituitary MRI showed a marked reduction in the size of pituitary adenoma with no compromise of optic chiasm. He remains on prednisolone and thyroxine replacement and his prolactin remains low.

3. A 70-year old man with known severe symptomatic aortic stenosis developed symptoms of a TIA. CT head identified a 1.8 cm pituitary macroadenoma. MRI pituitary showed no chiasmal compromise. Baseline pituitary function showed no deficiency. His cardiac and neurological symptoms with aortic stenosis necessitated urgent valve replacement. He was on cardiopulmonary bypass and had significant hypotensive episodes during surgery. His repeat MRI showed complete involution of the macroadenoma. He remains on hydrocortisone and has new thyroxine requirement while awaiting dynamic pituitary function testing.



Conclusion:
Sheehan's syndrome occurs when pituitary infarction occurs due to systemic hypotension in patients with an enlarged pituitary at the end of pregnancy. Our patients demonstrate that the same pathophysiology can cause hypopituitarism in males who have a pituitary adenoma rather than pituitary hyperplasia of pregnancy. The unusual pituitary portal circulation in setting of hypotension makes the pituitary critically ischaemic without haemorrhage when it is enlarged.

11.) Peculiar pathology of a sellar lesion and management dilemmas

Authors

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Biography:
Dr Fountas is a Post-CCT Fellow at the Department of Endocrinology, Queen Elizabeth Hospital, University Hospitals Birmingham NHS Foundation Trust and a Clinical Research Fellow at the Institute of Metabolism and Systems Research, College of Medical and Dental Sciences, University of Birmingham, Birmingham, UK.

Abstract

A 68-year-old man presented in 10/2017 with sudden headache, vomiting, left III nerve palsy, bilateral hemianopia and decreased left visual acuity. CT revealed a sellar mass (32 x 13 mm) with an area of hyperdensity suggestive of haemorrhage in a pituitary tumour. Hormonal investigations confirmed anterior hypopituitarism. Urgent transsphenoidal surgery was offered. Pathology showed an apoplectic gonadotropinoma (LH/FSH expression) and a tissue fragment separate from the adenoma representing a haemorrhagic tumour with biphasic appearances; a component of atypical cuboidal and columnar cells merging into a proliferation of spindle cells. Cuboidal and columnar cells were positive for synaptophysin and occasional cells expressed FSH beta-SU and LH beta-SU, while spindle cells were negative for these markers. Ki-67 was negligible in the adenoma and positive in up to 70% of spindle cells. The aggressive epithelial tumour was of unknown origin and was considered to be a metastatic deposit in the adenoma. Post-operatively, there was residual intrasellar tumour. Extensive investigations for primary malignancy were negative. In March-April 2018, radiotherapy was offered to the fossa (54 Gy, 30 fractions) aiming to maximise local tumour control. The patient had close clinical and imaging follow-up and in his most recent review (11/2019), there is no evidence of growth of the sellar lesion or of any primary malignancy.

In this challenging case, there is unusual and unexpected pathology posing management dilemmas. Pituitary metastasis can be the initial presentation of an unknown primary tumour in up to 20-30% of the patients; in about 3%, the primary tumour remains undetected despite extensive investigations. Exceptionally, a pituitary adenoma (functioning or not) may be the site of metastatic spread and very rarely present with pituitary apoplexy necessitating surgery. Metastases to pituitary adenoma are mainly found in patients with disseminated malignant disease and their underlying pathogenetic mechanism is not clear. Management of pituitary metastases is individualized, depending on stage of malignant disease and prognosis of the patients, with radiotherapy being the primary option.

12.) Pituitary macroadenoma and a colloid cyst of the third ventricle: dilemmas and options

Authors

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Biography:
Bessie Kwok is currently a Junior Clinical Fellow in Endocrinology at St Bartholomew's Hospital. She underwent her undergraduate training at University College London Medical School before completing her foundation training in Barts Health NHS Trust.

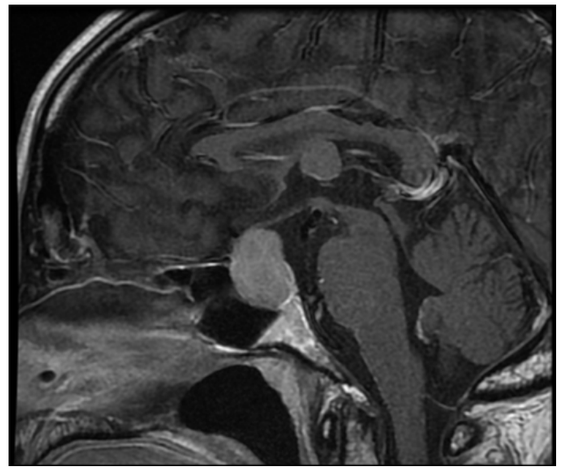
Abstract

A 73-year-old male was admitted with a 3-day history of severe lethargy, loss of appetite and generalised weakness. He was found to be profoundly hyponatraemia (Na+ 114 mmol/L). Further investigations revealed hypocortisolaemia (9am cortisol 150nmol/L [185 – 624]), mild prolactinaemia (516 mU/L [<278mU/L]), hypogonadotrophic hypogonadism (FSH 3.9 U/L [1.3-19.3], LH 4.6 U/L [1.2 – 8.6], testosterone 4.6 nmol/L [6.1 – 27.1]) and secondary hypothyroidism (fT4 9.1 pmol/L [10.5 – 24.5], TSH 1.82 mU/L [0.27 – 4.2]).

Subsequent neuroimaging demonstrated a solid heterogeneously enhancing 22 x 21 x 15mm pituitary macroadenoma extending into the suprasellar region and displacing the optic chiasm. An incidental finding was also made of an 11mm lesion in the anterosuperior aspect of the third ventricle with features in keeping with a colloid cyst.

He was commenced on hydrocortisone and the hyponatraemia resolved prior to discharge. Thyroxine was also later started. Past medical history of note included prostate cancer diagnosed four years previously, which was under surveillance but had not required treatment to date. Thus, testosterone replacement was withheld pending urological advice. He denied any acute decline in his vision but on formal testing visual acuity was 6/9 in both eyes with some bitemporal visual field loss.

Colloid cysts are histologically benign lesions that may result in obstructive hydrocephalus and sudden death. The indication for surgical intervention in asymptomatic patients with incidental lesions is not clearly defined although a cut-off of >7mm is often used in risk stratification scores. This case of a male patient with a clinically significant pituitary macroadenoma associated with panhypopituitarism and a coexistent diagnosis of a colloid cyst of the third ventricle, both of which require neurosurgical intervention, presents a difficult management dilemma. Surgical intervention on both lesions has the potential to influence the outcome in the other. The opinion of the neurosurgical faculty on the order, timing and technique/approach to this dual pathology would be greatly valued.



13.) Management of suprasellar pituitary macroadenomas with temporal extension

Authors

Mr Jarnail Bal[1], Dr James Ahlquist1, Mr Alireza Shoakazemi[1], Mr Jonathan Pollock[1]
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Biography:
Current RCS Skull Base fellow based between Queens Hospital and The Royal London Hospital

Abstract

Introduction

We present three patients with large macroadenomas with symptomatic suprasellar extension into the medial temporal region and discuss the management of this unusual growth pattern.

Cases

1. A 47 male presented with visual deterioration from a large macroadenoma with suprasellar extension and a unilateral temporal component. He underwent transsphenoidal (TS) debulking of a pituitary adenoma (Silent gonadotroph. Ki-67 3%) in 2015, then subsequent revision TS resection and a right sided craniotomy. Subtotal removal was achieved. He developed a symptomatic left sided internal capsular infarct following the transcranial debulking. He has subsequently been treated with Cabergoline and conventional radiotherapy.

2. A 62 female with episodes of complex partial seizures secondary to an extensive cystic pituitary adenoma with a large medial cystic temporal component. The patient underwent a TS debulking in 2013 and placement of an Ommaya reservoir for recurrent drainage of the temporal cyst. Histology demonstrated a corticotroph adenoma (silent). Subsequent imaging has shown a gradual increase in the solid component of the cyst. She has developed a new a spastic upper limb monoparesis. Her partial seizures are under control with medication. Following MDT discussion conventional radiotherapy is planned for the residual adenoma.

3. 65 Male who presented with a left sided hemiparesis secondary to an extensive suprasellar extension of pituitary adenoma. He underwent a burr hole aspiration of the temporal cystic component of the adenoma and an and TS debulking of the pituitary adenoma. The histology showed focal to expression LH, FSH and ACTH.

Conclusions

These patients have all had significant morbidity associated with a temporal suprasellar component of a pituitary adenoma and have presented significant management challenges. The findings in these patients are discussed.

POSTER ABSTRACTS

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1	Dopamine Agonists - a Novel Cure for Autoimmune Diabetes	*Dr Lorcan Cooper, Dr Anelia Sirkova, Sister Anna Hawkins, Dr Frederick Nkonge, Sister Funmi Akinalade, Dr Nemanja Stojanovic*
2	A whispering pituitary adenoma	*Dr Ankur Poddar, Dr Marie Wallner, Dr P John, Mr. Andrew Martin, Dr Gul Bano*
3	A case of testosterone replacement causing increase in Prolactin by Macroprolactinoma	*Dr Hessa Boharoon, Dr Oluwagbemiga Idowu, Dr Mark Sutton-Smith, Dr Zeeshan Yasin, Dr Jeannie Todd*
4	An Unusual case of Hypopituitarism	*Dr Vanitha Karunakaran, Dr Philip Rich, Mr Martin A J, Dr Gul Bano*
5	Giant pituitary macroadenoma with brainstem compression and the use of cabergoline	*Dr Bonnie Grant, Dr Aneliya Sirkova, Dr James Ahlquist, Mr Jonathan Pollock, Mr Alireza Shoakazemi, Dr. Frederick Nkonge, Dr Nemanja Stojanovic*
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7	The Role of Cross-Continental Telemedicine in the Management of Non-functioning Pituitary Adenoma: a Model for Future Global Health Collaboration and Partnership Schemes	*Miss Davina Jugnarain, Dr Mahamud Bashir, Dr Shirin Patel, Dr Dauda Balami, Dr Gideon Mlawa*
8	A macroprolactinoma in a young male presenting with acute neuropsychosis initially treated with aripiprazole and later with cabergoline; challenges in management and pharmacotherapeutic strategy	*Dr Ahmad Moolla, Dr David Hope, Dr Neil Hill, Prof Karim Meeran*
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10	A case Diabetes Insipidus Post Pituitary Apoplexy Surgery and it's outcomes	*Dr Shirine Patel, Dr Gideon Mlawa*
11	First Presentation of Hereditary Haemochromatosis in a Patient with Secondary Hypogonadism	*Dr Katharine Lazarus, Dr Mushtaq Rahman, Dr Shahir Hamdulay, Dr Charalampia Kyriakou, Dr Keith Steer*
12	Come out, come out, wherever you are…	*Dr Bijal Patel, Dr Sriranganath Akavarapu, Dr Anastasia Gontsarova, Dr Emma Hatfield, Dr Clara Limback-Stanic, Dr Niamh Martin, Mr Nigel Mendoza, Prof Karim Meeran*
13	Polycystic ovary syndrome – microprolactinoma – or both?	*Dr James MacFarlane, Dr Waiel Bashari, Dr Olympia Koulouri, Dr Russell Senanayake, Laura Serban, Prof Mark Gurnell*
14	Cushing's Disease: A difficult road to travel	*Dr Shamaila Zaman, Dr Ambreen Qayum, Dr Berit Inkster, Dr Mark Gurnell, Dr Florian Wernig, Dr Niamh Martin, Dr Jeannie F Todd, Mr Ramesh Nair, Prof Karim Meeran*
15	A Pituitary Mass - When there is more than meets the eye!	*Dr Desiree Seguna*

Notes:

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