Programme

Sixteenth Clinicopathological Conference on Pituitary Disease

Monday 10th February 2014

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

Conference organiser: Dr Stephanie E. Baldeweg, UCLH/ NHNN

A multidisciplinary approach to pituitary disease, with workshop discussions of cases by representatives from neurosurgery, endocrinology, ENT, paediatrics, radiotherapy, pathology and neuroradiology

Educational grants have been provided by:

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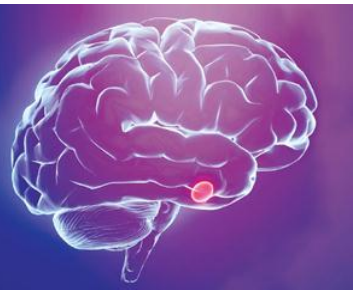
Faculty List



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Royal Free Hospital

Panel Members:

Neuropathologists:
Federico Roncaroli – London

Neurooncologists:
Naomi Fersht – London

Neuroradiologists:
Katherine Miszkiel – London
Andrew Platts – London

Endocrinologists:
Karim Meeran – London
Richard Quinton – Newcastle
Stephen Ball – Newcastle
Mark Gurnell – Cambridge
Niki Karavitaki – Oxford

Neurosurgoens:
Nicholas Thomas – London
Michael Buchfelder – Nuremberg
Richard Nelson – Bristol
Simon Cudlip – Oxford

Organising Committee:

Stephanie Baldeweg, Michael Powell, Joan Grieve, Gerard Conway, James Ahlquist, Mark Vanderpump

Agenda

08:15 **Registration**

09:15 **Welcome and Introduction:**
Dr Stephanie Baldeweg, Consultant Endocrinologist
University College London Hospital

09:20 **Key note lecture:**
The pituitary gland in 2013
Dr Mark Gurnell, Consultant Endocrinologist
Addenbrookes Hospital, Cambridge

10:00 **Forum 1 – Case Presentations – Pituitary – all sorts**
Chairs: Dr Mark Vanderpump and Mr Michael Powell

Case 1 – Successful pregnancy and headache treatment in a patient with acromegaly
Author(s): Angela Rogers, Wellcome Trust Clinical Training Fellow and John A. H. Wass, Professor of Endocrinology.

Case 2 – Multisystem Langerhans Cell Histiocytosis: Pituitary recovery post cladribine and 6-mercaptopurine
Author(s): Dr B.L.Carpenter, Professor A Chu, Professor Russell-Jones

Case 3 – Acromegaly with GH-positive neuronal choristoma and pituitary adenoma: a case of PANCH tumour
Author(s): Leong Quah, Ute Pohl, Jonathan Pollock & James Ahlquist
Neurosurgery & Neuropathology, Queen's Hospital Romford and Endocrinology, Southend Hospital, Essex.

Case 4 – A challenging case of microTSHoma – the role of functional pituitary imaging
Author(s): Olympia Koulouri[1], Carla Moran[1], Alison Melvin[2], David Halsall[3], Nagui Antoun[4], Andrew Hoole[5], Heok Cheow[4], Sarah Heard[5], Dan Gillett[5], Richard Mannion[6], Krish Chatterjee[1], Mark Gurnell[1]
Institute of Metabolic Science[1] and Departments of Clinical Biochemistry[3] , Radiology[4] , Physics[5] and Neurosurgery[6], University of Cambridge & Addenbrooke's Hospital, Cambridge, UK. Department of Endocrinology, Bedford Hospital[2], UK

11:00 **Tea, Coffee and Posters**

11:30 **Pituitary papers that changed my practice (not on my CV)**
Chairs: Dr Stephanie Baldeweg and Mr Michael Powell

- Mr Nicholas Thomas, Consultant Neurosurgeon, London Bridge Hospital
- Prof Karim Meeran, Professor of Endocrinology, Imperial College, London
- Mr Richard Nelson, Consultant Neurosurgeon, Frenchay Hospital, Bristol
- Dr Niki Karavitaki, Consultant Endocrinologist, Churchill Hospital, Oxford

12:30 **Forum 2 – Case Presentations – Unusually aggressive pituitary cases**
Chairs: Dr James Ahlquist and Miss Joan Grieve

Case 5 – A leopard that changed its spots: ACTH dependent Cushing's in a patient with a previous history of an apoplectic non-functioning pituitary macroadenoma
Author(s): Aikaterini Theodoraki, Joan Grieve, Stephanie Baldeweg

Case 6 – The role of chemotherapy in the management of invasive, aggressive pituitary adenomas
Author(s): Surya Rajeev[1], Helen White[1], Daniel Cuthbertson[1], David Husband[2], Kumar Das[3], Catherine Gilkes[4] and Christina Daousi[1]
[1]Deparment of Endocrinology, University Hospital Aintree, Liverpool
[2]Department of Oncology, The Clatterbridge Cancer Centre, Liverpool
[3]Department of Neuroradiology, The Walton Centre for Neurology and Neurosurgery, Liverpool
[4]Department of Neurosurgery, The Walton Centre for Neurology and Neurosurgery, Liverpool

13:00 **Lunch and Posters**

14:00 **The Pituitary Foundation**

14:10 **Forum 3 – Case Presentations – Fertility in pituitary disease**
Chairs: Prof Gerard Conway

Case 7 – Experience of managing fertility in male patients with hypogonadotrophic hypogonadism at our institution; pitfalls and lessons learnt
Author(s): Dr K Gunganah, Dr Kaniseya Nadarasa, Professor Korbonits, Professor W M Drake, Dr S Akker

Case 8 – Hyperprolactinaemia and infertility
Author(s): Nazia Rashid, K Majumdar, SE Baldeweg
University College London Hospitals NHS Foundation Trusts, London

Case 9 – Subclinical acromegaly and fertility
Author(s): S Chakrabarti[1], E Hatfield[1], B Jones[2], A Mehta[2], N Mendoza[3], N Martin[1], K Meeran[1]
1. Imperial Centre for Endocrinology, Charing Cross Hospital, Imperial College Healthcare NHS Trust, 2. Department of Neuroradiology, Charing Cross Hospital, Imperial College Healthcare NHS Trust, 3. Department of Neurosurgery, Charing Cross Hospital, Imperial College Healthcare NHS Trust.

Case 10 – Risking Progression of Nelson's in Pregnancy: An update from the 10th Pituitary CPC 2008
Author(s): Allum M1, Mendoza N2, Roncaroli F3, Mehta A4, Peters D1, Jones B4, Hatfield EC1, Meeran K 1, Martin NM1
1 Imperial Centre for Endocrinology, 2 Department of Neurosurgery, 3 Department of Neurohistopathology, 4 Department of Neuroradiology Charing Cross Hospital, Imperial College Healthcare NHS Trust, London.

15:10 **Hypogonadotrophic hypogonadism**

- **In men**
  Dr Richard Quinton, Consultant Endocrinologist,
  Royal Victoria Infirmary, Newcastle
- **In women**
  Prof Gerard Conway, Consultant Endocrinologist,
  University College London Hospitals

15:40 **National Pituitary Surgery Audit**
Mr Richard Nelson, Consultant Neurosurgeon,
Frenchay Hospital, Bristol

15:55 **Tea, Coffee and Posters**

16:15 **Introduction:**
Miss Joan Grieve, Clinical Lead for Neurosurgery, The National Hospital for Neurology and Neurosurgery, London

Key note lecture:
iMRI
Prof Michael Buchfelder, Professor of Neurosurgery,
University of Erlangen-Nuremberg

17:00 **Forum 4 – Case Presentations – Radiotherapy in pituitary disease**
Chairs: Dr Stephanie Baldeweg and Dr Naomi Fersht

Case 11 – Can RapidArc® IMRT improve radiotherapy to pituitary tumours in terms of side effects and tumour coverage?
Author(s): Dr Richard Crossley, Melissa Hill, Dr Bernie Foran

Case 12 – Improving the accuracy of delivery of Rapidarc IMRT to pituitary tumours: an analysis of the UCH set-up margins and identifying whether there is a need to account for independent optic nerve motion
Author(s): Rachel Lewis, Syed A Moinuddin, Naomi Fersht
Department of Clinical Oncology, UCLH, 235 Euston Road, London NW1 2BU

Case 13 – Proton Beam therapy for a pituitary chondrosarcoma
Author(s): Vanessa Wilshaw[1], Bernadette Foran[1], John Newell-Price[2], Saurabh Sinha[3], Richard Crossley[1]
Depts of [1]Oncology, [2]Endocrinology, [3]Neurosurgery; Pituitary MDT, Royal Hallamshire Hospital, Sheffield Teaching Hospitals NHS Foundation Trust, Sheffield; UK

17:45 **Poster and presentation prizes**
Dr Stephanie Baldeweg, Consultant Endocrinologist
University College London Hospital

18:00 **Close**

We would be most grateful if you could please complete your evaluation form and hand it in to a member of the CFS Events team as you leave.

Thank you.

Oral Presentations

No:	Authors	Title:
Case 1	Angela Rogers, Wellcome Trust Clinical Training Fellow and John A. H. Wass, Professor of Endocrinology.	Successful pregnancy and headache treatment in a patient with acromegaly
Case 2	Dr B.L.Carpenter, Professor A Chu, Professor Russell-Jones	Multisystem Langerhans Cell Histiocytosis: Pituitary recovery post cladribine and 6-mercaptopurine
Case 3	Leong Quah, Ute Pohl, Jonathan Pollock & James Ahlquist Neurosurgery & Neuropathology, Queen's Hospital Romford and Endocrinology, Southend Hospital, Essex.	Acromegaly with GH-positive neuronal choristoma and pituitary adenoma: a case of PANCH tumour
Case 4	Olympia Koulouri[1], Carla Moran[1], Alison Melvin[2], David Halsall[3], Nagui Antoun[4], Andrew Hoole[5], Heok Cheow[4], Sarah Heard[5], Dan Gillett[5], Richard Mannion[6], Krish Chatterjee[1], Mark Gurnell[1] Institute of Metabolic Science[1] and Departments of Clinical Biochemistry[3] , Radiology[4] , Physics[5] and Neurosurgery[6], University of Cambridge & Addenbrooke's Hospital, Cambridge, UK Department of Endocrinology, Bedford hospital[2], UK	A challenging case of microTSHoma - the role of functional pituitary imaging
Case 5	Aikaterini Theodoraki, Joan Grieve, Stephanie Baldeweg	A leopard that changed its spots: ACTH dependent Cushing's in a patient with a previous history of an apoplectic non-functioning pituitary macroadenoma
Case 6	Surya Rajeev[1], Helen White[1], Daniel Cuthbertson[1], David Husband[2], Kumar Das[3], Catherine Gilkes[4] and Christina Daousi[1] [1]Deparment of Endocrinology, University Hospital Aintree, Liverpool [2]Department of Oncology, The Clatterbridge Cancer Centre, Liverpool [3]Department of Neuroradiology, The Walton Centre for Neurology and Neurosurgery, Liverpool [4]Department of Neurosurgery, The Walton Centre for Neurology and Neurosurgery, Liverpool	The role of chemotherapy in the management of invasive, aggressive pituitary adenomas

Case 7	Dr K Gunganah, Dr Kaniseya Nadarasa, Professor Korbonits, Professor W M Drake, Dr S Akker	Experience of managing fertility in male patients with hypogonadotrophic hypogonadism at our institution; pitfalls and lessons learnt
Case 8	Nazia Rashid, K Majumdar, SE Baldeweg University College London Hospitals NHS Foundation Trusts, London	Hyperprolactinaemia and infertility
Case 9	S Chakrabarti[1], E Hatfield[1], B Jones[2], A Mehta[2], N Mendoza[3], N Martin[1], K Meeran[1] 1. Imperial Centre for Endocrinology, Charing Cross Hospital, Imperial College Healthcare NHS Trust, 2. Department of Neuroradiology, Charing Cross Hospital, Imperial College Healthcare NHS Trust, 3. Department of Neurosurgery, Charing Cross Hospital, Imperial College Healthcare NHS Trust,	Subclinical acromegaly and fertility
Case 10	Allum M1, Mendoza N2, Roncaroli F3, Mehta A4, Peters D1, Jones B4, Hatfield EC1, Meeran K 1, Martin NM1 1 Imperial Centre for Endocrinology, 2 Department of Neurosurgery, 3 Department of Neurohistopathology, 4 Department of Neuroradiology Charing Cross Hospital, Imperial College Healthcare NHS Trust, London.	Risking Progression of Nelson's in Pregnancy: An update from the 10th Pituitary CPC 2008
Case 11	Dr Richard Crossley, Melissa Hill, Dr Bernie Foran	Can RapidArc® IMRT improve radiotherapy to pituitary tumours in terms of side effects and tumour coverage?
Case 12	Rachel Lewis, Syed A Moinuddin, Naomi Fersht Department of Clinical Oncology, UCLH, 235 Euston Road, London NW1 2BU	Improving the accuracy of delivery of Rapidarc IMRT to pituitary tumours: an analysis of the UCH set-up margins and identifying whether there is a need to account for independent optic nerve motion
Case 13	Vanessa Wilshaw[1], Bernadette Foran[1], John Newell-Price[2], Saurabh Sinha[3], Richard Crossley[1] Depts of [1]Oncology, [2]Endocrinology, [3]Neurosurgery; Pituitary MDT, Royal Hallamshire Hospital, Sheffield Teaching Hospitals NHS Foundation Trust, Sheffield; UK	Proton Beam therapy for a pituitary chondrosarcoma

Poster Presentations

No:	Authors	Title:
Poster 1	Allum M1; Falinska A1; Todd JF1 1Imperial Centre for Endocrinology, Imperial College Healthcare NHS Trust , Hammersmith Hospital, Du Cane Road , London UK	An Unusual Case of Severe ACTH-dependent Cushing's Syndrome with Spontaneous Resolution and Sustained Remission
Poster 2	Dr Muhammad Asam (endocrine registrar) ,Dr Andy James (Consultant endocrinologist) both from Royal victoria infirmary Newcastle upon Tyne	Two important overlapping tumors in pituitary fossa
Poster 3	Ms. Jo Burgin, Dr. Seshadri Pramodh, Dr. Alex Bickerton	Late growth spurt: a rare presentation of a co-secreting pituitary tumour
Poster 4	Dr Bernadette Carpenter, Dr Anna Gateley, Dr Victoria Hordern, Dr Roselle Herring, Professor David Russell-Jones.	Craniopharyngioma: To operate or not to operate?
Poster 5	S Chakrabarti[1], B Jones[2], A Mehta[2], N Mendoza[3], N Martin[1], F Roncaroli[4], K Meeran[1] 1. Imperial Centre for Endocrinology, Charing Cross Hospital, Imperial College Healthcare NHS Trust, 2. Department of Neuroradiology, Charing Cross Hospital, Imperial College Healthcare NHS Trust, 3. Department of Neurosurgery, Charing Cross Hospital, Imperial College Healthcare NHS Trust, 4. Department of Neurohistopathology, Charing Cross Hospital, Imperial College Healthcare NHS Trust	Treatment and follow up of incidentally discovered acromegaly
Poster 6	A Falinska; R Tanday; JF Todd Imperial Centre of Endocrinology, Imperial College Healthcare NHS Trust Hammersmith Hospital	Resolution of diabetes insipidus following Sodium cromoglicate
Poster 7	Jeyaraman, K[1], Kaushal, R[1] [1]Department of Endocrinology, West Middlesex Hospital	Pituitary involvement in CLL
Poster 8	K Jeyaraman[1], S Zac-Varghese[1], B Jones[3], A Mehta[3], F Roncaroli[4], A Sandison[4], J Evanson[6], P Clarke[2], M Korbonits[5], N Martin[1] [1] Imperial Centre for Endocrinology, [2]Department of Head and Neck surgery, [3]Department of Neuroradiology, [4] Department of Neuropathology, Charing Cross Hospital, Imperial College Healthcare NHS Trust, Fulham Palace Rd, London W6 8RF [5] Department of Endocrinology, [6] Department of Neuroradiology, Barts and The London School of Medicine and Dentistry, Turner Street, London E1 2AD	Ectopic Prolactinoma - a case report and literature review

Poster 9	Jeyaraman, K1, Zac-Varghese, S1, Meegan, J1, Mendoza, N2, Mehta, A3, Roncaroli, F4, A Sandison4, W Grant5, Hatfield E1,Martin, N1, K Meeran1 1 Imperial Centre for Endocrinology, 2 Department of Neurosurgery, 3 Department of Neuroradiology, 4 Department of Neurohistopathology. 5 Department of Ear,nose and throat surgery, Charing Cross Hospital, Imperial College Healthcare NHS Trust, London.	Sphenoid Sinus Mucocele - An unusual mimic of a pituitary adenoma
Poster 10	Ahilan Kailaya-Vasan, Simon Carr, Showkat Mirza, Saurabh Sinha	We Love the '80s
Poster 11	L.Krishnan, S.Nada and A.M. Jennings	ACTH dependent Cushing's Syndrome: Nasal carcinoid or ectopic pituitary adenoma?
Poster 12	Y Ling, T Vakilgilani, A Mehta, B Jones, JF Todd Imperial Centre of Endocrinology, Imperial College Healthcare NHS Trust, Hammersmith Hospital	A case of memory impairment in Langerhans cell histiocytosis
Poster 13	Mohamed Ashif Majeed (1), John Clark (2) (please put the lead Author's name first) 1.StR Endocrinology , 2. Consultant Endocrinologist West Suffolk Hospital	Pituitary Mass In A Patient With Failing Vision And Breast Cancer
Poster 14	Kalpita Majumdar, Stephanie Baldeweg University College London Hospital	Unwell after acromegaly treatment – one pathology or more?
Poster 15	Author(s): Alexander Miras, Nicola Neary, Brynmor Jones, Amrish Mehta, Nigel Mendoza, Rami Fikri, Emma Hatfield, Niamh Martin, Karim Meeran	How many haemorrhages can a pituitary take?
Poster 16	Gideon Mlawa ,Eswari Chinnasamy, Darshi Sivakumaran and Gul Bano St Georges George`s Hospital, Thomas Addison Unit-London	Diabetes Inspidus Post Traumatic Head Injury
Poster 17	Dr V Oguntolu, H N Buch, A Viswanath, B M Singh	Incidental suprasellar meningioma in a patient with severe mental health disorder: What is the best management approach?
Poster 18	A S Powlson[1], N Antoun[2], J Tysome[3], R J Mannion[4], J D Pickard[4], M Gurnell[1] [1]Metabolic Research Laboratories, Institute of Metabolic Science, [2]Department of Radiology, [3]Department of Otolaryngology [4]Department of Neurosurgery, University of Cambridge & Addenbrooke's Hospital, Cambridge, CB2 0QQ, UK	Sibling apoplexy; lightning strikes twice
Poster 19	Nazia Rashid1, J Grieve2, SE Baldeweg1 1Department of Endocrinology University College London Hospital NHS Foundation Trust, London 2 The National Hospital for Neurology and Neurosurgery, Queen Square, London	Giant Macroprolactinoma

Poster 20	D Sivakumaran[1], G Mlawa[1], G Bano[1] Thomas Addison Unit, St George's Hospital, Blackshaw Road, London, SW17 0QT	An unusual Cause of Hypopituitarism
Poster 21	Tanday R, Falinska A, Mehta A, Jones B, Todd JF Imperial Centre of Endocrinology, Imperial College Healthcare NHS Trust, Hammersmith Hospital	Macroadenoma from Malopolska
Poster 22	Vakilgilani T, Ling Y, Todd JF, Meeran K Imperial centre of Endocrinology, Imperial college healthcare NHS trust Hammersmith Hospital	A case of cyclical Cushing's syndrome for 10 years
Poster 23	Zac-Varghese, S[1]., Jeyaraman, K[1]., Papadopoulou, D[1]., Jones, B[3]., Mehta, A[3]., Roncaroli, F[4]., Martin, N[1]., Hatfield, E[1]., Clarke, P[5]., K. Meeran[1], Mendoza, N[2] *1 Imperial Centre for Endocrinology, 2 Department of Neurosurgery 3 Department of Neuroradiology, 4 Department of Neuropathology, 5 Department of ENT surgery Charing Cross Hospital, Imperial College Healthcare NHS Trust, London.*	A stuffy nose
Poster 24	Zac-Varghese, S1., Papadopoulou, D1., Jeyaraman, K1., Jones, B3., Mehta, A3., Roncaroli, F4., Martin, N1., Hatfield, E1., Mendoza, N2., Bridges, N5., K. Meeran1 1 Imperial Centre for Endocrinology, 2 Department of Neurosurgery 3 Department of Neuroradiology, 4 Department of Neuropathology Charing Cross Hospital, Imperial College Healthcare NHS Trust, London. 5 Chelsea and Westminster Hospital, London.	Two cases of hypogonadism following transphenoidal pituitary surgery and induction of fertility
Poster 25	Zac-Varghese, S[1]., Jeyaraman, K[1]., Papadopoulou, D[1]., Cooke, R[1]., Nair, R[2]., Mehta, A[3]., Jones, B[3]., Roncaroli, F[4] ., Martin, N.[1], Hatfield, E[1]., Meeran, K[1] *1 Imperial Centre for Endocrinology, 2 Department of Neurosurgery 3 Department of Neuroradiology, 4 Department of Neuropathology Charing Cross Hospital, Imperial College Healthcare NHS Trust, London.*	Triphasic response following expanded endonasal transphenoidal surgery for sellar/suprasellar tumour

Oral Presentation Abstracts

Case 1

Title: Successful pregnancy and headache treatment in a patient with acromegaly.

Author(s): Angela Rogers, Wellcome Trust Clinical Training Fellow and John A. H. Wass, Professor of Endocrinology.

A 30 year old female presented with acromegaly in October 2011. She had mild clinical disease but experienced headache. Her growth hormone levels preoperatively were mean 62 mU/L, IGF-1 323 (normal range up to 186 ng/ml). The preoperative scan showed macroadenoma with cavernous sinus invasion on the left. She underwent endoscopic trans-sphenoidal pituitary surgery in December 2011 and post operatively her growth hormone levels were lower at 24 mU/L and the scan at three months showed residual tumour in the cavernous sinus.

She became pregnant six months later and within a short period of time began to experience worsening headaches, these became severe and so it was decided to start octreotide by subcutaneous injection. This considerably helped with the headaches but did not obviate them. As is characteristic in these circumstances the octreotide (100mcg subcutaneously) alleviated the headaches for an hour or two. Eventually the patient was taking this every three or four hours throughout the twenty-four hour period. However although the headaches were improved they remained considerable. At this stage therefore Bromocriptine was added in a dose of 10mg a day to reduce the lactotroph hyperplasia in the pituitary and thereby hopefully to improve the headaches albeit that the primary tumour was not prolactin secreting. This had the desired effect and a normal foetus was born at 38 weeks. Postpartum the Bromocriptine was stopped and the octreotide continued. This helped the headaches albeit that the acromegaly persisted despite regular octreotide treatment (growth hormone 15mU/L).

Conclusion:
This case demonstrates the successful treatment of headaches through pregnancy using octreotide and Bromocriptine. Octreotide can be used in pregnancy and is well known to alleviate headaches in acromegaly also in non-pregnant patients.

Case 2

Title: Multisystem Langerhans Cell Histiocytosis: Pituitary recovery post cladribine and 6-mercaptopurine.

Author(s): Dr B.L.Carpenter, Professor A Chu, Professor Russell-Jones.

Introduction:

Langerhans Cell Histiocytosis (LCH) is a rare disease in adults with an estimated prevalence of 1-2 cases per million and typically affects bone, lungs, pituitary or skin. Pituitary manifestations include failure of the anterior and/or posterior pituitary, with diabetes insipidus and gonadotrophin deficiency being most prevalent (1). Although radiotherapy has been shown to reduce pituitary LCH, pituitary dysfunction is considered permanent (2). We describe a case of post cladribine and 6-Mercaptopurine pituitary recovery.

Case Description:

A 24 year old caucasian female presented with diabetes insipidus and subsequently developed secondary amenorrhoea with progressive pituitary failure, which was treated with replacement hormone therapy. She later developed respiratory complications and a CT thorax demonstrated a fine reticular pattern affecting the mid and upper zones. She had a video-assisted lung biopsy, which identified histiocytic cells, stellate scarring and positive immunohistochemical stains for S100 and CD1a. She was diagnosed with multisystem LCH with lung and pituitary involvement and advised to stop smoking. Due to increasing respiratory dysfunction she was treated with 4 cycles of chlorodeoxyadenosine (cladribine or 2-CDA), a purine analogue which is toxic to monocytes and penetrates the CNS. This was followed with 1 year of 6-Mercaptopurine. At follow-up she stated 4 periods post treatment with a 10 year history of amenorrhoea and lung function tests had stabilised.

Discussion:

This case is unique in demonstrating clinical pituitary recovery post cladribine and 6-mercaptopurine in a patient with panhypopituitarism caused by LCH.

References:

1. M. Aricò et al, Langerhans cell histiocytosis in adults Report from the International Registry of the Histiocyte Society, European Journal of Cancer, Volume 39, Issue 16, November 2003, Pages 2341-2348.
2. Kaltsas GA et al. Hypothalamo-pituitary abnormalities in adult patients with Langerhans cell histiocytosis: clinical, endocrinological, and radiological features and response to treatment. J Clin Endocrinol Metab 2000;85:1370-6.

Case 3

Title: Acromegaly with GH-positive neuronal choristoma and pituitary adenoma: a case of PANCH tumour

Author(s): Leong Quah, Ute Pohl, Jonathan Pollock & James Ahlquist
Neurosurgery & Neuropathology, Queen's Hospital Romford and
Endocrinology, Southend Hospital, Essex.

Pituitary Adenoma with Neuronal Choristoma (PANCH tumour), is a rare form of pituitary pathology composed of a mixed pituitary adenoma/gangliocytoma. We describe a patient with acromegaly who had evidence of growth hormone synthesis in the neuronal component of a PANCH tumour.

A 55 year old woman was found to have facial features of acromegaly, confirmed biochemically: basal GH 13.56 ng/mL, GTT nadir 8.87 ng/mL, IGF1 97.2 nmol/L (9-40). Pituitary function was otherwise normal. MRI revealed a 25 mm mass in the pituitary fossa, with cavernous sinus extension; there were no unusual radiological features. She underwent endoscopic trans-sphenoidal surgery, during which the tumour was noted to have an unusual, slightly fibrous consistency. Intra-operative histological examination suggested that the tumour may be a ganglioglioma. Following surgery pituitary function remained intact: formal endocrine reassessment is awaited.

Histological examination of the tumour revealed islands of pituitary adenoma embedded in a neuropil substrate made up of ganglion-like cells, some of which showed dysplastic features (cytomegaly, binucleation and dysmorphism). Ganglion cells were embedded within both the adenoma and the neuroglial tissue. Immunohistochemistry confirmed that the adenoma cells were positive for GH & prolactin, with scattered cells positive for TSH also. In addition, a subpopulation of the ganglion cells also showed strong staining for prolactin, and weak staining for GH, TSH and ACTH. Both pituitary adenoma and ganglion cells were strongly positive for synaptophysin; chromogranin only stained rare small cells. Glial fibrillary acid protein staining was generally negative throughout. The neural tissue features were described as typical of a neuronal choristoma.

The combination of a pituitary adenoma with neuronal choristoma (PANCH) is a very rare form of pituitary pathology. The finding of neuronal tissue expressing GH and prolactin in association with a somatotroph adenoma is intriguing. The pathogenesis of this phenomenon is not clear. The combination of two distinct GH & prolactin-positive cell types, pituitary adenoma and gangliocytoma, occurring within a single tumour, suggests that the neuronal cell population may have arisen as a result of neuronal differentiation within a pituitary adenoma.

Case 4

Title: A challenging case of microTSHoma - the role of functional pituitary imaging.

Author(s): Olympia Koulouri[1], Carla Moran[1], Alison Melvin[2], David Halsall[3], Nagui Antoun[4], Andrew Hoole[5], Heok Cheow[4], Sarah Heard[5], Dan Gillett[5], Richard Mannion[6], Krish Chatterjee[1], Mark Gurnell[1]

Institute of Metabolic Science[1] and Departments of Clinical Biochemistry[3] , Radiology[4] , Physics[5] and Neurosurgery[6], University of Cambridge & Addenbrooke's Hospital, Cambridge, UK
Department of Endocrinology, Bedford hospital[2], UK

A 57 year old female was referred with hyperthyroxinaemia [FT4 23.5 pmol/l (RR 10-19.8)] and inappropriately raised TSH [TSH 7.7 mIU/L (RR 0.35-5.5)]. She had been given a putative diagnosis of primary hypothyroidism 6 years earlier, on the basis of an elevated TSH. However, administration of levothyroxine (up to 200mcg/day) failed to suppress TSH, despite FT4 concentrations reaching 35 pmol/L.

After excluding assay interference, the diagnosis was narrowed down to either TSHoma or Resistance to thyroid hormone (RTH). Biochemical/dynamic investigations aimed at discriminating between the two conditions were inconclusive. Serial conventional pituitary MRIs were normal, but on a single occasion showed suspicion for a small left sided microadenoma. Volume MRI was concordant with this finding and ^{11}C-Methionine PET/CT demonstrated an area of increased tracer uptake on the left side of the sella.

A trial of somatostatin analogue therapy was undertaken for both diagnostic and therapeutic purposes. This achieved TSH and FT4 suppression to such degree (TSH 0.57 mIU/L, FT4 8.9 pmol/l), that replacement with levothyroxine was commenced as part of a 'block and replace' regimen. The left sided hot-spot previously detected on PET/CT disappeared and the micro-lesion on volume MRI became barely visible.

Unfortunately, the patient developed gastrointestinal side effects and mild hyperglycaemia, and treatment was discontinued. Following this, thyrotoxicosis gradually reappeared again over several months (TSH 12.4 mIU/L, FT4 20.3 pmol/l, FT3 7.3 pmol/l). Repeat conventional MRI appearances remained 'normal'. However, volume MRI and PET/CT revealed the same findings as previously detected at baseline (left sided 'active' microadenoma).

The patient has been offered transsphenoidal exploration for her presumed microTSHoma. She is currently asymptomatic on adequate beta-blockade.

Questions to the panel:

1. How secure is the diagnosis at this stage?
2. Is TSS a reasonable next step?
3. Could functional pituitary imaging find a role in similar challenging cases?

Case 5

Title: A leopard that changed its spots: ACTH dependent Cushing's in a patient with a previous history of an apoplectic non-functioning pituitary macroadenoma

Author(s): Aikaterini Theodoraki, Joan Grieve, Stephanie Baldeweg

Introduction: ACTH dependent Cushing's with marked hypertension and hypokalaemia usually characterises Cushing's due to ectopic ACTH secretion. We present a patient who was initially diagnosed with an apopleptic non-functioning pituitary macroadenoma, and subsequently developed florid Cushing's with marked hypertension and hypokalaemia due to Cushing's disease.

Case Presentation: An 81 year old lady was admitted with one week history of double vision, headache, and nausea and vomiting in August 2012. She had a background history of primary hypothyroidism, peptic ulcer disease and melanoma excision. On examination partial 3rd cranial nerve palsy was present, on admission prolactin was raised at 3126mIU/L, gonadotrophins were suppressed and 9am cortisol was 160nmol/L. Pituitary MRI scan showed a pituitary macroadenoma with right parasellar and suprasellar extension, recent haemorrhage and optic chiasm displacement. She underwent pituitary hypophysectomy, and the histology showed a null cell adenoma. Glucagon stimulation testing 4months after the operation showed adequate cortisol reserve.

Ten months later the patient presented with a three month history of rapidly worsening leg weakness, facial plethora, weight gain, and a new diagnosis of hypertension and hyporkalaemia (K 2.6mmol/L). Investigations showed ACTH dependent Cushing's: 24 hr urine cortisol output was 2566nmol/24h, 9am cortisol was 877nmol/L, post dexamethasone cortisol 682nmol/L, midnight cortisol 1043nmol/L and ACTH 90.3ng/L. A pituitary MRI scan showed stable appearance of the pituitary disease. PET and gallium dotatate scans showed no ectopic source of ACTH. Inferior petrosal sinus sampling revealed a ratio of 15:1 after CRH stimulation on the right, and a ratio of 4.7:1 on the left. Repeat staining of pituitary tissue from the initial operation showed no ACTH staining. The patient was commenced on metyrapone and hydrocortisone was added once cortisol was controlled, with partial symptomatic improvement.

Conclusion: Hypertension and hypokalaemia can occur in pituitary dependent Cushing's. Cushing's disease developing in a patient with a non-functioning pituitary adenoma suggests a change in the behaviour of the tumour.

Questions to the panel:

1. What treatment would the panel suggest next?
2. Could the absence of ACTH staining in tissue harvested during the first operation be due to the apopleptic event?

Case 6

Title: The role of chemotherapy in the management of invasive, aggressive pituitary adenomas

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Invasive, aggressive pituitary adenomas are very rare but their management is extremely challenging requiring combined treatment strategies.

Case 1: A 34 year old lady presented in 1997 with acromegaly and acute pituitary apoplexy and underwent emergency transsphenoidal surgery (TSS) followed by radiotherapy and treatment with bromocriptine and long-acting somatostatin analogue (SSA). After 9 years of static residual adenoma on serial pituitary MRIs, she experienced several massive, symptomatic recurrences and underwent repeat TSS followed by three debulking craniotomies for massive tumour recurrence and severe brainstem compression. In 2009 she received a 12-month course of temozolomide 150 mg/kg/m2 given for 5 days in 28-day cycles, with a favourable radiological and clinical response which were maintained for 8 months after discontinuation of treatment. Tumour progression was initially treated with re-introduction of temozolomide for a further 3 months with no clinically meaningful response, followed by 3-month treatment with CCNU with an unfavourable response. Third line chemotherapy was prescribed in the form of oral etoposide 50 mg BD for 10 days in monthly cycles. She has received so far 23 cycles of etoposide which have been very well tolerated and accompanied but radiological tumour size reduction; biochemical control though of her acromegaly has not yet been achieved.

Case 2: A 40 year old female presented in 1996 with apoplexy and underwent TSS which confirmed the presence of a somatotroph adenoma. She received fractionated XRT in 1997 and stereotactic radiosurgery in 2003 because of ongoing disease activity. Further tumour progression and biochemical relapse of her acromegaly was treated with craniotomy in 2008 along with maximum doses of long acting SSA and dopamine agonists. She failed to respond to a 3 month trial of temozolomide with continuing tumour growth and worsening of her epilepsy and was then switched to CCNU regime. She has tolerated 9 cycles of this so far with good radiological response and improvement of her seizure control and function.
These two cases illustrate that optimal management of aggressive pituitary adenomas that fail to respond to multimodality treatment remains challenging. Because of the rarity of these tumours, the place of conventional and newer chemotherapeutic agents in their management can only be determined through multi-centre collaboration with data and experience exchange and longitudinal follow up.

Case 7

Title: Experience of managing fertility in male patients with hypogonadotrophic hypogonadism at our institution; pitfalls and lessons learnt

Author(s): Dr K Gunganah, Dr Kaniseya Nadarasa, Professor Korbonits, Professor W M Drake, Dr S Akker

Male hypogonodotrophic hypogonadism accounts for less than 1% of male infertility[1]. The success rate of inducing spermatogenesis and subsequent fertility with gonadotrophin replacement therapy is high[2]. Despite being highly effective and NICE approved since 2004[3] multiple logistical factors stand in the way of patient access to treatment.

We present 12 male patients with post-pubertal hypogonadotrophic hypogonadism from all causes. We have looked at the success rate of spermatogenesis, spontaneous conception, duration of treatment, type of treatment used, factors influencing fertility success rate as well as factors affecting access to treatment.

Out of the 12 patients, 4 had treated pituitary tumours, 4 had idiopathic hypogonadotrophic hypogonadism, 2 had lymphocytic hypophysitis, 1 had Kallman's syndrome and 1 had haemochromatosis. 83% of our patients had either successful conceptions and or spermatogenesis. Factors affecting chances of spermatogenesis and fertility included partner fertility issues, systemic conditions such as sarcoidosis and poor compliance to treatment. Factors affecting access to treatment included delay in referral, lack of local knowledge and experience in managing male infertility in hypogonadotrophic hypogonadism, cost of treatment and significant delays in funding approval from Comissioning groups in primary care.

We would like to present our case series in more detail as well as our protocol based on available evidence in the literature and our experience from the case series.

References:

1. Comhaire FH, de Kretser D, Farley TMM. Towards more objectivity in the management of male infertility. The need for a standardized approach. Int J Androl 1987;10:1–2.
2. Liu PY, Handerlsman J. The present and future state of hormone treatment for male infertility. Human Reproduction Update. 2003;9:9-23
3. NICE guidelines. Fertlity: assessment and treatment for people with fertility problems. February 2013

Case 8

Title: Hyperprolactinaemia and infertility

Author(s): ***Nazia Rashid, K Majumdar, SE Baldeweg***
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Introduction

Hyperprolactinaemia can be caused be variety of conditions including physiological (Pregnancy, breast feeding), pathological (prolactinomas) and functional (drugs such as antiemetics, antipsychotics and illicit). Periodic, heavy, and regular marijuana use can also cause clinically significant Hyperprolactinaemia over the long term.[1]

Hyperprolactinaemia in young women commonly manifest through amenorrhea and infertility. Fertility may be promoted with long-term use of dopaminergic agonists. The normalized PRL level allows the occurrence of spontaneous ovulatory cycles or the normalization of the defective luteal phase.[2]

Case History

19 years old female was found to have an incidental pituitary fossa tumour detected on CT head done after an alleged assault. She had no symptoms of headache or visual field problems but had long standing issues with secondary Amenorrhea. She also has history of excessive Marijuana use. She expressed her wish to get pregnant in recent future.

Visual fields were normal on examination.

Pituitary profile suggested Hyperprolactinaemia with levels at 1437 mIU/l however, the monomeric Prolactin after discarding the normal Macroprolactin level was only 204 mIU/L. Rest of Pituitary profile was unremarkable. FSH 11 iu/L, LH 77.2 iu/L, cortisol 451 nmol/L, IGF-1 28.9 nmol/L, Oestradiol 253 pmol/L, TSH 2.03 mIU/L, and T4 16.0 pmol/L. Subsequent Pituitary MRI showed enlarged pituitary gland with convex upper border abutting the optic nerves immediately before they join to form the optic chiasm. There was an impression of

< 1cm adenoma in the left side of anterior pituitary gland.

Recent Prolactin levels from December 2013 are 3400 miu/L with bioactive Prolactin of

690 miu/L.

Questions to panel:

1) What is the cause of Hyperprolactinaemia in this case?
2) What is cause of amenorrhea? Hyperprolactinaemia or PCO's
3) What treatment should be given to improve fertility? Cabergoline or Clomiphene

[1]The Endocrinologist. Ali A Rizvi (Impact Factor: 0.12). 10/2006; 16(6):308-310
[2]Middle East Fertility Society Journal, Volume 17, Issue 2 , Pages 63-69, June 2012

Case 9

Title: Subclinical acromegaly and fertility

Author(s): ***S Chakrabarti[1], E Hatfield[1], B Jones[2], A Mehta[2], N Mendoza[3], N Martin[1], K Meeran[1]***

1. Imperial Centre for Endocrinology, Charing Cross Hospital, Imperial College Healthcare NHS Trust, 2. Department of Neuroradiology, Charing Cross Hospital, Imperial College Healthcare NHS Trust, 3. Department of Neurosurgery, Charing Cross Hospital, Imperial College Healthcare NHS Trust,

A 40 year old lady presented to the neurologists with migraine. As part of her investigations, an MRI was performed, which revealed a 1.2 cm pituitary lesion, in contact with, but not compressing the left optic nerve.

On referral to our Endocrinology Unit, she had no symptoms of pituitary dysfunction. She had a previous history of hypertension treated with alpha methyldopa and impaired fasting glucose. She had regular menstrual periods although the cycle had shortened from 28 to 22-25 days over the past five months. Day 21 progesterone measurement indicated that she was ovulating. Prolactin was marginally raised at 681mU/L (NR 100-500 mU/L), with negative macroprolactin, but there were no other symptoms of hyperprolactinemia. IGF-1 was elevated at 52.9 mcg/L (NR 13-50mcg/L). She had no acromegalic symptoms. Remaining pituitary blood tests were normal, as was an HbA1C (31mmol/mol).

Dynamic testing revealed failure of growth hormone (GH) to adequately suppress, confirming acromegaly.

Time	**Glucose**	**GH (mcg/l)**
0 min	7.5	3.08
30 min	4.6	2.06
60 min	5.4	1.81
90 min	6.6	1.67
120 min	5.9	1.75

She had no visual field defect on formal perimetry.

This patient's current priority is fertility. She has regular ovulatory cycles, which could be compromised by pituitary surgery, and she has no current visual compromise. We have therefore chosen to medically manage her acromegaly, and she has been started on cabergoline 250mcg weekly to try and normalise prolactin and GH levels. Our plan is that she will stop cabergoline once pregnant, with the intention that it could be restarted in pregnancy if visual fields become compromised.

Our questions for the expert panel are:
Would you recommend this patient has pituitary surgery before trying for a pregnancy?
How should her acromegaly be managed, if at all, if she becomes pregnant without having pituitary surgery?

Case 10

Risking Progression of Nelson's in Pregnancy: An update from the 10th Pituitary CPC 2008

Author(s): Allum M[1], Mendoza N[2], Roncaroli F[3], Mehta A[4], Peters D[1], Jones B[4], Hatfield EC[1], Meeran K [1], Martin NM[1]

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A 21-year-old woman was referred in 2003 for investigation of possible Cushing's syndrome. ACTH-dependent Cushing's syndrome was confirmed on low dose dexamethasone suppression testing (LDDST) and inferior petrosal sinus sampling excluded an ectopic source of ACTH. Pituitary MRI was normal. Patient underwent trans-sphenoidal surgery (TSS) in Feb 2005. Histology confirmed a corticotroph adenoma. 18 months later she had recurrence of symptoms and LDDST confirmed recurrent Cushing's. Pituitary MRI showed a 4mm central cystic pituitary lesion. The patient elected to have bilateral adrenalectomy to preserve fertility. Plasma ACTH prior to adrenalectomy was 85.7 ng/L. Repeat MRI August 2007 showed an increase in size of the cystic pituitary lesion to 6mm. The patient then fell pregnant. Pituitary MRI in the second trimester did not show increase in size of the lesion and visual fields remained normal. The case was discussed at the 10thCPC in 2008 regarding the risks of the patient developing future Nelson's syndrome. The opinion of the expert panel was that she should have external beam radiotherapy following delivery, as there was significant risk of the lesion growing rapidly. Unfortunately, the baby was stillborn following premature rupture of membranes at 35 weeks gestation. We opted for a conservative approach to her management to optimise her chances of a future successful pregnancy.

The patient was monitored regularly with ACTH levels and pituitary MRIs. There was no change in the pituitary lesion on MRI. ACTH levels two hours post-hydrocortisone have remained stable at around 50ng/L. The patient became pregnant again in August 2011 and subsequently had an uncomplicated delivery of a healthy baby boy.

Our questions for the expert panel are:

- With hindsight, was pituitary radiotherapy advisable?
- Is a future conservative approach to her management reasonable?

Case 11

Title: Can RapidArc® IMRT improve radiotherapy to pituitary tumours in terms of side effects and tumour coverage?

Author(s): Dr Richard Crossley, Melissa Hill, Dr Bernie Foran

BACKGROUND:
Radiotherapy is used to control pituitary tumours especially where there is optic chiasm compression and a threat to vision. However, long term side effects in patients with benign disease undergoing therapy are a concern. RapidArc® Volumetric arc therapy (VMAT) is a type of rotational intensity modulated radiotherapy (IMRT) used in malignant tumours to reduce dose to organs at risk (OARs) and improve dose and coverage of the target tumour. Could this improve outcomes and toxicity in pituitary tumours treated with radiotherapy?

OBJECTIVE:
Demonstrate an improvement in the dose distribution to the pituitary target volume, whilst simultaneously showing an minimisation of dose to surrounding organ at risk by utilising RapidArc® VMAT.

METHODS:
We compared radiotherapy treatment plans for pituitary tumours before and after the establishment of RapidArc® planning. Treatment volumes, beam arrangements with colourwashes and dose-volume histograms were analysed for comparison.

RESULTS:
Planning with RapidArc® as compared to conventionally planned conformal radiotherapy results in a reduction in the area treated to higher doses, but also results in a larger area receiving a lower dose of radiation. The doses to the temporal lobes are reduced by using RapidArc®.
The dose distribution and homogeneity across the PTV was significantly better with RapidArc® as compared to conventionally planned radiotherapy.

CONCLUSIONS:
RapidArc® is a modern IMRT technique that results in better coverage of the tumour target and reduced doses to the OARs when treating pituitary tumours. Longer term follow up within a trial setting would be needed to confirm the improvements in long term outcomes and toxicity.

Case 12

Title: Improving the accuracy of delivery of Rapidarc IMRT to pituitary tumours: an analysis of the UCH set-up margins and identifying whether there is a need to account for independent optic nerve motion.

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Introduction: Radiotherapy to pituitary tumours at UCLH has been delivered with rapidarc IMRT for the last 4 years. This has enabled a dose distribution which has better confomality to the tumour and allows constraints to be set which avoid exceeding the tolerance of adjacent normal structures. As the dose distribution is so precise, it is vital that any variation between the planning scan and the executed treatment (error) is anticipated and accounted for. When treating a benign tumour, the avoidance of late effects is especially important.
The majority of pituitary tumours at UCLH are treated with 50.4Gy/28# (1.8Gy/#); a dose which is adequate to treat macroscopic disease. In our centre, the tolerance for optic chiasm and optic nerves is 50Gy at 2Gy/# (almost equivalent to the treatment dose when taking into account dose per fraction) when treating benign tumours. Therefore small errors can have a great impact on the optic apparatus.

Error can occur in a number of ways: volume delineation uncertainty (both due to imaging co-registration and inter-observer variability), internal organ movement and set-up error. When voluming the tumour and organs at risk, the GTV (gross tumour volume) is outlined using fused MRI as a guide. This is then expanded to the CTV (clinical target volume - to incorporate and likely areas of microscopic disease eg. the cavernous sinuses), a further PTV (planned target volume) margin is added to allow for set-up error and internal organ motion. A PRV (planned organ at risk volume) margin is added to organs at risk.

Current practice involves immobilising patients with a thermoplastic head shell to minimised set-up error. Patients are imaged daily with kv imaging, and after the first 3 days are shifted to account for any systematic error. On subsequent days they are shifted to zero before treatment after kv imaging. They also have a weekly cone beam CT to judge 3D set-up error and soft tissue matching, but are not shifted as a result.

At present the PTV margin for the pituitary is 3mm and the PRV margin for the organs at risk is also 3mm. This study aims to establish whether these margins are adequate or excessive, and also aims to investigate whether the optic nerve movement is significant, independent of set-up error.

Method: The clinical imaging database was searched to identify patients with brain tumours whose were treated with rapidarc IMRT with a thermoplastic shell and daily kv imaging between 2007 and 2012. Systematic and random errors were calculated and margins postulated. Within this group, patients with pituitary tumours were identified, and these patients had all also had weekly cone beam CT. These latter patients were reviewed to see whether optic nerve motion was significant, independent of bone set-up, and whether a PRV of 3mm was acceptable. The dose distribution to different PRV margins on the optic nerves was also calculated.

Results: 107 patients were identified with suitable kV imaging (3235 images) with a subset of 30 patients (169 images) with skull base lesions. 7 of these had pituitary tumours. When all the image sessions were used to calculate the margins, they were found to be 2.1mm, 2.5mm and 2.4mm in the vertical, longitudinal and lateral directions. When the first three kV image sessions alone were used, the margins were found to be 2.9mm, 4.0mm and 3.3mm in the vertical, longitudinal and lateral directions. When all kV images sessions were used to calculate the margins but the data from #4- was amended to take into account #1-3 systematic error adjustment, they were found to be 3.6mm, 4.5mm and 4.1mm in the vertical, longitudinal and lateral directions. Mean residual CBCT bone error was found to be 0.0mm (+/-0.7), -0.3mm (+/-1.0), 0.0mm(+/-0.9) in the vertical, longitudinal and lateral directions and 0.04deg.(+/-0.75), 0.13deg.(+/-1.08) and -0.15 (+/-0.95) in Pitch, Roll and Rotation.

Conclusion: With new, precise planning technology and delivery, the minimisation of error is paramount. The UCLH data shows that the 3mm PTV for pituitary tumours, with current immobilisation and imaging protocols is adequate. However, the optic nerves move independently to bone and our work will establish whether a PRV of 3mm is adequate, and whether additional interventions are be necessary to minimised optic nerve movement, for example fixing the patients' gaze.

Case 13

Title: Proton Beam therapy for a pituitary chondrosarcoma

Author(s): Vanessa Wilshaw[1], Bernadette Foran[1], John Newell-Price[2], Saurabh Sinha[3], Richard Crossley[1]
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Pituitary sarcomas are a rare tumour entity. They present complex management problems due to the location and surrounding vital structures. Evidence supports bi-modality treatment with surgery and radiotherapy. Here we discuss a 40-yr-old patient who presented with amenorrhoea and galactorrhoea. MRI revealed a 30mm pituitary lesion. She was treated with cabergoline and surgery was recommended. The patient opted for radiological surveillance and this continued for 3 yrs when the mass increased in size and caused opthalmoplegia due to a 4th and 6th nerve palsy. The patient underwent debulking surgery and histology revealed a grade 2 chondrosarcoma. The patient was referred for adjuvant proton beam therapy to reduce the risk of recurrence of the tumour.

Proton beam (particle) therapy is an emerging technique in radiotherapy. There are currently no centres in the UK offering this treatment and so patients have to have prior approval and funding to have this treatment overseas, primarily in the USA. Cases must initially be referred to a specialist panel who decide if treatment is appropriate and will approve NHS funding for the treatment as well as travel and subsistence costs.

Proton beam therapy is considered superior to traditional photon (x-ray) radiotherapy in 2 main situations. Firstly when a higher dose is needed for cancer control than can safely be delivered by conventional external beam radiotherapy. Secondly, it has preferred dose distribution properties to reduce the dose to surrounding critical structures such as the brainstem and optic chiasm which could cause significant co-morbidity. This is because of the properties of protons, meaning they deposit their energy at a very confined depth. Both of these factors were critical in this case due to the location and histology of the tumour.

The case will examine the evidence for proton beam therapy its relative merits and side effects

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