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Society for Endocrinology

Twenty-first Clinicopathological Conference on Pituitary Disease

Monday 4th February 2019

Royal College of Obstetricians and Gynaecologists, London, NW1 4RG

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They have had no input into the meeting organisation, the agenda or the selection of speakers, unless otherwise stated.

AGENDA

08:30 **Registration**

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

09:30 **Key Note Lecture: The pituitary in 2018**
Dr Niki Karavitaki, Senior Clinical Lecturer in Endocrinology, University Hospitals Birmingham

Forum 1 – Case Presentations: Acromegaly
Chairs: Mr Neil Dorward, Dr Stephanie Baldeweg

10:00 Is it only the hands? A case of low grade acromegaly
Dr Ziad Hussein, University College London Hospitals NHS Foundation Trust

10:15 The relevance of histological subtypes of growth hormone-secreting tumors in predicting response to treatment
Dr Isuri Kurera, SPR, St Georges University Hospitals NHS Foundation Trust

10:30 Therapy dilemmas in acromegaly due to a giant adenoma in a young woman
Dr Ali Hameed, ST6, Royal Victoria Hospital, Belfast

10:45 A massive invasive fibrous pituitary macroadenoma with biochemical evidence of growth hormone excess; what is the optimal treatment strategy?
Dr Ultan Healy, Post CCT Fellow in Endocrinology, Oxford University Hospitals Trust

11:00 **Exhibition, Refreshments and Poster Display**

11:30 **Clinical and pathological aspects of silent adenomas**
Prof Marta Korbonits, Consultant Endocrinologist, St Bartholomew's Hospital

Forum 2 – Case Presentations: Surgical corner
Chairs: Miss Joan Grieve, Dr Mark Vanderpump

12:00 Expanding the surgeons toolbox: development and evaluation of devices for the endoscopic transsphenoidal approach
Mr Hani Marcus, Pituitary Fellow, National Hospital for Neurology and Neurosurgery, London

12:15 To biopsy or not? A rare cause of a hypothalamic tumour
Dr Jodie Sabin, SPR Endocrinology, North Bristol NHS Trust

12:30 Should patients be offered surgical decompression for third nerve palsy following pituitary apoplexy? 3 illustrative cases
Mr Amr Mohamed, Pituitary Surgery Senior Clinical Fellow, Oxford University Hospitals NHS Foundation Trust

12:45 Surgical challenges associated with a rare case of pituicytoma
Dr Mikhail Harty, Clinical Fellow Neurosurgery, Southmead Hospital North Bristol NHS Trust

13:00 **Exhibition, Lunch and Poster Display**

13:45 **The Pituitary Foundation**
Mrs Menai Owen-Jones, CEO, The Pituitary Foundation

14:00 **Quality of life in pituitary disease**
Dr Nienke Biermasz, Consultant Endocrinologist, Leiden University Medical Center, The Netherlands

Forum 3 – Case Presentations: Cushing's syndrome/disease
Chairs: Dr Helen Simpson, Dr James Ahlquist

14:40 Nelson's syndrome: diagnostic and management challenges
Dr Athanasios Fountas, Clinical Research Fellow, Queen Elizabeth Hospital, University Hospitals of Birmingham NHS Foundation Trust

14:55 A case of overt Cushing's disease with resolution in a microadenoma after subclinical apoplexy
Dr Syed Bitat, ST5, Royal Free Hospital, London

15:10 Two cases of persistently unidentifiable (occult) sources of ectopic ACTH secretion post-adrenalectomy
Dr James MacFarlane, ACF ST3 Diabetes and Endocrinology, Norfolk And Norwich University Hospital

15:25 Immediate postoperative low cortisol in Cushing's disease doesn't always predict long term remission: Two cases of late recurrence of Cushing's disease many years after initial successful TSS
Dr Ambreen Qayum, Imperial College NHS Healthcare NHS Trust, London

15:40 **Exhibition, Refreshments and Poster Display**

16:00 **GIRFT in pituitary disease (the surgical perspective with a touch of endocrine)**
Mr Nick Phillips, Consultant Neurosurgeon, Leeds Teaching Hospitals

Forum 4 – Case Presentations: Pituitary all sorts
Chairs: Prof Pierre-Marc Bouloux, Dr Umasuthan Srirangalingam

16:30 Central diabetes insipidus as the inaugural manifestation of langerhans cell histiocytosis – a rare condition with an often missed presentation
Dr Win Htun Oo, Trust Registrar in General Medicine, Ipswich Hospital, East Suffolk & North Essex NHS Foundation Trust

16:45 Hypophysitis - an immune-related adverse event
Dr Nadeem Abbas, Consultant Endocrinologist & Consultant Physician, Hywel Dda University Health Board, Bronglais General Hospital

17:00 Tri-phasic response post extended endonasal transsphenoidal resection for suprasellar craniopharyngioma
Dr Raya Almazrouei, Endocrine SpR, Imperial College Healthcare NHS Trust, London

17:15 **Poster and case presentation prizes**
Dr Stephanie Baldeweg, Consultant Endocrinologist, University College London Hospitals

17:20 **Close**

FACULTY

PANEL & ORGANISERS

FACULTY

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

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

Dr Nienke Biermasz, Consultant Endocrinologist, Leiden University Medical Center, The Netherlands

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

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

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

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

Prof Marta Korbonits, Consultant Endocrinologist, St Bartholomew's Hospital

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Mr Nick Phillips, Consultant Neurosurgeon, Leeds Teaching Hospitals

Dr Helen Simpson, Consultant Endocrinologist, University College London Hospitals

Dr Mark Vanderpump, Consultant Endocrinologist, University College London Hospitals

ORGANISING COMMITTEE

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Consultant Endocrinologist,
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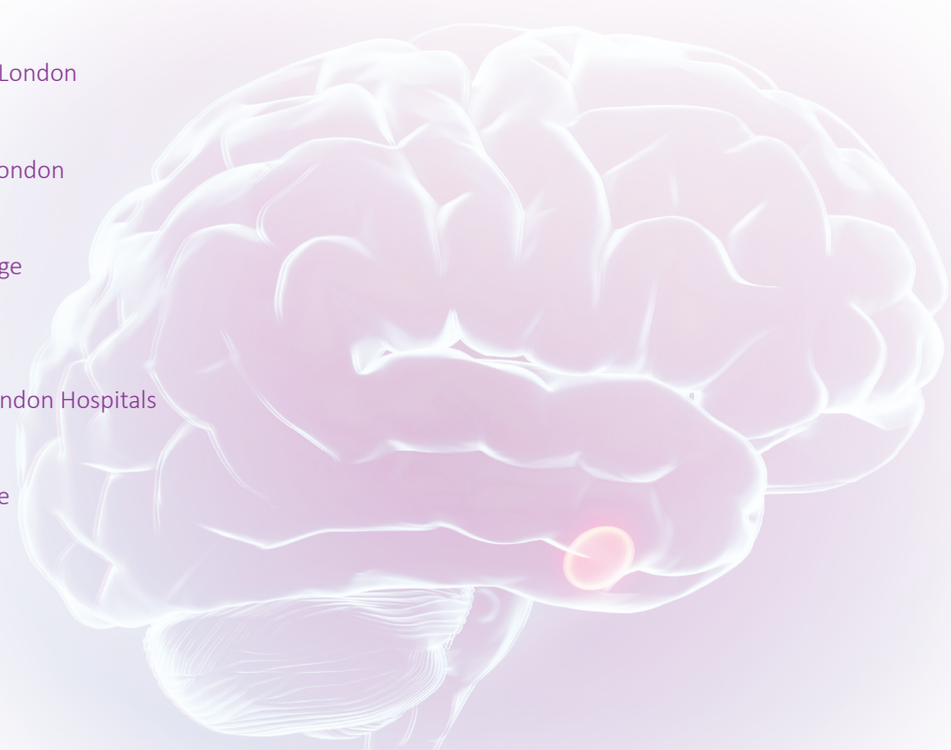

1.) Is it only the hands? A case of low grade acromegaly

AUTHORS:

Dr Ziad Hussein, Dr Helen Simpson, University College London Hospitals NHS Foundation Trust

Abstract

Acromegaly is a rare endocrine disease caused by over production of growth hormone. Skeletal complications are very common and mostly irreversible even after
biochemical remission.
A 53 year old lady was seen as a new patient in clinic recently She was originally diagnosed with acromegaly aged 22. She had two trans-sphenoidal surgeries performed in1987 and1996 also underwent several maxillofacial reconstructive surgeries in the past to correct her enlarged jaw and macroglossia, complicated by infection and poor healing. The patient was not seen in endocrinology clinics for more than 10 years until representing in 2018. Available letters suggest there were concerns about her not being in full remission. Interestingly she represented with enlarging fingers more marked distally mainly on the left side over several weeks. It was evident that she has symptoms of hypopituitarism including lack of energy and amenorrhoea for 22 years (She is a mother of two) with active skeletal changes including large hands with arthritic changes and onycholysis due to grossly enlarged finger pulps.
Initial biochemical tests suggested secondary hypoadrenalism (afternoon cortisol 33 nmol/L), Growth hormone 3ug/L, normal IGF1 17.7 nmol/L (NR 5.5-32.0), secondary hypothyroidism (TSH 1.48 mIU/L, FT4 11.5 pmol/L (NR 12-22) and hypogonadotropic hypogonadism (FSH 1.7IU/L, LH 0.6IU/L, Oestradiol <44pmol/L). Further investigations proved active acromegaly and adrenal insufficiency by OGTT and SST on 21/09/2018 respectively as below:

Time	Lab glucose mmol/L	Growth hormone µg/L
zero.	4.1	2
30 mins	5.5	1.7
60 mins	5.2	1.3
90 mins	4.0	1.5
120 mins	3.7	1.9

Time point	Cortisol nmol/L
Zero	102
30 min	141
60 min	154

She was started on hydrocortisone and thyroxine and feels much better. Plain radiograph of her hands demonstrated classical manifestations of acromegaly but interestingly the bone changes are far more extensive in the left distal phalanxes:

1. Large hands with soft tissue thickening.
2. Hypertrophy of the muscular attachments of the proximal phalanges.
3.Prominent metacarpal heads with widening of metacarpophalangeal joints.
4. Spade phalanx sign or tufting of terminal phalanges.

Conclusion:
Monitoring of acromegaly requires periodic evaluation of GH and IGF-1. Although the vast majority of patients show concordant results, discrepancy of the levels has been reported and there is increasing awareness of GH/IGHF-1 discordance. This case demonstrates normal age adjusted IGF-1 and her low grade active disease was confirmed by failure of growth hormone suppression during OGTT.
It is not very obvious why this patient developed disproportionate overgrowth of hands and fingers over short period of time. GH stimulates osteoblast proliferation and activity, promoting bone formation in two ways: via direct interaction with GHRs (activates (JAK)–(STAT) signalling pathway) and via an induction of endocrine and autocrine/ paracrine IGF-I. Our patient suggest here that GH rather than IGF-I was responsible for the marked skeletal changes. We are currently awaiting imaging to then decide the best long term management. We think she is fearful of treatment options and one priority here will be to explore her concerns and decide a strategy that will suit her best.

2.) The relevance of histological subtypes of Growth hormone-secreting tumors in predicting response to treatment

AUTHORS:

Dr Syed Basharat Andrabi, P Johns, P Rich, L Welsh, Mr Andrew Martin, Dr Gul Bano
St Georges University Hospitals NHS Foundation Trust, United Kingdom

Abstract

Introduction
Current treatment for the growth hormone-secreting tumors includes surgery, medical therapy with somatostatin analogs (SSA), dopamine agonists, or a GH receptor antagonist and radiotherapy. The treatment results in disease remission in approximately half of the patients. Histological subtypes, particularly densely (DG) versus sparsely (SG) granulated adenomas have been used as predictors of GH tumor response to different therapies. We describe a patient of Acromegaly where the clinical course and histology may help the management plan.

Case History
Twenty-nine years old male was diagnosed with Acromegaly at the age of 25. He had surgery that resulted in normalization of IGF-1 for five months postoperatively. The histology showed a sparsely granulated growth hormone cell pituitary adenoma. The subsequent increase in IGF-1 was initially treated with dopamine agonist but did not result in lowering of IGF-1. He was then treated with somatostatin analog Lanreotide, but this resulted in severe side effects without any improvement in symptoms or decline in IGF-1. Repeat MRI scanning showed residual tumor. 11C-methionine PET, revealed a functional residual tumor. He had a second surgery in October 2018.

Discussion
Histologically GH tumors can be densely granulated, showing keratins diffusely distributed throughout the cytoplasm, and a perinuclear distribution pattern. Sparsely granulated adenomas have characteristic dot-like keratin immunoreactivity, representing fibrous bodies.
SG tumors have been reported to occur in younger patients, tend to be larger but show no differences in tumor-invasion characteristics and may have lower GH/IGF-1 levels than DG tumor. DG tumors are reported to have a higher rate of remission in response to surgery as well as to medical therapy with SSAs as compared to SG. SG tumors can be associated with disease persistence. There is a significant difference in the rate of remission between sparse and dense tumor subtypes.

Conclusion
Histological GH tumor subtyping identifies different clinical phenotypes and biologic behaviors, providing prognostic significance for surgical success and response to medical therapies.

3.) Therapy dilemmas in acromegaly due to a giant adenoma in a young woman

AUTHORS:

Dr Ali Hameed, Dr Hamish Courtney, Royal Victoria Hospital, Belfast, UK

Abstract

A 45 year old woman presented early 2018 with unilateral hearing deficit and was noted to be acromegalic. This was confirmed biochemically (IGF1 147 nmol/l N 8.1-26.8). Her vision was normal. The remainder of her pituitary function revealed secondary hypothyroidism and hypogonadism. She demonstrated typical other features of longstanding acromegaly including severe osteoarthritis and sleep apnoea.
Imaging revealed a 5x6x6.5cm pituitary tumor with extension in all directions.
She was commenced on sandostatin LAR 20 mg monthly in April 2018 prior to other treatment modalities. Her IGF1 reduced to 39 within 3 months of treatment with some corresponding radiological improvement. There was little clinical change in terms of her headaches or sweating. However a CSF leak has also recently developed.
The opinion of the panel on future treatment approaches, surgically, medically and using radiotherapy to managing this large tumor and GH excess would be welcome.

4.) A massive invasive fibrous pituitary macroadenoma with biochemical evidence of growth hormone excess; what is the optimal treatment strategy?

AUTHORS:

Dr Ultan Healy, Dr Christine May, Dr Olaf Ansorge, Mr Simon Cudlip, Dr Bahram Jafar-Mohammadi, Dr Aparna Pal
Oxford University Hospitals Trust, Oxford, United Kingdom

Abstract

A 68 year old woman has attended the pituitary clinic at the Oxford University Hospitals Trust since March 2017. She was initially referred from the neurology service after presenting with a 4 year history of symptoms consistent with partial seizures. MRI brain revealed a large (62 x 52 x 44 mm) pituitary macroadenoma with suprasellar extension and expansion into the right temporal fossa. The tumour completely encased the right internal carotid artery. The hippocampus was compressed, presumably resulting in the presenting seizures. The optic chiasm was displaced superiorly. Visual fields were intact. There was no evidence of an endocrinopathy on examination or on directed history.

Initial biochemistry revealed elevated IGF1 at 59.2 nmol/L (4.5 – 22.5 nmol/L). Random growth hormone was 9.2mcg/L. Prolactin was normal at 361 mU/L (110 – 560 mU/L). Thyroid and Adrenal Axes were intact. Gonadotrophins were clearly deficient. Hba1c was in the pre-diabetes range at 46 mmol/mol.

Given that there were symptoms related to mass effect, the optic chiasm was threatened, and there was biochemical evidence of growth hormone excess; debulking surgery was attempted via transsphenoidal approach in May 2017. Intraoperatively the tumour was found to be extremely fibrous and difficult to resect; a small volume (16x6x4mm) of tumour was successfully removed. Histology was of an invasive pituitary adenoma with minimal growth hormone and prolactin expression (<1%). Post-operative MRI demonstrated tumour stability (63 x 52 x 41 mm).

Considering the presence of growth hormone staining cells and grossly elevated IGF1, Lanretotide Autogel 120mg was subsequently commenced and administered at an interval of 28 days. A growth hormone day curve at this point demonstrated a mean growth hormone of 2.15 mcg/L and IGF1 remained stable (51.4 nmol/L).

Follow-up MRI in October 2017 demonstrated a reduction in tumour size (58 x 45 x 35 mm) compared to initial postoperative imaging and further imaging has demonstrated tumour stability through to October 2018.

However, despite increasing the frequency of Lanreotide to 21 days and adding Cabergoline, titrated to 3mg per week; IGF1 remained grossly elevated at 60.4 nmol/L and a repeat growth hormone day curve demonstrated a mean value of 3.46 mcg/L. No clinical features of acromegaly have developed.

In summary, we present the case of a 68 year old woman with an invasive pituitary tumour which has proven difficult to resect. There is biochemical evidence of growth hormone excess, but no overt features of acromegaly. Somatostatin analogue therapy resulted in an initial reduction in tumour size, but growth hormone and IGF1 remain elevated. Optimal management of this case is unclear; surgical resection via craniotomy followed by radiotherapy is being considered. Long term medical therapy coupled with a more conservative watch-and-wait approach may also be reasonable.

5.) Expanding the surgeons toolbox: development and evaluation of devices for the endoscopic transsphenoidal approach

AUTHORS:

Mr Hani Marcus[1], Prof Danail Stoyanov[2], Mr Neil Dorward[1]
[1]National Hospital For Neurology And Neurosurgery, London, United Kingdom, [2]University College London, London, United Kingdom

Abstract

Background:
The purely endoscopic endonasal transsphenoidal approach represents among the best examples of "keyhole" surgery in the brain and may be associated with similar rates of tumour excision and remission rates, but with fewer operative complications and shorter hospital stay, than the standard microsurgical approach. Over the last decade, a group at the University of Pittsburgh have systematically expanded the endoscopic endonasal approach to access other ventral skull base lesions, which are challenging to treat with microsurgical approaches. Although the expanded endoscopic endonasal approach offers theoretical over standard microsurgical approaches to the ventral skull base, it is technically challenging to perform, and has not been widely embraced by neurosurgeons.

In order to better understand the technical barriers to the expanded endoscopic endonasal approach we recently surveyed members of the Society of British Neurological Surgeons. In all, approximately half of the respondents described difficulties with intra-operative visualisation, and in particular lack of depth perception, and the majority of respondents struggled when handling tissue using existing instruments, with one surgeon saying it was akin to "operating using chopsticks".

Aims and objectives:
In our research, we aimed to address the barriers to the expanded endoscopic endonasal approach by working with engineers to adapt existing devices, and develop new devices, tailored to the particular requirements of this approach. The objectives of the research were threefold: first, to combine an augmented reality display and a specially designed 3D endoscope to enhance the surgeon's vision; second, to introduce robotic instruments that allow for wrist-like dexterity, and limit the forces exerted, to enhance the surgeon's touch; and third, to use artificial intelligence developed using videos of existing experts to enhance the surgeon's judgement.

Methods and Results:
The development and evaluation of these individual devices for the expanded endoscopic endonasal approach was done in an ordered and logical manner, building on previous work, and in keeping with the IDEAL (Idea, Development, Exploration, Assessment and Long-term follow-up) framework for surgical devices: first, an augmented reality and 3D endoscope was developed and evaluated in a preclinical randomised controlled trial; second, a series of studies were performed to determine the working specifications for robotic instruments and a feasibility study performed; and third, artificial intelligence software was trained on a series of expert videos and to automatically detect the steps of endoscopic transsphenoidal surgery in real-time, with a feasibility study planned for the near future.

Discussion:
The intention of future work will be to replace the bulky multipurpose robots under development by other research groups today, with a platform consisting of a range of "smart" handheld instruments, all encompassing robotic qualities, and tailored to the endoscopic endonasal approach. We believe these handheld robotic instruments will be more acceptable to patients, surgeons, and healthcare institutions.

6.) To biopsy or not? A rare cause of a hypothalamic tumour

AUTHORS:

Dr Jodie Sabin, Dr Fong Chau
North Bristol NHS Trust, Westbury-on-Trym, United Kingdom

Abstract

We present a case of a 23 year old lady who was diagnosed with diabetes insipidus and hypopituitarism after presenting acutely to hospital with polydipsia. An MRI scan revealed a hypothalamic tumour involving the optic chiasm, felt radiologically to be an astrocytoma.

She had normal visual field testing. Surgical resection was not an option due to involvement of the optic pathways; biopsy also carried a significant risk to her vision. After lengthy discussions, she decided to opt for surveillance.

Over the next 4 months her symptoms rapidly progressed. She developed hyperphagia and gained 30kg of weight. She had increased somnolence, resulting in a road traffic accident after falling asleep driving. She also developed type 2 diabetes. Clinical psychology support was required due to the impact on her life and she also had several inpatient admissions due to hypernatraemia and difficulty managing diabetes insipidus.

A repeat MRI scan 4 months later showed increased oedema and bulk of the mass, so she proceeded to have a biopsy taken. She remained an inpatient following this, with worsening confusion and short term memory loss.

Biopsy results revealed a diagnosis of Langerhans Cell Histiocytosis (LCH). Further investigations (including PET-CT) found no other site of disease. There has been reduction in the size of hypothalamic mass following chemotherapy treatment but unfortunately no clinical improvement as yet.

LCH is a rare disease and less than 30% of cases occur in adults. The hypothalamic-pituitary region is affected in up to 50% of patients with LCH but it is rare for LCH to be isolated to hypothalamic-pituitary region only.

This case highlights the need to consider this rare diagnosis in patients presenting with diabetes insipidus, anterior pituitary dysfunction and hypothalamic-pituitary lesions. It also highlights the importance of MDT involvement and the difficulties around the decision and timing of biopsy.

7.) Should patients be offered surgical decompression for third nerve palsy following pituitary apoplexy? 3 illustrative cases

AUTHORS:

Mr Amr Mohamed, Mr Simon Cudlip
Neurosurgery Department, John Radcliffe Hospital, Oxford University Hospitals NHS Foundation Trust, Oxford, United Kingdom

Abstract

Introduction: Third nerve palsy is a recognised consequence of pituitary apoplexy, occurring in up to 25% of cases. Current guidelines suggest conservative management of ocular palsy without visual field defects.

Patients: In this presentation we will present 3 patients presenting with third nerve palsy but no visual field defects, associated with pituitary apoplexy, Together with the contrasting management strategies used as guided by patient specific factors.

Methods: Current guidelines suggest that isolated third nerve palsy associated with pituitary apoplexy should be managed conservatively. Two patients were considered for urgent surgery and underwent transsphenoidal decompression of pituitary apoplexy, a third was managed conservatively due to significant co-morbidity.

Results: The two patients undergoing urgent surgical resection of the pituitary apoplexy had an immediate recovery of the third nerve palsy within 24 hrs of surgery. The patient who was treated conservatively has improved gradually over the 6 weeks following surgery,

Conclusion: These cases illustrate the potential for accelerated recovery of third nerve palsy after pituitary apoplexy with surgical treatment. Is it appropriate to discuss the option of surgery with patients when appropriate, bearing in mind the current guidelines?

8.) Surgical challenges associated with a rare case of Pituicytoma

AUTHORS:

Dr. Mikhail Harty[1], Mr. Richard Nelson[1], Mr. Adam Williams[1], Dr. Kathreena Kurian[1], Dr. Kate Allen[2]
[1]Southmead Hospital North Bristol NHS Trust, Bristol, United Kingdom, [2]Royal United Hospitals Bath NHS Foundation Trust, Bath, United Kingdom

Abstract

Introduction/Background:
Pituicytomas are rare, solid, spindle cell tumours of low grade (WHO Grade I) that originate from supportive glial cells in the neurohypophysis (1). Less than 100 cases of pituicytoma have been documented in the literature (2–5). Most cases of pituicytoma are only partially resected and the diagnosis is usually not expected in the treatment of pituitary lesions. In their review of cases, Feng et al. (5) found that gross total resection occurred in 42% of transsphenoidal surgeries, 33% of craniotomies, and 100% of extended transsphenoidal surgeries, but post-operative morbidity was high with craniotomies. The vascularity of the tumour often precludes complete resection warranting radiotherapy or a second surgery to achieve cure.

Case:
We present the case of a 31-year-old man who presented as a consequence of decreased libido and absent ejaculate. His clinical examination was significant for bitemporal superior quadrantanopia, normal visual acuity, and normal fundoscopic examination. His initial biochemistry revealed low testosterone (undetectable and <0.4 nmol/L) and inappropriately low gonadotrophins (LH 1.0 IU/L, FSH 0.3 IU/L). He was euthyroid and had normal cortisol (9 am, 305 nmol/L) but slightly elevated prolactin (631 mIU/L) consistent with a stalk effect. MRI revealed a pituitary macroadenoma with suprasellar extension, displacing the optic chiasm. He underwent attempted endoscopic endonasal transsphenoidal resection of the lesion which was firm, vascular and adherent to surrounding structures, resulting in limited debulking only.

Histopathology revealed a tumour consisting of oval cells with spindle syncytial cytoplasm. Immunohistochemistry showed strong positivity for TTF-1 and background positivity for GFAP. Immunohistochemistry was negative for S100, vimentin, SSR2, EMA, synaptophysin, chromogranin, and CAM5.2. It was also negative for pituitary hormones LH, FSH, TSH, GH, ACTH.

He developed panhypopituitarism postoperatively and was started on replacement cortisol and L-thyroxine, though he did not have diabetes insipidus. His visual fields returned to normal. However, an MRI at 3 months' follow-up, demonstrated that the residual tumour volume remained a threat to his vision. A perioperative note was made of feeding vessels from multiple sources including the optic chiasm. He underwent craniotomy and resection of the tumour which was freed from the optic nerves, chiasm, and the internal carotid arteries. The pituitary stalk was not salvageable. Complete resection of the tumour was achieved, but with a resulting bitemporal hemianopia and diabetes insipidus that was treated with oral desmopressin.

Discussion
This case highlights the unexpected diagnosis of this rare tumour and the particular surgical difficulties. Despite a benign pathological classification, these tumours are adherent and have significant vascularity that poses significant challenges to the surgeon and the risk of postoperative morbidity for the patient can be high.

9.) Nelson's syndrome: diagnostic and management challenges

AUTHORS:

Dr Athanasios Fountas[1,2], Dr Santhosh Nagaraju[1], Dr Han Seng Chew[1], Dr John Ayuk[1], Dr Paul Sanghera[1], Dr Shahzada Ahmed[1], Dr Niki Karavitaki[1,2]

[1]University Hospitals of Birmingham NHS Foundation Trust, Birmingham, United Kingdom
[2]Birmingham Health Partners, Birmingham, United Kingdom

Abstract

A 12-year-old Caucasian boy presented with a 6-month history of excessive weight gain, headaches, tiredness. He had signs of hypercortisolaemia and investigations were consistent with severe ACTH-dependent Cushing's syndrome [UFC:2400 mmol/24h (<350), ACTH 174 ng/L (<46), no suppression of cortisol on low dose dexamethasone suppression test]. Although pituitary imaging revealed a left-sided microadenoma, given the severity of his clinical picture, urgent bilateral adrenalectomy was performed (1992). Eight months later (1993), he developed generalized pigmentation; increased ACTH levels and adenoma enlargement were found. Nelson's syndrome was diagnosed and was managed by transsphenoidal adenomectomy (1993) and conventional radiotherapy (45 Gy in 30 fractions, 1995). Pathology showed pituitary adenoma with abundant ACTH expression and no significant mitotic activity. Serial pituitary imaging revealed empty sella and ACTH decreased to 38.5 ng/L (morning sample, off hydrocortisone). No clinical, biochemical or imaging progression of the Nelson's was detected in the next years. In 2012, ACTH started rising again (random sample 81.4 ng/L, on hydrocortisone) and over the subsequent 18-month period, it increased by 8-fold (random sample 634.4 ng/L, on hydrocortisone). Pituitary MRI (2014) revealed a 17.8 mm lesion extending into the left cavernous sinus. Second transsphenoidal surgery was performed (May 2014), complicated by CSF leak requiring four surgical interventions. Pathology revealed ACTH-staining pituitary adenoma with Ki-67 4%. Post-operatively, ACTH decreased (262.9 ng/L, morning sample, off hydrocortisone) and imaging showed 6x7 mm residuum in the left cavernous sinus. This was further treated with stereotactic radiosurgery (Cyberknife, 21 Gy in one fraction, April 2015). In October 2015, he developed acute third cranial nerve palsy which was attributed to radiation toxicity and significantly improved over time. In December 2017, he developed pigmentation and ACTH was again increased (300.7 ng/L before morning hydrocortisone, suppressed to 266.3 ng/L two hours after hydrocortisone administration). Pituitary imaging showed stable appearances and a conservative approach with careful observation was adopted. However, seven months later (July 2018), ACTH had increased further (720.3 ng/L before morning hydrocortisone, suppressed to 623.4 ng/L two hours after hydrocortisone administration); MRI revealed growth of tumour surrounding the anterior aspect of the left internal carotid artery. Spinal MRI showed a 19 mm intradural extramedullary meningioma and no metastases. Further surgery was considered of high risk and treatment with pasireotide will be initiated.
Nelson's syndrome is a challenging condition with prevalence between 8% and 47% (influenced by diagnostic criteria, referral bias of the reporting centres and length of follow-up after adrenalectomy). The interval between adrenalectomy and diagnosis of Nelson's is 0.5-24 years and its pathogenesis remains unknown. Tumours in Nelson's can be locally aggressive and malignant transformation with distant metastases has also been reported. Notably, our patient was diagnosed with Nelson's progression 17 years after initial treatment illustrating the unpredictable behaviour of these tumours. Management options show wide variation and include observation, surgery, various modalities of radiation therapy and pharmacotherapy. Given the limited published literature, there are significant uncertainties on the diagnostic criteria, optimal management and follow-up protocols, as well as on the outcomes of these patients necessitating large scale collaborative studies.

10.) A case of overt Cushing's disease with resolution in a microadenoma after subclinical apoplexy

AUTHORS:

Dr Syed Bitat, Dr Bernard Khoo, Dr Efthimia Karra, Dr Ahmed Yousseif, Dr Dipesh C Patel
Royal Free Hospital, London, United Kingdom

Abstract

Case history: A 24 year old Spanish female was referred by GP with a three year history of classical symptoms of glucocorticoid excess including weight gain, purple striae, easy bruising and alteration in face appearance. Oligomenorrhoea and headache were also reported. She was diagnosed with Cushing's disease but did not undergo treatment due to perceived operative risks and the possibility of cyclicity. Compatible with this, when the patient was reviewed in our service, her menstrual cyclicity had normalised, the only complaint was of mild hirsutism. She was otherwise well with no significant history of alcohol nor medication use.

On examination, BP 135/83 and BMI was raised at 37 kg/m2. She exhibited multiple violaceous striae of greater than 1cm in width. No bruising, nor myopathy was evident. Visual fields to confrontation testing were within normal limits.

Investigations: FBC- Normal, Normal renal, liver function and bone profile.
24 hour urinary cortisol level was 121 and repeat was 57 (Normal 0-250 nmol/day)
Overnight Dexamethasone suppression test cortisol (ONDST) 122nmol/l, ACTH 19.9ng/L (Normal <46), Repeated ONDST showed value of 68nmol/l. Low dose dexamethasone suppression test 2+48h cortisol 15nmol/l.
LH 21 U/L, FSH 8.4 U/L, Oestradiol 176 pmol, prolactin 387 mUnit/L, Testosterone 1.5 nmol/L, TSH 0.72 mUnit/L, FT4 15.7 pmol, 9 am cortisol 227 nmol/L in August 2017 , IGF1 38.7 nmol/L (Normal 11-46 nmol/L). HbA1C 39.9 mmol/mol.
MRI of Pituitary in 2016 in Spain showed 6X7 mm microadenoma with evidence of haemorrhage. Serial scanning few months later showed pituitary microadenoma of 6 x 7 x 6 mm. A subsequent scan in the UK in July 2017 showed 5 mm microadenoma in the right half of the pituitary gland with changes consistent with haemorrhage.

Follow up: Patient weight remained stable over the year around 102kg to 104kg. Blood pressure remained within normal limits during clinic visits. Her purple striae became paler. HbA1c remained normal. She had normal bone density.
She became spontaneously pregnant soon after presenting in the UK with an initial uncomplicated pregnancy. She underwent an emergency caesarean section for preeclampsia in May 2018. There was no evidence of hypopituitarism pre or post-partum.
Repeat MRI imaging in 2018 showed changes on the right side had regressed. Persistent changes were visible compatible with small areas of haemorrhage within the pituitary gland.
Is this expected over 2 years after initial haemorrhagic changes?

Discussion: Pituitary apoplexy, a rare clinical syndrome which complicates 2%–12% of pituitary adenomas, especially nonfunctioning tumors. According to epidemiological studies, its prevalence is about 6.2 cases per 100 000 inhabitants and incidence 0.17 episodes per 100 000 per year. Subclinical (asymptomatic) apoplexy is much more frequent than acute apoplexy and up to 25% of all pituitary tumours display haemorrhagic and/or necrosis areas either on imaging or at autopsy. Clinically silent ACTH adenomas may be particularly prone to necrosis, hemorrhage and cyst transformation. These complications occur in 30%–64% of cases. Our case had florid clinical evidence of Cushing's and a microadenoma with subsequent biochemical and clinical evidence of cure without intervention due to auto apoplexy.

11.) Two cases of persistently unidentifiable (occult) sources of ectopic ACTH secretion post-adrenalectomy

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Abstract

Clinical case 1: A 40 year old man presented in 2013 with a short history of symptoms suggestive of Cushing's syndrome (CS) including 20kg weight gain, facial plethora and red bruised skin. He was hypertensive (180/100mmHg). Work-up for an acute episode of chest pain revealed a non-traumatic fracture of his 10th posterior rib with osteoporosis subsequently being confirmed by DEXA. Biochemistry was in-keeping with ACTH-dependent CS: elevated 24-hour urinary cortisol (3615 and 3618 nmol/24 hours) [50 – 300 nmol/24 hours], abnormal low dose dexamethasone suppression test (982 nmol/L suppressing to 627 nmol/L) [<50 nmol/L], elevated waking midnight cortisol (550 and 701 nmol/L) and elevated waking midnight ACTH (50 and 123 ng/L) [<47 ng/L].
Given the severity of disease he was initiated on metyrapone pending further investigations to identify the source. CT adrenals and dynamic MRI pituitary were unremarkable. Inferior petrosal sinus sampling was in-keeping with a peripheral source of ACTH with a basal central-to-peripheral ACTH ratio of 1.1:1 and a CRH-stimulated ratio of 1.2:1 [1]. PET scanning and CT CAP revealed no source of ectopic ACTH.The patient's clinical status declined over the following 6 months; particularly his mobility. Severe derangements in water handling lead to inpatient stays. Given the burden of disease an emergency bilateral adrenalectomy was performed.
He has gone on to have serial imaging in numerous modalities: Octreotide scans (March 2015, May 2016 and May 2018), CT full body (May 2016 and May 2018), repeat MRI pituitary (Jan 2015 and May 2018) but none have been able to identify a target. Despite the negative imaging his ACTH remains persistently elevated (409 suppressing to 264ng/L after maintenance steroids).

Clinical case 2: A 43 year old woman presented in 2008 with a 6 month history of acne and hirsuitism on a background of 12 months of diabetes and hypertension. Biochemistry was suggestive of ACTH-dependent CS: elevated 24-hour urinary cortisol (>5849 nmol/24 hours x 2) [50-300 nmol/24 hours], failure to suppress cortisol after a low dose dexamethasone suppression test (1898 rising to 2298 nmol/L at 48 hours) and elevated 09:00 ACTH (125 and 119 ng/L)
MRI scanning and petrosal sinus samplings were both suggestive of a peripheral ACTH source (basal central: peripheral ratio of 1:1; unchanged with CRH-stimulation) [1].
The patient underwent elective bilateral adrenalectomy after appropriate blockade. Despite 10 years follow-up and serial imaging (CT CAP 2008, 2010, 2012, 2014 and 2017, Octreotide scan 2009 and 2012 and MIBG 2009) the source of the ectopic ACTH remains occult. Early morning ACTH remains elevated at 288ng/L suppressing to 101ng/L on maintenance dose steroids. The patient is clinically very well.
Existing case series have shown that 9.3% of ectopic ACTH sources remain occult. [2]

Conclusion: We present 2 cases of CS with occult ACTH secretion that have been followed up for 5 and 10 years respectively. We would like to stimulate discussion as to:

- How should these patients be followed up?
- Which imaging modalities are indicated?
- Is serial imaging in this context is an efficient use of resources?

12.) Immediate postoperative low cortisol in Cushings disease doesn't always predict long term remission: Two cases of late recurrence of Cushing's disease many years after initial successful TSS

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Abstract

Background: There is no consensus on the criteria to define cure after transsphenoidal surgery (TSS) for Cushing's disease (CD). Low postoperative serum cortisol is regarded by many Centres as a tool to predict the probability of long-term remission. Studies have shown late recurrence rate of 11-25% with long-term follow up in patients who achieve remission after surgery. We present two cases of late CD recurrence many years after postsurgical remission.

Case 1: 62 years old female had TSS for CD in 2000. Post surgically, her day four 9 am cortisol was <50nmol/l. Her HPA axis eventually recovered evidenced by a normal stress response to insulin-induced hypoglycaemia. A 2.5 mm residual right-sided pituitary lesion remained stable on serial MRI scans. In Sept 2015, her afternoon Cortisol was 492 nmol/L with ACTH of 24.7ng/l, but she remained clinically well with no obvious symptoms suggestive of hypercortisolemia. In 2016, 16 years after her initial TSS, she had clinical evidence of hypercortisolemia. Investigations confirmed CD recurrence with 3 raised late-night salivary cortisol and cortisone levels, failed LDDST, loss of diurnal cortisol variation and elevated 24hr urinary free cortisol. She therefore underwent a second TSS in 2017. Her day four postoperative cortisol was 95 nmol/L and she was started on prednisolone replacement. A cortisol day curve six weeks post-operatively suggested that she was in full remission. She could not have an insulin tolerance test because of a previous myocardial infarction in 2013. A glucagon stimulation test showed a sub-optimal cortisol response (peak cortisol of 212 nmol/L), but several months later, she had a completely normal adrenal response to synacthen stimulation with a peak cortisol level of 607 nmol/l. Therefore, her steroid replacement was gradually weaned off. She continued to lose weight and developed clinical symptoms in keeping with hypocortisolemia. A subsequent metyrapone suppression test showed an insufficient 11-DOC rise (160 nmol/L) with ACTH of 17.2 ng/l, supporting a diagnosis of secondary adrenal insufficiency. She was restarted on prednisolone replacement and remains clinically well.

Case 2: 51 years old lady had TSS for CD with macroadenoma in 2007. Preoperative MRI did not demonstrate cavernous sinus invasion. Post surgically, her day 4 cortisol was 57nmol/l. She was maintained on steroid replacement, as her HPA axis did not recover. Histology confirmed a corticotroph adenoma with Ki67 proliferation index of 1 to 2%. 10 years later, her glycaemic control worsened with no other clinical suggestion of hypercortisolemia. Investigations, however, confirmed CD recurrence evidenced by failed overnight DST, failed LDDST, elevated late-night salivary cortisol and cortisone and loss of diurnal cortisol variation. MRI pituitary showed a new right-sided hypo-enhancing lesion of 5 mm suggestive of possible tumour recurrence that was confirmed to be metabolically active by C-11 methionine PET CT scanning. In November 2018, she underwent a second TSS which confirmed a corticotroph adenoma histologically. Day 4 postoperative cortisol was 28 nmol/L. She was initiated on steroid replacement and remains in full remission clinically.

Conclusion: These two cases illustrate the need for life long follow up of CD patients even if immediate postoperative low cortisol levels predict long term remission. Furthermore, the first case highlights clear limitations of SST in assessment of HPA axis recovery even several months after CD surgery. SST results need to be interpreted with caution in the context of CD possibly due to adrenal hyperplasia secondary to long term ACTH exposure.

13.) Central diabetes insipidus as the inaugural Manifestation of langerhans cell histiocytosis – a rare condition with an often missed presentation

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Abstract

Introduction:
Langerhans Cell Histiocytosis (LCH) is a rare granulomatous histiocytic mainly affecting children with annual incidence of 1 in 200,000, whilst in adults it is even rarer with reported incidence at 1-2 in 1,000,000. It has a variable clinical presentation ranging from isolated skin or bone disease to a systemic disease with infiltration most frequently in bones, lungs and skin. Involvement of the hypothalamic-pituitary axis is rare and usually presents following skeletal and visceral involvement. We present an adult subject whose symptoms of Cranial DI (CDI) predated his systemic involvement by almost 5 years and was missed at multiple stages.

Case summary:
A 28-year old healthy male first reported symptoms of polyuria and polydipsia to his GP and despite drinking upto 20 litres of fluid daily with frequent nocturia, his symptoms were largely ignored for nearly 5 years. He was subsequently referred in late 2017 to Dermatology for widespread skin lesions in axillae, groins and scalp (initially diagnosed as severe hydradenitis suppurativa) and to Gastroenterology for bowel symptoms - colonic biopsies showed evidence of chronic inflammatory process but the diagnosis was inconclusive and he was treated as Crohn's disease. His next referral was to the Head & Neck clinic for neck swelling which on biopsy showed extensive infiltrate of histiocytoid cells with eosinophilic cytoplasm suggestive of LCH. This led to re-review of his clinical history and a referral to haematology and endocrinology for his CDI-related symptoms.

This lead to a multi-modal work-up for LCH including PET-CT which showed increased uptake in axillae, groins and perianal region, reflecting the lesions previously presumed as hydradenitis suppurativa. Colonic biopsies were now identified to be suggestive of LCH but skeletal survey and bone marrow biopsies were normal.

Endocrine work-up included normal anterior pituitary functions; pituitary MRI with contrast showed thickened pituitary stalk and loss of posterior pituitary T1 hyperintense signal. Water deprivation test following overnight fluid abstinence showed baseline serum osmolality (SO) concentrated to 312 mosm/kg and urine osmolality (UO) still dilute at 328 mosm/kg with 5.5 litres diuresis overnight indicative of CDI. This was further proven by a positive response to DDAVP with SO normalising to 291 and UO going up to 697. He was started on DDAVP intranasal spray and since then has had a remarkable resolution to his hyperosmotic symptoms. His LCH is being currently treated with 12 cycles of cytarabine and after 5 cycles of chemotherapy, his skin and neck lesions have mostly disappeared and pituitary MRI shows partial resolution of stalk infiltration

Conclusion:
CDI is the most common endocrine abnormality, occurring in 12% of children with LCH and 30% of adults with LCH. Established DI is generally permanent. This case summary offers the following important learning points:
i) All subjects with a diagnosis of cranial DI should be extensively investigated for infiltrative aetiologies including LCH.
ii) In subjects with a primary diagnosis of LCH, endocrine investigations into putative pituitary dysfunction should always be considered.
iii) CDI as the first manifestation of LCH is extremely rare but always probable..

14.) Hypophysitis - an immune-related adverse event

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Abstract

An 83 year old male having previously been operated for a malignant melanoma subsequently developed new liver and lung metastases. He was commenced on duel immunotherapy treatment i.e Ipilimumab and Nivolumab by the oncologist in February 2017.

He presented to A&E in May 2017 with headaches, nausea and extreme fatigue but denied visual symptoms. Initial investigations showed a random cortisol of 41nmol/L. Full pituitary hormone profile results were consistent with anterior hypo-pituitarism, low LH 0.4, FSH 2.6, testosterone <0.1, TSH 0.04, T4 4.7, low ACTH 2.3ng/L, cortisol 41, prolactin <10, low normal IGF1 6.5nmol/L(FBC, LFTs, U&E's were normal). Patient did not have a previous personal or a family history of endcrinopathies.

CT Head scan showed diffuse enlargement of pituitary gland with no discrete adenoma. This was also confirmed on MRI pituitary. Visual field assessment showed no field loss bilaterally.

Patient was treated with intravenous and then oral hydrocortisone and Levothyroxine treatment was added later on. After the Oncologist review, immunotherapy treatment was changed to single agent only i.e Nivolumab which has been continued(due to a positive treatment response on his metastases with almost complete resolution of lung metastases and marked reduction of the liver lesions. He is also now on Testosterone replacement and remains well. Patient continues to remain on the above pituitary hormone replacements.

Repeat MRI pituitary scan after 6months showed a significantly reduction in size of enlarged pituitary and an 18 months follow up scan has shown a complete resolution of pituitary enlargement.

There are several reported cases of Ipilimumab induced autoimmune hypophysitis in literature and clearly this patient's hypo-pituitarism was secondary to his immunotherapy treatment. Guidelines surrounding the initial management of autoimmune hypophysitis recommend replacing pituitary hormones in the standard fashion i.e hydrocortisone, levothyroxine +/- testosterone) and repeating an MRI scan to reassess the size of pituitary enlargement. Case series suggest an enlargement of pituitary gland with no discrete pituitary adenoma is the classical MRI finding. Usually immunotherapy-induced autoimmune hypophysitis is mostly irreversible but over time it may be worth re-assessing the hormone axis periodically as there can be some recovery more so in the thyroid and gonadal than with ACTH.

15.) Tri-phasic response post extended endonasal transsphenoidal resection for suprasellar craniopharyngioma

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Abstract

Background: Water balance disorders post neurosurgery is well recognized and may lead to hypo or hypernatremia. Tri-phasic response is reported with incidence of around 1.1%. The biochemical profile suggests transient central diabetes insipidus (DI) followed by syndrome of inappropriate antidiuretics hormone secretion (SIADH) and then permanent central DI. The pathophysiology is due to early hypothalamic dysfunction, subsequent release of vasopressin from the degenerating pituitary and, finally, depletion of vasopressin stores. Patients with craniopharyngioma are at higher risk of developing DI postoperatively. We present a case of craniopharyngioma with tri-phasic response postoperatively and highlight the importance of close electrolytes monitoring in such cases as they at risk of developing sudden rapid sodium changes.

Case: A 58 year-old lady presented with a one month history of progressive left eye visual loss. Apart from that, she has no headache, polydipsia, polyuria or symptoms related to hormonal excessive secretion or deficiency. Humphrey Visual Filed testing demonstrated a left superotemporal field defect. Subsequent MRI showed 12x10mm suprasellar lesion which compresses the optic chiasm on the left and as well as left optic nerve. The lesion extends into the sella and appears separate from the pituitary gland. The imaging appearance was in keeping with craniopharyngioma. Preoperative pituitary profile was normal with hypo-gonadal axis suggestive of postmenopausal state. The patient underwent extended endonasal transsphenoidal resection with histological findings in keeping with an adamantnomatous craniopharyngioma. Postoperatively she developed CSF leak and lumbar drain was in place for 3 days. Day 2 postoperatively, she developed transient DI with urine output of more than 1 litter in four hours. Her plasma sodium went up to 150 mmol/L with serum osmolality of 310 mOsm/kg. She required one dose of sc desmopressin (0.5mg). Day 5 postoperatively, her sodium level dropped to 132 mmol/L and further rapidly dropped to 114 mmole/L within 48 hours by day 7. During this phase, measured paired serum osmolality was 245 mOsm/Kg and urine osmolality was 570 mOsm/Kg with serum sodium of 116 mmol/L. She was managed successfully with fluid restriction. The SIADH phase lasted seven days. By day 11, her sodium level went up to 130 mmol/L from 114 mmol/L in the day before. She started to have symptoms of thirsty and mouth dryness associated with polyuria again. Her fluid intake was 650 ml and urine output was 3515 ml within 24 hours. At this point, fluid restriction was stopped and she was allowed to drink to thirst. She maintained normal sodium with symptoms suggestive of persistent DI and she was started on desmopressin. Water deprivation test done later confirmed persistent DI with loss of ability to concentrate urine in response to water deprivation.

Conclusion: Despite careful monitoring, the rapid changes in ADH activity are difficult to predict, and extremes in plasma osmolality occurred even with active management.

POSTER ABSTRACTS

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2	To Breast-feed or not to Breast-feed?	*P Behary, A Buckley, F Wernig, R Scott*
3	When steroids are not enough	*D Henderson, A De Silva, M De Bono, S Sinha*
4	Abnormal TFTs – a macro-cause for concern	*A Sharma, P Behary, S Zaman, N Khalid, A Qayum, J Todd*
5	Reduced cerebral perfusion and neurological deficit are reversed by transsphenoidal decompression in a patient with pituitary apoplexy following cardiac surgery	*A Powlson, R Dhillon, A Helmy, T Das, N Evans, D Scoffings, J Tysome, K Allinson, R J Mannion, M Gurnell*
6	Symptomatic hyperprolactinaemia from a large macroprolactinoma involving the clivus: A case report	*A Sheikh, K Rajaguru, F Kaplan*
7	A pituitary diagnostic dilemma – infective, inflammatory or neoplastic?	*C C Attard, G Conway*
8	A Lactotroph Pituitary Carcinoma with Spinal Metastasis	*T Saeed, R Pofi, J Halliday, L Anguelova, C May, A Pal, O Ansorge, A Grossman, N Warner, S Cudlip, B Jafar-Mohammadi*
9	Diabetes Insipidus a rare side effect of immune checkpoint inhibitor (ICI) therapy	*S Andrabi, G Maltese, A Fusi, G Bano*
10	Cushing's syndrome or not? Diagnostic challenges in a patient with generalised epilepsy treated with carbamazepine	*M Moustaki, F Lam, S Baldeweg*
11	High grade sella sarcoma mimicking a pituitary adenoma	*T Crabtree, H Ali, S Sandhu, R Vijayan, G Dow, R Stanworth*
12	TSHoma presenting with gynaecomastia and increased IGF-1 levels with good therapeutic response to somatostatin analogue.	*Y Manova, M Moustaki, S Baldeweg*
13	Whole body FDG PET CT and pituitary uptake: diagnostic dilemmas in oncology patients	*M Ahmad, M Asia, S Nagaraju, J Ayuk, S Chavda, S Ahmed, N Karavitaki*
14	PANCH tumour: a rare enigmatic entity	*K Lithgow, M Asia, L Vamvakopoulos, L Senthil, N Gittoes, J Ayuk, U Pohl, S Ahmed, N Karavitaki*
15	Bronchial typical carcinoid tumour metastatic to pituitary in a patient with primary hyperparathyroidism and negative MEN1 testing	*K Lithgow, A Fountas, R Batra, S Chavda, J Ayuk, U Pohl, G Tsermoulas, I Geh, P Sanghera*

Notes:

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