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Nineteenth Clinicopathological Conference on Pituitary Disease

Thursday 2nd February 2017

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

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AGENDA

PROGRAMME OF EVENTS

08:20 **REGISTRATION**

09:00 **Welcome and Introduction**
Dr Stephanie Baldeweg, Consultant Endocrinologist, University College London Hospitals and National Hospital for Neurology and Neurosurgery

09:10 **Key note lecture: The pituitary in 2016**
Dr Miles Levy, Consultant Endocrinologist, Leicester Royal Infirmary

09:40 **FORUM 1: CASE PRESENTATIONS - MALIGNANCIES IN THE PITUITARY**
Chair: Dr Ahlquist and Mr Dorward

CASE 1: PITUITARY CARCINOMA
Authors: N Nabil, P Rich, A Martin, P Johns, L Welsch, G Bano
St Georges, University of London

CASE 2: PITUITARY METASTASES FROM RENAL CELL CARCINOMA: REPORT OF THREE CASES
Authors: F Solda[1] , N Dorward[2], J Grieve [2], T Wongwarawipat[3], SE Baldeweg[4], N Fersht[1]
[1] Department of Oncology, University College London Hospitals NHS Foundation Trust
[2] UCL Institute of Neurology and National Hospital for Neurology and Neurosurgery
[3] The Whittington Hospital NHS Trust
[4] Department of Endocrinology, University College London Hospitals NHS Foundation Trust

CASE 3: APOPLEXIA IN PITUITARY TUMOR AS THE FIRST MANIFESTATION OF THE LUNG CANCER
Authors: A Obradovic & K Tucic
General Hospital Subotica, Serbia

CASE 4: PITUITARY SPINDLE CELL ONCOCYTOMA: A RECENTLY RECOGNISED TUMOUR ENTITY AND CHALLENGES IN MANAGEMENT
Authors: J Yousaf[1], N Karavitaki[2], M Carey[4], S Chavda[5], S Ahmed[3], A Paluzzi[1]
[1] Department of Neurosurgery, University Hospital Birmingham NHS Foundation Trust
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[3]ENT department, University Hospital Birmingham NHS Foundation Trust
[4]Department of Pathology, University Hospital Birmingham NHS Foundation Trust
[5]Department of Radiology, University Hospital Birmingham NHS Foundation Trust

CASE 5: A RARE, MALIGNANT, SELLAR TUMOUR; ACUTE PRESENTATION, RAPID RECURRENCE AND SUCCESSFUL MULTI-MODALITY MANAGEMENT OF A MYOEPITHELIAL TUMOUR
Authors: A Powlson[1], O Koulouri[1], A Dean[2], S Jefferies[3], R Mannion[4], N Burnet[3], M Gurnell[1]
[1]Wellcome Trust-MRC Institute of Metabolic Science & Departments of
[2]Pathology
[3]Oncology
[4]Neurosurgery University of Cambridge & Addenbrooke's Hospital, Cambridge, UK

CASE 6: MALIGNANT RETINAL MELANOMA PRESENTING WITH METASTASIS IN THE PITUITARY 20 YEARS AFTER INITIAL DIAGNOSIS
Authors: Syed Zaidi, Harit Buch, Swarupish Chavda, Yit Yang, Alessandro Paluzzi, Niki Karavitaki, Shahzada Ahmed
Queen Elizabeth Hospital Birmingham, University Hospitals Birmingham NHS Foundation Trust

11:00 **TEA, COFFEE AND POSTERS**

11:30 **Genetic screening in pituitary disease: Whom and when?**
Prof Márta Korbonits, Honorary Consultant in Endocrinology, Barts and the London School of Medicine

12:00 **FORUM 2: CASE PRESENTATIONS – PITUITARY ALL SORTS**
Chair: Miss Grieve and Prof Bouloux

CASE 7: WHEN THE GOING GETS TOUGH THE TOUGH GETS GOING
Authors: M Giordano Imbroll[1], J Grieve[2] & M Gruppetta[1]
[1]Mater Dei Hospital, Malta
[2]The National Hospital for Neurology & Neurosurgery, London

CASE 8: RESISTANT ACROMEGALY IN A CASH-STRAPPED NHS
Authors: A Pal[1], A Grossman[2], B Jafar-Mohammadi[1], S Cudlip[1], S Khan[1]
[1]Oxford University Hospitals Foundation Trust
[2]OCDEM, Churchill Hospital

CASE 9: PITUITARY STONE – A RARE ENTITY AND A MANAGEMENT CHALLENGE
Authors: J Benjamin[1], F Rasul[2], J Ahlquist[3]
[1]Queen's Hospital, Romford
[2]Kings College Hospital
[3]Southend Hospital

CASE 10: AN UNUSUALLY UNPLEASANT COURSE FOR A PROLACTINOMA
Authors: F Ahmed[1], N Karavitaki[2], S Cudlip[1], A Olaf[1], A Grossman[3]
[1]Oxford University Hospitals Foundation Trust
[2]Institute of metabolism and systems research,
[3]OCDEM, Churchill Hospital

12:55 **Update on vision and driving and on retinal imaging (OCT) in chiasmal compression**
Dr Gordon Plant, Consultant Neurologist, National Hospital, Queen Square, Moorfields Eye Hospital and St Thomas' Hospital, London

13:25 **LUNCH AND POSTERS**

14:25 **FORUM 3: CASE PRESENTATIONS - FUNCTIONING TUMOURS**
Chair: Dr Baldeweg and Mr Powell

CASE 11: LIMITATIONS OF OCTREOTIDE THERAPY IN THYROTROPIN SECRETING PITUITARY ADENOMA (TSH-OMA)
Authors: T Rehman, O Kirresh, R Herring, D R Jones
Royal Surrey County Hospital

CASE 12: A CHALLENGING CASE OF PROLACTINOMA IN A CHILD
Authors: W A Bashari, NG Burnett, G Horan, R Mannion, M Gurnell, C Acerini, HL Simpson
Cambridge University Hospital

CASE 13: THE CHALLENGES OF THE MANAGEMENT OF A MACROPROLACTINOMA IN A PATIENT WITH PARANOID SCHIZOPHRENIA
Authors: DCD Hope[1], SE Baldeweg[2], JE Ostberg[1]
[1]Watford General Hospital
[2]University College London Hospitals and National Hospital for Neurology and Neurosurgery

CASE 14: CUSHING'S DISEASE – THE POTENTIAL PITFALLS OF ADRENAL AUTONOMY
Authors: N Bolding[1], RJ Gorrigan[2], S Shaho[1], CE Stiles[1], WM Drake[1]
[1]Barts Health
[2]Hammersmith Hospital

15:20 **The Pituitary Foundation**
Ms Menai Owen-Jones, CEO, The Pituitary Foundation

15:30 **Cushing's Disease: Practice makes (near) perfect**
Mr Simon Cudlip, Consultant Neurosurgeon, John Radcliffe Hospital, Oxford

16:00 **TEA, COFFEE AND POSTERS**

16:30 **FORUM 4: CASE PRESENTATIONS - THIRST/ DIABETES INSIPIDUS**
Chair: Dr Vanderpump and Dr Srirangalingam

CASE 15: DILEMMAS IN DIABETES INSIPIDUS
Authors: B Cooke[1], PJ Kettle[2], AC Chu[3], CH Courtney[4]
[1]Specialty Trainee Endocrinology, Royal Victoria Hospital, Belfast
[2]Consultant Haematologist, Dept. of Haematology, Belfast City Hospital, Belfast
[3]Professor of Dermatology, Imperial College, London
[4]Consultant Endocrinologist, Royal Victoria Hospital, Belfast

CASE 16: A RARE CAUSE OF DIABETES INSIPIDUS
Authors: CN Uduku[1], F Wernig[1], AH Sam[1], C Slattery[2], N Martin[1], K Meeran[1], E Hatfield[1]
[1]Imperial Centre for Endocrinology
[2]Dept. of Neurology, Imperial College

17:00 **Poster and presentation prizes**
Dr Stephanie Baldeweg, Consultant Endocrinologist, University College London Hospitals and National Hospital for Neurology and Neurosurgery

17:10 **CLOSE**

FACULTY

PANEL & ORGANISERS

FACULTY

Dr James Ahlquist
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Ms Menai Owen-Jones
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Dr Umasuthan Srirangalingam
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University College London Hospitals

Dr Mark Vanderpump
Consultant Endocrinologist
Royal Free Hospital, London

ORGANISING COMMITTEE

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Consultant Endocrinologist

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Mr N Dorward, Consultant Neurosurgeon

Miss J Grieve, Consultant Neurosurgeon

Mr M Powell, Consultant Neurosurgeon

Dr M Vanderpump, Consultant Endocrinologist

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Prof Will Drake - London
Prof Ashley Grossman - Oxford
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Prof Karim Meeran - London
Dr Umasuthan Srirangalingam - London
Dr Francesca Swords - Norwich
Dr Mark Vanderpump - London

1.) PITUITARY CARCINOMA

AUTHOR(S):

N Nabil, P Rich, A Martin, P Johns, L Welsch, G Bano
St Georges, University of London

Abstract

Pituitary carcinomas are defined as pituitary tumors with subarachnoid, brain, or systemic metastasis. Most pituitary carcinomas develop from invasive relapsing or previously operated or irradiated invasive adenomas. Diagnosis is often made years after the first manifestation. We report a case of pituitary carcinoma in a female patient who had malignant growth and rapid progression of the disease from the the first presentation.

Case report

A 64 years old lady presented with change in her vision. She had history of intermittent headaches, retro-orbital pain and fatigue for the past 3 months. She was seen in the eye clinic and had right total 3rd nerve palsy. Her visual acuity and fields were normal. CT and MRI scans on the day of presentation showed enlargement of the pituitary fossa by a soft tissue mass with a suprasellar component that impinged on the left side of the optic chiasm. There was effacement of the right cavernous sinus without overt invasion with erosion of the sella floor and haemorrhage in the sphenoid sinus. Her endocrine investigations showed that she had secondary hypoadrenalism, hypothyroidism, hypogonadism and a marginally high prolactin. She was started on hormone replacement. A month after presentation she complained of sudden visual loss in the left eye. A repeat MRI scan showed rapid tumour growth. She had endoscopic debulking surgery. Biopsies were taken from the centre of the tumour and a reasonable amount of tumour was removed.Her histology showed highly-malignant tumour with very brisk proliferative activity, anaplastic cytological features and a complex immunophenotype. Conclusion was a malignant neoplasm with epithelial and neuroendocrine features and the absence of a primary tumour elsewhere, the histological features were compatible with a pituitary carcinoma. She had CT thorax, abdomen and pelvis; which did not show a primary tumour but demonstrated several vertebral body metastases, subsequently confirmed on MRI of the spine. Despite debulking of the pituitary tumour a repeat MRI scan three weeks after surgery showed suprasellar disease progression with greater mass effect on the optic chiasm. She was given palliative radiotherapy to painful spine bone metastasis. Patient developed metastasis in brain, lungs, lymph nodes and bones. She declined further treatment.

2.) PITUITARY METASTASES FROM RENAL CELL CARCINOMA: REPORT OF THREE CASES

AUTHOR(S):

F Solda[1] , N Dorward[2], J Grieve [2], T Wongwarawipat[3], SE Baldeweg[4], N Fersht[1]
[1] Department of Oncology, University College London Hospitals NHS Foundation Trust
[2] UCL Institute of Neurology and National Hospital for Neurology and Neurosurgery
[3] The Whittington Hospital NHS Trust
[4] Department of Endocrinology, University College London Hospitals NHS Foundation Trust

Abstract

Purpose: Symptomatic metastases to the pituitary gland from renal clear cell carcinoma (RCC) are rare and mainly occurring in elderly patients with advanced stage of disease. We present our experience with 3 patients treated with adjuvant radiotherapy.

Methods and Materials: Between March 2011 and April 2016, three patients (Pt 1, 2 and 3 respectively 61, 70 and 75 years' old males) received radiotherapy after confirmation of pituitary metastases from RCC. Symptoms at presentation included headaches (Pt 1 and 2), bitemporal hemianopia (Pt 1) and panhypopituitarism (Pt 3).
In Pt 1, primary RCC was identified on imaging after TSS (partial resection) for a pituitary lesion originally thought to be an adenoma. In Pt 2 and 3, diagnosis was confirmed on a trans-sphenoidal biopsy 8 and 5 years after primary diagnosis of RCC respectively. Pt 2 had obtained a previous complete response (lung metastases) on sunitinib and Pt 3 received chemotherapy with sorafenib up to 18 months before histological confirmation of pituitary metastatic disease.Adjuvant radiotherapy was delivered to the whole brain (WBRT 30 Gy in 10#) in Pt 1 while a stereotactic irradiation of the pituitary fossa/residual disease was performed in Pt 2 and 3 (25 Gy in 5# and 20 Gy in 5# respectively).

Results: Pt 1 rapidly progressed presenting acutely with non-obstructive hydrocephalus 2 months after WBRT. Local and diffuse systemic progression was confirmed on follow up imaging. He died of progressive disease 6 months after treatment. Pt 2 showed radiological stability of disease on last imaging although complaining of persistent headaches (18 months OS from treatment). Nine months after treatment, Pt 3 is alive with stable disease on follow up imaging.

Conclusion: While tumour invasiveness can make resection difficult, the use of targeted agents (sorafenib and sunitinib) is debated in pituitary metastatic RCC. Stereotactic radiotherapy can be beneficial in controlling progressive or residual tumours after incomplete surgery.

3.) APOPLEXIA IN PITUITARY TUMOR AS THE FIRST MANIFESTATION OF THE LUNG CANCER

AUTHOR(S):

A Obradovic & K Tucic
General Hospital Subotica, Serbia

Abstract

Background: Metastasis to the pituitary gland are rare events. Lung cancer account for approximately two thirds,and usually indicate widespread malignant disease.

Case presentation: A 53-year old female presented with headache that rapidly progressed in severity one month ago; nausea and vomiting, fatigue, general malaise, III cranial nerve palsy causing double vision, left eye ptosis, left pupil dilatation and pathological pupillary light reflex , right arm hemiparesis . CT scan presented native hiperdense (68HU) pituitary lesion characterised as macroadenoma with internal bleeding. After eight days on follow up MRI scan dimensions of the lesion significantly increased with suprasellar extension and compression of optic chiasma. Abdominal CT verified heterogenoeus 132x82mm tumor of the left suprarenal gland and secondary deposit in S7 of liver. Neck CT revealed pathological conglomerate lymph nodes. Chest X ray presented parahylar pleural extended opacity and following lung CT detailed . The most part was affected by subtotal neoplastic infiltration of the right lung middle lobe without signs of bronchial obstruction. Neck lymph node biopsy was diagnostic for lung adenocarcinoma with EGFR gene mutation and thyrosine kinase inhibitors- gefitinib has been used last 10 months. Result of therapy's been subtotal regresion of pituitary tumor and two-fold regression of all metastatic lesions.

Conclusion: Patient have survival benefits and good quality of life with significant reduction in size of the primary tumor and metastasis. Regarding metastasis to a pituitary adenoma, althout tumor to tumor metastasis is extremly rare, nevertheless the infiltration of preexisting nonfunctioning adenoma cannot be totally excluded.

Key words : pituitary metastases, apoplexy, lung carcinoma, thyrosine kinase inhibitors, nonfunctioning adenoma

4.) PITUITARY SPINDLE CELL ONCOCYTOMA: A RECENTLY RECOGNISED TUMOUR ENTITY AND CHALLENGES IN MANAGEMENT

AUTHOR(S):

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[5]Department of Radiology, University Hospital Birmingham NHS Foundation Trust

Abstract

Introduction

Pituitary spindle cell oncocytoma (SCO) is a rare non-adenomatous neoplasm accounting for 0.1-0.4% of sellar tumours with only 26 cases described in the published literature. We report a case of SCO and discuss areas requiring attention in the management of these tumours.

Case report

A 48-year old male presented in 8/2014 with bitemporal hemianopia and hypopituitarism. MRI revealed a homogenously enhancing sellar tumour with large suprasellar extension into the third ventricle. The tumour was thought to be consistent with a pituitary macroadenoma and the patient underwent three unsuccessful attempts of resection through transsphenoidal and transcranial routes. The procedures were abandoned on all occasions due to high tumour vascularity and severe intraoperative haemorrhage. Histology was consistent with SCO. In 9/2016, he presented with progressively worsening headaches, lethargy, confusion, visual deterioration and urinary incontinence. MRI revealed tumour progression with obstructive hydrocephalus. Preoperative cerebral angiogram was performed but embolisation of the tumour vessels was felt to be unsafe. The patient underwent bifrontal craniotomy and interhemispheric approach on 15/09/2016. A soft and highly vascular tumour in the third ventricle was debulked and subtotal excision was achieved.

Discussion

SCOs have been formally recognised as a distinct entity by the WHO 2007 classification of brain tumours. Their pathogenesis is uncertain and although they are classified as WHO grade 1, their clinical course may be aggressive. They present a challenge at multiple levels. Thus, given that their presentation and imaging features do not differ from those of macroadenomas, pre-operative suspicion of the diagnosis is very difficult. Importantly, high tumour vascularity may be associated with significant intraoperative blood loss making surgical resection extremely challenging with potential adverse sequelae. Radiotherapy has been employed for residual mass with mixed results. Based on the limited published experience, SCOs are also associated with high recurrence rates necessitating close follow-up.

5.) A RARE, MALIGNANT, SELLAR TUMOUR; ACUTE PRESENTATION, RAPID RECURRENCE AND SUCCESSFUL MULTI-MODALITY MANAGEMENT OF A MYOEPITHELIAL TUMOUR

AUTHOR(S):

A Powlson[1], O Koulouri[1], A Dean[2], S Jefferies[3], R Mannion[4], N Burnet[3], M Gurnell[1]
[1]Wellcome Trust-MRC Institute of Metabolic Science & Departments of
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[3]Oncology
[4]Neurosurgery University of Cambridge & Addenbrooke's Hospital, Cambridge, UK

Abstract

A 59-year-old woman presented with headaches, vomiting, polydipsia/polyuria and four days of progressive visual impairment. She was found to have a bitemporal hemianopia and pan-hypopituitarism. MRI demonstrated a sellar mass with suprasellar extension and chiasmal compression. Hormone replacement was commenced and the lesion was debulked as an emergency via craniotomy. Early postoperative MRI showed substantial debulking of the suprasellar component, with decompression of the chiasm, and with a small amount of unenhancing residual tissue. There was significant improvement in vision post-operatively, but without complete resolution.

Three weeks after surgery, she re-presented with severe headache and photophobia, and a new CNIII palsy. MRI revealed rapid recurrence of the tumour within the surgical bed, with involvement of the cavernous sinus, and with enhancing tissue now eroding into the clivus. High dose dexamethasone was commenced with urgent MDT review of the serial imaging and histopathology. CT chest, abdomen and pelvis did not demonstrate any other sites of disease.

Secondary review of the pathology concluded that this was a malignant tumour (MIB-1 75%), with immunophenotype and focal clear cell morphology suggestive of a myoepithelial tumour. Urgent RT was given (20Gy, 5#), followed by six cycles of carboplatin, etoposide and cyclophosphamide (CEC) chemotherapy.

Ten months after her initial presentation, following surgery, radiotherapy and chemotherapy, the patient is clinically well with no residual neurological deficit. Recent MRI demonstrated no evidence of residual or recurrent disease.

This case highlights the importance of a multidisciplinary approach to the management of pituitary disease, particularly with aggressive, rapidly progressive tumours.

6.) MALIGNANT RETINAL MELANOMA PRESENTING WITH METASTASIS IN THE PITUITARY 20 YEARS AFTER INITIAL DIAGNOSIS

AUTHOR(S):

Syed Zaidi, Harit Buch, Swarupish Chavda, Yit Yang, Alessandro Paluzzi, Niki Karavitaki, Shahzada Ahmed
Queen Elizabeth Hospital Birmingham, University Hospitals Birmingham NHS Foundation Trust

Abstract

A 45-year old female was referred to our Department in November 2015 with a short history of deterioration of the visual fields in her only seeing Right eye and a sellar mass extending to the optic pathways. Her medical history included Left-sided retinal melanoma treated with stereotactic radiosurgery in 1995 and polymyalgia rheumatica on steroids. Pituitary MRI showed a haemorrhagic pituitary fossa lesion with suprasellar extension and involvement of the Right optic chiasm and proximal optic nerve. Irregular extension into the Right subarachnoid space and the interpeduncular cistern were also noted. She had secondary hypothyroidism, normal prolactin and IGF-I. Following discussion in our pituitary MDT meeting, debulking of the lesion through an endoscopic transsphenoidal approach was performed (November 2015), with patient's vision improving from counting fingers only, preoperative to 6/6 within a few days postoperatively. Pathology confirmed malignant melanoma negative for BRAF, cKIT and NRAS mutations and pituitary MRI showed a small amount of residual tissue. Post-operatively, she remains on Levothyroxine and her ACTH reserve off steroids is intact. Further follow-up and imaging (brain, neck, chest, abdomen and pelvis) have not been consistent with disease progression up to 12 months post surgery and she remains on close monitoring.

Although malignant melanoma is known to have a high propensity to metastasize to the brain, melanoma metastasizing to the pituitary gland is very rare. To our knowledge, there is no report in the literature of pituitary gland involvement detected 20 years after initial diagnosis. Surgery facilitates the diagnosis and provides symptomatic improvement. Radiotherapy has been considered a further treatment approach for sellar disease and the prognosis remains uncertain.

7.) WHEN THE GOING GETS TOUGH THE TOUGH GETS GOING

AUTHOR(S):

M Giordano Imbroll[1], J Grieve[2] & M Gruppetta[1]
[1]Mater Dei Hospital, Malta
[2]The National Hospital for Neurology & Neurosurgery, London

Abstract

Ms Z.O. first presented in 2007 at the age of 28 years with left sided visual field defect, headache and amenorrhoea. Past medical history revealed her to have activated protein C resistance resulting in DVT post delivery and homozygous MTHFR gene mutation. On examination she had coarse facial features, spade like hands and increased sweating. Pituitary profile at the time showed the following results:
IGF1 – 1259ng/ml (114-494);
GH – 111ng/ml (0-10);
TSH – 1.1uIU/ml;
FSH – 3.4IU/l;
LH – 0.4IU/l;
Prolactin – 2101mIU/L;
Cortisol – 297nmol/l.
MRI showed a 35x24x19mm pituitary adenoma with suprasellar extension in midline compressing the bottom of the third ventricle. She had transphenoidal resection of the growth hormone producing pituitary adenoma in June 2007, confirmed histologically.

Post operatively she did well and received Thyroxine replacement only. MRI post op showed a small residual tumour on the right side of sella. Menstruation resumed regularly only to stop again in 2008. IGF1 at the time was 704ng/ml (114-494). MRI showed significant enlargement of the residual adenomatous tissue within the pituitary gland, without optic chiasm compression. She was at this point started on Octreotide LAR® 20mg every 4 weeks, increased to 30mg every 4 weeks and eventually Pegvisomant, increased gradually to 20mg dly in view of inability to control IGF1. MRI January 2011 showed the residual pituitary adenoma had grown to 16x20x17mm.

In June 2011 she had her second transphenoidal hypophysectomy of her growth hormone pituitary adenoma followed by radiotherapy in view of the high Ki67 proliferation index (focally up to 5%). MRI post op showed no evidence of residual tumour and IGF1 was 72ng/ml (67-228).
Pegvisomant and Octreotide LAR® were stopped post operatively.

She is now on cortisol and thyroxine replacement. She is however very reluctant to start hormone replacement in the form of HRT in view of her previous DVT.

8.) RESISTANT ACROMEGALY IN A CASH-STRAPPED NHS

AUTHOR(S):

A Pal[1], A Grossman[2], B Jafar-Mohammadi[1], S Cudlip[1], S Khan[1]
[1]Oxford University Hospitals Foundation Trust
[2]OCDEM, Churchill Hospital

Abstract

This gentleman was diagnosed with Acromegaly in July 2005 at 27 years of age by his GP with an IGF-1 of 150 nmol/L and random GH of 3.87. CT scan showed 1.9 cm Macroadenoma.

His Acromegaly remained uncontrolled on Octreotide and he had transsphenoidal surgery in May 2006. Histology confirmed atypical Somatotroph adenoma (MIB-1 index ~5%). He was not cured after surgery and his GH and IGF-1 stayed significantly above normal range despite maximum doses of Lanreotide and Cabergoline. He developed Cavernous Sinus Syndrome with right 3rd nerve palsy due to progression of adenoma in the right cavernous sinus.

After repeat transsphenoidal surgery in August 2007, maximum Lanreotide and Cabergoline were unable to control his Acromegaly. With external beam radiotherapy in 2008, his IGF-1 and GH levels returned to normal for a few months before IGF-1 rose again above reference range. His acromegaly remained uncontrolled and gamma Knife Surgery in 2012 only served to improve his symptoms of Cavernous Sinus Syndrome.

Lanreotide was stopped (2013) after 8 years of therapy due to intractable diarrhoea. He remains symptomatic with headaches and hyperhidrosis. At the age of 36 he has hypothyroidism, hypogonadism, morbid obesity, type 2 diabetes, hypertension, osteoarthritis, obstructive sleep apnoea with type 2 respiratory failure and heart failure with systolic and diastolic dysfunction. IGF-1 and GH remain elevated (IGF-1 55.3nmol/L normal range 14.2-46.5).

He has had two requests for Pegvisomant funding rejected as his case was deemed not to represent an "exceptional situation". Given recent progression of his cardiac dysfunction a third request for Pegvisomant funding is being submitted.

Discussion points

1) Difficult to control Acromegaly.
2) Cavernous Sinus Syndrome
3) Pegvisomant and how to obtain in a cash-strapped NHS – what constitutes exceptionality?

9.) PITUITARY STONE – A RARE ENTITY AND A MANAGEMENT CHALLENGE

AUTHOR(S):

J Benjamin[1], F Rasul[2], J Ahlquist[3]
[1]Queen's Hospital, Romford
[2]Kings College Hospital
[3]Southend Hospital

Abstract

Pituitary stone is a rare entity characterised by significant calcification in the pituitary fossa. It was first described in the 19th century. The pathophysiology remains unclear. In clinical practice the most common cause of a calcified sellar mass is craniopharyngioma; less commonly such calcifications are due to pituitary adenoma.

We describe the case of a 56 year old man who presented with a few months' history of nonspecific headache associated with nausea. He was also found to have visual field deficits on formal perimetry testing. MRI showed a large sellar mass abutting the optic chiasm. Pre-operative prolactin was 5699 mU/L. He remained refractory to cabergoline therapy.

He underwent trans-sphenoidal hypophysectomy in September 2016. Intraoperatively the lesion was found to be hard and calcified. During the procedure there was no descent of the diaphragm sellae.

Immunohistochemistry was positive for prolactin within neoplastic cells. The amorphous extracellular material in the form of psammoma bodies was strongly positive. Ki67 index revealed a neoplasm of low proliferative potential. Therefore a prolactinoma with massive calcification – a pituitary stone – was diagnosed.

This case presents both a diagnostic challenge and an on-going management dilemma. Following debulking surgery of a 'hard, calcified mass' there has not been a significant improvement of the visual field deficits.

The appearances of a calcified mass are not well described and were initially managed on the basis of a hormonal profile. Due to the paucity of literature relating to pituitary stone it is not clear how best this patient should be managed in the future. Is there a role for re-do surgery or radiotherapy? Should the patient be managed expectantly?

10.) AN UNUSUALLY UNPLEASANT COURSE FOR A PROLACTINOMA

AUTHOR(S):

F Ahmed[1], N Karavitaki[2], S Cudlip[1], A Olaf[1], A Grossman[3]
[1]Oxford University Hospitals Foundation Trust
[2]Institute of metabolism and systems research,
[3]OCDEM, Churchill Hospital

Abstract

A 68 year-old man was seen in 2008 with a history of collapses, preceded by one year history of lethargy, weight loss, loss of pubic and chest hair, and visual deterioration. An urgent MRI demonstrated a pituitary macroadenoma with a fluid level consistent with pituitary apoplexy. The serum prolactin was 55,278mU/L. At trans sphenoidal surgery (TSS) a tumour was excised which on immunochemistry showed features of a pituitary adenoma with sporadic prolactin expression and some TSH co expression, a raised Ki-67 of 3-5% but no other atypical features . His visual fields fully resolved, but he was left pan hypopituitary and required full replacement therapy; serum prolactin was 35,633mU/l but was normalised on cabergoline. He remained well at regular follow-up until 2014, when he reported visual deterioration and bitemporal hemianopia on examination. His serum prolactin had risen to 32,558 Mu/L. A new MRI revealed considerable increase in size of the residual tumour with chiasmal compression, and he was immediately treated with high-dose cabergoline 1mg daily. However, his visual fields remained abnormal and his serum prolactin failed to fall, so he had repeat TSS in February 2015 when the tumour was found to be cystic in consistency; three-month post-TSS his vision was normal but the prolactin remained markedly elevated. By September 2015 his vision had again deteriorated with a prolactin level rising to 107,269 mU/L; a new MRI revealed a progressive increase in the size of the r esidual tumour with a significant suprasellar extension. Repeat TSS with debulking of the tumour was performed, with histology now showing evidence of an atypical pituitary adenoma with a very high mitotic rate and Ki-67 20-30%Ki-67 While awaiting radiotherapy planning a new MRI at 3 months showed dramatic tumour regrowth and thus following another TSS he is now undergoing external beam radiotherapy.
This case is unusual in a number of ways: the disease was well controlled after the first surgery and was quiescent for 6 years, but then showed a relatively sudden dramatic change with tumour regrowth requiring three operations within a one-year window; there has been a failure to control the prolactin level with high-dose cabergoline and surgery, and we note the change in histology from a typical to an atypical adenoma.

11.) LIMITATIONS OF OCTREOTIDE THERAPY IN THYROTROPIN SECRETING PITUITARY ADENOMA (TSH-OMA)

AUTHOR(S):

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Abstract

We present a 67 year old woman who underwent radioactive iodine ablation therapy for presumed thyrotoxicosis in 1994. TSH was always measurable; (TSH 1.66, T4 28.6, T3 13.2). Significant other history included ITP and paroxysmal atrial fibrillation.

Endocrinology opinion was sought by her GP in 2011 as her TSH level remained elevated on high dose Levotyroxine-T4 (225 micrograms). Referral TFTs showed TSH 7.6, T4 28.4, T3 5.9. Thyroid ultrasound did not show any discernible thyroid gland. Heterophile antibodies were negative and alpha subunit of glycoprotein was elevated at 4.55. MRI pituitary fossa revealed a large pituitary adenoma extending up to the right optic chiasm with normal visual fields. Pituitary profile showed LH 23, FSH 62, Prolactin 231, Cortisol 193, IGF-1 12.2.

Thyrotropin releasing hormone test (TRH) showed blunted TSH response suggestive of TSH-oma (0 min. 6.1, 20 min. 17.3, 60 min. 14.5) and she underwent T3 suppression test which showed preserved peripheral tissue responses in keeping with TSH-oma.

She started on Octreotide with biannual MRI and visual field testing. Following 3 months Octreotide therapy there was marked improvement in TSH level from 10 down to 1.6.Repeat MRI showed reduction in adenoma size from 2.1 x 1.7 cm down to 1.8 x 1.4 cm.

Unfortunately the patient is experiencing episodes of recurrent hypoglycaemia and disabling GI symptoms leading to temporary cessation of Octreotide. What are the next therapeutic strategies? She is reluctant to consider surgery.

12.) A CHALLENGING CASE OF PROLACTINOMA IN A CHILD

AUTHOR(S):

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Abstract

Case report:
An 11-year-old pre-pubertal boy was undergoing clinical surveillance for MEN-1. Whilst no mutation was initially found, he had a extensive family history consistent with MEN1 with his father having parathyroid carcinoma and malignant gastrinoma, and grandmother primary hyperparathyroidism and prolactinoma. At diagnosis he had a prolactin of over 3000mIU/L and 1.5cm pituitary adenoma with suprasellar extension. He was started on bromocriptine, then cabergoline which was titrated up. Unfortunately he remained refractory to treatment and on cabergoline dose of 1.5mg twice week his prolactin was still increasing with an enlarging adenoma which was encroaching the optic chiasm, resulting in a visual field defect. Concomitantly he was diagnosed with growth hormone deficiency and started on rGH replacement.

As medical management was not slowing tumour growth he underwent endoscopic transphenoidal surgery with a good surgical result. Histology confirmed prolactinoma.
Unfortunately surveillance MRI imaging demonstrated regrowth and residual tumour extending into the right cavernous sinus with an elevated prolactin. After discussion it was felt that further surgery would not be curative and he was referred for consideration of external beam radiotherapy. In view of long term side effects and his young age he has been accepted for proton beam therapy.
During this period genetic testing in his father identified a mutation compatible with MEN-1, which was confirmed in him also- a splice site mutation (c.784-9G>A) identified in intron 4 of the MEN-1 gene.
To date he has no other features of MEN1 on biochemical and radiological surveillance.

Conclusion:
This case highlights several challenges and we will discuss:
• MEN1 surveillance in childhood
• Therapeutic options of aggressive prolactinoma in childhood and when to consider surgical intervention
• Complications of radiotherapy compared to proton beam therapy

13.) THE CHALLENGES OF THE MANAGEMENT OF A MACROPROLACTINOMA IN A PATIENT WITH PARANOID SCHIZOPHRENIA

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Abstract

A 39 year-old man with paranoid schizophrenia presented with low total testosterone and symptoms of hypogonadism including gynaecomastia. Symptom-onset preceded commencement of antipsychotic medications which included Risperidone 3mg daily and Trazadone 50mg daily. Investigations revealed hypogonadotropic hypogonadism: FSH 0.9U/L, LH 2.3U/L, testosterone 6.6nmol/L, 9am cortisol 250nmol/L. The prolactin was significantly raised at 13,354 mU/L (45-375mU/L), no macroprolactin, thyroid and Growth hormone levels normal. MRI pituitary showed a pituitary adenoma 12x8.2mm, partly extending into the left cavernous sinus with no suprasellar extension. Cabergoline 500ug x1/week reduced prolactin to a nadir of 2,554 mU/L over 12 months. A 3 month MRI showed no change in size of the macroprolactinoma. Hypogonadotrophic hypogonadism persisted and thus testosterone replacement therapy was commenced, initially at 5mg daily, uptitrated to 30mg daily. At this time Aripriprazole 10mg daily was substituted for previous antipsychotic medications. Four months after commencing testosterone replacement, prolactin levels rose to 5,260mU/L with testosterone of 27nmol/L, oestradiol 344 (50-200pmol/L). Repeat MRI pituitary showed no change in prolactinoma size and prolactin fell to 3,143mU/L after discontinuation of testosterone replacement, although this was subsequenty restarted at a lower dose of 10mg when the patient developed hypogonadal symptoms. Prolactin levels rose again on three further occasions and correlated with titrating testosterone up against hypogonadal symptoms. Gynaecomastia became a significant problem and the patient underwent mastectomy, with resultant complications. This case highlights the challenges of treating prolactinomas in patients undergoing testosterone replacement who are 'high aromatizers', where oestrogen stimulation of lactotrophic cells exacerbates hyperprolactinaemia with inadequate suppression with dopaminergic treatment. These patients may undergo unnecessary repeated imaging or medication changes. The patient was treated more aggressively with high dose Cabergoline, 1g x3/week; hypogonadal symptoms were treated with lower dose testosterone. Second line treatment such as radiotherapy or surgery can also be considered in macroprolactinomas resistant to medical therapy.

14.) CUSHING'S DISEASE – THE POTENTIAL PITFALLS OF ADRENAL AUTONOMY

AUTHOR(S):

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Abstract

A 54 year-old female presented to her GP with a 9 year history of poorly controlled hypertension (requiring five antihypertensive agents) and type 2 diabetes mellitus, associated with central weight gain, low mood and poor wound healing. On examination she had clinical evidence of glucocorticoid excess.

Cushing's syndrome was confirmed on low dose dexamethasone suppression testing (2+0 cortisol 857 nmol/l, 2+ 48 cortisol 346 nmol/l). Cushing's day curve demonstrated loss of circadian rhythm, with a mean cortisol of 702 nmol/l. Serum ACTH was detectable at 45 ng/l and inferior petrosal sinus sampling confirmed pituitary-dependent Cushing's disease. Pituitary MR imaging demonstrated a markedly enlarged, predominantly empty pituitary fossa, with a sliver of tissue on the left hand side, raising the possibility of a previous macroadenoma which had auto-infarcted. Adrenal imaging showed bilateral marked nodular hyperplasia. The relative values of the ACTH and cortisol levels was thought to indicate that the adrenals had developed a degree of s
emi-autonomy.

Medical therapy was commenced with 8 hourly metyrapone 750mg , 750mg, 1000mg. The patient has been referred for conventional fractionated external beam radiotherapy, in light of the fact that trans-sphenoidal surgery is unlikely to be curative and highly likely to result in a CSF leak due to the anatomy. It is likely she will require unilateral or bilateral adrenal surgery in the future.

This case demonstrates that long-term ACTH stimulation of the adrenal glands can result in significant adrenal hyperplasia with autonomy and highlights the potential value of adrenal imaging, even in cases of proven ACTH-dependent pituitary disease.

Does the panel agree with our management strategy?

15.) DILEMMAS IN DIABETES INSIPIDUS

AUTHOR(S):

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Abstract

A 31 year old lady presented with a 4month history of polydipsia and tiredness. There was no significant medical history. Examination was normal.

Initial investigations:

Serum Sodium	Serum osmolality	Urine osmolality	fT4	TSH	Prolactin	FSH	LH	Oestradiol	Serum Cortisol
156 mmol/L	324 mOsm/kg	162 mOsm/kg	5.9 pmol/L	3.8 mU/L	2966	0.4 U/l	<0.2 U/l	65 pmol/L	251 nmol/L

Her presentation was consistent with diabetes insipidus and response to DDAVP confirmed this as cranial DI. Pituitary MRI revealed an enhancing left hypothalamic lesion measuring 1.5 x1.7cm with surrounding oedema abutting the pituitary stalk and extending across the midline to the right hypothalamus. CSF and tumour markers were unremarkable. CT imaging showed lung consolidation and lytic bone lesions in multiple ribs. Thyroxine was also commenced.

Her hypothalamic lesion was deemed difficult to biopsy and while a rib biopsy attempt was made, this was unsuccessful. Following MDM discussion and a working diagnosis of histiocytosis she was treated with prednisolone 20mg

On repeat imaging at 3 months the pituitary lesion had decreased from 13 to 9mm with resolution of the surrounding oedema.
Given the likely diagnosis of Langerhans Cell Histiocytosis she was commenced on Cladribine.
Serial pituitary imaging revealed continued improvement and following Cladribine a course of mercaptopurine was commenced. Repeat endocrine assessment confirmed on-going diabetes insipidus, hypogonadism and secondary hypothyroidism.
At last review, 2 years since last chemotherapy she remained symptomatically well with only mild nodular enhancement of the hypothalamus. This case illustrates the dilemma of parasellar lesions without a tissue diagnosis. In this case both the pattern of multiorgan involvement and radiological findings pointed to Langerhans Cell Histiocytosis. The infrequency with which it is encountered together with the preference for histological evidence makes for a diagnostic challenge in a patient with pituitary disease.

16.) A RARE CAUSE OF DIABETES INSIPIDUS

AUTHOR(S):

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Abstract

A 42-year-old man was admitted following a collapse. He reported polydipsia, polyuria, dizziness and diplopia. He was severely dehydrated and had generalised weakness. Blood tests showed a serum sodium of 170 mmol/L, serum osmolality of 314 mosm/kg and secondary hypothyroidism. Brain MRI revealed brainstem, thalamic and pituitary infiltration, raising the suspicion of a neoplastic or inflammatory process. He was commenced on dexamethasone to relieve brainstem oedema, oral DDAVP for diabetes insipidus and levothyroxine. Cerebrospinal fluid analysis showed an insignificant white count of 7 cells/ml (85% mononuclear), protein 0.49 g/L and low ACE levels.

18F-FDG PET-CT showed high uptake within the intracranial lesions and bilaterally in the distal femora and right mid humeral shaft. Plain radiographs identified proximal tibial and distal femoral medullary sclerosis. Improved MRI appearances following four weeks of dexamethasone treatment favoured an inflammatory aetiology. The x-ray changes in combination with the findings on PET-CT and MRI are pathognomonic of Erdheim-Chester disease, a rare form of on non-Langerhans histiocytosis, thought to be derived from the monocyte-macrophage lineage. Erdheim-Chester disease is characterized by multifocal osteosclerotic lesions of the long bones. Approximately 25% of cases manifest with cranial diabetes insipidus secondary to pituitary infiltration. Interferon-alpha is the treatment of choice in symptomatic cases. The BRAF inhibitor vemurafenib has been successfully used in patients with refractory Erdheim-Chester disease with a BRAF V600E mutation who did not respond to interferon-alpha.

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3	*"Solda' F1, Brown N1, Dorward N2, Pollock J R3, Sioftanos G4, Bouloux PM5, Fersht N1 1Department of Oncology, University College London Hospitals NHS Foundation Trust, London, UK 2Department of Neurosurgery, UCL Institute of Neurology and National Hospital for Neurology and Neurosurgery, London, UK 3Department of Neurosurgery, Queens Hospital, Romford, UK 4Department of Oncology, Southend University Hospital, Prittlewell Chase, Westcliff-on-Sea, UK 5Department of Endocrinology, Royal Free London NHS Foundation Trust, London, UK"*	ADULT ATYPICAL TERATOID/RHABDOID TUMOUR (AT/RT) OF THE PITUITARY FOSSA: A CASE REPORT
4	*Ahmed Al-Sharefi, Suhel Ashraff , Sath Nag and Vijayaraman Arutchelvam*	A PITUITARY EVOLUTION THEORY: ARE NFPA REALLY NON-FUNCTIONAL OR JUST A PART OF THE EVOLVING CONTINUUM?
5	*Dr Vasiliki Tsampasian, Core Medical Trainee, Department of Diabetes & Endocrinology, Norfolk and Norwich University Hospital, Dr Ketan Dhatariya, Consultant Physician, Department of Diabetes & Endocrinology, Norfolk and Norwich University Hospital*	A CASE OF CUSHING'S DISEASE ACCOMPANIED BY RATHKE'S CLEFT CYST
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13	*P Machenahalli1, N Hill1, A Mehta3, N Mendoza2, P Lewis4, C Pusey5, NM Martin1, K Meeran1,F Wernig1, E Hatfield1. Department of Endocrinology1, Neurosurgery2, Radiology3, Histopathology4, Nephrology5. Imperial College Healthcare NHS Trust*	IGG4 RELATED HYPOPHYSITIS-A DIAGNOSTIC CHALLENGE
14	*Punith Kempegowda1,3, Andrea Mason1, Neil Gittoes1, Andy Toogood1, John Ayuk1, Rosalind Mitchell2, Niki Karavitaki1,3 1Departments of Endocrinology and 2Neurosurgery, University Hospital Birmingham NHS Foundation Trust, 3Institute of Metabolism and Systems Research, School of Medical and Dental Sciences, University of Birmingham*	FUNCTIONING GONADOTROPH ADENOMAS: A HEADACHE
15	*Olivia Dunscombe 1, Omer A Jamall 1, Claire Feeney 1,2, Joanna Zaw-Linn 1, David J Sharp 1, Anthony P Goldstone 1,2 1 Division of Brain Sciences, Imperial College London, Hammersmith Hospital, London, UK 2 Imperial Centre for Endocrinology, Imperial College Healthcare NHS Trust, St. Mary's Hospital, London, UK*	PREVALENCE AND ASSOCIATIONS OF TSH DEFICIENCY IN ADULTS AFTER TRAUMATIC BRAIN INJURY USING THE TSH INDEX
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